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THE INVOLVEMENT OF *Pseudomonas* BACTERIA IN INDUCED SYSTEMIC RESISTANCE IN PLANTS (REVIEW)

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This article reviews the most recent results of studies on the mechanism of induced systemic resistance (ISR) elicited in plants by non-pathogenic bacteria of the genus *Pseudomonas*. Several examples of *Pseudomonas* strains eliciting resistance against fungal phytopathogens in different species of crop plants are presented. Literature data dealing with bacterial elicitors and the effect of their interaction with plant receptors are quoted. Special focus is focused on the controversial issue of the correlation between the synthesis of pathogenesis-related proteins (PRs) and ISR.

Brief characterization of plant resistance types. Over a long period of time environmental pressure on plants induced the development of mechanisms enabling plants to combat the organisms negatively affecting them. One of these is resistance. Although plants do not have an immune system characteristic for mammals, this term applies to plants as well. In this case, resistance is defined as "the insusceptibility of a plant to infection". Two types of resistance can be distinguished: constitutive (passive), and induced (acquired, active). Constitutive resistance is created by anatomical and physiological barriers. In turn, the state of enhanced plant defense, appearing as a consequence of the activity of a biotic or abiotic factor, is termed induced resistance. Usually enhanced resistance is not limited to the site at which a particular agent acts. A signal induced at one site spreads systemically to other organs, as a result of which the whole plant is prepared for an attack by a pathogen [1, 2]. Two kinds of induced resistance with systemic range can be distinguished: systemic acquired resistance (SAR) and induced systemic resistance (ISR). SAR is induced by pathogens eliciting necrotic changes in the plant tissues. Their presence within tissues results in increased synthesis of salicylic acid (SA), which in turn activates the expression of pathogenesis-related (PR) genes. The products of the expression of these genes participate in the destruction of the plant pathogen [3, 4]. On the other hand, ISR is mediated by non-pathogenic bacteria and fungi that stimulate plant growth. These are known as the plant growth-promoting rhizobacteria (PGPR) and fungi (PGPF). They inhabit the root system, in which resistance is induced. The signaling pathway initiated in the underground plant organs, embraces the above ground parts as well. Jasmonic acid (JA) and ethylene (ET) play an important role in this pathway [5, 6]. These plant hormones participate in the activation of genes, whose transcription and translation lead to the formation of compounds that have negative effect on pathogens and plant pests. Activation occurs only after the plant organism is attacked by the phytopathogen [7, 8]. Genes respond more rapidly in the presence of JA and ET and their expression is both enhanced and faster. Resistance thus seems to have economic advantages for the overall plant defense response [9]. PGPR strains, after effective colonization and induction of resistance, maintain a plant in a state of elevated readiness to the occurrence of a pest in the general sense. Thus, there is no constitutive production of compounds by the plant which limits energy losses at the metabolic level [10]. The expression of induced systemic resistance (ISR) has been observed for different plant species, not only in dicotyledons (string bean, *Arabidopsis*, carrot, tobacco, radish and tomato), but also in monocotyledons (corn, rice) [11]. It appears that the mechanism of this plant resistance is effective in combating bacteria, fungi, viruses [12], nematodes [13] and insects [14].

ISR inducers. The participation of *Pseudomonas* bacteria, as well as of other bacteria classified to the PGPR in the induction of systemic resistance, is related to the production by these non-pathogenic bacteria of so-called elicitors (inducers, determinants), activating the defense responses of plant cells. In the case of an immune response to pathogens these compounds have been termed pathogen-associated molecular patterns (PAMPs). Frequently in multicellular organisms recognition of PAMPs by pathogenesis-related proteins (PRs) also enhances a SAR response [16]. A considerable diversity of the elicitors involved in ISR has been observed. They include the building blocks of bacterial cells as well as extracellular compounds synthesized by the microbes [17]. In the case of *Pseudomonas* they are: siderophores, flagellin, lipopolysaccharides (LPS) [18], an N-alkylated ben-

zoamine derivative [19], SA, 2,3-butanediol, [20] and antibiotics, including 2,4-diacetylphloroglucinol and pyocyanin [21, 22]. Two important aspects of ISR induction should be pointed out – the time needed for this type of resistance to develop and the number of bacterial cells required for its initiation. ISR can be elicited only when the number of bacterial cells reaches a minimal value equal to 10^5 CFU/g (colony forming unit/g) of plant root and time of root colonization by the bacteria is not shorter than a few days [23].

Interaction between inducers and plant receptors. The mechanism of ISR induction is still unclear. It is thought that the induction of resistance in plants by rhizobacteria is analogous to the mechanism of resistance elicited by pathogens in eukaryotic cells. In both cases receptors are recognized by elicitors [24]. The induction of ISR is not a random process. A certain specificity in recognition and binding of PGPR elicitors to the corresponding root receptor of a given plant species is observed [25]. It appears that some bacterial strains, such as *P. fluorescens* WCS417r, demonstrate a high effectiveness of the binding in different plant species, whereas others, e.g. *P. fluorescens* WCS358, are characterized by narrow specificity. The former strain induces resistance via LPS in such plants as *Arabidopsis* [26] carnation [27], radish [28]. In turn, flagellin and LPS of *P. fluorescens* WCS358 are effective inducers of resistance in Arabidopsis but not in the pea and tomato [18]. Initially it was thought that a flagellin conservative motif of all PGPR could be recognized by the most plants. However, studies using two different *Pseudomonas* strains contradict this theory. *P. fluorescens* WCS358 and WCS374 strains both have a flagellum but only the former induces ISR in *Arabidopsis thaliana* [26]. As yet, the nature of the receptor binding a given elicitor is not known. It has been assumed that flagellin may be identified by the receptor LRR-NBS (leucine-rich repeat-nucleotide binding site). In *Arabidopsis* LRR-NBS binds to the most conserved domain of flagellin, triggering a mitogen-activated protein (MAP) kinase pathway. Ultimately, several phosphorylations lead to the formation of a protein belonging to the WRKY family that is a group of proteins being transcription factors for genes encoding proteins involved in defense mechanisms of the plant cell [29].

ISR signaling pathway elicited by P. fluorescens WCS417r. The binding between a root receptor and the PGPR determinant results in activation of ISR signaling pathway. *A. thaliana,* ecotype Columbia (Col) is a model plant for which this process is studied in detail. This particular species is distinguished by the best studied genetic profile of the interactions between a microorganism and a plant [30]. To induce resistance, *Arabidopsis* roots were colonized by *P. fluorescens* WCS417r. A characteristic trait of the strain is its effectiveness in inhibiting the growth of pathogens of not only the model plant [6, 31].

Local response – ET and MYB72-dependent signaling pathway. ET is one of the first elements of a signaling pathway initiated by *P. fluorescens* WCS417r in the *Arabidopsis* root. This compound was found to be indispensable for the occurrence of resistance in the above ground plant organs [32]. Besides ET, gene *MYB72* also played an important role in the early signaling steps of ISR [33]. It appears that *P. fluorescens* WCS417 elicits the activation of the transcription of *MYB72* (as well as of 96 other genes) in the root. *MYB72* belongs to the *R2R3-MYB* family of genes in the *Arabidopsis* genome and encodes the transcription factor TF MYB 72. The expression of these genes results in the formation of proteins R2R3-MYB, whose presumable function is related to tolerance to stress, regulation of cell death and resistance to pathogens [34]. It has been reported that the presence of gene *MYB72* is required in the initial stages of ISR induction. This was confirmed in studies using plant mutants carrying the recessive form of the gene, in which bacteria did not elicit ISR [8]. It was also observed that overexpression of gene *MYB72* did not translate to increased level of resistance to such pathogens as *P. syringae* pv *tomato*, *Hyaloperonospora parasitica*, *Alternaria brassicicola* and *Botrytis cinerea*. This allows concluding that initiation of *MYB72* requires the participation of an additional factor for initiating ISR. In view of the fact that under *in vitro* conditions an interaction between MYB72 and ethylene-insensitive3-like 3 protein (EIL3) has been observed, the latter has been suggested to be the putative factor. EIL3 is a transcription factor of the EIN3 family. Close homologs of EIN3 are transcription factors EIL1 and EIL2 participating in regulation of the ethylene-dependent signaling pathway [35].

*Systemic signaling response***.** The signaling pathway locally initiated by *P. fluorescens* strain WCS417r in the root spreads to the above ground plant organs. The major molecules involved in the expression of ISR in the stem and leaves are jasmonic acid (JA) and ET [36]. This is indicated by the results of studies using mutated plants with defects in genes participating in the cellular response to the above compounds. Mutations were introduced into such genes as *Etr1*, encoding ET receptor (a dominant mutation in this gene led to decreased susceptibility of tissues to the hormone) [35], and *Jar1* coding an enzyme involved in regulating JA activity in the signaling pathway [37]. Expression of ISR was not observed in any of the obtained mutants [36]. Of importance in the discussed phenomenon was that ISR did not cause increased synthesis of JA and ET, but only increased susceptibility of the plant to these hormones [38]. Studies have revealed that important components of the SAR pathway: SA and protein non-expressor of pathogenesis-related genes 1 (NPR1), are also implicated in the resistance induced by rhizobacteria. NPR 1 is considered a key regulatory protein in cross–talk between SAR and ISR [39]. It has been reported that even

though the ISR signaling pathway in *Arabidopsis* is independent of SA, NPR1 is one of its indispensable elements [36]. JA, ET and NPR1 form a specific signaling cascade in which the action of JA is followed by ET. In turn, these hormones interact with the final element of the pathway, protein NPR1. The occurrence of a signaling pathway involving ET and JA is observed only in plants carrying the dominant form of gene *ISR1*, located on chromosome III. It was shown that the product of the transcription and translation of this gene is a component of the ET-dependent signaling pathway [40]. Ecotypes of *Arabidopsis* carrying a recessive mutation in the *ISR1* gene locus (RLD and Wassilewskija) are susceptible to attack by the pathogen *P. syringae* pv. *tomato*, as opposed to ecotype Columbia (*ISR1/ISR1*). This is related to the fact that interruption of the signaling pathway at any point results in enhanced susceptibility of plants to the deleterious action of phytopathogens [40].

Other signaling pathways. Even though, similarly to *P. fluorescens* WCS417r, four other PGPR strains have been described to induce resistance in *A. thaliana* via the chemical messengers JA and ET [41], the course of the signaling pathway presented above is not a universal ISR scheme. JA and NPR1 are indispensable components of the signaling pathway initiated in the same plant by *P. fluorescens* CHAO in response to attack by *Perenospora parasitica* [21]. Another exception is the role of SA in the induction of ISR. This situation is observed in the tobacco plant, in which the mediation of the rhizobacterium *P. aeruginosa* 7NSK2 induces resistance to infection by the tobacco mosaic virus [42]. The above examples serve as proof of interactions between plant signal transduction pathways, which allows adjusting the defence strategy of plants to the changed environmental conditions. A close cross-talk between the jasmonate and salicylate plant defense pathways is known to have a strong influence on the molecular and enzymatic profile of the plant and on some plant parasites [43].

Effects of ISR in plants. The final and also the most important elements of resistance induced in plants PGPR involve the synthesis of compounds protecting the plants cells from being harmed by a pathogen and/or destroying the cells of the pathogen destruction. The synthesis of these compounds in plant tissues is a result of activated transcription of the genes encoding them. However, as studies have shown, the process is not the immediate result of colonization of *Arabidopsis* roots by *P. fluorescens* WCS417r. The same phenomenon also occurs following challenge inoculation by the plant pathogen – 81 genes in the leaves show an augmented expression pattern, very likely as a result of the activity of signaling pathway molecules [9]. Following pathogen attack, JA-dependent genes characterized by over-expression of the motif 5'CACATG 3' in the promotor region of gene are activated in ISR-expressing plant cells. This sequence is a binding site for the transcription factor MYC2 which plays a crucial role in regulating the transcription of these genes. MYC2 is probably involved in activating the transcription of genes, but only in response to an attack by a plant pathogen whose destruction is dependent on jasmonic acid-regulated genes. Otherwise, no up-regulation of MYC2 expression is observed [14]. It has been postulated that its activity is inhibited by the jasmonate-zim-domain (JAZ) suppressor protein. In situations other than described, the repressor is shunted into a pathway leading to its degradation in the proteasome [44]. The final outcomes of ISR are structural and biochemical changes in plant cells. Their aim is to impede penetration into the tissues and inhibit pathogen growth, and thus prevent the development of the disease caused by a specific plant pathogen [45].

Structural and biochemical modifications. Structural modifications occurring in the cell wall, being the response of a plant to attack by pathogen, consist in the physical and mechanical strengthening of the wall through the deposition of various compounds, making it impossible for pathogens to penetrate the plant tissues. These substances stiffen the cell wall, thus creating an impenetrable barrier. This involves the deposition of callose and infiltration of phenolic compounds at the site of infection by the plant pathogen [46]. Certain rhizobacteria, as a consequence of ISR induction, cause lignification of the cell wall, as observed in the case of bean [47].

Besides structural alterations in the cell wall, ISR also results in enhanced synthesis of various secondary metabolites participating in defense mechanisms (Table). These include the phytoalexins which are a chemically diverse group of low-molecular-weight compounds including sesquiterpenoids synthesized by the *Solanaceae*, and isoflavonoids in individual representatives of the *Papilionaceae*. The role that phytoalexins play in resistance is related to their toxic action against pathogens. Their presence inhibits the formation and germination of fungal spores and growth of hyphae, and restricts the growth of bacteria and viruses. In plant tissues, in which ISR was expressed, increased activity of phenylalanine ammonia-lyase (PAL), participating in resistance responses, was found [48]. The enzyme is also involved in splitting off an ammonia molecule from (S)-phenylalanine, leading to the formation of E-cinnamonic acid which, in turn, is a lignin precursor. PAL thus plays an important role in the formation of a mechanical barrier. This enzyme is also involved in the synthesis of the phytoalexins mentioned above [49].

An important role in combating pathogens is carried out by pathogen-related proteins (PR). Some of them, thanks to their hydrolytic activity, participate in the degradation of the cell wall of fungi: proteins of the family PR-2 – (β-1,3-glucanase), PR-3, PR-4, PR-8, PR-11 (endochitinases) and PR-7 (endoproteinase). Others, such as protease inhibitors (PR-6), are capable of deactivating toxic polypeptides secreted by par-

Compounds participating in the destruction of plant pathogen cells in ISR

PGPR	Pathogen	Plant	Protective compound*	References
Pseudomonas aeruginosa 7NSK2 *Pseudomonas fluorescens* CHAO	*Botrytis cinerea*	Grape	Phytoalexins	[48]
Pseudomonas fluorescens	*Fusarium oxysporum* f.sp. *cubense,*	Banana (*Musa* sp.)	PO, PAL, chitinase, β-1,3-glucanase, phenolic compounds	[56]
	Alternaria palandui,	Onion (*Allium cepa* var *aggregatum*),	PO, PPO, chitinase, β-1,3-glucanase	[57]
	Pyricularia grisea,	Finger millet (*Eleusine coracana*),	Chitinase, β-1,3-glucanase, PO, PPO	[23]
	Pythium apha-nidermatum	Tomato (*Lycopersicon esculentum*), Paprika (*Capsicum annuum*)	PAL, PO, PPO, phenolic compounds	[11]
Pseudomonas fluorescens *Pseudomonas aeruginosa*	*Erysiphe pisi*	Pea (*Pisum sativum*)	Phenolic compounds	[59]
Bacillus pumilus IN937a and IN937b	*Sclerotium rolfii,* *Ralstonia solanacearum,* *Colletotrichum gleosporioides*	Tomato (*Lycopersicon esculentum*), Paprika	SOD, PO	[58]

* PAL – phenylalanine ammonia lyase, PO – peroxidase, PPO – polyphenol oxidase, SOD – superoxide dismutase.

asites within the tissues [50]. The action of peroxidases (PR-9), in turn, results in increased production of lignin and its deposition in the cell wall, with subsequent formation of a mechanical barrier [51]. The correlation between the synthesis of PR proteins and ISR is controversial. In the case of *Arabidopsis*, *P. fluorescens* WCS417r cells did not induce the synthesis of these proteins similarly as in the case of radish [52, 53]. The results of these studies allow concluding that resistance in the case of *P. fluorescens* WCS417r is not related to the production of pathogen-related proteins. This conclusion is, however, contradicted by the results of numerous experiments with other strains of bacteria (Table). An example is *P. fluorescens* CHA0, whose presence induced the synthesis of proteins of the PR-1a, PR-1b and PR-1c families in tobacco [54]. *P. fluorescens 63–28*, in turn, caused increased β-1,3-glucanase and chitinase activity in the pea [46]. Mariutto et al. [55], on the other hand, found an increased transcript level of lipoxygenase, an enzyme involved in the synthesis of jasmonic acid, in tomato plants inoculated with *P. putida* BTP1. In these studies no stimulation of PAL activity was observed.

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The phenomenon of induced systemic resistance (ISR) described above rouses the high hopes of many researchers representing different fields of science.

The mechanism of this resistance is still, however, insufficiently elucidated. There are many unknowns about this resistance, beginning with its induction, through the course of the signaling pathway, to a fungistatic-type substance. Answers to these questions may enable the practical application of the mechanism of this resistance in biological plant protection.

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