Converging Strategies, Diverging Paths: A Comparative Perspective on Plant and Animal Immune Systems

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Abstract

For plants and animals to survive against infections, immunity is a crucial evolutionary adaptation. Despite having split from a common unicellular ancestor more than a billion years ago, both plant and animal lineages have evolved sophisticated immune systems that are adapted to their unique biological and ecological environments. The origin, evolutionary paths, mechanisms, and contemporary functional differences between the immune systems of plants and animals are examined in this review. Molecular recognition, signalling pathways, and systemic responses are highlighted in a comparative analysis of the divergent and convergent immunological strategies. Knowing these differences offers a framework for translational research in biomedicine and agriculture as well as insight into host-pathogen interactions.

Keywords: animals, plants, immune system, host-pathogen, origin, mechanism

1. Introduction

An essential biological system, the immune system is in charge of identifying and neutralizing harmful threats. In contrast to mammals, who have both innate and adaptive immunity, plants only have innate immunity. Their different evolutionary forces and physiological structures are reflected in the difference of their immunological systems. This essay explores the genesis, development, workings, and contemporary distinctions between the immune systems of plants and animals.

2. Origin of Immune Systems

2.1 Common Ancestral Mechanisms

Basic pathogen recognition machinery is shared by a eukaryotic ancestor of both plants and animals [1]. Among them are conserved pattern recognition receptors (PRRs) that may identify MAMPs (microbe-associated molecular patterns) [2].

2.2 Divergence and Lineage-Specific Evolution

Immune strategies evolved separately as a result of the evolutionary division between the kingdoms of plants and animals. Adaptive immunity in animals began with the appearance of mobile phagocytes and then lymphocytes. However, plants developed cell-autonomous defences and structural barriers [3].

3. Evolution of Immune Mechanisms

3.1 Plants

A very complex two-layered defensive system makes up plant immunity. When pattern recognition receptors (PRRs) on the cell surface identify conserved microbial characteristics known as microbe-associated molecular patterns (MAMPs), such bacterial flagellin or fungal chitin, the first layer, known as Pattern-Triggered Immunity (PTI), is triggered [4]. Reactive oxygen species (ROS) generation, transcriptional reprogramming, and MAPK

cascades are examples of downstream signaling processes that result from this. Plants trigger a second layer called Effector-Triggered Immunity (ETI) when pathogens get around PTI by introducing effectors into host cells. Intracellular nucleotide-binding leucine-rich repeat (NLR) proteins regulate ETI by identifying these effectors and triggering a strong, frequently localized, hypersensitive reaction that results in systemic acquired resistance and programmed cell death [5].

3.2 Animals

The animal immune system exhibits remarkable complexity through its bifurcated structure comprising innate and adaptive immunity. Innate immunity serves as the first line of defence and includes non-specific mechanisms such as phagocytosis by macrophages, antigen presentation by dendritic cells, and activation of pattern recognition receptors like Toll-like receptors [6]. This arm provides immediate, but general, protection. In contrast, adaptive immunity—exclusive to jawed vertebrates—is highly specific and features immunological memory. It relies on somatic recombination of antigen receptor genes in B and T lymphocytes via V(D)J recombination, generating diverse antigen recognition capabilities. These components enable lifelong protection and vaccine-based immunity.

4. Functional Components

Table 1. Comparative Overview of Plant and Animal Immune Systems

Component	Plants	Animals	References
Recognition receptors	PRRs (e.g., FLS2), intracellular NLRs	PRRs (e.g., TLRs), BCRs, TCRs	[4, 5] [7]
Signaling pathways	MAPK cascades, calcium flux, salicylic acid and jasmonic acid signaling	NF-κB, JAK-STAT, cytokine signaling	[7, 8]
Effector response	Reactive oxygen species (ROS), phytoalexins, cell wall reinforcement, hypersensitive response (HR)	Phagocytosis, antigen presentation, antibody secretion, cytotoxic killing	[7, 9]
Memory	Priming (no true memory; epigenetically regulated)	Long-lasting immunological memory via B and T cells	[7, 9]
Systemic response	Systemic acquired resistance (SAR), mediated by mobile signals (e.g., methyl salicylate)	Systemic inflammation, lymphocyte migration via the circulatory and lymphatic systems	[7, 8, 10]
Cellular mobility	Static cells; no mobile immune cells	Mobile immune cells (e.g., macrophages, neutrophils, lymphocytes)	[10] [7]
Receptor diversity	Germline-encoded PRRs and NLRs, some diversification via gene duplication	Somatic recombination in BCRs and TCRs enables immense diversity	[7, 11]
Immune strategy	Innate immunity only, multi-layered (PTI and ETI)	Innate and adaptive immunity	[3, 5] [7]

Table 1. presents a side-by-side comparison of key immunological components in plants and animals. It highlights differences in recognition, signaling, response strategies, and systemic coordination across both kingdoms.

5. Current Functional Status

Plant and animal immune systems, despite having evolved independently, are remarkably complicated in their current functional architecture. These variations reflect the organism's physiology, motility, evolutionary restrictions, and environmental stresses.

5.1 Complexity and Specialization

High levels of compartmentalization and organizational complexity define the animal immune system. The growth, activation, and circulation of immune cells, such as macrophages, dendritic cells, and lymphocytes, are supported by specialized organs such the bone marrow, thymus, spleen, and lymph nodes [7]. Rapid, systemic immune responses are made possible by the vascular and lymphatic network that connects these mobile cells. Plant cells, on the other hand, have solid cell walls that prevent them from moving, thus each cell must recognize and react to infections on its own. However, plants need a complex set of signaling processes to compensate. Long-distance signals including salicylic acid, jasmonic acid, azelaic acid, and reactive oxygen species enhance intercellular communication through plasmodesmata [8, 12]. These signals coordinate systemic acquired resistance (SAR), allowing plants to mount whole-organism defences despite structural immobility.

5.2 Immune Memory and Learning

The ability of vertebrate adaptive immunity to retain immunological memory—in which memory B and T cells survive an infection and facilitate quick, targeted reactions when the same antigen is encountered again—is one of its defining characteristics. Vaccine tactics are based on this long-term protection. On the other hand, plants can experience "defense priming," a process whereby exposure to pathogens or elicitors strengthens the plant's future defensive ability, even if they lack clonal growth and antigen-specific memory. Epigenetic changes like histone acetylation or DNA methylation are frequently responsible for this primed state, which can last for a long period [9, 13]. While not as accurate as adaptive immunity, this significantly increases plant resilience in a manner similar to memory.

5.3 Pathogen Counter-Strategies

Co-evolution is fueled by pathogens' selective pressure on host immune systems. Numerous bacterial and fungal pathogens in plants release effector proteins that directly block intracellular nucleotide-binding leucine-rich repeat (NLR) proteins or pattern recognition receptors (PRRs), thereby blocking the PTI and ETI pathways [5, 14]. Similar mechanisms have been evolved by pathogens in animals: bacterial effectors, like Salmonella's AvrA and Yersinia's YopJ, acetylate or degrade components of innate immune pathways, preventing antigen presentation, NF-κB activation, or Toll-like receptor (TLR) signalling [15, 16]. By downregulating MHC molecules or altering epitopes to prevent T-cell recognition, viral pathogens frequently elude immune detection. The evolutionary arms race between host immunity and pathogen virulence strategies is highlighted by these advanced countermeasures.

6. Comparative Immunity: Key Differences

6.1 Cellular Mobility

Cellular mobility is the most obvious difference between plant and animal immunity. Specialized leukocytes, such as neutrophils, macrophages, dendritic cells, natural killer cells, and lymphocytes, are found in vertebrates. These

cells move through blood and lymph constantly to examine tissues, quickly move to infection sites, and mount systemic defenses (Murphy & Weaver, 2016). Millions of cells can be recruited on a minute scale through chemokine gradients, integrin-mediated adhesion, and endothelial extravasation; this frequently results in the clonal expansion of antigen-specific T and B cells [17]. In contrast, immune surveillance is cell-autonomous in plants because they are sessile organisms with fixed cells inside rigid cell walls. Each cell triggers pattern-triggered immunity (PTI) when it detects danger, and it may use vascular or airborne signals like salicylic acid, pipecolic acid, and methyl jasmonate to boost defences systemically [10, 12]. All following defence reactions take place in situ, but warning messages can also be sent via electrical waves and calcium fluxes at speeds that are comparable to those of animal nerves. Glutamate-triggered calcium waves are used by plants as quick defensive signals that go through their bodies at a speed comparable to nerve impulses [18]. Calcium channels and electrical signals play a part in long-distance signaling after injury [19]. ROS and calcium in systemic fast signaling, which has transmission speed similarities to animal nervous systems [20]

6.2 Molecular Diversity

The molecular diversity of recognition receptors is a second difference. Vertebrates use somatic V(D)J recombination and junctional diversification in immunoglobulin and T-cell receptor genes to achieve remarkable receptor breadth, estimated at 10^13 specificities [21]. Plants, on the other hand, have germline-encoded intracellular nucleotide-binding leucine-rich repeat (NLR) proteins and lack lymphocyte-like mechanisms. Through gene duplication, unequal crossing-over, and domain reshuffling, diversity develops over evolutionary rather than somatic time, creating "integrated decoys" that imitate effector targets Plant populations maintain broad surveillance through large NLR repertoires and presence/absence polymorphisms, despite the fact that this strategy cannot customize receptors to each new infection within an individual's lifetime [22].

6.3 Evolutionary Innovation

And finally, there are fundamental differences in evolutionary innovation. About 500 million years ago, whole-genome duplications and horizontal capture of recombinase genes from transposons are thought to have produced the dual-arm (innate + adaptive) immune architecture that is specific to jawed vertebrates[23, 24]. Comparative genomics shows that all of the major angiosperm clades already had hundreds of NLR loci, indicating convergent evolution toward modular sensors long before animals developed adaptive immunity. In contrast, plants expanded their NLR superfamily much earlier[25, 26]. These disparate inventions highlight how life experiences and mobility limitations shape unique but equally potent immune responses.

7. Applications and Translational Relevance

Understanding the evolution and mechanisms of plant and animal immunity offers vast translational applications in both agriculture and human health. In crop science, leveraging plant immune components like pattern recognition receptors (PRRs) and NLR genes has facilitated the creation of transgenic and genome-edited plants with durable resistance to pathogens[27, 28] In biomedical research, animal immunology has led to breakthroughs in monoclonal antibody therapies, vaccines, and cancer immunotherapies[7, 29]. Synthetic biology now bridges both systems, enabling the transfer of immune traits across kingdoms to develop sustainable disease control strategies [30, 31].

8. Conclusion

Animals and plants have evolved complex immune systems tailored to their respective lives, despite following different evolutionary trajectories. Comparative immunology identifies both distinctive adaptations and common ancestral traits. Plants acquired robust, multi-layered innate defences, whereas mammals evolved an adaptive immune system that allowed for memory and specificity. Understanding these distinctions promotes creative approaches to agriculture and health while also deepening our understanding of immunity.

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References

[1] F. M. Ausubel, "Are innate immune signaling pathways in plants and animals conserved?," (in eng), *Nat Immunol,* vol. 6, no. 10, pp. 973-9, Oct 2005, doi: 10.1038/ni1253.

[2] C. Zipfel and G. Felix, "Plants and animals: a different taste for microbes?," (in eng), *Curr Opin Plant Biol,* vol. 8, no. 4, pp. 353-60, Aug 2005, doi: 10.1016/j.pbi.2005.05.004.

[3] P. N. Dodds and J. P. Rathjen, "Plant immunity: towards an integrated view of plant-pathogen interactions," (in eng), *Nat Rev Genet,* vol. 11, no. 8, pp. 539-48, Aug 2010, doi: 10.1038/nrg2812.

[4] T. Boller and G. Felix, "A renaissance of elicitors: perception of microbe-associated molecular patterns and danger signals by pattern-recognition receptors," (in eng), *Annu Rev Plant Biol,* vol. 60, pp. 379-406, 2009, doi: 10.1146/annurev.arplant.57.032905.105346.

[5] J. D. Jones and J. L. Dangl, "The plant immune system," (in eng), *Nature,* vol. 444, no. 7117, pp. 323-9, Nov 16 2006, doi: 10.1038/nature05286.

[6] R. Medzhitov and C. Janeway, "Innate immune recognition: mechanisms and pathways," (in eng), *Immunol Rev,* vol. 173, pp. 89-97, Feb 2000, doi: 10.1034/j.1600-065x.2000.917309.x.

[7] K. Murphy and C. Weaver, *Janeway's immunobiology*, 9th edition. ed. New York, NY: Garland Science/Taylor & Francis Group, LLC, 2017, pp. xx, 904 pages.

[8] Z. Q. Fu and X. Dong, "Systemic acquired resistance: turning local infection into global defense," (in eng), *Annu Rev Plant Biol,* vol. 64, pp. 839-63, 2013, doi: 10.1146/annurev-arplant-042811-105606.

[9] U. Conrath *et al.*, "Priming: getting ready for battle," (in eng), *Mol Plant Microbe Interact,* vol. 19, no. 10, pp. 1062-71, Oct 2006, doi: 10.1094/MPMI-19-1062.

[10] M. Heil and J. Ton, "Long-distance signalling in plant defence," (in eng), *Trends Plant Sci,* vol. 13, no. 6, pp. 264-72, Jun 2008, doi: 10.1016/j.tplants.2008.03.005.

[11] Z. Q. Shao *et al.*, "Large-Scale Analyses of Angiosperm Nucleotide-Binding Site-Leucine-Rich Repeat Genes Reveal Three Anciently Diverged Classes with Distinct Evolutionary Patterns," (in eng), *Plant Physiol,* vol. 170, no. 4, pp. 2095-109, Apr 2016, doi: 10.1104/pp.15.01487.

[12] M. Hartmann and J. Zeier, "N-hydroxypipecolic acid and salicylic acid: a metabolic duo for systemic acquired resistance," (in eng), *Curr Opin Plant Biol,* vol. 50, pp. 44-57, Aug 2019, doi: 10.1016/j.pbi.2019.02.006.

[13] A. Martinez-Medina *et al.*, "Recognizing Plant Defense Priming," (in eng), *Trends Plant Sci,* vol. 21, no. 10, pp. 818-822, Oct 2016, doi: 10.1016/j.tplants.2016.07.009.

[14] J. Win *et al.*, "Effector biology of plant-associated organisms: concepts and perspectives," (in eng), *Cold Spring Harb Symp Quant Biol,* vol. 77, pp. 235-47, 2012, doi: 10.1101/sqb.2012.77.015933.

[15] R. M. Jones, H. Wu, C. Wentworth, L. Luo, L. Collier-Hyams, and A. S. Neish, "Salmonella AvrA Coordinates Suppression of Host Immune and Apoptotic Defenses via JNK Pathway Blockade,"

(in eng), *Cell Host Microbe,* vol. 3, no. 4, pp. 233-44, Apr 17 2008, doi: 10.1016/j.chom.2008.02.016.

[16] S. Mukherjee *et al.*, "Yersinia YopJ acetylates and inhibits kinase activation by blocking phosphorylation," (in eng), *Science,* vol. 312, no. 5777, pp. 1211-4, May 26 2006, doi: 10.1126/science.1126867.

[17] E. C. Butcher and L. J. Picker, "Lymphocyte homing and homeostasis," (in eng), *Science,* vol. 272, no. 5258, pp. 60-6, Apr 05 1996, doi: 10.1126/science.272.5258.60.

[18] M. Toyota *et al.*, "Glutamate triggers long-distance, calcium-based plant defense signaling," (in eng), *Science,* vol. 361, no. 6407, pp. 1112-1115, Sep 14 2018, doi: 10.1126/science.aat7744.

[19] S. A. Mousavi, A. Chauvin, F. Pascaud, S. Kellenberger, and E. E. Farmer, "GLUTAMATE RECEPTOR-LIKE genes mediate leaf-to-leaf wound signalling," (in eng), *Nature,* vol. 500, no. 7463, pp. 422-6, Aug 22 2013, doi: 10.1038/nature12478.

[20] S. Gilroy *et al.*, "A tidal wave of signals: calcium and ROS at the forefront of rapid systemic signaling," (in eng), *Trends Plant Sci,* vol. 19, no. 10, pp. 623-30, Oct 2014, doi: 10.1016/j.tplants.2014.06.013.

[21] N. Chaudhary and D. R. Wesemann, "Analyzing Immunoglobulin Repertoires," (in eng), *Front Immunol,* vol. 9, p. 462, 2018, doi: 10.3389/fimmu.2018.00462.

[22] A. C. Barragan and D. Weigel, "Plant NLR diversity: the known unknowns of pan-NLRomes," (in eng), *Plant Cell,* vol. 33, no. 4, pp. 814-831, May 31 2021, doi: 10.1093/plcell/koaa002.

[23] M. F. Flajnik and M. Kasahara, "Origin and evolution of the adaptive immune system: genetic events and selective pressures," (in eng), *Nat Rev Genet,* vol. 11, no. 1, pp. 47-59, Jan 2010, doi: 10.1038/nrg2703.

[24] G. W. Litman, J. P. Rast, and S. D. Fugmann, "The origins of vertebrate adaptive immunity," (in eng), *Nat Rev Immunol,* vol. 10, no. 8, pp. 543-53, Aug 2010, doi: 10.1038/nri2807.

[25] D. M. Prigozhin and K. V. Krasileva, "Analysis of intraspecies diversity reveals a subset of highly variable plant immune receptors and predicts their binding sites," (in eng), *Plant Cell,* vol. 33, no. 4, pp. 998-1015, May 31 2021, doi: 10.1093/plcell/koab013.

[26] Y. Liu *et al.*, "An angiosperm NLR Atlas reveals that NLR gene reduction is associated with ecological specialization and signal transduction component deletion," (in eng), *Mol Plant,* vol. 14, no. 12, pp. 2015-2031, Dec 06 2021, doi: 10.1016/j.molp.2021.08.001.

[27] J. L. Dangl, D. M. Horvath, and B. J. Staskawicz, "Pivoting the plant immune system from dissection to deployment," (in eng), *Science,* vol. 341, no. 6147, pp. 746-51, Aug 16 2013, doi: 10.1126/science.1236011.

[28] S. Lacombe *et al.*, "Interfamily transfer of a plant pattern-recognition receptor confers broad-spectrum bacterial resistance," (in eng), *Nat Biotechnol,* vol. 28, no. 4, pp. 365-9, Apr 2010, doi: 10.1038/nbt.1613.

[29] I. Mellman, G. Coukos, and G. Dranoff, "Cancer immunotherapy comes of age," (in eng), *Nature,* vol. 480, no. 7378, pp. 480-9, Dec 21 2011, doi: 10.1038/nature10673.

[30] T. Roudaire, M. C. Héloir, D. Wendehenne, A. Zadoroznyj, L. Dubrez, and B. Poinssot, "Cross Kingdom Immunity: The Role of Immune Receptors and Downstream Signaling in Animal and Plant Cell Death," (in eng), *Front Immunol,* vol. 11, p. 612452, 2020, doi: 10.3389/fimmu.2020.612452.

[31] B. J. Staskawicz, M. B. Mudgett, J. L. Dangl, and J. E. Galan, "Common and contrasting themes of plant and animal diseases," (in eng), *Science,* vol. 292, no. 5525, pp. 2285-9, Jun 22 2001, doi: 10.1126/science.1062013.