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Evolution of views on plant immunity: from Flor's "gene-for-gene" theory to the "zig-zag model" developed by Jones and Dangl

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Abstract: The study of plant defence mechanisms in response to pathogens in the mid-20th century resulted in Harold Flor's gene-for-gene interaction hypothesis, which became recognised as central to the study of phytoimmunity. According to this theory, the outcome of interactions in plant – pathogen phytopathosystems – i.e. compatibility or incompatibility – is controlled genetically in interacting organisms and determined by the presence of specific genes in both pathogen and plant: resistance genes in the plant and avirulence genes in pathogen. The latest achievements in phytoimmunology, obtained with the help of modern molecular biology and bioinformatics methods, have made a significant contribution to the classical understanding of plant immunity and provided grounds for a modern concept of phytoimmunity consisting in the "zig-zag model" developed by Jonathan Jones and Jefferey Dangl. Plant immunity is currently understood as being determined by an innate multi-layer immune system involving various structures and mechanisms of specific and non-specific immunity. Recognition by plant membrane receptors of conservative molecular patterns associated with microorganisms, as well as molecules produced during cell wall disruption by pathogen hydrolytic enzymes forms a basic non-specific immune response in the plant. Detection of pathogen effector molecules by plant intra-cellular receptors triggers a specific effector-triggered immunity, resulting in the development of the hypersensitive response, systemic resistance and immune memory of the plant. Virulence factors and pathogen attack strategies on the one hand, and mechanisms of plant immune protection on the other, are the result of one form of constant co-evolution, often termed an "evolutionary arms race". This paper discusses the main principles of Flor's classical "gene-for-gene interaction" theory as well as the molecular-genetic processes of plant innate immunity, their mechanisms and participants in light of contemporary achievements in phytoimmunology.

Keywords: *plant innate immunity; molecular patterns; effectors; receptors; pattern-triggered immunity; effector-triggered immunity*

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Эволюция взглядов на иммунитет растений: от закона H.H. Flor «ген-на-ген» до «зигзаг модели» J. Jones и J. Dangl

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Резюме: *Изучение защитных механизмов растения в ответ на воздействие патогена привело к созданию в середине прошлого века концепции «ген-на-ген взаимодействия» (H.H. Flor), которая на сегодняшний день признана классической теорией фитоиммунитета. Согласно данной теории, исход взаимоотношений в фитопатосистеме «растение – патоген» – совместимость или несовместимость, находится под генетическим контролем взаимодействующих организмов и определяется наличием специфических генов патогена и растения-хозяина. Достижения последних лет в области фитоиммунологии, полученные благодаря новейшим методам молекулярной биологии и*

биоинформатики, существенно дополнили и углубили классические взгляды на иммунитет растений и обосновали современную концепцию фитоиммунитета «зигзаг модель» (J. Jones и J. Dangl). Согласно современным воззрениям, защита растительного организма от воздействий патогена определяется функционированием многоуровневой врожденной иммунной системы с участием различных структур и механизмов специфического и неспецифического врожденного иммунитета. Распознавание мембранными растительными рецепторами консервативных молекулярных паттернов, ассоциированных с микроорганизмами, а также молекул, возникающих вследствие атаки гидролитическими ферментами патогена клеточных стенок хозяина, определяет базовый неспецифический иммунитет растения. Детекция эффекторных молекул патогена внутриклеточными рецепторами растения запускает специфический эффектор-индуцируемый иммунитет, включающий развитие реакции сверхчувствительности, системной устойчивости и иммунной памяти растения. Факторы вирулентности и стратегии нападения патогенов, с одной стороны, и участники, и механизмы иммунной системы растений, с другой, являются результатом постоянного совместного эволюционирования, что напоминает «гонку вооружения и обороны» между противоборствующими сторонами. В статье обсуждаются молекулярно-генетические процессы врожденного иммунитета растений, их механизм и участники в свете современных достижений фитоиммунологии.

Ключевые слова: *врожденный иммунитет растений, молекулярные паттерны, эффекторы, рецепторы, паттерн-активируемый иммунитет, эффектор-индуцируемый иммунитет*

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INTRODUCTION

In natural habitats, plants have to co-exist with a large variety of microorganisms, many of which are pathogenic. In order to survive, a plant must quickly recognise a pathogen and activate relevant defence mechanisms. Together, these myriad defences activated following microbiological attack constitute an integrated plant immune system – phytoimmunity.

In the middle of the 20th century Harold Flor advanced the “gene-for-gene interaction" hypothesis, which postulated that the outcome of host-pathogen interactions in a phytopathosystem – i.e. in terms of compatibility or incompatibility – falls under the genetic control of interacting organisms and determined by the presence of specific genes in both parasite and plant [1]. A more thorough understanding of the molecular-genetic mechanisms of plant immunity has only recently become possible due to the introduction of innovative methods of molecular biology and bioinformatics into phytoimmunology. The modern science of plant resistance represents many of the classical concepts of Flor’s theory, while at the same time introducing new concepts and broadening the scope of phytoimmunology following the successes of more recent studies. The key immunological achievements of the late 20th – early 21st century include Charles Janeway’s Pattern Recognition Principle, describing innate recognition by multicellular organisms of conservative structures (patterns) associated with microorganisms [2]. Innate recognition is based on detection by membrane receptors of “alien” molecular structures, which are inherent to microorganisms, but are absent from the host plant. These receptors, known as Pattern-Recognising Receptors (PRRs), have been found in all multicellular organisms from invertebrates (sponges, insects) and plants through to mammals (mice, humans) [3]. Thus, C. Janeway’s principle of pattern recognition is universal and characteristic of innate immunity in all multicellular organisms. The molecules recognised by PRR are invariant and conservative for each class of microorganisms and are denoted in respect to their origin either as Pathogen-Associated Molecular Patterns (PAMP) or Damage-Associated Molecular Patterns (DAMP). Their detection results in the activation of a series of basic, non-specific plant defence responses (Pattern-Triggered Immunity – PTI): generation of reactive oxygen species (ROS) and nitric oxide (NO), synthesis of phytoalexins, lignification of cell walls and callose deposition, as well as a number of other mechanisms [4].

The evolution of pathogens in response to basic immune defences resulted in the emergence of protein effectors (products of avirulence genes – *Avr*-genes), as well as systems for facilitating their transport directly into the cell; via Type III secretion systems acting through the “molecular syringe” principle, the effectors are delivered to the cytoplasm, bypassing the cell wall and membrane in order to block PTI development. The evolution of plants, in its turn, gave way to the emergence of intracellular or cytoplasmic Nucleotide Binding Domain Leucine-Rich Repeat Domain-containing Receptors (NLRs) [5] (products of resistance genes – *R*-genes), which detect specific protein effectors,

directly or indirectly, allowing for the induction of Effector-Triggered Immunity (ETI). In ETI "gene-for-gene" interaction is implemented; a specific protein effector correlates with an R-gene product – a complementary NLR. Modern concepts of plant immunity are summarised in the "zig-zag" model proposed in 2006 by Jonathan Jones and Jeffery Dangl [6]. This model, as an addition to Harold Flor's classical "gene-for-gene" theory, puts together multi-level plant immunity responses of differing specificity and amplitude and highlights continuous evolutionary adaptation of partners in the course of plant-microbial interactions.

CLASSICAL THEORY OF PHYTOIMMUNITY

The study of plant defence mechanisms in response to pathogens in the middle of the 20th century resulted in the formation of phytoimmunity theory, which has been recognised as central to the study of plant immunity. According to this theory, the outcome of interactions in phytopathosystems is under genetic control [7]. This means that during co-evolution, the host plant and its parasite develop complementary gene pairs: resistance (*R)* gene in the plant and avirulence (*Avr)* gene in the parasite. The study of these genes in flax rust prompted American phytopathologist Harold Flor to develop the aforementioned "gene-for-gene" concept. He found that the linen plant's (*Linum usitatissimum* L.) resistance to pathogen *Melampsora lini* is a consequence of the interaction of specific host and pathogen genes and arises only in the presence of a dominant *R*-gene allele in the plant and a dominant *Avr*-gene allele in the pathogen. All other combinations of these genes, for instance loss or alteration of the respective gene by one of the partners, do not prevent the development of the disease [8]. This type of resistance is monogenetically controlled, with the pathogen and its host typically possessing the same geographical centers of origin and evolving in parallel. The "gene-for-gene" theory informed the prominent Russian botanist Nikolai Vavilov's studies into the conjugated evolution of plant and parasite in their joint habitat [9]. The most frequently cited biochemical explanation of "gene-for-gene" theory is advanced by Peter Albersheim, who proposed that dominant alleles of resistance genes control synthesis of receptor proteins on the cell wall or membrane of host plants, whereas dominant alleles of avirulence genes code for the synthesis of glycosyltransferase enzymes, which produce hydrocarbon chains or elicitors on the surface of the pathogen cell wall [10]. Four types of factors were described in the "gene-for-gene" theory: 1) avirulence genes and their products (elicitors) 2) resistance genes and their products (receptors) 3) signal transductors carrying the information to the genome; 4) immune response genes and their products (PR-proteins, phytoalexins, lignin, etc.). The former two groups are specific; the latter ones are non-specific [1]. Thus, in compliance with the classical view of plant immunity, the interaction of products of *R*- and *Avr*-genes, receptors and elicitors, results in the development of the hypersensitive response (HR) and determines plant resistance to the pathogen. Flor's classical theory of "gene-for-gene interaction" illustrates the *Pseudomonas syringae* – tomato (*Solanum lycopersicum* L.) phytopatho-system. The *Avr*-gene of pathogen *P. syringae* is the *AvrPtoB* gene; the *R*-gene of the tomato plant is the *Pto* gene. Plant species with a dominant *R*-gene are resistant to pathogen strains with a dominant *Avr*-gene; when the *Avr/R* pair interacts, the hypersensitive response develops, and the host and pathogen are incompatible. Should any component of the *Avr/R* pair be absent or altered, they become compatible and the disease develops [11].

MODERN CONCEPT OF PLANT INNATE IMMUNITY

The application of innovative methods of molecular biology and genetics, which in many respects predicted success in recent studies of phytoimmunology, made a significant contribution to our understanding of defence mechanisms, thus supporting the modern concept of plant immunity. According to this concept, plant immunity is the result of a multi-layer innate immune system having various structures and mechanisms of both specific and non-specific immunity.

When plant and microorganism interact, the latter needs to first overcome the plant's constitutive defences, such as a powerful wax layer and rigid cell wall, as well as defence compounds including cuticular lipids, antimicrobial enzymes and secondary metabolites [12]. The next barrier in the way of a pathogen is the plant's innate immunity system; to overcome it, the pathogen has to either avoid recognition by the plant or suppress the defence response of the host. In order to recognise pathogens and their metabolites, plants use a conservative method, which is found in animal organisms as well – membrane receptors. Receptors detect specific molecular ligands, which are conservative and characteristic of pathogens, but are alien to plants. These molecules, as mentioned above, were identified for the first time by Charles Janeway and termed Pathogen-Associated Molecular Patterns (PAMPs) [13]. In reality, PAMPs are inherent to all microorganisms, regardless of their pathogenicity, thus the term MAMP (Microbial-Associated Molecular Pattern) is often used interchangeably [14]. Plants are also capable of detecting fragments formed through damage by microbial lysis of such plant structures as the cuticle or cell wall. Such products fall within the Damage Associated Molecular Pattern (DAMP) classification [15]. Detection of molecular patterns – PAMP and DAMP – is performed by Pattern Recognition Receptors (PRRs), which trigger immune signaling.

This process results in the activation of a number of defence responses leading to the prevention of disease progression. This immunity mechanism, known as Pattern-Triggered Immunity (PTI) [6, 16] presents the first level of plant innate immunity. virulent pathogen strains can suppress defence responses elicited by PRRs via the triggering of effectors, which act as virulence factors for susceptible hosts [17]. Subsequently, detection of these effectors forms the second level of innate phytoimmunity – Effector-Triggered Immunity (ETI) [6].

Basic, non-specific resistance is conferred through PTI (the first level of resistance), while the second level, ETI, induces immunity characterised by the development of the hypersensitive reaction and resulting in systemic resistance. The development of systemic resistance provides long-term protection against a wide variety of pathogens [18]. Plants may also utilise trans-generational immune memory; that is, stress caused by disease acting on one generation could bring about effective adaptation of the next generation to the same stress [19].

Pattern-Triggered Immunity – PTI

Microbial patterns or PAMPs are conservative molecular structures, which are characteristic of practically all classes of microorganism, regardless of their pathogenicity. In addition to pathogens, symbionts and endophytes also display PAMPs, resulting in an initial recognition as “alien” by the plant, which subsequently triggers an innate immune response [4, 6]. Findings suggest that the presence of both human and plant enterobacteria pathogens can activate the non-specific immune response in plants. Based on these data the authors presume that such atypical plant microorganisms have PAMPs that can be recognised by plants [20, 21]. Distinct groups of microorganisms exhibit different PAMPs: flagellin and elongation factor are found in bacteria, chitin in fungi and virulence factor NSP (Nuclear Shuttle Protein) in viruses [22-24].

PRR receptors, which specifically recognise PAMPs, include Receptor-Like Kinases (RLKs) and Receptor-Like Proteins (RLPs). RLKs are trans-membrane receptors containing an Extracellular Do-main (ECD), transmembrane domain and intracellular serine/threonine kinase domain. The ex-tracellular domains are highly variable, providing the means to recognise a wide range of PAMPs including lipopolysaccharides, peptides and polysaccharides. The transmembrane sector is characterised by an α-spiral fragment, which permeates the membrane only once. The cytoplasmatic domain formed by the C-terminal sector contains a kinase fragment, where the residues of serine and threonine are auto-phosphorylated with the formation of a dimer complex following receptor activation by a ligand. Plants have a large number of RLKs. For example, in *Arabidopsis*, there are more than 410, and more than 640 in rice [25] Plant RLKs are structurally simillar to animal Receptor-Tyrosine Kinases (RTKs) [26].

RLP receptors contain only extracellular and transmembrane domains. Since they lack an intracellular activation domain, they need to interact with adaptor molecules to transfer the signal. RLPs are close in structure to Toll-Like Receptors (TLR), which recognise MAMPs in animal cells [27]. Animal TLRs, with the help of adaptor proteins, activate IRAK (Interleukin-1 Receptor Associated Kinase) or RIP (Receptor-Interacting Protein), which trigger the expression of antimicrobial defence molecules [28]. These kinases, which belong to the same class of non-RD kinases as plant kinases, are linked to innate immune reactions in representatives of both kingdoms [29]. The number of RLPs in plants is much lower than RLKs – in *Arabidopsis* there are about 170, while in rice the equivalent figure is 90 [25].

RLPs and RLKs can be classified into different subfamilies according to domains or motifs in their ECDs. The N-terminal domain of Leucine-Rich Repeats (LRRs) is mainly involved in the recognition of proteins and peptides found in bacteria and viruses.

Currently, a large number of pathogenic molecular patterns and corresponding receptors are known, but three ligand-receptor pairs are characterised in detail. The investigation into pattern-recognising receptors began with the discovery of the Xa21 protein, which was found in rice (*Oryza sativa* L.) and the respective molecular pattern of bacterial phytopathogen *Xanthomonas oryzae pv. oryzae* (Xoo). This receptor specifically binds to Ax21, a sulphated 17-amino-acid peptide of the N-sector of proteins secreted by a given pathogen [30]. A well-known pair is receptor FLS2 (Flagellin-Sensitive 2) which is found in *Arabidopsis thaliana* (L.) Heynh., tomato (*S. licopersicum*) and tobacco (*Nicotiana tabacum* L.) and interacts with the 22-amino-acid peptide of the N-terminal segment of flagellin – flg22 [23]. Another well studied pair of PRR-MAMPs is EFR (EF-Tu Receptor) and prokaryotic elongation factor EF-Tu. The epitope of this molecular pattern is formed by the first 18 amino-acids of the N-terminal (elf18) [31].

The lysine domain (LysM) is present in receptors that recognise bacterial peptidoglycan and fungal chitin [16]. An example of a kinase that participates in the recognition of fungal ligands is *Arabidopsis* CERK1 (Chitin Elicitor Receptor Kinase 1) with three extracellular LysM domains [32], LYP4 and rice LYP6 [33].

The Lectin RLK family includes receptors with a lectin domain, the presence of which allows recognition of bacterial lipopolysaccharides [34]. Another family of PRRs is represented by receptors containing the Epidermal Growth Factor (EGF) -like domain, whose presence determines the detection of oligogalacturonides [35].

Besides PAMPs, DAMPs – damage associated molecular patterns – may also signal the attack of a

pathogen. They include oligogalacturonides formed as a result of the influence of pectolytic enzymes of phytopathogens on pectin in the plant cell wall. Destruction of plant cell walls by microbial enzymes results in the formation of oligomers with a specific and constant structure [36]. DAMPs are detected by membrane receptor kinases and interact with the plant cell wall – WAK (Wall Associated Kinases) [37]. Kinases in this family were shown to determine the integrity of oligogalacturonides produced from plant cell walls and in the case of their integrity distortion are able to activate signal cascades and the transcription of resistance genes [35].

Association of receptor and ligand is known to cause changes in the conformation of receptor molecules, which fosters enhancement of affinity between receptor molecules. As receptor kinases laterally diffuse along the membrane, they quickly and readily form dimer complexes, and in some cases, form complexes with co-receptors. At the expense of the approximation of two receptors, kinase centers are activated, and receptor molecules begin to phosphorylate each other using serine and threonine residues in the area of the kinase center. Phosphorylation results in the formation of the active site – a platform serving to interact with further components of the signal chain. Thus, in the course of interaction, the flg22:FLS2 kinase domain of the FLS2 receptor is quickly phosphorylated when stimulated by the flg22 peptide and phosphorylated FLS2 is immediately dimerised with BAK1 kinase (Brassinosteroid Intensive 1 (BRI1)-Associated Kinase – BAK1) [38]. The crystal structure of the FLS2 ectodomain was determined in combination with flg22 and BAK1: the FLS2 ectodomain was found to have 28 LRR; binding to flg22 occurs in LRR3-16, and with BAK1 in the region of LRR23-26 [39]. It should be noted that BAK1 kinase participates in the perception of signals and the regulation of many other PRRs, as well as playing an important role in the regulation of the plant immune response. The phosphorylation of kinase BAK1 has been shown to occur in different amino acid residues [40]. The importance of BAK1 is reinforced by the fact that often this kinase acts as a target for some pathogen effectors, for example, AvrPto and AvrPtoB [41]. Following BAK1 inhibition via these effectors, pattern-triggered immunity is suppressed.

To further transduce the signal, another important component of the PRR complex is activated – Receptor-Like Cytoplasmic Kinase (RLCK) BIK1 (Botrytis-Induced Kinase 1). Mutual trans-phosphorylation of kinase domains BIK1 and FLS2/BAK1, which takes place within 30–60s of signal perception, results in conformational changes; in the long run, phosphorylated BIK1 is released to activate further signal components [42]. Moreover, BIK1 has been shown to trigger a cascade of reactions from several receptor complexes. Along with FLS2 / BAK1, BIK1 receives the signal from EFR and CERK1 [43].

In *Arabidopsis*, BIK1 induces two synchronous cascades of activation in mitogen activated protein-kinases, which contain MKK4/MKK5-MPK3/ MPK6 and MEKK1/MKK1/MKK2-MPK4 [44, 45]. These cascades induce the activation of transcription factors in the WRKY family [46]. WRKY family proteins have a DNA-associating domain of about 60 amino-acids and a conservative region WRKYGQK with a unique zinc-finger domain, with residues of cysteine and histidine. The presence of a DNA-associating domain allows these WRKY trans-factors to interact with cis-elements of the W-box (TTGACC/T motif), which is present in the promoter of defence reaction genes [47]. WRKY transcription factors are involved in the activation of the expression of RBOH-NADPH oxidase providing an oxidative burst in *Nicotiana benthamiana* [48]. Transcription factors, which are under control of WRKY family genes, are found in many plants. They participate in the formation of reactions providing resistance to biotic [49] and abiotic stressors, low temperatures and dehydration in particular [50, 51].

Signal transduction following pathogen stress activates the following defence mechanisms: change in calcium ion concentration, ROS and NO production, expression of defence enzyme genes and production of antimicrobial molecules, such as phytoalexins. At the same time, as part of PTI development, the biosynthesis of Salicylic Acid (SA) or Jasmonic Acid (JAC) and ethylene can be triggered, depending on the type of nutrition and the attack strategies of the pathogen [52].

It should be noted that the mechanism of the first non-specific level of innate immunity accounts for the development in plants of a response to the presence of symbionts and endophytes, as these microorganisms also possess PAMPs/MAMPs.

Recent studies in the field of plant immunity highlight the important role of Post-Translational Modifications of proteins (PTMs) in the processes of rapid cell reprogramming and the regulation of defence responses to the perception of PAMPs [53]. The activity of kinases BAK1 and BIK1 decreases following the action of phosphatases PP2A and PP2C38, which leads to a decrease in the intensity of ROS production [54, 55]. In addition, the activity of kinase complexes in the perception of PAMPs and signal transmission can be regulated with E3 ubiquitin ligases. It is assumed that ubiquitination of receptor kinases is necessary with a prolonged PAMP stimulus to “restart” the cell signaling [56, 57].

Effector-Triggered Immunity – ETI

The second level of plant protection is specific effector-triggered immunity (ETI), which corresponds to the classical theory of “gene-for-gene interaction”. This immunity is conferred through the activity of plant R-proteins, which

recognise a number of pathogen effectors (Avr-proteins) and activate the hypersensitive response, which is fundamentally different from defence reactions in PTI [6, 58].

During evolution, plant pathogens, in their attempts to suppress the immunity of host plant, developed the ability to secrete specific protein molecules – effectors – which are transported directly to the cell through a universal Type 3 Secretion System (TTSS). Effectors of one pathogen are numerous and, presumably, functionally interchangeable. The effectors' target is most often plant kinases, which participate in the perception of molecular patterns and/or signal transfer. Thus, in *Pseudomonas syringae*, effectors AvrPto and AvrPtoB were detected, each targeting the FLS2–BAK1 complex, whose function is described above [59]. Another studied effector, *P. syringae pv. tomato* DC3000 – tyrosine phosphatase HopAO1, acts in plant cells on the kinase domains of FLS2 and EFR, suppressing the development of PTI [60]. It was experimentally established that the activity of effectors may be directed at receptor-like kinases containing a lysine motif (LysM-RK), thus, two tomato kinases – Bti-9 and SlLyk1 – are targets for the AvrPtoB effector [61]. Effectors may also target other defence mechanisms in the plant – the effector Pep1 was shown to exert an effect on apoplastic peroxidase in maize plants, reducing the generation of ROS [62].

Effectors (*Avr*-gene products) are recognised by receptors (products of *R*-genes), which are intracellular nucleotide binding domain leucine-rich repeat domain-containing receptors (NLRs) [5]. Their structure is similar to that of Nod-like receptors in mammals [63] recognising microbial molecular patterns. Both receptors, plant and animal, are intracellular proteins containing central nucleotide-associating domains involved in activation and multimerisation, as well as LRR domains [16]. The group of scientists headed by Y. Kadota demonstrated that R-proteins of mammalian Nod-like receptors and plant NLRs, along with structural similarity, perform analogous functions. It was also established that the stability of these receptors, both in plants and in animals, is regulated by a chaperone complex containing HSP90 (Heat Shock Protein 90) and SGT1 (Suppressor of G-Two Allele of Skp1) [64]. Both NLRs in plants and in animals are classified in accordance with the architecture of the N-terminal domain. Two major classes of cytoplasmatic NLRs are described: CC-type NLRs (CNLs) contain coiled coil motifs and TIR-type NLRs (TNLs) possess N-terminal homology with the intracellular TIR-domain. Homology between the TIR in plant protein receptors and Toll proteins in *Drosophila* and IL-1R (receptor of interleukin-1) in mammals allows the presumption of a universal execution of mechanisms and immune signal transmission pathways for all multicellular organisms. This is one more in a number of pieces of evidence proving the existence of homologous structures and mechanisms in animals and plants, thereby confirming the conservative nature and significance of these processes in the immunity of all organisms.

In plants, effectors may be "recognised" via immediate association with NLRs or indirectly, via adaptor proteins. Such indirect recognition is accounted for by the guard hypothesis, which holds that NLRs are associated with adaptor-proteins acting as targets for microbial effectors. Interaction of effector with adaptor-protein modifies the latter, and as a result is recognised by an R-protein, which in its turn induces the activation of ETI defence reactions [65]. In *Arabidopsis,* the ETI activation system was explored, which confirmed the guard hypothesis. Effectors AvrRpt2, AvrRpm1 and AvrB, products of relevant *Avr*-genes of bacteria *P. syringae pv. tomato* and *P. syringae pv. maculicola*, modify target protein RIN4 (RPM1-Interacting Protein4) in *Arabidopsis*. AvrRpm1 and AvrB modify RIN4 via phosphorylation, and AvrRpt2 via proteolysis. Conformation of the RIN4 protein is checked by receptor proteins RPS2 (Resistance to *Pseudomonas Syringae* 2) and RPM1 (Resistance to Pseudomonas *Syringae pv. Maculicola* 1) – products of respective *R*-genes. Therefore, phosphorylation and proteolysis of adaptor proteins, respectively, defines their recognition by receptors and activates RPM1- and RPS2-dependent immunity in ETI [66].

Within the framework of the "guard" hypothesis another scenario is probable – the so-called "decoy" model – whereby a mediator protein acts not as a target for a pathogen effector, but only as its structural analogue, which competes for association with the effector [67]. This situation is illustrated with the Prf protein, intracellular receptor of tomato plants of NLRs type, which forms a complex with mediator protein kinase Pto. Pto kinase has a structural analogy with kinase domains FLS2 and CERK1, which act as targets for AvrPto and AvrPtoB [68].

In addition to this, there is one more model of interaction between effectors and specific receptors activating ETI – the "bait-and-switch" model. NLRs may associate effectors only after they have formed a complex with a mediator protein. This may be accompanied by dramatic enhancement of the affinity of the "plant mediator protein /effector to R-protein" complex [69].

The mechanisms of further signal transfer conveyed by NLRs have not been completely studied, but the following signal transduction model is proposed: activated NLRs move to the nucleus and immediately interact with transcription factors to trigger the expression of defence genes – NLRs such as tobacco N-protein, barley MLA10 protein (*Hordeum vulgare* L.) and protein RPS4 *Arabidopsis*

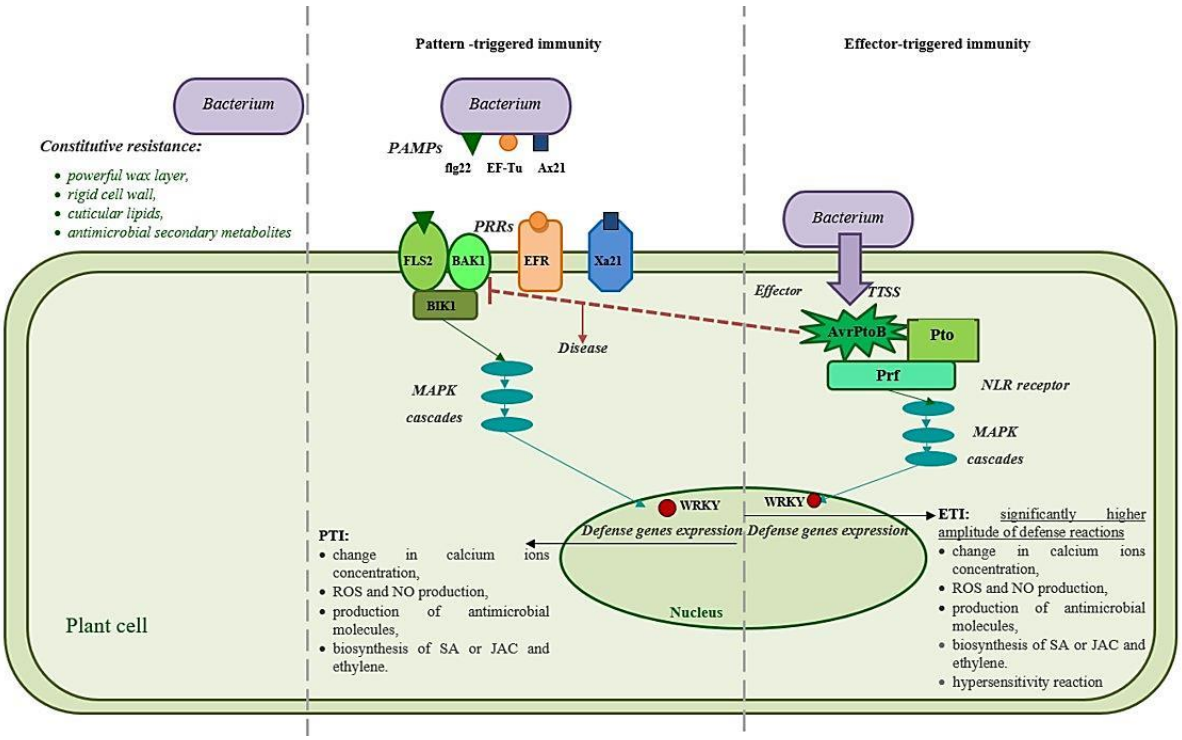
are synthesised in the cell cytoplasm, but in order to function and activate plant defence genes, they need to accumulate in the nucleus [70, 71].

Following NLR activation, effector-activated immunity reactions partially correspond with the reactions of non-specific immunity, including the activation of subsequent MAPK cascades and WRKY transcription factors. This stage of signal transduction can also be a target for effectors; for example, the bacterial effector PopP2 is capable of acetylating certain transcription factors of WRKY, which disrupts their ability to bind to promoters of defence genes [72]. During normal activation of WRKY factors, further molecular events occur, such as a change in ion streams, ROS and NO accumulation in addition to the activation of a transcription of the group of genes that produce PR proteins, for biosynthesis of salicylic and jasmonic acids, as well as ethylene, resulting in the activation of the hypersensitive response and development of systemic resistance [73]. It is important to note that the intensity of analogous molecular events in PTI and ETI differs considerably [74]. Effector-triggered immunity is characterised by a significantly higher amplitude in the development of immune response defence reactions, which, according to some researchers, proves that quantitative differences are more significant for hypersensitive response induction than qualitative differences [6].

The "Zig-zag model" proposed by Jones and Dangl in 2006 reflects a continuous process of improvement in pathogen "attack" strategies and the mechanisms of the plant immune response as a result of evolutionary adaptation during plant-microbe interactions. The model was put forward as an illustration of evolution in the relations within the "*P. syringae* – tomato plant" phytopathosystem (Picture). Perception of bacterial molecular patterns (in this case – flg22) is carried out by a pattern-recognising receptor (FLS2), which is accompanied by its interaction with BAK1 kinase and brings about the development of non-specific pattern-triggered immunity (PTI). In the course of joint evolution, the phytopathogen acquires the ability to secrete effectors into plant cells(in the case of *P. syringae* – AvrPtoB). An important role in this process is played by the type-3 secretion system, which ensures effectors are delivered directly into the cytoplasm, bypassing the cell wall and membrane. It may be assumed that the emergence in pathogens of such a system is conditioned by the necessity to transport effectors directly into the cell to suppress pattern-triggered immunity.

As mentioned above, the AvrPtoB effector is targeted at FLS2–BAK1 complex, which leads to the suppression of pattern-triggered immunity and fosters disease development. In the course of further co-evolution the necessity to survive resulted in emergence in plants of effector detection

"Zig-zag model" of innate immunity system



Evolution in the relations plant-microbe interactions within phytopathosystem
"P. syringae – tomato plant"

Эволюция растительно-микробных взаимоотношений в фитопатосистеме
«P. syringae – растение томата»

systems. Emergence in plants of *R*-genes, whose products are able to recognise the effector and activate defence reactions results in a second specific level of immunity. Resistant tomato plants possessing the product of *R*-genes (Prf kinase) recognise bacterial effectors with the help of an intermediate (Fen kinase) to induce enhanced protection (ETI). To counteract this, *P. syringae* acquires new functions in AvrPtoB, at the expense of developing the E3 ligase domain, which marks Fen for degrading, again resulting in the development of disease. In turn, tomato plants generate a new intermediate protein – Pto kinase – which is resistant to degradation and triggers ETI inducing the hypersensitive response.

It should be noted that long-term joint evolution of plant and pathogen resulted in the development of diverse alternative interaction strategies in both parties. As mentioned above, bacteria are characterised by large effector sets, while plants have “side tracks” both on the level of detection (Fen kinase and Pto kinase), and on the level of signal transduction. The WRKY domain in the NLR RRS1 (Resistance to Ralstonia Solanacearum 1) was shown to be acetylated by the bacterial effector Pop2P, along with the WRKY transcription factors. The authors of the study suggested that during evolution, the WRKY domain was inserted into the protein RRS1 as “bait” for recognising effector activity and subsequently inducing ETI [75].

CONCLUSION

The analysis of contemporary scientific publications in this area, which presents and discusses diverse molecular-genetic aspects of plant immunity taking into account evolution processes, allowed the authors to infer the following. Successful colonisation of plants requires the pathogen, after overcoming the constitutive barriers, to combat the two-layer innate immunity of the plant. The first level is non-specific innate immunity (PTI), based on recognition of PAMPs/DAMPs using PRR. Patterns of plant immune response formed as a result of plant cell wall attack by hydrolytic enzymes of the pathogen. PAMPs are invariant and conservative for a given class of microbes and their changes in the course of evolution seem unlikely as the vast majority are structural elements of the organisms (cell walls, flagella). Therefore, PAMP modifications in the course of evolution may negatively affect pathogen survivability. However, a small number of changes which do not disturb cell structure do exist, giving the pathogen a chance to avoid recognition by the plant via PAMPs.

An important evolutionary acquisition in pathogen strategy is the development of effectors. In order to suppress PRR-mediated defence reactions and PTI some pathogens developed the ability to synthesise effectors and deliver them directly to the plant cell cytoplasm via Type III secretion. Significant modifications of effectors or elimination of one or even several of them will pose no threat to the vital functions of the microbe, as it synthesises a large number of effectors, which are functionally interchangeable and often derived from older effectors

Plants, through the course of their own evolution, developed the ability to detect pathogen effector proteins (or effector-induced changes in the structure/function of their own proteins) via cytoplasmatic receptors, triggering effector-activated specific resistance – ETI – which is the second level of innate immunity characterised by faster and more intense defence reactions to pathogens, as well as development of the hypersensitive response, systemic resistance and immune memory.

The above discussion prompts the conclusion that pathogen virulence factors and plant resistance mechanisms have confronted each other throughout their evolution and are being constantly improved as a result: enhancement of plant resistance to pathogens is a result of pathogens successfully modifying their virulence factors or acquiring the ability to avoid immune detection. In its turn, enhancement of plant resistance to pathogens is a result of successful plant evolution in respect to their ability to recognise the “opponent”, involving various additional molecules and alternative mechanisms (adaptor proteins, co-receptors, ubiquitination mechanisms, etc.). Thus, the plant provides an effective defence response in a timely and adequate manner. Evolutionary processes of plant and pathogen take place together and react to one another. Following testing for feasibility of structures and mechanisms they become permanent during the evolution and are used by various organs even under stresses of a different nature. A detailed study of all the links and elements of the relationship, which determines the establishment of innate immunity, is far from complete. This is particularly true for in plants especially. Studies addressing these issues will be conducted in the near future.

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Contribution

Tat'yana N. Shafikova, Yuliya V. Omelichkina carried out the experimental work. The authors on the basis of the results summarized the material and wrote the manuscript. All authors have equal author's rights and bear equal responsibility for plagiarism.

Conflict of interests

The authors declare no conflict of interests regarding the publication of this article.

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