PLANT IMMUNITY THROUGH THE PRISM OF EVOLUTION: COMPARATIVE ANALYSIS OF RESISTANCE GENES IN MONOCOTYLEDONOUS AND DICOTYLEDONOUS SPECIES

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Abstract. The article explores the evolution of plant immunity, comparing immune responses in monocotyledonous (Monocotyledonae) and dicotyledonous (Dicotyledonae) crops using winter wheat (*Triticum aestivum*) and sunflower (*Helianthus annuus*) as models. It analyzes receptor systems crucial for immune signaling. In dicots, membrane receptors (RLK, RLP) and intracellular NLR receptors with TIR domains (TNL), together with co-receptors RNL (NRG1, ADR1), form the TNL–EDS1–PAD4–SAG101 network responsible for hypersensitive response (HR) to pathogens. In cereals, particularly wheat, TNL and RNL receptors are largely reduced due to evolutionary adaptation to their thick, lignified cell walls, which act as barriers against necrotrophic pathogens. Instead, cereal immunity relies mainly on receptors with CC-NBS-LRR (CNL) domains and RLKs specialized in recognizing biotrophic pathogens. The study highlights that the evolution of plant immune systems reflects the balance between structural adaptations and pathogen pressure. Dicotyledonous plants have retained a full complement of immune components, ensuring resistance to a wide spectrum of pathogens, including necrotrophs and hemibiotrophs. Monocots, by contrast, have compensated for the loss of TNL receptors through the enhanced use of CNL and RLK pathways. Understanding these evolutionary differences is essential for modern biotechnology, crop breeding, and plant protection. The findings provide a theoretical foundation for improving the immune potential of cultivated plants and guide future research in agronomy and molecular biology.

Keywords: biotrophs; dicots; disease resistance receptors; monocots; necrotrophs; pathogens; sunflower; winter wheat.

INTRODUCTION

Agriculture continually faces challenges from phytopathogens – including fungi, bacteria, viruses, and other microorganisms – that cause substantial crop losses and reduce product quality [1, 16, 65].

In the context of global climate change, agricultural intensification, and the emergence of new pathogen races, a comprehensive understanding of plant immunity mechanisms is becoming increasingly vital [28, 67].

Oilseed crops such as sunflowers are particularly affected by a wide spectrum of fungal pathogens, which exert strong evolutionary pressure on plant defense systems [84].

The evolution of the plant immune system reveals profound diversification between the two major taxa: monocotyledons and dicotyledons. These differences arise from adaptations to specific ecological niches and genomic architectures that have shaped the evolution of receptor systems and pathogen recognition mechanisms. The earliest ancestors of angiosperms likely possessed a limited repertoire of PRR- and NLR-like proteins. Throughout evolution, both monocot and dicot species have experienced substantial changes in the diversity of encoded proteins and resistance gene domains [8]. These evolutionary shifts have led to the development of unique immune systems within each plant group, enabling tailored defensive responses to pathogenic threats [25, 52].

The evolution of immune systems in plants is closely tied to environmental changes and the specific types of pathogens they encounter. This has enabled different plant types to develop optimal defense mechanisms against particular threats. The diversification of immune systems across plant groups reflects complex evolutionary adaptations to various pathogens, shaping their ability to resist diseases and survive under diverse environmental conditions. Therefore, a comparative analysis of immune responses in monocots and dicots, which differ significantly in their morphological and physiological traits, as well as in the structural and functional characteristics of their disease resistance genes, is particularly important [56, 57].

This includes both the evolutionary foundations and the structural-functional distinctions between the two primary classes of plant immune receptors: intracellular NLRs and membrane-bound PRRs [6].

The evolution of immune genes in different plant groups is often accompanied by extensive gene duplications, whole-genome duplications (WGD), and local amplifications of resistance genes, facilitating the formation of gene clusters. These clusters serve as important centers for the generation of novel pathogen-specificities [22, 46, 47, 54, 91].

In dicotyledonous plants, particularly in *Arabidopsis thaliana* and other members of the Brassicaceae family, the evolution of immune genes has been accompanied by several large-scale genome duplications [19]. A classic example is the resistance gene cluster, especially the TNL receptor cluster in *Arabidopsis*. Numerous instances of gene duplication and amplification have been identified, contributing to an expanded capacity for pathogen recognition, particularly through effector-triggered immunity (ETI) mechanisms. These genome duplications also enhance genetic variation within populations, enabling plants to

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adapt more effectively to emerging pathogens [39, 71, 81].

Genome doubling and local amplification of resistance genes have also been documented in monocotyledonous plants, including *Oryza sativa* (rice), *Zea mays* (maize), and *Triticum aestivum* (wheat). In these species, whole-genome duplications (WGD) and local amplifications of RLK and CNL receptor genes have significantly enhanced their capacity to recognize biotrophic and hemibiotrophic pathogens, such as *Magnaporthe oryzae*, the causative agent of rice blast disease. In some cases, complex resistance gene clusters have been identified, where certain genes are involved in pathogen recognition while others are responsible for triggering the corresponding immune responses. Whole-genome duplication and local gene amplification serve as key mechanisms of evolutionary adaptation, representing critical stages in the development of plant immunity. These processes not only help preserve the essential functions of immune receptors but also actively contribute to the emergence of novel specificities, thereby enabling plants to combat a wide range of pathogens effectively [4, 27, 40, 90].

Plant pathogens exhibit a wide range of life strategies. Bacteria proliferate in the apoplast – the intercellular space – which they access through natural openings (such as stomata) or via mechanical damage to plant tissues. Nematodes and aphids feed by inserting their stylets directly into plant cells. Fungi may penetrate epidermal cells or spread their hyphae across the surface, intercellularly, or even intracellularly. Some pathogenic and symbiotic fungi, as well as oomycetes, form specialized feeding structures called haustoria that invaginate the host cell's plasma membrane. At the site of interaction, the haustorium, extracellular matrix, and host membrane establish a highly complex interface. Regardless of the pathogen type, all of them deliver effector molecules (virulence factors) into the plant cell to suppress host defence mechanisms and facilitate infection [33, 58, 77-79].

Phytopathogens vary significantly in their feeding modes, infection strategies, and interactions with plant cells. Based on these characteristics, they are classified into three main groups: biotrophs, necrotrophs, and hemibiotrophs. Biotrophs (e.g., *Blumeria graminis, Puccinia* spp.) feed on living plant cells by forming specialized structures such as haustoria. They avoid triggering host cell death and instead manipulate host metabolism to maintain cell viability. Necrotrophs (e.g., *Botrytis cinerea, Fusarium* spp.) induce cell death by releasing toxins, hydrolytic enzymes, or triggering apoptosis, and subsequently feed on the dead tissue. Their aggressive infection strategy relies on the active destruction of host cellular structures. Hemibiotrophs (e.g., *Zymoseptoria tritici, Colletotrichum* spp.) employ a mixed strategy: during the initial phase of infection, they behave like biotrophs, later transitioning to a necrotrophic lifestyle. This flexibility enables them to evade early plant immune responses [18, 26, 55, 57].

The plant immune system is a multilayered and highly dynamic system. The first line of defence is PAMP-triggered immunity (PTI), which is initiated upon recognition of pathogen-associated molecular patterns (PAMPs) by specialized membrane-bound pattern recognition receptors (PRRs) [13]. If pathogens succeed in circumventing this barrier, a second layer of defense is activated – effector-triggered immunity (ETI), mediated by intracellular receptors encoded by resistance (R) genes. In addition to these mechanisms, plants also possess non-specific (horizontal) resistance, which is associated with a range of physiological and biochemical processes, including the activity of transporters, regulators of programmed cell death, and stress response factors [14, 68, 85, 88]. In addition to receptor-mediated immunity, plants activate downstream biochemical defense mechanisms, including the biosynthesis and accumulation of phytoalexins - inducible antimicrobial secondary metabolites produced following PTI/ETI activation. Although phytoalexin biosynthesis pathways were beyond the primary receptor-centered scope of this review, they represent an important complementary component of plant resistance against pathogens.

This study aims to compare the evolution of immune mechanisms in monocotyledonous and dicotyledonous plants, with a focus on pathogen–cell interaction types, domain organization of immune receptors, and the structure of encoded proteins. Winter wheat (*Triticum aestivum*) and sunflower (*Helianthus annuus*) were used in this review as representative model species to illustrate major evolutionary trends in immune system organization between monocotyledonous and dicotyledonous plants, respectively. However, some immune traits discussed herein may be more characteristic of Poaceae species rather than universally applicable to all monocots, whereas sunflower-derived observations should be interpreted as model-based tendencies rather than universal features of all dicotyledonous plants.

The analysis is based on bioinformatic data regarding genetic resistance sources in winter wheat and sunflower to major diseases. This research examines the complex and dynamic process of immunity development in winter wheat cultivars and sunflower hybrids, considering interactions with diverse pathogen types, including biotrophs, necrotrophs, and hemibiotrophs. Scientific exploration of this process necessitates a detailed analysis of the evolution of both structural and inducible immunity, with particular emphasis on the molecular mechanisms underlying pathogen recognition and the activation of defense responses. In our study, we presented genes responsible for winter wheat and sunflower resistance, which demonstrate diverse mechanisms of plant defense against pathogens. These genes include both specific and nonspecific components of the immune response, which allow plants to respond effectively to

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different types of pathogens, such as biotrophic, necrotrophic, and hemibiotrophic pathogens [31, 34].

For the comparative analysis of winter wheat and sunflower immunity based on structural and functional considerations and characterization of encoded proteins and domain type, known pathogen resistance genes covering different types of trophic interaction were used: biotrophic, hemibiotrophic, necrotrophic, viral, and bacterial pathogens. Genes were searched and annotated based on open-access publications, NCBI, Ensembl Plants, PRGdb, as well as Pfam and InterPro databases to determine the domain structure of proteins. We also took into account the classification of immune genes by domain type (TNL, CNL, RLK, RLP, etc.), immune response type (PTI, ETI), and mechanism of action (HR, antioxidant response, transporters, etc.) [41, 62, 66, 70].

The genes were grouped by pathogen type (biotrophs, hemibiotrophs, necrotrophs, viruses, bacteria), which allowed for a systematic comparison of immune strategies in wheat and sunflower. For each gene, the following were determined: type of resistance (race-specific / general/partial/polygenic); immune response (ETI, PTI, systemic resistance, etc.); domain type (TNL, CNL, RLK, RLP, WRKY, NAC, etc.); functional role of the protein; mechanism of immune response (effector recognition, transportation, barrier action, antioxidant activity). The data were presented in the form of tables for visual comparison (Tables 1, 2). Below are tables containing information on some resistance genes, their domains, functions, and mechanisms of action used for bioinformatic analysis of genetic resistance sources. The results of the analysis formed the basis for building a comparative model of the immune response of winter wheat and sunflower against the main types of pathogens [21, 64].

Table 1. Pathogen resistance genes of winter wheat: examples of pathogens, domain type, and protein function

Pathogen type	Resistance gene	Pathogen	The type of resistance	The immune response	The type of domain	The function of protein	The mechanism of the immune response
0	**1**	**2**	**3**	**4**	**5**	**6**	**7**
Biotrophs	Sr31	*Puccinia graminis f. sp. tritici*	Race specific	ETI, HR	CNL	Recognizing the effectors	Recognition of effectors through HR (hypersensitivity) activation
Biotrophs	Sr50	*Puccinia graminis*	Race specific	ETI	CNL	Effector recognition AvrSr50	Interaction with the AvrSr50 effector to activate HR
Biotrophs	Lr26	*Puccinia triticina*	Race specific	ETI	CNL	Effector recognition	Activation of response through effector recognition
Biotrophs	Pm3	*Blumeria graminis f. sp. tritici*	Race specific	ETI/HR	CNL	Effector recognition AVRPM3	Activates HR for plant defense
Biotrophs	Pm60	*Blumeria graminis*	Race specific	ETI	CNL	Effector recognition	The AVRPM3 effector triggers HR
Biotrophs	Sr57 (Lr34/Yr18)	*Puccinia graminis / Blumeria graminis*	General	Partial resistance	ABC transporter	Transport of metabolites and phytohormones antibiotic activity	Prevention of pathogen spread, resistance to multiple diseases
Biotrophs	Lr67	*Puccinia triticina, Blumeria graminis*	General	Delay in pathogen development	SLC (glucose transporter)	Disruption of glucose transport, inhibition of pathogen growth	Protects by inhibiting pathogen metabolism
Hemibiotrophs	Stb6	*Zymoseptoria tritic*	Race specific	ETI	RLP	Recognition of the AvrStb6 effector	Recognition of the AvrStb6 effector, activation of a local response
Hemibiotrophs	Stb16q	*Zymoseptoria tritic*	Race specific	ETI	CNL	Recognition effector	Stimulates local defense mechanisms
Hemibiotrophs	Stb17	*Zymoseptoria tritic*	Partial resistance	ETI + PTI	CNL	Complex response to the pathogen	Coordinated activation of effectors for a deeper response
Necrotrophs	Sb1	*Bipolaris sorokiniana*	Nonspecific	PTI	RLK	PAMP recognition	Initiation of primary defense through receptors
Necrotrophs	Fhb1	*Fusarium graminearum*	Partial resistance	Without HR; barrier defense	NB-LRR / WRKY	Cell wall reinforcement, delayed penetration	Activation of the antioxidant system

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Table 1. Pathogen resistance genes of winter wheat: examples of pathogens, domain type, and protein function (continued)

0	1	2	3	4	5	6	7
Necrotrophs	Fhb7	*Fusarium graminearum*	General	DON detoxification	GST	Detoxification of the mycotoxin DON	–
Necrotrophs	TaNAC032	*Fusarium graminearum*	General	Antioxidant response	NAC	Induction of defense genes	–
Viruses	Bdv2	*Barley yellow dwarf virus*	General	Limitation of spread	NLR (likely)	Formation of a phloem barrier	Prevention of viral spread within the plant
Viruses	Cmc4	*Wheat streak mosaic virus*	General	Suppression of viral replication	NPR (regulator)	Recognition of viral proteins	Ensuring viral detection and inhibition of replication
Bacteria	TaNPR1	*Pseudomonas syringae, Xanthomonas translucens*	General	Systemic resistance	NPR (regulator)	Induction of PR genes via SA signaling	Mechanism of systemic plant defense
Bacteria	TaRPM1	*Pseudomonas syringae*	Partial resistance	ETI	CNL	Recognition of bacterial effectors	Activation of local immune response
Bacteria	QTL 3B, 6D	*Pseudomonas, Xanthomonas*	General	Polygenic	–	Enhancement of physiological resistance	Stability against bacterial infections

Note: The data in the table on resistance genes were taken from the NCBI, Ensembl Plants, PRGdb databases, and Krattinger *et al.* (2019) [37].

Table 2. Sunflower resistance genes against pathogens

Pathogen type	Resistance gene	Pathogen	The type of resistance	The immune response	Domain type	Protein function	Immune response mechanism
Biotrophs	Pl5	*Plasmopara halstedii*	Race-specific	ETI	TNL	Recognition of the pathogen effector	Activation of HR via effector-dependent detection
Biotrophs	Pl8	*Plasmopara halstedii*	Race-specific	ETI	TNL	Recognition of pathogenic proteins	Initiation of local hypersensitive response
Biotrophs	PlARG	*Plasmopara halstedii*	Race-specific	ETI	TNL	Recognition of the AvrPlARG effector	Triggering of HR through specific interaction
Necrotrophs	HaNAC1	*Alternaria helianthi*	General	Antioxidant	NAC	Transcription factor regulating defense genes	Activation of ROS-dependent defense
Necrotrophs	HaWRKY10	*Macrophomina phaseolina*	General	JA/ET-dependent	WRKY	Regulation of gene expression under stress	Activation of JA/ET-dependent signaling pathways
Hemibiotrophs	HaPR5	*Phoma macdonaldii*	Partial	PTI	Thaumatin-like	Osmotic lysis of fungal cells	Weakening of the pathogen cell wall
Hemibiotrophs	HaHSF3	*Sclerotinia sclerotiorum*	Partial	Antioxidant	HSF-type DBD	Transcriptional activation of antioxidant genes	Reduction of oxidative stress in cells
Viruses	HaTIR1	*Sunflower mosaic virus*	General	PTI	F-box	Involvement in protein degradation	Auxin-mediated defense, hypothetically
Bacteria	HaPR1	*Pseudomonas syringae*	General	SA-dependent	PR1	Activity against bacteria	Induction of defense genes involving salicylic acid
Bacteria	QTL 10LG, 13LG	*Xanthomonas campestris*	Polygenic	Polygenic	QTL	Complex physiological resistance	Enhancement of cell wall barrier function and oxidative defense

Note: The data in the table on resistance genes were taken from the NCBI, Ensembl Plants, and PRGdb databases.

Structural and functional organization of immunity receptors in wheat and sunflower

The plant immune system is based on two main levels of defense: recognition of pathogen-associated molecular patterns (PAMPs or PTI) and recognition of pathogen effectors (ETI) [50].

Accordingly, receptors are divided into two major groups: surface-localized receptors (RLK, RLP) and intracellular receptors with NB-ARC domains (CNL, TNL, RPW8-NL) [8, 75]. In monocot and dicot plants, these receptors have distinct structures, evolutionary origins, pathogen specificities, and signaling mechanisms [7, 30, 48].

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Our generalization on the evolution of immunity mechanisms in monocots and dicots, based on a structural-functional analysis and characterization of resistance gene-encoded proteins and domains in winter wheat and sunflower, is presented in Table 3. It logically explains the structural, functional, and evolutionary aspects of plant immunity receptors, focusing on differences between monocots (winter wheat) and dicots (sunflower). The table includes five main types of plant receptors that play a key role in defending against pathogens. Each type has unique features, advantages, and limitations, as well as varying prevalence between monocots and dicots [57].

Overall, CNL and RLK receptors are universal and present in both plant groups, underscoring their importance in the plant immune system. At the same time, TNL, RPW8-NL, and RLP receptors are more common in dicots and are limited in monocots. This reflects evolutionary adaptations of plants to different environmental conditions and pathogens and is linked to differences in the structure of the cell wall and epidermis between monocot and dicot species. It's also worth noting that the differing activation mechanisms, functional specificities, and the contrasting epidermal and cell wall structures may influence their effectiveness in pathogen defense [66].

Comparative studies on the structure of encoded proteins and domain types in resistance genes of winter wheat and sunflower showed that CNLs are the most universal cytosolic receptors, found in all higher plants. TNL and RPW8-NL cytoplasmic receptors are specific to dicots and are absent in grasses due to the loss of the EDS1-dependent signaling system. RLK membrane receptors are conserved and found in both dicots and monocots. On the cell surface, RLP receptors are more frequent in dicots, likely due to the evolutionary expansion of functional partners [10, 29, 49, 61].

RLK receptors are critical for PTI (pattern-triggered immunity) in grasses due to their structural advantages and functional features. RLK (Receptor-Like Kinases) receptors possess several key characteristics that enable them to effectively recognize PAMPs (pathogen-associated molecular patterns). Their structure includes: an extracellular LRR domain that directly recognizes PAMPs, such as flagellin, chitin, or peptides; a transmembrane segment that integrates the receptor into the cell membrane; and a cytoplasmic kinase that activates a signaling cascade, initiating the immune response. One of the main advantages of RLKs is their ability to independently initiate a primary immune response (PTI) without the need for co-receptors, which distinguishes them from RLP receptors that require additional co-receptors for PTI activation [50].

In grasses, RLK receptors play a particularly critical role due to the large number of functional RLKs specialized in recognizing various types of PAMPs, such as flagellin, chitin, or peptides. Grass cells have dense, multi-layered cell walls rich in cellulose, which makes pathogen penetration more difficult. This means that grasses benefit from a strong surface immune response, such as PTI, which enables early detection and restriction of pathogen development. An example in winter wheat is the FLS2 receptor, belonging to the RLK class. This receptor recognizes a fragment of bacterial flagellin – flg22, a conserved PAMP protein in many bacterial pathogens, including *Pseudomonas syringae*. In response to flg22 recognition, FLS2 in wheat initiates phosphorylation of signaling proteins, activating PTI – the primary

Table 3. Comparative analysis of immunity receptors in monocot (winter wheat) and dicot (sunflower) plants (with examples and evolutionary distribution) based on the structure of encoded proteins and domain types of disease resistance genes

Receptor Type	Domains	Most Effective Against	Main Plant Group	Key Partner Proteins	Reason for Advantage / Limitation	Examples	Distribution: Monocots	Distribution: Dicots
CNL (CC-NLR)	CC, NB-ARC, LRR	✅ Biotrophs, Hemibiotrophs	Universal	-	The most common, autonomous activation of ETI	*RPS2*, *RPM1* (Arabidopsis thaliana; *R3a* (Potatoes); *Sr33*, *Sr50* (Wheat)	Present	Present
TNL (TIR-NLR)	TIR, NB-ARC, LRR	✅ Biotrophs	Dicots	EDS1, PAD4, SAG101	Absent in cereals, dependent on signaling partners	*RPP1*, *RPP4* (Arabidopsis thaliana)	Absent (lost)	Present
RPW8-NL	RPW8, NB-ARC, LRR	✅ Biotrophs, partially Hemibiotrophs	Dicots	EDS1, іноді NRG1	Rare in cereals; RPW8 - "proton-like" domain	*ADR1*, *NRG1* (Arabidopsis thaliana)	Very rare/absent	Present
RLK	LRR, TM, кіназний домен	✅ Necrotrophs, Hemibiotrophs	Universal	-	Independent signaling capability, critical for PTI	*XA21* (rice), *FLS2*, *EFR* (Arabidopsis thaliana)	Present (very important)	Present
RLP	LRR, TM	✅ Biotrophs, Hemibiotrophs	More commonly dicots	SOBIR1, BAK1	Require co-receptors, flexible specificity	*Cf-4*, *Cf-9*, *Ve1* (Tomatoes)	Rare / poorly studied	Present (highly active)

Note (Explanation of Evolutionary Distribution Reasons): 1. CNLs dominate in grasses because they do not require partner proteins (such as EDS1), and ETI is maintained even with the loss of the TNL/EDS1 system. 2. TNL-associated signaling pathways are substantially reduced or modified in most grasses compared with dicots, which likely contributed to the strong reduction of TNL and RPW8-NL receptor repertoires. These signaling systems are either lost or inactive in monocots. 3. RLKs are crucial for PTI in grasses because their kinase domain is active, allowing them to initiate signaling independently, without co-receptors (e.g., XA21 in rice). 4. RLPs dominate in dicots because dicots possess a rich co-receptor system (SOBIR1, BAK1), enabling RLPs to trigger highly specific signaling (e.g., the Cf-system in tomatoes).

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immune response, which includes the production of reactive oxygen species (ROS), reinforcement of the cell wall, and the expression of defense genes [63].

LP receptors are specific to dicots because they are membrane receptors that contain only an extracellular recognition domain (typically LRR) and a transmembrane region, but lack a cytoplasmic kinase domain, and therefore cannot independently transmit a signal. To activate a defense response, RLPs require co-receptors from the RLK family, such as SOBIR1 or BAK1, which provide phosphorylation of signaling proteins. Evolutionarily, dicot plants have developed a highly modular PRR system that includes a wide range of RLPs and co-receptors, allowing them to flexibly respond to a variety of apoplastic pathogens. Additionally, the thinner and more structurally variable cell wall in dicots acts as a weaker barrier compared to grasses and is more easily affected by pathogens that release effectors into the apoplast. This likely contributed to the enhanced role of RLP receptors in dicots as a means of rapid recognition of external danger signals. An example in sunflower is the HaRxL23 gene, which encodes a protein recognized by an RLP-like receptor in interaction with the SOBIR1 co-receptor. This system enables recognition of effectors from the pathogen *Plasmopara halstedii*, the causative agent of downy mildew. As a result, the PTI response is activated, including cell wall strengthening, ROS formation, and expression of defense genes, aimed at limiting pathogen invasion in the apoplast [32].

In cereal crops like wheat, CNL-type receptors (CC-NLRs) have become the key foundation of effector-triggered immunity (ETI) due to their structural autonomy and evolutionary adaptation. Structurally, CNL receptors consist of: an N-terminal coiled-coil (CC) domain – initiating the immune response; a central NB-ARC domain – binding and hydrolyzing ATP/GTP for activation; and a C-terminal LRR domain – responsible for specific recognition of pathogen effectors. In contrast to TNL receptors, which require partner proteins such as EDS1, PAD4, or SAG101 to transmit a signal, CNLs function independently of these cofactors. This is critical for grasses, which, through evolution, have lost or retained only non-functional copies of these signaling components. Evolutionarily, this loss has led to CNLs becoming the dominant (and often the only) class of intracellular receptors capable of initiating ETI in grasses. Due to their autonomy, they provide a fast and efficient response to biotrophic pathogen intrusion. Examples include CNL genes Sr33 and Sr50, which encode receptors in winter wheat that confer resistance to the stem rust pathogen (*Puccinia graminis*), activating a localized hypersensitive response and blocking pathogen development [43].

TNL and RPW8-NL receptors are rare or absent in monocot plants, particularly in grasses, due to their structural dependence on specific signaling components and evolutionary constraints related to the reduction of key partner proteins. TNL receptors (TIR-NLRs) contain a TIR (Toll/Interleukin-1 Receptor-like) domain at their N-terminus, which is unable to initiate an immune response on its own. For full functionality, TNLs require specific partner proteins - EDS1, PAD4, and SAG101 - which form a signaling complex to transmit the presence of a pathogen [5].

A similar situation applies to RPW8-NL receptors, which also depend on components of the EDS1 signaling pathway, particularly the NRG1 and ADR1 proteins, and do not function autonomously. In our view, monocot plants (including all cereal crops) underwent an evolutionary modification and partial reduction of components associated with TNL signaling, likely rendering TNL and RPW8-NL receptors non-functional or obsolete. As a result, these receptor classes have either disappeared from their genomes or remain as non-functional copies. In contrast, dicots have retained a full set of signaling components, which has enabled the development of a broad repertoire of TNL and RPW8-NL receptors [59].

An example in sunflowers is the HaTNL1 receptor, which belongs to the TNL class and contributes to effector-triggered immunity (ETI) against the biotrophic pathogen *Plasmopara halstedii*, the causal agent of downy mildew. HaTNL1 contains a typical TIR domain, and its function is mediated through an EDS1-dependent signaling pathway, similar to TNLs in other dicot species. Additionally, sunflower has been found to contain ADR1/NRG1 homologs - components of the RPW8-NL signaling system - which are activated upon pathogen recognition and enhance the ETI response. These genes are actively expressed in response to pathogen infection, especially in resistant lines. Thus, sunflower, as a typical dicot species, possesses functional TNL and RPW8-NL systems, which provide effective control of biotrophic pathogens through interaction with signaling partners EDS1, PAD4, and NRG1 [21].

Evolutionary differentiation of the immune system in monocots and dicots: structural and functional features of receptors and plant defense strategies.

To explain the identified features - why RLK receptors are critical for PTI in grasses, why RLP receptors are specific to dicots, why CNL (CC-NLR**)** receptors form the basis of immunity in grasses, and why TNL and RPW8-NL receptors are rare or absent in monocots - it is necessary to consider the morpho-anatomical differences between plant types, the structure of the cell wall**,** the nature of pathogens, and the type of immune response that has evolved. The study of the structural and functional organization of receptor proteins and the domains of disease resistance genes reveals a deep differentiation of the immune system in monocot and dicot plants. This process is not uniform; it depends on environmental conditions, pathogen pressure, and the functional needs of the cell [17].

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To better understand these evolutionary trends, we analyzed the immune strategies of winter wheat (monocot) and **s**unflower (dicot) as model species that interact with different trophic groups of pathogens - biotrophs, necrotrophs, and hemibiotrophs**.** This comparative approach allows us to uncover the dynamics of both structural and induced immunity at the molecular level, and to highlight the relationships between receptor type, cell architecture, signal transduction pathways, and the evolution of the plant immune system. Understanding these mechanisms is crucial for developing resistant cultivars and hybrids capable of effectively withstanding various pathogens, as well as for designing novel approaches in plant protection [57].

We conducted a comparative analysis of immune receptors in monocots and dicots**,** focusing on pathogen types (biotrophs, necrotrophs, hemibiotrophs) and their interactions with plant cells, explaining why certain receptors are more effective against specific pathogens. Table 4 presents different types of plant immune receptors, their effectiveness against pathogens, and their functional features in monocot and dicot species. The data in the table demonstrate the key roles of different receptor types in defending against biotrophic, necrotrophic, and hemibiotrophic pathogens. The diversity of these receptors across monocot and dicot plants reflects evolutionary defense strategies and helps to elucidate their functional mechanisms. However, it is important to note that some receptors, such as TNL and RPW8-NL, are limited to dicot plants, which may be critical for resistance gene selection and the development of new plant varieties [64].

Analysis of the data in Table 4 revealed that cytosolic receptors CNL (CC-NLR) and TNL (TIR-NLR) are the primary defense mechanisms against biotrophic pathogens. Both types recognize intracellular effectors and activate a hypersensitive response (ETI), halting pathogen development at early stages. The RPW8-NL receptor**,** which also activates defense against biotrophs, is specific to dicot plants and is associated with anti-haustorial activity, which is important for defending against pathogens that form haustoria. RLK (Receptor-Like Kinase) and RLP (Receptor-Like Protein) are membrane-bound receptors that recognize necrotrophs and hemibiotrophs by interacting with pathogen-associated molecular patterns (PAMPs) such as chitin and flagellin, initiating pattern-triggered immunity (PTI)**.** This leads to cell wall reinforcement and oxidative stress activation**.** Unlike RLKs, RLP receptors require co-receptors for full activity, which enhances their effectiveness in dicots [79].

TNL and RPW8-NL receptors are specific to dicots, indicating evolutionary differences in immune mechanisms between monocots and dicots. This limits the defense spectrum of monocot plants, particularly against pathogens that deploy effectors requiring these receptors for recognition. In contrast, CNL and RLK receptors are present in both monocots and dicots**,** suggesting a more universal role in protecting against various pathogen types. Thus, examining receptors in terms of their effectiveness against pathogen types shows that cytosolic NLR receptors (CNL, TNL, RPW8-NL) are critically important for biotrophic pathogens**,** which rely on living host cells. The loss of TNL and RPW8-NL receptors in grasse**s** has made them dependent on CNLs for ETI. At the same time, membrane-bound PRR receptors like RLKs are essential against necrotrophs, where an early membrane response is critical to halt pathogen progression. RLP receptors**,** which function in dicots – usually in tandem with RLKs – provide sensitive recognition of apoplastic effectors [51].

In contrast to infections caused by either biotrophs or necrotrophs, plants require combined immune defenses against hemibiotrophic pathogens – RLK/PTI at early stages, followed by NLR/ETI at later stages. The analysis of receptor effectiveness at the cellular level demonstrates that membrane and cytosolic classes represent the multi-layered structure of the plant immune system, where PTI serves as the baseline barrier, and ETI acts as a powerful, amplified mechanism to combat adapted pathogens. This interaction is often described as "immune cascade feedback," where successful pathogen penetration despite PTI activates ETI as a second line of defense [92].

The first defense line (membrane receptors RLK and RLP) initiates early cellular responses, such as ROS production, cell wall reinforcement, and defense gene expression (PR genes). The second line of defense (cytosolic receptors CNL, TNL, RPW8-NL) initiates localized programmed cell death, which restricts pathogen spread through a hypersensitive response (HR) (see Table 5).

Table 4. Comparison of receptors in terms of their effectiveness against different pathogen types

Receptor Type	Most Effective Against	Reason	Presence in Monocots / Dicots
CNL (CC-NLR)	Biotrophs, hemibiotrophs	Recognize effectors acting inside the cell; activate ETI (hypersensitive response), halt pathogen development at early stages	Present in both
TNL (TIR-NLR)	Biotrophs	Similar to CNLs but function only in dicots; activation via the EDS1–PAD4 complex is required for full ETI	Absent in monocots / present in dicots
RPW8-NL	Biotrophs, partially hemibiotrophs	Specific to dicots; associated with anti-haustorial activity - defense against haustoria-forming pathogens	Absent in monocots / present in dicots
RLK	Necrotrophs, hemibiotrophs	Recognize PAMPs (e.g., flagellin, chitin); initiate PTI, trigger oxidative stress, and cell wall reinforcement	Present in both
RLP	Biotrophs, hemibiotrophs	Recognize apoplastic effectors; require co-receptors (BAK1, SOBIR1) to activate immune response; especially important in dicots	Limited in monocots / widely represented in dicots

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Table 5. Comparison of receptor effectiveness against pathogens at the cellular level

Receptor type	Localization	Role at the cellular level
CNL, TNL, RPW8-NL	Cytoplasm/nucleus	Detect effectors introduced by the pathogen into the cell → trigger a hypersensitive response (HR): programmed death of infected cells
RLK, RLP	Plasma membrane	Detect PAMPs or apoplastic effectors at the cell surface → activate PTI: cell wall reinforcement, ROS production, and defense gene expression

The data presented in Table 5 help illustrate how different receptors recognize pathogens at the cellular level and how their roles in defense vary depending on the type of plant. CNL, TNL, and RPW8-NL receptors are localized in the cytoplasm and nucleus, where they detect pathogen effectors that have entered the cell. These receptors trigger a hypersensitive response (HR), resulting in the programmed cell death of infected cells. While CNL receptors are common to both plant groups, TNL and RPW8-NL receptors are restricted to dicots. RLK and RLP receptors, located on the plasma membrane, detect PAMPs or apoplastic effectors acting at the cell surface. They activate pattern-triggered immunity (PTI), which involves cell wall reinforcement, ROS production, and defense gene expression. These receptors are found in both monocots and dicots [86, 87].

To provide a holistic view of plant immunity in the context of pathogenesis, we constructed a logical defense cascade: "pathogen type → receptor type → cellular response," explaining why specific immune pathways are activated depending on the biology of the pathogen and the plant cell. Biotrophic pathogens feed on living cells, penetrate slowly, often form haustoria to absorb nutrients, and suppress host defenses via effectors. Since biotrophs benefit from host cells staying alive, the plant counters by inducing localized self-destruction (HR) to stop pathogen progression. The defense cascade for biotrophs is as follows: A biotrophic pathogen releases effectors that enter the plant cell. These effectors are recognized by intracellular NLR receptors such as CNL, TNL, or RPW8-NL. Recognition triggers effector-triggered immunity (ETI), which includes a hypersensitive response (HR). This results in localized cell death, restricting pathogen spread. Simultaneously, PR genes (pathogenesis-related) are activated, enhancing the plant's immune defense and halting pathogen development [3].

Examples of such defense mechanisms include:

- Wheat's interaction with the biotrophic pathogen *Puccinia* spp. (causal agent of rust), recognized by intracellular CNL-type receptors, such as the Sr33 and Sr50 resistance genes [42].
- Arabidopsis thaliana's defense against *Erysiphe* spp. (causal agent of powdery mildew), characterized by

  recognition involving the RPW8 gene, which encodes a protein with an RPW8-NL domain [82]
- (see Table 6).

Unlike biotrophs, necrotrophic pathogens secrete toxins and CWDEs (cell wall-degrading enzymes) to kill plant cells and feed on the dead tissue. In this case, HR (hypersensitive response) only facilitates infection by the necrotrophic fungus, so the plant should not activate ETI. Instead, an early surface-level defense response is required – such as cell wall reinforcement, ROS production, lectin activation, and others. The defense cascade against necrotrophs begins when the pathogen releases PAMPs (pathogen-associated molecular patterns) such as chitin or flagellin. These molecules are recognized by membrane-bound receptors, particularly RLKs (e.g., FLS2, which detects flagellin), or RLPs in combination with co-receptors like SOBIR1. This recognition triggers PTI (pattern-triggered immunity), which includes the generation of reactive oxygen species (ROS), lignins, callose, and antimicrobial proteins. This mechanism protects the plant by preventing pathogen entry and limiting its development [76].

For example, the pathogen *Botrytis cinerea* releases flagellin, which is recognized by the FLS2 receptor (an RLK type) on the plant's plasma membrane. This triggers PTI, including ROS production, lignification, and the accumulation of antimicrobial molecules, which restrict the pathogen's further spread. Another example is *Sclerotinia sclerotiorum*, which interacts with the RLP23 receptor, requiring the SOBIR1 co-receptor to activate PTI. This response enhances plant protection by strengthening cell walls and producing antimicrobial proteins to help contain the disease [53].

In contrast, hemibiotrophic pathogens initially behave like biotrophs (evading immunity), then transition to a necrotrophic phase. During the early infection stages, PTI is essential to halt pathogen entry. If the pathogen progresses to its necrotrophic stage, ETI is activated via intracellular effectors. The defense cascade against hemibiotrophs proceeds as follows: 1.

Table 6. Types of pathogens and their interaction with plant cells

Pathogen type	Feeding mechanism	Examples	Mode of entry	Plant immune strategy
Biotrophs	Feed on living tissues	*Erysiphe graminis*, *Puccinia* spp.	Appressorial invasion, from haustoria	Effector recognition → ETI via NLR receptors
Necrotrophs	Induce cell death	*Botrytis cinerea*, *Fusarium* spp.	Secretes toxins, enzymes	PTI, oxidative burst, cell wall reinforcement
Hemibiotrophs	Initially biotrophic, later necrotrophic	*Zymoseptoria tritici*, *Colletotrichum* spp.	Sequential phase transition, complex strategy	Combined PTI/ETI response

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In the early phase, the pathogen releases PAMPs, which are recognized by RLK or RLP receptors on the cell surface. This triggers PTI, including cell wall strengthening, ROS production, and activation of antimicrobial molecules; 2. In the later phase, the pathogen releases effectors that are recognized by intracellular NLR receptors, primarily of the CNL type. This initiates ETI, leading to hypersensitive response (HR), localized cell death, and activation of PR genes. As a result, the combined immune response involves cell wall reinforcement, HR, and defense gene expression, helping to limit pathogen development [89].

Examples of such mechanisms include: 1. Infection by *Zymoseptoria tritici* triggers PTI in wheat during the early stages of infection through PAMP recognition by RLK or RLP receptors; 2. Infection by *Colletotrichum* spp., which releases effectors recognized by intracellular CNL receptors in dicot plants, initiates ETI, HR, and PR gene expression as part of the plant's defense response. A comparative summary of immune responses in monocot and dicot plants to different types of pathogens is presented in Table 7. The data illustrate the differences in plant immune mechanisms against various pathogen types – biotrophs, necrotrophs, and hemibiotrophs – taking into account receptor types, cellular responses, and the presence of these mechanisms in monocot and dicot species [69].

In response to biotrophic pathogens, plants activate effector-triggered immunity (ETI), which involves a hypersensitive response (HR) and the activation of salicylic acid (SA)-dependent pathways. In this defense process, intracellular NLR receptors (CNL, TNL, RPW8-NL) play a central role. However, TNL genes are found only in dicots, which limits the range of ETI responses in monocots. In response to necrotrophic pathogens, plants rely on pattern-triggered immunity (PTI), which activates cell wall reinforcement, reactive oxygen species (ROS), and jasmonic acid/ethylene (JA/ET) signaling pathways. This immune response involves membrane-bound receptors RLK and RLP, in cooperation with co-receptors (e.g., BAK1). This type of immunity is present in all plants as a basal, evolutionarily conserved system [90].

Against hemibiotrophic pathogens, plants mount a combined response - PTI at early stages, followed by potential activation of ETI. This defense involves RLK + CNL receptors, and sometimes TNLs in dicots. These pathogens require a complex immune strategy, as they change their feeding mode during infection. Thus, the type of pathogen determines the type of immune response: 1. Biotrophs primarily activate ETI, Necrotrophs activate PTI, Hemibiotrophs require both PTI and ETI; 2. The type of receptor correlates with the pathogen type: NLRs are effective against biotrophs, and RLK/RLPs are effective against necrotrophs; 3. Evolutionary differences between monocots and dicots include: The absence of TNL genes in most monocots may necessitate alternative immune strategies; The RPW8 domain, specific to dicots, enhances ETI; Selection of resistance genes for breeding should take into account both the pathogen type and the plant's taxonomic group [45].

Physiological and anatomical basis of immunity in monocotyledonous and dicotyledonous plants

A comparative analysis of the protein structures encoded by disease resistance genes in winter wheat and sunflower shows that dicotyledonous plants have evolved a more flexible and diverse PRR (pattern recognition receptor) system. This allows them to adapt more rapidly to new pathogens. We hypothesize that this is due to differences in the structure of the cell wall and epidermis, which are often targets for specialized pathogen effectors. There are significant differences in the cell wall and epidermal structure between monocots and dicots, which determine their susceptibility to various types of pathogens [15].

The cell wall of dicots is richer in pectins, especially homogalacturonans, which are easily degraded by pectinases produced by necrotrophs. The primary wall in dicots is also more flexible and enriched with proteins and pectic components. The epidermis is less lignified, making it more vulnerable to pathogen-derived enzymes. Consequently, we suggest that dicots are more frequently affected by necrotrophic and hemibiotrophic pathogens that destroy the cell wall to release nutrients [73].

Table 7. Immune responses in monocot and dicot plants to biotrophic, necrotrophic, and hemibiotrophic pathogens: receptors, mechanisms, examples

Pathogen type	Receptor	Immune response	Cellular response	Monocots	Dicots	Gene examples	Pathogen examples
Biotrophs	CNL / TNL / RPW8-NL	ETI	HR, cell death, SA signaling	CNL present, TNL absent	Є (CNL, TNL, RPW8-NL)	*Sr33*, *Sr50* (wheat); *RPS2*, *RPW8*	*Puccinia* spp., *Erysiphe* spp.
Necrotrophs	RLK / RLP + Co-receptors	PTI	ROS, cell wall reinforcement, antimicrobial proteins (JA/ET)	present	present	*Htn1*, *Xa21* (rice); *RLP30*, *SOBIR1*	*Botrytis cinerea*, *Bipolaris* spp., *Sclerotinia* spp.
Hemibiotrophs	RLK + CNL	PTI → ETI	Cell wall reinforcement → HR	present	TNL also present	*Fhb1* (wheat); *RPM1*, *RPS5*	*Zymoseptoria tritici*, *Fusarium graminearum*

Note: TNL receptors (intracellular) are predominantly found in dicots; CNLs occur in both dicots and monocots; RPW8-NL receptors are present in dicots (e.g., *Arabidopsis*); RLK and RLP are cell surface receptors, widely distributed across all angiosperms, though some types may be species-specific; Sr33, Sr50 – rust resistance genes (CNL type) in wheat; RPW8 – powdery mildew resistance gene in *Arabidopsis* (RPW8-NL type); Xa21 – receptor-like kinase (RLK) in rice, active against bacterial pathogens; Htn1 – RLK gene in maize, conferring resistance to northern corn leaf blight; Fhb1 – CNL-like gene providing partial resistance to Fusarium head blight in wheat; RPM1, RPS5, RPS2 – TNL genes in *Arabidopsis*.

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In contrast, monocot cell walls contain more hemicelluloses (mainly xylans) and lignin, creating a more rigid and resistant structure to enzymatic degradation. They also have fewer pectins, resulting in fewer substrates for necrotrophic pectinases. The cuticle of the monocot epidermis is thicker, enhancing the barrier against mechanical and enzymatic penetration. Therefore, we conclude that monocots are more frequently infected by biotrophs, which form specialized structures such as haustoria to enter cells without destroying them. Based on this comparative analysis, we hypothesize that due to differences in the structure of the cell wall and epidermis – and consequently the vulnerability to necrotrophic and hemibiotrophic pathogens-dicotyledonous plants have evolutionarily developed a powerful PRR system with an extensive network of RLP receptors and corresponding co-receptors, as well as a full set of intracellular NLR receptors: TNL, RPW8-NL, and CNL [9].

RLP (receptor-like proteins), which lack intrinsic kinase activity, play a key role in rapidly recognizing extracellular pathogen effectors and enzymes – particularly from necrotrophs. Their function is mediated through interaction with co-receptors: SOBIR1, which acts as a kinase-active adapter, and BAK1, which initiates immune signaling cascades. This architecture enables dicot plants to effectively respond to early pathogen-associated molecular patterns (PAMPs) or effectors released into the apoplast. Furthermore, dicots have developed a system of intracellular immune surveillance involving TNL receptors – proteins with a TIR domain – closely associated with the EDS1/PAD4/SAG101 signaling complex. Activation of this complex initiates programmed cell death (HR – hypersensitive response), which is an effective defense strategy against hemibiotrophs that require living host cells [32].

RPW8-NL receptors (also known as RNLs) play a particularly important role as “immune helpers.” They function in a complex with EDS1 proteins and specific partners – NRG1 or ADR1 – enhancing immune responses and providing broad-spectrum non-specific resistance. This system enables dicots to effectively coordinate both local and systemic defense responses against a wide range of pathogens, particularly those that destroy cellular structures. Table 8 presents our comparative characterization of NLR receptor classes in dicots and monocots, along with their key protein partners. As seen in the data, dicots have developed a more differentiated NLR system that includes three main classes of receptors: TNL, CNL, and RNL. TNL receptors with a TIR domain activate the hypersensitive response (HR) through the EDS1/PAD4/SAG101 signaling complex, effectively counteracting hemibiotrophs. RNLs (RPW8-NL), acting as “immune helpers,” amplify TNL signals in interaction with ADR1 or NRG1. In contrast, most grass species have lost or strongly reduced TNL and RNL receptor repertoires. Their primary role in pathogen recognition is performed by CNL receptors. We believe this structural difference reflects evolutionary adaptation to different types of pathogens and cell wall characteristics [80].

TNL receptors (with a TIR domain) are completely absent in monocotyledonous plants, which is a well-documented case of evolutionary loss of this class. In contrast, dicotyledonous plants actively utilize TNLs, particularly against biotrophic and hemibiotrophic pathogens. Their effectiveness is associated with the EDS1/PAD4/SAG101 signaling cascade. CNL receptors are the most conserved class, present in both monocots and dicots. This class provides a universal effector-triggered immune response. Examples of CNLs can be found in major crops: *Sr33* in wheat, *RPM1* in *Arabidopsis*.

RNL receptors (RPW8-NL) act as “immune helpers”, amplifying TNL-derived signals through partner proteins ADR1 or NRG1. These receptors are also absent in monocots, as they are closely linked to the TNL-based system. Genome analysis confirms this distribution, highlighting the diversification of LRR-RLK gene families in cereal crops such as wheat [90].

Table 8. Comparative characteristics of NLR receptor classes in dicotyledonous and monocotyledonous plants

NLR receptor class	Domain organization	Representative protein	Partner proteins	Main function	Type of immunity	Monocots / dicots
TNL	TIR-NBS-LRR	RPS4, RPP1	EDS1, PAD4, SAG101	Activation of HR, local cell death, and effector-triggered immunity	ETI (effector-triggered immunity)	Absent / Present
CNL	CC-NBS-LRR	RPM1, Sr33, Pm3	Often independent, occasionally involves ADR1	Direct activation of the immune response cascade via kinase-like domains	ETI, basal immunity	Present / Present
RPW8-NL (RNL)	RPW8-NBS-LRR або NBS-LRR	ADR1, NRG1	EDS1 (through TNL), NRG1/ADR1	"Immune helpers" – amplification of TNL signaling, systemic resistance	Supportive role in ETI and the TNL/NLR system	Absent / Present
NL (foundational NLRs)	NBS-LRR (lacking N-terminal domain)	Predicted NLRs	-	Likely auxiliary or reduced function	Unclear/evolutionary remnants	Present / (limited, mostly inactive)

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NL (basic NLRs) represent a potentially rudimentary or not yet fully characterized group. They may be present in both plant groups, but their functionality is low or unknown. They are likely remnants of the ancestral NLR system or potential precursors of other classes. Thus, dicotyledonous plants possess a more complete, complex, and differentiated NLR receptor system, including TNLs, RNLs, and CNLs. This corresponds to their higher susceptibility to necrotrophs and hemibiotrophs, which require precise effector recognition and HR activation. Monocots, in contrast, have retained CNLs as their primary immune tool, which aligns with their biotrophic defense strategy and more structurally protected tissues. Based on the comparative analysis of protein structures controlled by disease resistance genes in winter wheat and sunflower, we hypothesize that in monocotyledonous plants, due to the specific structure of the cell wall and epidermis – and as a result, the dominance of biotrophic and hemibiotrophic pathogens – an immune system has evolved that is predominantly composed of RLK receptors with kinase domains and cytosolic CNL receptors with coiled-coil (CC) domains [12].

RLK proteins, such as FLS2 and EFR, are capable of independently initiating defense signaling cascades upon recognition of PAMPs (e.g., flagellin), without the need for partner proteins. CNL receptors with CC domains function effectively without TIR domains, triggering local immune responses without inducing excessive cell death. Due to the reduction of the TNL class, monocots rely less on effector-triggered hypersensitive responses (HR). Moreover, the high density of the lignin- and hemicellulose-rich cell wall in monocots limits the efficiency of extracellular pathogen attack, reducing the need for a complex RLP recognition system. To summarize and integrate the data, Table 9 provides a comparative overview of immune receptor systems in dicotyledonous and monocotyledonous plants, using winter wheat and sunflower as examples, with consideration of cell wall structure and epidermal architecture. As shown in the table, the structural features of the cell wall, epidermis, and receptor systems in dicots and monocots are the result of evolutionary adaptations to specific types of pathogens [89].

Dicotyledonous plants have a more developed system for recognizing necrotrophs and hemibiotrophs through cytosolic TNL and RPW8-NL receptors and membrane-bound RLP receptors. In contrast, monocotyledonous plants have evolved with a focus on biotrophs and hemibiotrophs, and possess dominant surface-localized RLK receptors and cytosolic CNL proteins. In dicots, TNL receptors, which activate the EDS1/PAD4/SAG101 signaling pathway, and RPW8-NL receptors, which function through EDS1 and NRG1/ADR1, play key roles. This allows effective responses against necrotrophic and hemibiotrophic pathogens. In monocots, TNL receptors are either completely absent or highly reduced, which reflects adaptation to biotrophs that do not trigger HR. The absence of the EDS1 signaling pathway diminishes their defense capability against hemibiotrophs [43].

RNL receptors (RPW8-NL), which serve as "immune helpers", are underdeveloped in monocots because their function is not direct pathogen recognition but the amplification of immune signaling from TNL receptors (TIR-NLRs). Since most monocot species, particularly grasses, lack functional TNL receptor systems and exhibit partial reduction or modification of components associated with TNL signaling, the classical TNL–EDS1–PAD4–SAG101 pathway is substantially diminished compared with dicots. In dicots, RLP receptors are well developed. Although they lack their own kinase domains, they require co-receptors (SOBIR1, BAK1) to activate immune cascades, enabling rapid recognition of necrotrophic effectors. In monocots, RLP receptors are rare, and their ability to recognize extracellular pathogens is weakened. While dicots also possess RLK receptors, they are not dominant. Instead, other receptor types play the major role in pathogen recognition. In monocots, RLK receptors dominate; notably, FLS2 and EFR, which possess intrinsic kinase activity and can initiate defense responses independently of co-receptors [5, 89].

In dicotyledonous plants, the full set of NLR immune receptors (TNL, CNL, RNL) is preserved, along with a well-developed system of RLP receptors that work effectively against necrotrophs through partnerships with SOBIR1 and BAK1. The hypersensitive response (HR) is used adaptively depending on the type of pathogen. In contrast, monocotyledonous plants have evolutionarily lost TNL and most RNL receptors, focusing instead on CNL receptors and membrane-bound RLKs with kinase activity. In monocots, HR is primarily effective against biotrophs, while necrotrophs are suppressed through

Table 9. Features of receptor systems in dicotyledonous and monocotyledonous plants

Aspect	Dicots (TNL, RPW8-NL, RLP)	Monocots (RLK, CNL)
1. Presence of TNL, RPW8-NL	Present. TNLs → activate EDS1/PAD4/SAG101. RPW8-NLs → act via EDS1 + NRG1/ADR1.	Absent or almost completely lost. Loss of the EDS1 signaling pathway.
2. RLP receptors	Well developed. Lacks a kinase domain, relies on SOBIR1 and BAK1.	Rarely present. Poor adaptation to extracellular recognition.
3. RLK-receptors	Present but not dominant.	Dominant. FLS2 and EFR possess intrinsic kinase activity and are independent of co-receptors.
4. Cell wall	Flexible, with lower lignin content → more susceptible target for necrotrophs/hemibiotrophs.	Rigid, rich in lignin and silicon → hinders extracellular penetration.
5. Typical pathogens	More commonly, necrotrophs and hemibiotrophs.	More commonly, biotrophs and hemibiotrophs. Necrotrophs are less effective.

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alternative defense mechanisms. To illustrate the role of NLR receptors depending on pathogen type (in the context of monocots and dicots), we conducted a comparison of the immune responses in winter wheat and sunflower across pathogen types, presented in Table 10. The data clearly demonstrate functional differences in immune responses between dicot and monocot plants against various pathogens [12].

Dicots possess a full range of NLR receptors (TNL, CNL, RNL), allowing effective responses to both biotrophic and hemibiotrophic infections. They also have a robust RLP system, which plays a critical role in recognizing necrotrophs. In contrast, monocots lack TNL and RNL receptors and rely mainly on CNL and RLK receptors, showing reduced activity against necrotrophs due to a weakly developed RLP system. Biotrophic and hemibiotrophic pathogens are effectively suppressed by the hypersensitive response (HR), which is mediated by intracellular NLRs – particularly TNL and CNL classes. Dicots retain the full complement of these receptors, including RNL "helper" proteins, while in monocots, due to the loss of TNL and RNL, CNL receptors dominate [43].

Against necrotrophs, dicots activate extracellular RLP receptors, while monocots rely primarily on RLK receptors. In monocots, HR is generally avoided in these cases, as it can actually facilitate necrotrophic infection. This highlights the functional adaptation of immune systems in angiosperms to different types of pathogens. Overall, the immune strategy of dicotyledonous plants is more flexible and specialized, particularly against necrotrophs, due to their broader receptor repertoire. Monocots, having lost TNL and RNL receptors, have adapted to biotrophic pathogens by enhancing the role of RLK and CNL receptors, but show limited effectiveness against necrotrophs. These evolutionary changes reflect a deep structural and functional divergence between the two major groups of flowering plants [45].

Dicotyledonous plants are generally more frequently affected by necrotrophic and hemibiotrophic pathogens, which release large amounts of toxins and enzymes. In dicots, a well-developed system of RLP receptors, in complex with partner proteins SOBIR1 and BAK1, provides effective protection against necrotrophic pathogens. Necrotrophs act aggressively on dicot crops, which is why, using sunflower as a model, we analyzed the effectiveness of the membrane-bound RLP system, together with SOBIR1 and BAK1, against this type of pathogen. Membrane-bound RLP receptors lack intrinsic kinase activity, but they recognize extracellular pathogen-derived molecules, including toxins and enzymes. SOBIR1 initiates the signaling cascade, while BAK1 amplifies the signal, forming a strong immune response without triggering hypersensitive cell death (HR). This response would be detrimental in the case of necrotrophic infection [2].

This system is especially important for dicots because their cell walls contain less lignin and more pectins and cellulose, making them more susceptible to enzymatic degradation by pathogens. Since necrotrophs pose a significant threat to dicots, the RLP–SOBIR1–BAK1 complex serves as a crucial line of defense. In contrast, monocotyledonous plants, which have more rigid, lignin- and hemicellulose-rich cell walls and greater mechanical protection against necrotrophs, have partially lost the RLP system. Instead, they have focused on RLK receptors and CNLs. Dicots, meanwhile, have developed RLPs as a key mechanism of extracellular immune response. This allows them to mount an effective defense without resorting to HR, which would otherwise enhance pathogen success [74, 83].

The RLP–SOBIR1–BAK1 system in dicots is an evolutionary adaptation to necrotrophic pathogens, enabling plants to avoid hypersensitive response while still activating robust immune defenses. To understand the evolutionary differences in immunity between dicotyledonous and monocotyledonous plants and to explain the development of immune response specialization, we conducted a comparison of immune receptors and signaling cascades in these groups. The systemic differences in immune system architecture between different types of angiosperms (winter wheat and sunflower) are presented in Table 11. In monocotyledonous plants, the immune system is primarily based on RLK and CNL receptors, which are effective against biotrophic and hemibiotrophic pathogens. These receptors do not require partner proteins and do not trigger a detrimental hypersensitive response (HR) during necrotrophic infections [72].

In contrast, dicotyledonous plants have actively evolved TNL, RNL (RPW8-NL), and RLP receptors, which provide a more flexible immune response to a wide spectrum of pathogens, including hemibiotrophs and necrotrophs. This is associated with their less rigid cell walls, which demand more complex recognition and defense mechanisms. Dicots have retained a greater diversity of immune receptor types (TNL, RLP), giving them higher evolutionary flexibility and

Table 10. Comparison of immune responses in dicotyledonous and monocotyledonous plants by pathogen types

Pathogen type	Pathogen target	Role of cell death (HR)	Main activated receptors	Dicots	Monocots
Biotrophs	Live in living cells	+ HR is highly effective - blocks the pathogen	TNL, CNL, RNL	+ TNL, CNL, RNL (Complete set)	+ CNL (TNL and RNL are lost)
Hemibiotrophs	Start as biotrophs, then kill the cell	± HR is effective if timely	TNL, CNL, RNL	+ All classes of NLRs are involved	± CNLs are dominant, a limited set
Necrotrophs	Kill cells and feed on dead tissue	− HR is harmful - promotes infection	RLP + SOBIR1/BAK1, RLK	+ RLPs are well developed, TNL is often suppressed	− RLPs are poorly represented, + RLKs are dominant

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Table 11. Comparative characteristics of the immune system in dicotyledonous and monocotyledonous plants

Characteristic	Dicots	Monocots
Types of pathogens	Necrotrophs, Hemibiotrophs	Biotrophs and hemibiotrophs
Cell wall	Pectins, less lignin	Hemicelluloses, high lignin content
Major membrane receptors	RLP (SOBIR1, BAK1), RLK	RLK (with intrinsic kinase activity)
Cytosolic receptors	TNL, RPW8-NL, CNL	Only CNL
Cytosolic response partner proteins	EDS1, PAD4, SAG101, NRG1	-
Hypersensitive response (HR)	Commonly mediated by TNL	Limited, localized via CNL
Evolutionary flexibility of receptors	High, diverse types	Lower, narrow specialization

broader protection. Monocots, having lost TNL and RNL, have simplified their immune systems, adapting them to their specific anatomy (denser cell walls, reduced DAMP recognition). The evolutionary loss of EDS1/PAD4/SAG101 partner proteins in monocots has limited their ability to initiate HR, but also reduced the risk of harmful cell death during necrotrophic infections [24].

In practical terms, this means that dicotyledonous plants (e.g., sunflower) possess a full set of intracellular receptors - TNL, CNL, and RNL - which enables them to effectively respond to biotrophic and hemibiotrophic pathogens via HR activation. Against necrotrophs, they rely on membrane-bound RLP and RLK receptors, which activate defense responses without triggering HR, which is critical for preserving cell integrity. Monocot plants (e.g., wheat, rice) have lost TNL and most RNL receptors during evolution, greatly limiting their ability to induce HR. Their defense relies primarily on CNL receptors (responsible for local intracellular immune signaling) and RLK receptors such as *FLS2*, which enable pathogen recognition at the cell surface. Due to the underrepresentation of the RLP system, their defense against necrotrophs is less efficient compared to dicots [24].

Thus, the immune architecture exemplified by sunflowers shows that in dicots, developing both NLR-dependent and PRR-dependent (RLP/RLK) systems through breeding is a promising strategy for creating resistant cultivars and hybrids, with a special focus on RLPs for protection against necrotrophs. In monocots (wheat, rice, maize), strategies should focus on increasing the diversity of CNL receptors through introgression, enhancing RLK-mediated immunity (e.g., via gene editing or transgenic approaches involving *FLS2*, *Xa21*), and potentially transferring the RLP/SOBIR1 system from dicots, provided that molecular compatibility allows it [20].

The data obtained indicate significant evolutionary divergence in the immune systems of dicotyledonous and monocotyledonous plants, driven not only by genetic architecture but also by morpho-anatomical features and the predominant types of pathogens in their environments. In dicots such as sunflower, the presence of TNL and RNL receptors indicates a functional EDS1–PAD4–SAG101 signaling cascade, which is essential for recognizing biotrophic effectors and initiating the hypersensitive response (HR). This mechanism is effective against pathogens that require living host cells and was likely evolutionarily conserved due to strong selective pressure from such pathosystems [3, 6, 24].

In contrast, cereals, including winter wheat, exhibit a reduction or complete loss of the TNL and RNL classes.This evolutionary pattern may partially reflect differences in cell wall composition and tissue architecture between monocots and dicots. The higher lignin and silica content often observed in grasses may contribute to enhanced structural protection against some pathogens; however, monocots remain susceptible to numerous necrotrophic pathogens. Therefore, the reduction of TNL/RNL classes should be viewed as a multifactorial evolutionary process involving genomic, ecological, and pathogen-driven selective pressures rather than as a direct consequence of cell wall structure alone. This anatomical strategy likely reduces the selective need for intracellular effector receptors, leading to the preferential development of CNL receptors and surface-level RLK/RLP systems, with RLKs being more dominant in monocots. These observations reflect the evolutionary extinction of TNLs in cereal families, representing a case of functional compensation via an enhanced role of plasma membrane PRR receptors. This immune system restructuring should not be viewed as a deficiency but as an adaptation to specific pathogen pressures and ecological conditions [20, 52, 81].

The evolution of the immune system in winter wheat illustrates a gradual shift from effector-dependent, race-specific resistance toward more stable, non-specific resistance, which relies on a combination of metabolic, physiological, and structural barrier mechanisms. Such transformation is crucial in the context of the high adaptability of pathogens. Modern breeding strategies should aim to integrate ETI (effector-triggered immunity), PTI (pattern-triggered immunity), and non-specific defense pathways to establish a durable, multi-layered immune response. Recent advances in genome editing open up promising opportunities for accelerating such integrative approaches to breeding. In the context of applied breeding, this highlights the importance of considering the evolutionary specificity of immune pathways when integrating resistance genes from different plant families. In particular, the transfer of TNL-dependent genes into cereals may be ineffective due to the absence of the functional protein environment required for their activity [11].

A key evolutionary factor behind the loss of TNL receptors in cereals is likely the alteration of signaling within the ETI (effector-triggered immunity) pathway,

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which depends on cooperation with EDS1 and NRG1/ADR1 proteins. In dicot plants, this cascade is well conserved and enables a rapid, localized hypersensitive response (HR) upon recognition of intracellular effectors. However, in cereals, where these cofactor proteins are lost or significantly modified, the TNL-dependent ETI pathway cannot function properly. Instead, cereals reinforce other elements of non-specific immunity, especially PRR-mediated PTI responses. RLK and RLP receptors play a compensatory role in cereals by recognizing pathogen-associated molecular patterns (PAMPs) on the cell surface. These receptors have expanded significantly in cereals, including classes such as LRR-RLK, WAK, and LYP, enabling detection of a broad range of pathogens at early stages of infection. In contrast, dicots rely on a combined PTI and ETI strategy, which provides two-tiered protection involving both HR and systemic resistance [5, 60].

Interestingly, despite TNL reduction, some cereals retain functional CNL receptors (e.g., *Sr33*, *Sr50*, *Lr34*) that contribute to both race-specific and non-specific resistance. Of particular interest are ABC transporters (*Sr57/Lr34*, *Sr58/Lr46*), which regulate cellular physiology and suppress pathogen development without direct effector recognition. This points to the evolution of alternative broad-spectrum resistance mechanisms in cereals, including metabolic pathways, transcriptional reprogramming, and hormonal regulation. Recognizing these differences is critically important when developing cultivars with broad-spectrum resistance. The "gene stacking" strategy, i.e., combining PRR receptors and CNL genes in cereal genomes, holds promise for improving resistance to both biotrophic and necrotrophic pathogens. In dicots, by contrast, the preservation of TNL genes allows effective utilization of classical effector-dependent ETI pathways in breeding programs [36, 73].

Thus, the evolutionary dynamics of plant immunity demonstrate not only the loss and modification of individual receptors but also a flexible adaptation to diverse ecological scenarios. Future studies should focus on identifying novel companion proteins and signaling components that can compensate for lost mechanisms, thereby opening new horizons for transgenic and genomic breeding approaches. It is important to emphasize that the reduction of TNL genes in cereals should not be viewed as a weakening of the immune system, but rather as an evolutionary reorientation. This strategy was likely adaptive in specific ecological and pathogenic environments, where the priority shifted toward rapid detection of pathogens at the cell surface rather than in the cytoplasm. Through the expansion of RLK and RLP receptors, cereals have broadened their PAMP-recognition repertoire and can efficiently coordinate defense responses at the earliest stages of infection [20, 52].

Additional flexibility is provided by the structural plasticity of CNL receptors. In cereals, some of these receptors exhibit substantial domain variability and can participate in the recognition of a wide range of pathogen effectors. This creates potential for the formation of hybrid proteins with novel specificities – a phenomenon actively exploited in modern plant bioengineering. In particular, gene-editing technologies such as CRISPR/Cas9 enable the reconstruction of NLR receptors with desirable properties, including the integration of effector-binding domains. In dicot plants, the retention of the TNL/EDS1/NRG1 module represents an energetically costly but strategically justified investment, especially under continuous pressure from biotrophic pathogens that manipulate host cellular processes. In contrast, cereals – which frequently encounter biotrophic and hemibiotrophic pathogens – have adapted to avoid HR and instead regulate the cellular environment (e.g., by enhancing cell wall barriers, suppressing ROS production, or modulating phytohormone signaling) [23, 35].

This functional division has practical implications: the selection of effective resistance genes for transgenic approaches must consider not only the pathogen type but also the host plant type. The transfer of TNL receptors into cereals is often ineffective due to the absence of compatible signaling partners, whereas PRR receptor integration between dicots and cereals has proven more successful, as demonstrated by cases involving EFR and XA21. Consequently, the study of cross-species compatibility of signaling modules and the engineering of receptor cascades remains a promising direction in modern phytopathology. In conclusion, the study of immune receptors in dicot and cereal crops offers broad opportunities not only for understanding the evolution of plant immunity but also for developing novel breeding strategies that combine race-specific and non-specific resistance within a single genomic architecture [38].

CONCLUSIONS

The earliest ancestors of angiosperms likely possessed a limited set of PRR- and NLR-like proteins. However, over the course of evolution, dicotyledonous and monocotyledonous plants have undergone significant changes in their repertoire of pathogen resistance genes, leading to the development of unique immune response mechanisms adapted to different types of pathogens. The immune potential of cultivated plants is realized through the integration of genetic defense mechanisms with optimized agronomic technologies, especially mineral nutrition systems, as shown in studies on winter wheat [44].

In dicotyledonous plants, due to certain structural weaknesses in the cell wall and epidermis, which often serve as targets for specialized pathogen effectors, a more complex immune system evolved. This includes a wide array of genes encoding membrane-bound RLP-type receptors, which interact with partner proteins

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SOBIR1 and BAK1, as well as RLK receptors with intrinsic kinase domains.

Additionally, dicots are characterized by the presence of all types of cytosolic receptors, including TNLs (TIR-NLRs), which function in conjunction with EDS1, PAD4, and SAG101; RPW8-NLs, which interact with EDS1 and sometimes with NRG1; and CNLs (CC-NLRs) with kinase domains. These receptors enable dicot plants to respond effectively to a wide range of pathogens, particularly biotrophs and hemibiotrophs.

In contrast, monocotyledonous plants, due to structural advantages in their cell wall and epidermis – including compact cellular architecture and rigid walls that hinder effector penetration – have lost some of these immune components. Specifically, membrane-bound RLP receptors and cytosolic TNL and RPW8-NL receptors are absent in monocots. Nevertheless, structural barriers alone do not provide complete protection, and many economically important monocot crops remain vulnerable to necrotrophic pathogens such as *Fusarium* spp., *Bipolaris* spp., and *Rhizoctonia* spp. Therefore, anatomical traits should be considered only one of several factors influencing immune system evolution.

However, monocots have retained RLK receptors with kinase domains that mediate an effective primary immune response, as well as CNL receptors with kinase domains that participate in flexible responses to pathogens. These adaptations allow monocot plants to effectively counter specific pathogen threats, such as necrotrophs. Thus, the evolution of immunity in cereals and dicot plants reflects deep adaptation to diverse environmental conditions and pathogen types, shaped in parallel with the development of cell wall structures and specific receptor molecules.

The evolutionary dichotomy between dicot and cereal crops in terms of immune receptors highlights a profound adaptation to differing pathogen pressures, cell wall architecture, and signaling landscapes. The retention of the TNL module in dicots is evolutionarily justified in the context of biotrophic pathogen defense, whereas in cereals, evolution has promoted the enhancement of PRR-mediated immunity (PTI) and the modification of CNL cascades for a more flexible ETI response.

Modern biotechnological approaches enable the redesign of the immune system in cereal and oilseed crops by integrating novel receptors, adapting signaling pathways, and constructing synthetic immune modules. These strategies offer promising opportunities to develop multifunctional cultivars with broad-spectrum resistance to various pathogen groups – an urgent need in the context of climate change and the evolution of pathogenic races.

Conflict of interest. There is no actual or potential conflict of interest in relation to this article.

Abbreviations. ADR1 – Activated Disease Resistance 1, BAK1 – BRI1-Associated Receptor Kinase 1, CNL – Coiled-Coil Nucleotide-Binding Leucine-Rich Repeat Receptor, DAMP – Damage-Associated Molecular Pattern, EDS1 – Enhanced Disease Susceptibility 1, ETI – Effector-Triggered Immunity, HR – Hypersensitive Response, JA – Jasmonic Acid, NLR – Nucleotide-Binding Leucine-Rich Repeat Receptor, PAD4 – Phytoalexin Deficient 4, PAMP – Pathogen-Associated Molecular Pattern, PRR – Pattern Recognition Receptor, PTI – Pattern-Triggered Immunity, RLK – Receptor-Like Kinase, RLP – Receptor-Like Protein, RNL – RPW8-NLR, ROS – Reactive Oxygen Species, SA – Salicylic Acid, SAG101 – Senescence-Associated Gene 101, SOBIR1 – Suppressor of BIR1, TNL – Toll/Interleukin-1 Receptor-NLR.

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