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A Review on Molecular Mechanism of Plant Immunity against Fungal Pathogens

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

The molecular mechanisms of plant immunity, with a particular focus on how plants defend themselves against fungal pathogens. Plant immunity is a complex, multi-layered system involving pattern-triggered immunity (PTI) and effector-triggered immunity (ETI), which together form a robust defense against a wide array of pathogens. Advances in genomics and transcriptomics have significantly enhanced our understanding of these immune mechanisms by identifying key resistance (R) genes and uncovering the transcriptional networks that regulate immune responses. Proteomics and metabolomics further elucidate the functional aspects of immunity, revealing how proteins and metabolites are mobilized during pathogen attack. The advent of gene editing technologies, particularly CRISPR-Cas9, has opened new avenues for enhancing plant immunity by enabling precise modifications of genes associated with disease resistance. The ever-evolving nature of fungal pathogens, driven by genetic diversity and environmental changes, poses ongoing challenges. Emerging pathogens and the breakdown of existing resistance in crops underscore the need for durable resistance strategies, which can be achieved through the pyramiding of multiple R genes, susceptibility gene knockouts, and the harnessing of beneficial plant microbiomes. As climate change exacerbates the spread and virulence of fungal pathogens, developing climate-resilient crops that can withstand both abiotic stresses and pathogen pressures is becoming increasingly important. Future research should prioritize understanding the molecular dynamics of plant-pathogen interactions, leveraging new technologies for crop improvement, and fostering interdisciplinary collaboration to address these challenges. Ultimately, translating these scientific advances into practical applications will be crucial for ensuring global food security and sustainable agricultural systems in the face of mounting environmental and biological threats.

Keywords: Plant immunity; fungal pathogens; resistance genes; proteomics; disease resistance; metabolomics.

1. INTRODUCTION

A. Plant Immunity

Plants possess a sophisticated immune system to combat a wide variety of pathogens such as fungi, bacteria, and viruses. Unlike animals, plants rely on a cell-autonomous defense mechanism, which is generally divided into two layers: pattern-triggered immunity (PTI) and effector-triggered immunity (ETI). PTI is the first line of defense, initiated by the recognition of pathogen-associated molecular patterns (PAMPs) through pattern recognition receptors (PRRs). This recognition triggers immune responses such as the production of reactive oxygen species (ROS) and the activation of mitogen-activated protein kinases (MAPKs) [1]. ETI, on the other hand, is a more specific and powerful response, triggered when intracellular nucleotide-binding leucine-rich repeat (NLR) proteins detect pathogen-secreted effectors, leading to a strong immune response like localized cell death, known as the hypersensitive response (HR) [2]. This dual defense strategy allows plants to detect and combat a wide spectrum of pathogens.

B. Importance of Studying Plant-Fungal Interactions

Fungal pathogens are significant agricultural threats, causing diseases such as rusts, smuts, blights, and wilts. They employ numerous strategies to bypass plant defenses, particularly through the secretion of effector proteins, which manipulate plant processes to facilitate infection [3]. This constant evolutionary battle between plants and fungal pathogens makes continuous research necessary. Advances in molecular biology, genomics, and biotechnology have deepened the understanding of these plant-pathogen interactions, providing insights into immune mechanisms and fungal strategies [4].

C. Objectives of the Review

This review will focus on the critical components of plant immunity, the role of fungal effectors in evading these defenses, and recent advancements in understanding plant-fungal interactions through modern technologies such as gene editing and molecular modeling [5].

2. FUNGAL PATHOGENS AND THEIR IMPACT ON PLANTS

A. Major Fungal Pathogens in Agriculture

Fungal pathogens are key contributors to plant diseases, leading to significant agricultural losses globally. These pathogens have various lifestyles, including biotrophic, hemibiotrophic, and necrotrophic modes of infection, each with distinct infection strategies (Table 1). Biotrophic fungi, such as *Puccinia* spp. (rusts) and *Blumeria graminis* (powdery mildew), depend on living host tissue, forming structures like haustoria that extract nutrients while evading plant immune responses [6]. For instance, *Puccinia graminis* f. sp. *tritici*, which causes wheat stem rust, has led to severe yield losses during periodic epidemics. Hemibiotrophic fungi, such as *Magnaporthe oryzae* (rice blast) and *Colletotrichum* spp. (anthracnose), begin their infection as biotrophs but switch to a necrotrophic phase, killing host cells and feeding on the dead tissue. *Magnaporthe oryzae* is notorious for causing rice blast, a disease that can lead to complete crop failure [7]. Necrotrophic fungi, like *Botrytis cinerea* (gray mold) and *Sclerotinia sclerotiorum* (white mold), kill host tissue actively and thrive on decaying matter. *Botrytis cinerea* affects over 200 plant species, including grapes, strawberries, and tomatoes, leading to post-harvest losses.

B. Economic and Ecological Consequences of Fungal Infections

The economic impact of fungal pathogens is immense, with an estimated 10-15% loss in global food production annually, translating to billions of dollars in revenue loss [8]. For example, wheat stem rust has historically caused devastating epidemics, with losses reaching millions of tons of wheat in regions such as North America, Australia, and Africa. Similarly, rice blast caused by *Magnaporthe oryzae* can lead to yield losses of up to 50% during epidemics, posing a threat to food security in rice-producing countries. Beyond yield losses, fungal infections drive up costs related to disease management, such as fungicide applications and crop rotations. For instance, managing late blight disease caused by *Phytophthora infestans* in potato and tomato crops requires frequent fungicide use, which significantly raises production costs [9]. Ecologically, fungal pathogens disrupt ecosystems by altering plant community dynamics and reducing biodiversity. Overuse of fungicides can result in resistant pathogen strains, leading to reduced efficacy of chemical controls and necessitating higher doses, with potential environmental consequences [10].

C. Overview of Plant Defense Responses

Plants have evolved complex defense systems to detect and counter fungal pathogens, primarily through two layers of immunity: pattern-triggered immunity (PTI) and effector-triggered immunity (ETI). PTI is activated when pattern recognition receptors (PRRs) on plant cells recognize pathogen-associated molecular patterns (PAMPs), such as chitin from fungal cell walls. For instance, *CERK1* in *Arabidopsis* recognizes chitin, initiating a defense response involving reactive oxygen species (ROS), mitogen-activated protein kinases (MAPKs), and antimicrobial compounds like phytoalexins [11]. ETI is triggered when intracellular nucleotide-binding leucine-rich repeat (NLR) receptors recognize specific effector proteins secreted by the pathogen to suppress PTI. This often leads to localized cell death (hypersensitive response) to prevent pathogen spread. For example, the recognition of the fungal effector AVR-Pia by the rice NLR protein RGA5 activates a robust ETI response against *Magnaporthe oryzae*. Additionally, plants can trigger systemic acquired resistance (SAR), a broad-spectrum defense mechanism associated with salicylic acid (SA) and the expression of pathogenesis-related (PR) genes, providing long-lasting protection against future infections [12]. The dynamic interaction between plant immune receptors and fungal effectors drives the ongoing evolutionary arms race between plants and their fungal pathogens.

D. Pattern Recognition Receptors (PRRs) and Pathogen-Associated Molecular Patterns (PAMPs)

Pattern recognition receptors (PRRs) are essential membrane proteins that detect pathogen-associated molecular patterns (PAMPs), crucial for initiating plant defense responses. These receptors are conserved across plant species and enable the early detection of pathogens [13-15]. Structurally, PRRs have an extracellular domain that recognizes PAMPs, a transmembrane domain for membrane anchoring, and an intracellular kinase domain that transmits signals into the cell [16]. For instance, the LRR receptor kinase FLS2 detects the bacterial flagellin-derived peptide flg22, while CERK1, a LysM receptor kinase, recognizes chitin from fungal pathogens, triggering defense responses. PAMPs, such as

bacterial lipopolysaccharides, fungal chitin, and viral double-stranded RNA, are conserved across pathogen classes, making them prime targets for plant immunity. Upon PAMP detection, PRRs activate signaling cascades, including mitogen-activated protein kinases (MAPKs) and the production of reactive oxygen species (ROS), amplifying defense signals. This leads to the expression of defense-related genes, production of antimicrobial compounds like phytoalexins, and reinforcement of the cell wall with callose deposition, fortifying the plant's defenses against pathogens [17].

E. Effector-Triggered Immunity (ETI)

Effector-triggered immunity (ETI) forms the second layer of plant defense, activated when intracellular nucleotide-binding leucine-rich repeat (NLR) proteins recognize specific pathogen effectors. ETI is more specific and robust than pattern-triggered immunity (PTI), leading to a stronger defense response. Pathogens secrete effector proteins to suppress PTI and promote infection by interfering with plant signaling pathways and manipulating the host immune system [18]. NLR proteins detect these effectors, triggering ETI. NLR proteins consist of nucleotide-binding (NB) domains, leucine-rich repeats (LRRs), and signaling domains like Toll/interleukin-1 receptor (TIR) or coiled-coil (CC) domains. Upon effector recognition, NLR proteins activate downstream signaling pathways, leading to a robust immune response. For example, the tomato NLR protein Pto recognizes the bacterial effector AvrPto, initiating a kinase cascade and causing the hypersensitive response (HR), a form of programmed cell death at the infection site [19]. Similarly, in rice, NLR protein RGA5 detects the fungal effector AVR-Pia, triggering a strong ETI response against *Magnaporthe oryzae*. ETI is frequently associated with HR, a localized cell death response that limits pathogen spread by cutting off its nutrient supply. Beyond local defense, ETI can activate systemic acquired resistance (SAR), providing long-lasting immunity against a broad range of pathogens. SAR is linked to the accumulation of salicylic acid (SA) and the expression of defense-related genes [20]. The ongoing evolutionary arms race between pathogens evolving new effectors and plants developing new NLR variants drives the diversification of plant immune responses.

Table 1. Fungal pathogens and their impact on plants

Section	Key Points
Introduction to Plant Immunity	Plants rely on pattern-triggered immunity (PTI) and effector-triggered immunity (ETI) to defend against pathogens. PTI is activated by pathogen-associated molecular patterns (PAMPs), while ETI is triggered by effector recognition [1,2].
Major Fungal Pathogens in Agriculture	Fungal pathogens such as *Puccinia spp.*, *Magnaporthe oryzae*, and *Botrytis cinerea* contribute to significant losses in crops like wheat, rice, and tomatoes, through distinct biotrophic, hemibiotrophic, and necrotrophic infection strategies [6,7].
Economic and Ecological Consequences	Fungal diseases cause 10-15% global food production loss annually. Management costs, yield losses, and reduced biodiversity impact both agricultural ecosystems and economic stability. Examples include *Puccinia graminis* and *Magnaporthe oryzae* [8,9].
Plant Defense Responses	Plant defense is based on two layers: pattern recognition receptors (PRRs) detect PAMPs in PTI, while nucleotide-binding leucine-rich repeat (NLR) proteins detect pathogen effectors in ETI. Local and systemic responses, such as the hypersensitive response (HR) and systemic acquired resistance (SAR), are key [11,12].
Fungal Effector Molecules	Pathogens secrete effector molecules to suppress PTI and evade ETI, targeting key immune receptors and signaling pathways. Effectors like AVR-Pii from *Magnaporthe oryzae* and AVR3a from *Phytophthora infestans* manipulate host defense [18,19].
Role of Hormones in Immunity	Fungal pathogens manipulate plant hormone signaling (SA, JA, ET) to weaken host immunity. Examples include *Verticillium dahliae* manipulating JA signaling and *Gibberella fujikuroi* overproducing gibberellins [34].
Impact of Emerging Pathogens	Climate change and global trade have contributed to the rise of new fungal pathogens, such as *Fusarium oxysporum* TR4, and the spread of previously controlled diseases. This threatens food security, especially in staple crops like bananas and wheat [63,64].
Durable Resistance Strategies	Strategies like pyramiding R genes, using CRISPR-Cas9 for susceptibility gene knockouts, and integrating plant microbiomes for enhanced immunity show promise for durable resistance against fungal pathogens [55,66].

3. MOLECULAR MECHANISMS OF PLANT IMMUNITY

Plants rely on intricate molecular mechanisms to detect and respond to pathogens, initiated by the recognition of pathogen-associated molecular patterns (PAMPs) or effector molecules through specific receptors (Table 2). These signaling pathways involve receptor-like kinases (RLKs), receptor-like proteins (RLPs), and key defense hormones such as salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) [21]. The interaction between these molecules ensures the immune response is effective while minimizing damage to the plant. The first step in plant immunity is the perception of pathogens by pattern recognition receptors (PRRs) on the cell surface, such as FLS2 and CERK1, which detect microbial signatures like flagellin and chitin. This recognition triggers a series of intracellular signaling events, leading to the activation of reactive oxygen species (ROS) and mitogen-activated protein kinases (MAPKs), which initiate the transcription of defense-related genes and the production of antimicrobial compounds [22,23]. RLKs and RLPs are critical components of plant immunity, functioning as sensors for external signals. RLKs, like FLS2 and BAK1, transduce signals upon ligand recognition, initiating defense cascades [24]. RLPs, such as RLP23, rely on associated kinases like SOBIR1 for signaling, expanding the range of recognized PAMPs [25]. Salicylic Acid, Jasmonic Acid, and Ethylene SA is linked to defense against biotrophic pathogens, playing a crucial role in systemic acquired resistance (SAR) by priming the plant for future attacks [26]. JA is involved in defending against necrotrophic pathogens and herbivores, while ET often works with JA to enhance defenses against pathogens like *Botrytis cinerea* and insects [27]. Cross-talk between pathways is essential for a balanced immune response. SA and JA often exhibit antagonism, allowing plants to prioritize defenses against specific pathogens, while JA and ET synergize to combat necrotrophs [28]. Additionally, hormones like abscisic acid (ABA) interact with these pathways, further refining the plant's response to both biotic and abiotic stresses [29]. This complex interaction allows plants to mount flexible, effective defenses tailored to the specific threats they face.

Table 2. Molecular mechanisms of plant immunity

Molecular Mechanism	Description	Examples	Function in Plant Immunity
Pattern Recognition Receptors (PRRs)	PRRs are membrane-bound receptors that detect pathogen-associated molecular patterns (PAMPs) on the pathogen's surface.	FLS2 detects bacterial flagellin; CERK1 recognizes fungal chitin [22].	Initiates pattern-triggered immunity (PTI), leading to ROS production and defense gene activation [22,23].
Effector-Triggered Immunity (ETI)	ETI is initiated when intracellular NLR proteins recognize specific pathogen effectors.	AVR3a from *Phytophthora infestans* is recognized by NLR proteins [19].	Triggers localized cell death (hypersensitive response) and stronger, targeted defense [19].
Mitogen-Activated Protein Kinase (MAPK) Pathways	MAPK signaling cascades are key intermediaries that transmit signals from PRRs and NLRs to activate downstream defense responses.	Activation of MAPKs following detection by PRRs like FLS2 and CERK1 [23].	Phosphorylates transcription factors to induce pathogenesis-related (PR) genes and antimicrobial compound production [23].
Hormonal Regulation and Cross-talk	Plant hormones such as salicylic acid (SA), jasmonic acid (JA), and ethylene (ET) modulate immune responses depending on the type of pathogen.	SA promotes defense against biotrophs; JA and ET work synergistically against necrotrophs [27].	Coordinates systemic resistance and tailors immune responses to specific pathogen types [26,27].
Post-Translational Modifications (PTMs)	PTMs, such as phosphorylation and ubiquitination, modify immune proteins, impacting their activity and stability during immune signaling.	Phosphorylation of MAPKs in PTI and ubiquitination of NLR proteins in ETI [23,43].	Regulates protein activation, stability, and interactions critical for signal transduction and immune responses [43].

Molecular Mechanism	Description	Examples	Function in Plant Immunity
RNA Interference (RNAi)	RNAi silences specific genes involved in pathogen virulence by degrading corresponding mRNA.	RNAi targeting of *Fusarium graminearum* genes in barley [54].	Reduces pathogen virulence by silencing key pathogen genes required for infection [54].
Systemic Acquired Resistance (SAR)	SAR is a long-lasting, broad-spectrum immune response activated throughout the plant following a localized infection.	SAR is commonly associated with the accumulation of salicylic acid and the expression of PR genes [12].	Provides systemic protection against future pathogen attacks by priming the immune system across the plant [12].
Proteomics and Metabolomics in Defense	Proteomics studies the defense proteins, while metabolomics examines the small molecules produced during immune responses.	Metabolomics identified phytoalexin camalexin in *Arabidopsis* during infection by *Pseudomonas syringae* [45].	Provides insights into the functional proteins and defense metabolites that directly combat pathogen infection [45].

4. FUNGAL PATHOGEN STRATEGIES TO OVERCOME PLANT IMMUNITY

Fungal pathogens have evolved sophisticated mechanisms to bypass plant immune defenses, essential for their survival and colonization of host plants [30]. Key strategies include the secretion of effector molecules, manipulation of host defense pathways, and rapid adaptation. Effectors are small proteins or compounds secreted by pathogens that interfere with the plant's immune system. For instance, the *Magnaporthe oryzae* effector AVR-Pii suppresses pattern-triggered immunity (PTI) by targeting plant receptor complexes [31]. Similarly, in effector-triggered immunity (ETI), effectors like *Phytophthora infestans* AVR3a suppress the hypersensitive response (HR), a form of programmed cell death crucial for plant defense [32]. Fungal pathogens also manipulate plant hormone signaling, particularly salicylic acid (SA), jasmonic acid (JA), and ethylene (ET), to weaken plant defences [33]. *Verticillium dahliae* inhibits JA signaling, reducing plant resistance against necrotrophic pathogens, while *Gibberella fujikuroi* overproduces gibberellins, weakening rice plants through excessive growth [34]. Additionally, fungal effectors disrupt key signaling pathways like MAPK cascades to block immune responses and facilitate infection [35]. Fungi also exhibit high genetic variation, which allows them to evolve quickly in response to plant resistance mechanisms. Pathogens like *Puccinia graminis* f. sp. *tritici* can rapidly evolve new races, such as Ug99, that overcome resistant wheat varieties [36]. This genetic adaptability is often driven by mutations, recombination, and horizontal gene transfer, enabling the emergence of new virulent strains. Effector genes, located in dynamic genomic regions, evolve rapidly, contributing to the pathogen's ability to evade plant immune receptors [37,38]. This ongoing evolutionary arms race highlights the constant adaptation between fungal pathogens and their plant hosts.

5. ADVANCES IN UNDERSTANDING PLANT IMMUNITY THROUGH MOLECULAR TECHNIQUES

Molecular techniques have dramatically advanced the understanding of plant immunity by providing insights into genetic, biochemical, and physiological processes that govern plant defenses. Genomic and transcriptomic approaches, such as next-generation sequencing (NGS), have enabled researchers to uncover key immune genes, like nucleotide-binding leucine-rich repeat (NLR) genes, which recognize pathogen effectors and trigger defense responses [39,40]. Whole-genome sequencing has identified novel resistance (R) genes, such as the rice *Xa21* gene, offering resistance to *Xanthomonas oryzae* [41]. Transcriptomics, through RNA sequencing (RNA-seq), reveals how gene expression changes during pathogen attacks. It identifies differentially expressed genes and highlights the role of non-coding RNAs like microRNAs (miRNAs) in modulating plant immune responses [42]. Meanwhile, proteomics, employing mass spectrometry, has identified proteins involved in defense, such as PR proteins and those associated with cell wall reinforcement, and studied post-translational

modifications like phosphorylation in immune signaling [43]. Metabolomics offers a look into the metabolic shifts that accompany immune responses, showing how metabolites like camalexin in *Arabidopsis* accumulate in response to pathogen attack, contributing to defense mechanisms [44,45]. Additionally, gene-editing technologies like CRISPR-Cas9 have revolutionized functional genomics by allowing precise modifications of immune-related genes. CRISPR has been used to generate disease-resistant crops, such as rice resistant to bacterial blight through *OsSWEET13* gene editing, demonstrating the potential of these tools in crop improvement [46]. Advanced techniques like base editing and prime editing offer even greater precision for enhancing plant immunity [47].

6. PRACTICAL APPLICATIONS AND IMPLICATIONS

Research into plant immunity has significant implications for agriculture, particularly in developing strategies to enhance crop resistance against fungal pathogens [48,49]. Traditional breeding techniques have long played a key role in this effort, focusing on selecting plants with desirable traits, such as resistance genes, to develop more robust crop varieties. For example, the *Lr34* gene in wheat has provided durable resistance to rust pathogens, while rice variety IR36 has successfully resisted rice blast disease [50]. Molecular markers have also accelerated the breeding process through marker-assisted selection (MAS), allowing for more precise selection of resistance genes [51]. Genetic engineering has further revolutionized crop resistance, enabling the direct manipulation of plant genomes [52]. Transgenic approaches, such as introducing chitinase genes into crops, enhance resistance by degrading fungal cell walls [53]. RNA interference (RNAi) is another promising technology, used to silence critical pathogen genes and reduce their virulence, such as in the case of *Fusarium graminearum* resistance in barley [54]. CRISPR-Cas9 and other genome editing tools allow precise modifications to enhance immunity, as seen in tomato plants edited for resistance to powdery mildew [55]. Challenges remain, particularly with the evolution of new pathogen races and regulatory hurdles surrounding genetically modified crops. However, techniques like pyramiding resistance genes and knocking out susceptibility genes offer promising solutions for developing durable resistance [56,57]. The deployment of resistant crop varieties, coupled with integrated disease management strategies, could reduce reliance on chemical fungicides and enhance sustainable agricultural practices [58].

7. CHALLENGES AND FUTURE D

The study of plant immunity and the development of strategies to combat fungal pathogens are critical for global food security, but they face several challenges. The complexity of plant-fungal interactions, the diversity of fungal lifestyles, and the constant evolution of pathogens make it difficult to develop long-lasting resistance [59]. Fungi, such as biotrophic *Puccinia* spp. and necrotrophic *Botrytis cinerea*, employ diverse infection strategies, while plants exhibit highly specific immune responses based on resistance (R) genes, influenced by environmental factors [60,61]. The co-evolutionary arms race between plants and fungi further complicates resistance efforts, as seen in the evolution of new virulent races of pathogens like *Puccinia graminis* f. sp. *tritici* (Ug99), which overcome previously effective R genes [62]. New fungal pathogens and the resurgence of previously controlled diseases pose significant threats to agriculture. Factors like climate change and the global movement of plant material contribute to the spread of pathogens, such as *Fusarium oxysporum* TR4, which threatens banana production worldwide [63]. Climate change may exacerbate these threats by altering the distribution and severity of fungal diseases, accelerating pathogen evolution, and breaking down resistance in crops [64]. This presents a direct threat to global food security, particularly in crops like wheat, rice, and maize, which are essential for billions of people [65]. Addressing these challenges requires a comprehensive research agenda. Future research should focus on understanding the molecular mechanisms of plant immunity, identifying new R genes, and exploring the role of plant microbiomes in enhancing resistance [66,67]. Developing durable resistance strategies, such as pyramiding R genes and using gene editing technologies, is crucial for staying ahead of evolving pathogens. Additionally, creating climate-resilient crops that can withstand both abiotic stress and pathogens will be increasingly important in the face of climate change [68]. Interdisciplinary collaboration and supportive policy frameworks are also essential to ensure the successful deployment of resistant crop varieties and contribute to sustainable agricultural practices.

8. CONCLUSION

The intricate dynamics of plant immunity, particularly in the face of evolving fungal pathogens, present both significant challenges and opportunities for modern agriculture. The complexity of plant-fungal interactions, the emergence of new and more virulent pathogens, and the ongoing arms race between plant defenses and pathogen strategies underscore the need for continued research and innovation. Advances in molecular techniques, including genomics, proteomics, and gene editing, offer promising avenues for enhancing crop resistance. The success of these strategies hinges on a deep understanding of plant immune mechanisms, the development of durable resistance, and the integration of sustainable practices. As we confront the realities of climate change and global food security, interdisciplinary collaboration and supportive policy frameworks will be crucial in translating scientific breakthroughs into practical solutions that ensure resilient agricultural systems and stable food supplies for the future.

DISCLAIMER (ARTIFICIAL INTELLIGENCE)

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc) and text-to-image generators have been used during writing or editing of this manuscript.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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