

## **Basal Resistance of Plants Against Bacteria: from Discovery to Molecular Characterisation\***

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Basal resistance (BR, a local resistance against pathogens induced by general elicitors) is a multifaceted defence mechanism appearing in many shapes and with many given names, such as induced (acquired) resistance (protection, defence), as well as a part of innate immunity. Here, we give a historical overview of concepts, present results and an outlook associated with bacteriological studies in the Plant Protection Institute (PPI). The topics on BR briefly covered are its elicitors, light and temperature dependence, changes in plant physiology, transcription and protein expression during it and its effect on bacteria. We emphasize the importance and relevance of its quick form, early BR, which might be a plant defence mechanism in nature against all kinds of pathogenic and saprophytic bacteria.

Keywords: apoplast proteins, bacteria, gene transcription, hypersensitive response, induced resistance, susceptible response.

### *BR prehistory for PPI*

Very early observations of plants recovering from a virus disease (Wingard, 1928) suggested that these immune [free, exempt from disease; in the followings, square brackets will serve for definitions] plants acquired a kind of resistance [ability to repress an invading pathogen] even to a second, challenge infection (Price, 1936). Results of sequential artificial infection studies added that the extent of resistance depended on the preexisting level of establishment of the pathogen when used as pretreatment. Although the emergence of new 'protective substances' of plant origin was suggested as a mechanism of the immunity (Price, 1936), direct antagonism between the pretreatment and challenge strains could also have been an explanation (Wallace, 1944). Works with virus strains causing hypersensitive reaction [HR, a blend of resistance and plant cell death] showed that direct antagonism could be excluded in pathogen-free parts of plants acquiring resistance close to (Yarwood, 1960; Ross, 1961a) or distant from (Ross, 1961b) to the HR lesions. Here made PPI its mark, explaining resistance with changes in activities of oxidative enzymes (Farkas et al., 1960). A work using constituents of a pathogen instead of an

\* This article pays tribute to the late Professor Zoltán Klement.

intact one as a pretreatment (Loebenstein, 1962) pointed to further characteristics of acquired resistance: there was no need for a living and growing pathogen for induction; direct antagonism was unlikely even at the same location; time – measured in a few days – was required for the resistance to develop.

### *The ‘late’ BR*

A study similar to this, but the first with a bacterial pathogen system, was reported by PPI scientists (Lovrekovich and Farkas, 1965). It was possible because of a simple and revolutionary leaf injection technique to artificially introduce bacteria (and other substances) into leaf intercellular spaces, making single cell-level reactions accessible to investigations for the first time (Klement, 1963). Lovrekovich and Farkas (1965) used heat-killed *Pseudomonas syringae* pv. *tabaci* cells as a pretreatment – elicitor of the resistance – and the same but live bacterium as a challenge inoculum in the same tissue in susceptible tobacco hosts. The time needed for the protection to unfold against the susceptible response (SR) was, again, above one day. The study was further important for the following reasons: i) It showed that heat-killed cells of a range of bacterial species elicited the same effect, suggesting the presence of general elicitors [common, constant bacterial components recognised as non-self by plants, usually triggering defence responses but not the HR. Of note, it was known from Klement’s studies that time that bacterial metabolic activity was required to induce HR (Klement et al., 1964)]. ii) As the resistance induced was ineffective against the toxin-containing bacterial culture filtrates, its efficacy against the active stage of a living pathogen, i.e. not against the symptoms, was inferred.

With the studies discussed above, outlines of [a local resistance induced by general elicitors] emerged. For simplicity, we will use the collective term basal resistance (BR) to refer to it. BR had been given several other names that reflect an elevated level of resistance (‘protection’, ‘defence’, ‘immunity’) to subsequent pathogenic challenge and the acquisition of this trait (‘induced’, ‘acquired’) after being exposed to general elicitors. In this article we deal only with local (not systemic) resistance phenomena.

### *Directions of interest generated worldwide*

Klement’s pioneering work on HR as well as Lovrekovich and Farkas’s on BR generated directions of interest for details of these bacterial-plant interactions worldwide.

Efficacy of general elicitors (heat-killed bacterial cells) against another type of pathogen, tobacco mosaic virus was soon shown (Loebenstein and Lovrekovich, 1966). The two directions (i.e. HR and BR) met in a study where general elicitors protected a plant tissue from undergoing the HR induced by incompatible [pathogenic on other plant(s), but not on the plant in question] bacteria (Lozano and Sequeira, 1970; Novacky et al., 1973).

A direction begun to pursue chemical identification of the general elicitor(s). This led to large glycoprotein constituents (Sequeira et al., 1972), lipid A part of lipopolysaccharide (LPS) (Graham et al., 1977), protein-LPS complexes (Mazzucchi et al., 1979), extracellular polysaccharides (EPS) (Mazzucchi et al., 1984) as candidate compounds. (EPS may have had a mechanism different from elicitors – Z. Klement, personal communication.)

Studies on the effects of BR on bacteria had shown repeatedly that BR interferes with bacterial multiplication, especially in the first 6–8 hours of challenge (Lozano and Sequeira, 1970; Sequeira and Hill, 1974). A morphological phenomenon, perhaps underlying problems in bacterial division, was observed during the quest for plant inhibitory substances: long chains of bacterial cells apparently unable to separate in the intercellular washing fluid (IWF) of pretreated plants. The respective inhibitor has not been found (Rathmell and Sequeira, 1975).

Leading the next path of scientific curiosity, measuring induced changes in plant metabolism, Klement found that incompatible bacteria (HR) caused more intense respiration than saprophytic ones (BR) (Németh and Klement, 1967). The most comprehensive technique to approach plant-bacterial interactions, electron microscopy, was not available in Hungary that time, but elsewhere in the world several studies were reported on ultrastructural changes following infiltration of saprophytic or pathogenicity mutant bacteria. Because of space limitations, we can only mention two phenomena, possibly connected with BR: immobilisation of nonpathogenic bacteria, but not compatible ones, to the plant cell wall (Sequeira et al., 1977), which could be correlated with activity of plant agglutinins (Sequeira and Graham, 1977); the highly localised apposition of material, e.g. callose, inside cell wall and outside the cytoplasmic membrane, called papilla (Sequeira et al., 1977).

Little was known about the environmental conditions. Light requirement for BR was shown (Sequeira et al., 1972).

Directions taken by Zoltan Klement with respect to BR stemmed from the fact that the HR and the SR became related by being similarly affected by pretreatment with general elicitors. Klement maintained that the two responses differ in only the threshold ratio of bacterial cells to plant cells: while for the HR it was 1:1, for the SR-related necrosis 50–100:1 (Klement et al., 1978; Klement, 1982). Likeness of HR and SR – an unsettled topic still today, see e.g. Tao et al., 2003 – was exemplified by their need for the so-called induction period. This is a time period (usually 1–3 hours) after infection/inoculation during which a bacterium becomes able to induce either response. When the bacterium is somehow – e.g. by artificial injection of antibiotics into the infected tissue – disturbed during induction time, no HR (Klement and Goodman, 1967) or SR (Klement et al., 1978) occurs.

### *The ‘early’ BR*

Now, what if the plant itself has a mechanism to disturb the bacterium during induction time? This would be of huge advantage in nature since preventing the bacterium from initiating interactions would doom it to starvation and extinction. Time for such a plant mechanism to act is much shorter than the gaps between pretreatment with a general elicitor and a challenge in BR studies discussed above. Klement’s investigation on the nature of the ‘HR inducer’ (Klement, 1977) necessarily using short gaps led, as a by-product, to the discovery of a BR mechanism that was characterised by its earliness (measurable effect within 6 hpi) (Klement and Burgyán, 1978; Burgyán and Klement, 1979). Some details on distinction between this ‘early’ and the known ‘late’ BR in tobacco were worked out in terms of light requirement (Hevesi et al., 1981) and partial elicitor characterisation

(Mehiar et al., 1981). However, no scientist outside his co-authors seemed to pay attention. Fundamental experiments were missing to show that such a mechanism operates in nature, i.e. that a BR induced by a bacterial cell inhibits the HR induced by the very same bacterial cell. Even Klement would turn to other subjects, like bacterial genetic determinants of the HR (Somlyai et al., 1986) or a bacterial cause of apoplexy of apricot (Klement et al., 1984). The next push on early BR came by chance, when Klement failed to demonstrate the HR for students when he visited his friend Klaus Rudolph in Göttingen, Germany. Klement felt the failure embarrassing, since HR is normally very robust. Having checked all the details, he found that on the day he injected the tobacco plants with an incompatible *P. syringae* pv. *phaseolicola* strain for the show, there was unusually high temperature, 30 °C. He felt that the early BR (Burgyán and Klement, 1979) might be responsible for the failure. He began to build a new working group and demonstrated that during a transition from 20 °C to 30 °C, the length of induction time for HR in *P. syringae* pv. *phaseolicola* remained 2.5 hours, but the time needed for early BR to develop shortened from 6 h to 2 h. Such a quick, bacterially induced BR may prevent another response (the HR) induced by the same bacterium later, during induction time. Indeed, various incompatible bacteria with longer induction time than plant BR development time were unable, and reversibly, those with shorter induction time were able to induce HR (Klement et al., 1999). Thus, there is a time race between invading bacteria and plants, reserving a place for early BR in nature, i.e. during a single course of a plant-bacterium interaction.

#### *Current results – in a broadened context*

With this new system, early BR, Klement's expanding workgroup assumed the old directions discussed above – and overviewed below. Before proceeding with present results, we should note that studies on BR (HR as well, albeit it had already been intense) gained new impetus by a major turn in animal/human immunity sciences in the late 1990s (reviewed by Kopp and Medzhitov, 1999). Namely, an ancient, cell-autonomous resistance, called innate immunity, underlying the well-known adaptive immunity was discovered in animals. Most importantly for us, receptor proteins of innate immunity were found to have a structure strikingly similar to the plant's resistance (R-) proteins. The respective ligands in animals, pathogen associated molecular patterns (PAMPs), include several types of conserved and typical bacterial components like LPS/endotoxin, peptidoglycan, CpG DNA or flagellin. These ligands correspond functionally and structurally to the general elicitors for plants (Nürnberger and Brunner, 2002). Plant R-proteins are activated by specific microbial avirulence proteins, which normally results in HR. Thus, in animals, R-protein-like host molecules interact with general elicitor-like bacterial ones. Translated to plant 'language', innate immunity mixes HR with BR. The contradiction is probably only seeming: we simply do not know enough about avirulence-like proteins of bacterial pathogens of animals and about BR receptors in plants, except for one case: flagellin interacts an R-protein-like receptor, Fls-2 in *Arabidopsis* (Zipfel et al., 2004).

## GENERAL ELICITORS OF EARLY BR

We set out to find general elicitor(s)/PAMP(s) of early BR in tobacco, using the HR inhibition biotest and chitinase markers (see later). We used known bacterial elicitors of plant responses reported to interfere with bacterial growth. Flagellin (Zipfel et al., 2004) was very similar to heat-killed bacteria in most aspects of early BR (G. J. Varga unpublished). The recently discovered general elicitor, CSP (cold shock protein, Felix and Boller, 2003, here data on induced resistance are lacking) led to a later/weaker BR at 24 hours post-inoculation (hpi). LPS was even slower, with significant effect only at 48 hpi. Peptidoglycan proved the least potent general elicitor (G. J. Varga unpublished). Thus, only flagellin from the elicitors investigated so far seems to be compatible with early BR. Although other bacterial components are likely to have a role (some are now under study), this finding is an important step forward that obviates the use of exceedingly complex elicitors (bacterial cells), enabling more mechanistic and traceable studies in the future.

## EFFECTS OF EARLY BR ON BACTERIA

Quickness of early BR strongly suggested that BR inhibits HR through an effect on the HR-inducing bacteria. [For late BR a similar inference was already made in 1965 (Lovrekovich and Farkas, 1965)]. As multiplication rates may not necessarily correlate with HR induction ability, direct measurements of bacterial *hrp* gene [of these genes, upregulated in the presence of plants, are required for HR and SR] activity were made. It was found that *hrp* genes of *P. syringae* pv. *syringae* infiltrated into tobacco leaves were activated, but much more so in plants where BR was compromised by inhibition of eukaryotic protein synthesis (Bozsó et al., 1999). Moreover, *hrp* gene activity was suppressed in plants pretreated with general elicitors (Ott, 2002). Disturbed *hrp* gene function may contribute to the fact that early BR delays SR symptoms and multiplication of the virulent *P. syringae* pv. *tabaci* in tobacco. This pathogen, unable to avoid its own recognition, must have (and has) means to suppress BR in its host (Ott et al., 1998). To embark on finding bacterial suppressors, genetic tools, IVET (*in vivo* expression technology) plasmids were constructed that proved valuable in determining several genes that are specifically turned on during *in planta* growth (Czelleng et al., 2004). Whether BR mechanisms inhibiting bacterial growth and *hrp* gene expression are indiscriminate or have parts specifically tailored against different types of bacteria, remains to be elucidated.

## INDUCED CHANGES IN PHYSIOLOGY DURING EARLY BR

Classical approaches on plant metabolism during BR, such as ultrastructural studies and measurements of pro-oxidative changes provided some information. For example, no specific symptom, not even papilla formation could be attributed to early BR (Ott et al., 1998). Also, a peroxidase- and H<sub>2</sub>O<sub>2</sub>-dependent tissue staining was described as correlating well with early BR (Bozsó et al. 2005). To find more inclusive and basic information, new experiments started at two levels, gene transcription and protein expression.

## TRANSCRIPTIONAL CHANGES DURING EARLY BR

To identify the genes and to characterise these changes we have used different methods, such as large (microarray) and small-scale (real-time reverse transcription polymerase chain reaction, RT-PCR) techniques on various model plants (tobacco, *Medicago truncatula*, *Arabidopsis*). In concert with another study where transcriptional changes of about 3% of genes after a general elicitor (flagellin) treatment was detected (Navarro et al., 2004), we can say that the activity changes are very complex and influence almost the whole repertoire of plant physiological processes. For example, the microarray results have pointed out that at time of early BR, genes of different isoenzymes (e.g. peroxidases, Bozsó et al., 2002) with the same or similar function are activated or repressed which suggests a sophisticated regulation of defence mechanism. Comparison of gene activity changes during early BR and the HR uncovered that the activity of many genes are similarly altered, albeit with higher amplitude in HR (Bozsó et al. unpublished). More detailed studies will help us find those genes that are specific for the particular type of resistance response. The activity changes of many genes during BR are temporal. After bacterial inoculation the activation of several genes increases 1–3 hpi and following a peak at 6–12 hpi the transcription level decreases nearly to the basal level. This temporal pattern is correlated well with the activation and termination of early BR. Of course the protection does not come to an end and there are other genes that retain their expression at a higher level and another set of genes exists that increase and maintain their transcription at a later (24–48 hpi) phase of the defence, corresponding to late BR (Szatmári et al. unpublished; Bozsó et al., 2005). Genes with modified activity during BR have diverse functions and can be associated directly and indirectly with defence processes. Strong activation of cell protection genes (e.g. glutathion S-transferase, epoxide hydrolase), cell wall fortification (lignin synthesis, proline or glycine-rich proteins), defence-related genes (PR proteins, various elicitor-inducible genes) and a lot of genes with unknown functions were found. Genes with a potential role in this transcriptional reorganisation, members of signalling pathways (e.g. kinases, phosphatases and transcription factors), also were found to be down- or up-regulated (Bozsó and Szatmári unpublished). The coordination of gene activity during BR is still unclear because the best known signal molecules of plant resistance responses, such as salicylic acid, methyl jasmonate and ethylene did not have a significant effect. However, the compatible pathogen can block the induction of several BR-associated genes (Szatmári et al. unpublished), which seems to be a crucial step of the pathogenesis (Jakobek et al., 1993). More detailed transcriptional analysis and functional genomic studies of identified genes, such as over- and decreased expression of the particular genes or groups of genes will lead to the precise identification of the important genes and plant processes required for development of the BR.

## EARLY BR-ASSOCIATED APOPLASTIC PROTEINS

Plant pathogenic bacteria do not enter living plant cells. Consequently, plant molecules effective in resistance transverse the apoplast. Non-denaturing electrophoresis of apoplastic proteins from general elicitor-treated and control tobacco plants led to the identification of

two novel chitinases as markers of early BR. BR chitinases were transcriptionally induced, and unlike for PR- (pathogenesis-related) proteins, environmental stresses or major defence hormones seem to have minor or no influence. Interestingly, expression of chitinases and efficacy of early BR are quantitatively correlated. Chitinase expression is suppressed by *P. syringae* pv. *tabaci*, but only to a limited extent, which may explain efficacy of pre-established early BR on this pathogen (Ott et al., 2006). In pepper a 22 kDa chitinase and a 32 kDa peroxidase, both of apoplastic origin, can also be of use to mark the BR. These markers are neither entirely qualitative, as they are constitutively expressed at a low level (Besenyi E. unpublished). Roles of early BR-related chitinases against bacteria is being elucidated.

#### ENVIRONMENTAL CONDITIONS FOR EARLY BR

Although darkness does not inhibit development of early BR (Burgyán and Klement, 1979), we have shown that light maintained early BR in terms of resistance strength and chitinase marker expression in tobacco, especially during the transition period (approx. 18–36 hours post-inoculation) to the late BR (Ott et al. 2006). Similar light dependence was affirmed in pepper (Besenyi E. unpublished).

The same temperature-dependence could be observed in pepper as in tobacco in the range of 20 °C to 30 °C. Temperatures lower than 20 °C were also examined for a particular reason: plant pathogenic bacteria, such as the opportunistic pathogen *P. syringae* pv. *syringae*, exhibit increased virulence at temperatures lower than their *in vitro* growth optimum (Smirnova et al., 2001). They can even multiply in such conditions and cause serious ‘cold-weather’ diseases in various plants, as PPI’s scientists have shown (Klement et al., 1984; Süle and Seemüller, 1987). Klement’s hypothesis was that the lack of EBR/HR is a key for the success of *P. syringae* pv. *syringae* in the cold. It was corroborated by the finding that at 5 °C neither EBR nor HR was detectable for at least 2–3 days post-inoculation. This may enable certain psychrophile pathogens to colonise plants (Besenyi et al. 2005). Thus BR, a complex defence response were shown to be vulnerable to extreme ambient conditions. Reasons for this, developmental, energetic or other, await experimental solution.

## Acknowledgments

We dedicate the following lines to Zoltán Klement, the late professor, the philanthropist who has overseen and coordinated the field of basal resistance against bacteria until recent times. Unlike many scientists of his calibre, Prof. Klement addressed everybody in a true democratic way. It was especially striking in his dealings with younger and less experienced colleagues. He made discussion partners feel on an equal footing, which broke barriers towards intimacy, liberated deep spirits and a free flowing of thoughts. This talent of his was nourished by a natural – and rare – interest in others intellect, something that can perhaps never be learned. His key of a great success was based on a magnanimous openness we shall desperately miss. This work was supported by OTKA AT049318.

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