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·特邀综述·

植物免疫的转录后调控

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摘要 病原体侵染严重威胁植物的正常生长发育, 是造成作物减产的主要因素之一。植物免疫系统在植物抵抗病原体侵染中发挥核心作用。自2006年提出植物免疫系统主要由模式触发的免疫(PTI)和效应子触发的免疫(ETI)两层防御体系组成以来, 大量的研究工作聚焦于解析PTI和ETI中的关键受体/共受体、PTI和ETI信号通路的组分及其作用机制、植物免疫激素水杨酸和茉莉素的合成与信号转导, 逐步形成了以病原体识别、活性氧爆发、Ca^{2+}内流、MAPK级联信号转导及下游防御基因诱导表达为核心的复杂免疫调控网络。近年的研究表明, 植物免疫相关基因的表达不仅受到转录调控, 其mRNA的稳定性、翻译效率和翻译产物也受到多种转录后调控机制的影响, 包括可变剪接、m^6A修饰、小RNA、uORF和R-motif。该文概述了植物免疫系统的组成和主要的调控通路及其组分, 详述了转录后调控对植物免疫的影响及病原体对相关调控作用的干扰机制, 梳理了转录后调控元件在作物中的应用, 为保障粮食安全、提高作物抗病性以及分子育种元件筛选提供参考。

关键词 植物免疫, 可变剪接, m^6A修饰, 小RNA, uORF

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农业是现代社会的基础。自人类开始种植作物以来, 植物病害就严重威胁农作物的产量, 某些严重的植物病害甚至能对人类社会造成巨大的影响。斯里兰卡曾是全球最大的咖啡产地, 19世纪末遭受了严重的咖啡锈病疫情后, 其咖啡产量大幅下降(Mou et al., 2025)。在咖啡产业受到打击后, 英国殖民者开始转向种植茶树。这使得斯里兰卡成为红茶的主要出口国, 并推动了英国下午茶文化的普及(McCook and Vandermeer, 2015)。由致病疫霉(*Phytophthora infestans*)菌导致的马铃薯(*Solanum tuberosum*)晚疫病对爱尔兰造成灾难性影响, 导致约100万人死亡, 并迫使200万人移居美国(Goss et al., 2014)。大量的爱尔兰人移民美国, 使美国的人口结构发生改变, 深刻影响了美国的现代政治体系。20世纪初, 由尖孢镰刀菌(*Fusarium oxysporum*)引起的香蕉枯萎病导致当时的主要香蕉品种大蜜哈几乎灭绝(漆艳香等, 2023), 继而口感较差的香芽蕉取代大蜜哈成为主要的栽培种。然而, 20世纪60年代对香芽蕉致病的尖孢镰刀菌4号生理小种开始在全球扩散, 这使香蕉产业再次面临严重威胁(李华平等, 2019)。

在植物与病原体旷日持久的“军备竞赛”中, 及时、精准且高效地调动防御基因的表达是生存的关键。长期以来, 植物免疫研究聚焦于模式触发的免疫(pattern-triggered immunity, PTI)和效应子触发的免疫(effector-triggered immunity, ETI)过程中免疫信号的触发、转导及下游转录调控的作用。然而, 抗病相关基因转录本丰度与最终蛋白质水平间常存在显著差异, 这揭示了一个关键的调控层面——转录后调控在塑造植物免疫反应中不可或缺的地位。它如同免疫交响乐中精细的指挥棒, 确保防御反应乐曲在正确的时间、地点以恰当的强度奏响。随着植物免疫研究的不断深入, 转录后水平的精细调控机制逐渐被揭示。转录后调控是植物在面临病原威胁时快速响应的关键环节, 其不仅能快速调整已有mRNA和蛋白质的水平与功能, 还可增加基因表达的多样性和可塑性, 对于植物快速响应病原菌入侵信号至关重要。因此, 深入解析植物免疫中转录后调控机制如何精确调控免疫基因的表达并塑造植物最终抗病表型, 及病原体如

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何通过破坏这些调控过程进而抑制免疫过程, 成为当前植物病理学和作物抗病改良研究最活跃且富有前景的领域之一。

1 植物免疫系统

在与病原体长期共演化过程中, 植物发展出一套精密的免疫系统(图1)。植物细胞表面存在一类模式识别受体(pattern recognition receptors, PRRs)分子, 通过其细胞外结构域监测病原体入侵。主要机制是识别病原菌表面的一些保守分子(如几丁质、多肽和鞭毛蛋白)——病原相关分子模式(pathogen-associated molecular patterns, PAMPs), 以及植物本身在受到机械损伤、病原体侵袭或其它应激条件下释放的内源性分子(如细胞壁降解产生的寡聚糖、细胞死亡释放的DNA片段和胞外小分子eATP (extracellular ATP))——损伤相关分子模式(damage-associated molecular patterns, DAMPs), 进而激活下游的免疫反应(Boutrot and Zipfel, 2017; Hou et al., 2019)。由PRR介导的免疫反应被称为病原相关分子模式触发的免疫反应(PTI)。根据其结构和功能, PRRs主要分为两大类: 受体激酶(receptor-like kinases, RLKs)和受体样蛋白(receptor-like proteins, RLPs)。受体激酶是一类跨膜蛋白, 通常包含1个细胞外配体结合域、1个跨膜域和1个细胞内激酶域。它们在植物免疫反应中起信号识别及转导作用, 通过磷酸化下游蛋白来激活免疫反应。例如, FLS2可识别细菌鞭毛蛋白中1个高度保守的22个氨基酸肽段flg22 (Chinchilla et al., 2006)、EFR (elongation factor Tu receptor)可识别细菌延伸因子EF-Tu中的18个氨基酸保守肽段elf18 (Zipfel et al., 2006)、CERK1 (chitin elicitor receptor kinase 1)可识别真菌细胞壁的几丁质等。受体样蛋白与受体激酶类似, 也具有细胞外配体结合域和跨膜域, 但缺乏细胞内激酶域。它们通常通过与受体激酶或其它信号蛋白互作来传递信号(Iizasa et al., 2010)。PRRs在识别PAMPs或DAMPs后, 通常会与胞内共受体, 如BAK1 (brassinosteroid insensitive 1-associated kinase 1) (Heese et al., 2007; Sun et al., 2013)和SOBIR1 (suppressor of BIR1-1)互作(Liebrand et al., 2013), 进一步激活受体样细胞质激酶(receptor-like cytoplasmic kinases, RLCKs), 触发MAPK (mitogen-activated protein kinase)级联反应, 诱导防御基因的表达(Lin et al., 2014)。胞内的RLCK, 如BIK1 (botrytis-induced kinase 1)在接收到PAMPs信号后, 可通过磷酸化钙离子通道蛋白CNGC (cyclic nucleotide-gated ion channels)等介导Ca^{2+}内流(Tian et al., 2019)。Ca^{2+}作为第二信使促进下游一系列信号的激活(Xu et al., 2022)。此外, BIK1还可磷酸化质膜定位的RBOHD (respiratory burst oxidase homolog D), 使其处于预激活状态, RBOHD在与Ca^{2+}结合后激活其酶活性, 导致活性氧(reactive oxygen species, ROS)爆发(Zhang et al., 2020; Jones et al., 2024)。ROS不仅具有直接的抗菌作用, 还可以作为信号分子传递免疫信号(Kadota et al., 2015; Qi et al., 2017)。

PTI是植物免疫系统的第一道防线。病原体通过分泌效应子(effectors)干扰PTI的关键步骤进而促进感染, 如干扰PRR的功能、抑制MAPK级联反应、干扰激素信号通路和调控基因表达。丁香假单胞菌(*Pseudomonas syringae*)作为植物免疫与病原菌互作研究的经典模式细菌, 其效应子干扰植物免疫的分子机制已得到深入解析。例如, 效应子AvrPto通过直接靶向受体激酶FLS2和EFR抑制其激酶活性(Xiang et al., 2008); 也可通过靶向共受体BAK1干扰其与PRRs的相互作用, 进而破坏免疫复合体的形成, 抑制PTI的信号转导(Shan et al., 2008)。AvrPtoB具有E3泛素连接酶活性, 可介导FLS2和CERK1的降解(Göhre et al., 2008; Gimenez-Ibanez et al., 2009)。HopAI1具有磷酸苏氨酸裂解酶活性, 通过去除MAPKs的磷酸基团抑制其激酶活性(Zhang et al., 2007)。HopU1具有单ADP-核糖基转移酶活性, 可以直接结合并催化RNA结合蛋白GRP7 (glycine-rich protein 7)发生ADP-核糖基化修饰。该修饰改变了GRP7的构象和功能, 抑制其与靶标*FLS2*和*EFR* mRNA的相互作用, 导致这些PRR的mRNA无法有效翻译, 降低受体蛋白的水平, 从而抑制PTI信号的激活(Fu et al., 2007; Nicaise et al., 2013)。AvrRpt2具有半胱氨酸蛋白酶活性, 通过加速生长素信号通路关键抑制因子Aux/IAA蛋白的降解, 促进生长素的信号转导, 抑制抗病基因的表达(Cui et al., 2013)。

植物通过识别病原效应子, 产生由效应子触发的免疫(ETI), 即植物免疫的第二道防线。与PTI的广谱

性不同，ETI能够实现对特定病原体的精准识别与防御。植物体内的NLR (nucleotide-binding leucine-rich repeat)蛋白是一类能识别病原效应子的细胞内免疫受体，通常由3个功能模块构成：分别是位于N端的卷曲螺旋或Toll/白细胞介素1受体结构域(Toll/interleukin 1 receptor (TIR) domain)、中间的核苷酸结合结构域(nucleotide-binding site, NBS)及C端的高度可变亮氨酸重复序列(leucine-rich repeat receptors, LRR)结构域(Ellis et al., 2000; 杨程惠子等, 2020)。NLR蛋白中的LRR结构域主要负责病原菌效应子的特异性识别，并具有高度的序列多样性和变异性。按照N端结构域特征，可将NLR蛋白划分为3类：卷曲螺旋形CNL、Toll受体型TNL和类似于RPW8的RNL。部分NLR蛋白在识别病原效应子后会形成多蛋白复合物，被称为抗病小体(resistosome)，发挥离子通道或催化酶作用。例如，CNL蛋白ZAR1 (HopZ-activated resistance 1)可以识别被黄单胞菌(*Xanthomonas campestris*)效应子AvrAC尿苷酰化修饰的PBL2 (PBS1-like protein 2) (Wang et al., 2015a)，进而与RKS1 (resistance related kinase 1)结合形成五聚化ZAR1-RKS1-PBL2^{UMP}的抗病小体(Wang et al., 2019a, 2019b)。进一步研究表明，该抗病小体定位于细胞膜，作为钙离子通道介导Ca^{2+}内流，触发细胞的程序性死亡(programmed cell death, PCD) (Bi et al., 2021)。小麦(*Triticum aestivum*)中的Sr35结构与ZAR1类似，也可发挥钙离子通道活性(Förderer et al., 2022)。拟南芥(*Arabidopsis thaliana*)中的TNL蛋白RPP1能直接识别霜霉病卵菌*Hpa* Noco2 (*Hyaloperonospora arabidopsidis* Noco2)分泌的效应子ATR1 (*Arabidopsis thaliana* recognized 1)，识别后通过寡聚化形成四聚体，并激活NAD^+水解酶活性，触发免疫反应(Ma et al., 2020)。除典型的TNL/CNL外，植物体内还存在大量仅含TIR结构域的截短型TNL (Meyers et al., 2002)，可在病原菌入侵产生的NAD^+/ATP作用下诱发相分离(phase separation)，激活其本身的NAD^+水解酶活性。例如，拟南芥中的TIR蛋白RBA1 (response to the bacterial type III effector protein HopBA1)及TX14 (AT2G32140)在与NAD^+结合后均可诱导其TIR结构上的无序区发生构象改变，介导头尾相对的TIR-TIR互作，引发相分离并形成NAD^+催化中心，进而诱发细胞死亡(Song et al., 2024)。除直接识别效应子，还有部分NLR蛋白可间接识别效应子，即识别效应子所诱导的另一种蛋白质修饰，从而触发免疫应答。这种识别类型被称为诱饵模型(decoy model) (van der Hoorn and Kamoun, 2008)。例如，在拟南芥中，效应子AvrRpt2可靶向RPM1的互作蛋白RIN4 (RPM1-interacting protein 4)并切割其磷酸化位点，这种磷酸化修饰会使RIN4被CNL蛋白RPS2识别，并最终触发免疫应答(Toruño et al., 2019)。

ETI通常会在感染位点诱导宿主细胞的局部快速程序性死亡，即超敏反应(hypersensitive response, HR)。HR不仅通过形成局部坏死病斑对病原物进行物理隔离，还能够通过激活抗病激素水杨酸(salicylic acid, SA)的生物合成，触发系统获得性抗性(systemic acquired resistance, SAR) (Zavaliev and Dong, 2024)。NPR1 (nonexpresser of pathogenesis-related genes 1)是SA信号通路的核心组分。在免疫未被激活时，NPR1以多聚体形式存在于细胞质中。在病原菌侵染导致SA含量升高后，细胞内的氧化还原状态改变，促进NPR1从多聚体形式还原为二聚体形式，进入细胞核行使功能(Kumar et al., 2022)。在核内，NPR1通过与TGA类转录因子(TGA transcription factors)形成异源复合体，发挥转录共激活因子作用，诱导病程相关蛋白基因(*PR* genes)等防御基因的表达(Yan and Dong, 2014)。这种转录调控网络不仅能增强局部组织的抗病性，还可通过SA系统信号转导，在未被直接感染的远端组织中诱导出广谱抗病性，最终形成植物的系统抗病能力。

PTI与ETI并非独立存在，而是相互协同，彼此增强。在拟南芥的PRR受体突变体中，由效应子AvrRpt2及AvrRps4等诱导的ETI严重受损。在拟南芥中过表达AvrRps4也可促进flg22诱导的ROS产生、胼胝质积累及免疫基因表达等多种生理过程。PTI与ETI通过Ca^{2+}和ROS等共同信号及MAPK等途径触发转录重编程，促进免疫相关基因表达，实现对病原体的有效防御(Ngou et al., 2021; Yuan et al., 2021; 王伟和唐定中, 2021)。

植物免疫系统的激活依赖于转录调控这一关键节点，其通过整合PTI和ETI信号、SA、茉莉酸(jasmonic acid, JA)等免疫激素，以及ROS和Ca^{2+}等小分子信号通路，触发防御基因的转录重编程以形成动态免

疫响应。WRKY、TGA、NAC (NAM/ATAF1,2/CUC2)、MYC和ERF (ethylene-response element binding factor)等关键转录因子家族成员通过协同调控免疫基因网络，介导植物对病原菌侵染的转录层面应答(Aerts et al., 2022)。WRKY转录因子通过特异性识别W-box顺式作用元件调控下游靶基因，其中WRKY33被MPK3/MPK6磷酸化后形成反式激活复合物，同步增强自身及其靶基因*PAD3* (*PHYTOALEXIN DEFICIENT 3*)的转录活性，促进植保素(亚麻荠素)的生物合成(Mao et al., 2011)。SA信号转导中的核心转录共因子NPR1可与包括TGA1、TGA2、TGA3、TGA5和TGA6在内的多个TGA转录因子家族成员互作，激活TGAs的转录活性。TGAs识别as-1顺式作用元件，促进*PR1*等防御基因的表达(Després et al., 2000, 2003; Zhang et al., 2003; Kumar et al., 2022)。NAC转录因子在植物免疫调控中扮演双重角色，既可激活抗病通路，也可抑制免疫反应。气孔是多种病原体侵染植物的天然通道。植物可通过关闭气孔增强其对病原体的抗性，该过程被称为气孔免疫。NAC053通过转录激活*BBX11*，正向调控拟南芥的气孔免疫，提高对丁香假单胞菌番茄致病变种DC3000 (*P. syringae* pv. *tomato* DC3000)的抗病性(Luo et al., 2025)。丁香假单胞菌通过分泌一种被称为冠菌素(coronatine)的JA类似物，促进气孔打开，增强对植物的致病性。这一过程由ANAC19、ANAC55和ANAC72三种NAC蛋白介导，它们抑制SA合成酶关键基因*ICS1* (*ISOCHORISMATE SYNTHASE 1*)的表达，诱发病原菌侵染时气孔重新打开(Zheng et al., 2012)。JA及其衍生物在植物应对生物胁迫过程中扮演关键角色，通过复杂的信号转导网络，协调植物对植食性昆虫和病原微生物的多层次防御反应。JA介导的免疫应答主要通过2个转录调控模块实现：即MYC主导的防御反应通路和ERF调控的防御反应通路。在植物遭受机械损伤或昆虫取食时，JA信号系统迅速启动MYC防御途径。这一过程涉及JAZ (jasmonate ZIM-domain)抑制蛋白的泛素化降解，从而释放被抑制的MYC类转录因子。MYC蛋白通过其保守的碱性螺旋-环-螺旋结构域识别靶基因启动子区G-box元件，特异性激活包括*VSP2* (*VEGETATIVE STORAGE PROTEIN 2*)在内的一系列防御相关基因的转录(Lorenzo et al., 2004; Kazan and Manners, 2013)。当植物遭遇坏死性病原体侵染时，JA信号系统优先激活ERF家族转录因子介导的防御通路。与MYC通路不同，ERF蛋白通过结合GCC-box (AGCCGCC)顺式作用元件，特异性调控*PDF1.2* (*PLANT DEFENSIN 1.2*)等抗病基因的表达(Pré et al., 2008)。转录因子SARD1 (SAR DEFICIENT 1)、CBP60g (CALMODULIN BINDING PROTEIN 60g)、NTL9 (NTM1-LIKE 9)、CHE (CCA1 HIKING EXPEDITION)、TCP8 (TEOSINTE BRANCHED 1/CYCLOIDEA/PCF 8)和TCP9可结合到*ICS1*的启动子上，调控SA的合成(Zhang et al., 2010; Wang et al., 2015c; Zheng et al., 2015)。植物免疫系统除受到一些典型的转录因子家族调控外，也受到生物钟的转录调控。约一半免疫响应基因的表达具有昼夜节律特征(周冕，2022)，包括PRR基因*FLS2*及NLR基因*RPP4*等(Bhardwaj et al., 2011)。生物信息学分析发现，约有464个抗病相关基因启动子上含有生物钟核心基因蛋白CCA1 (CIRCADIAN CLOCK ASSOCIATED 1)及LHY (LATE ELONGATED HYPOCOTYL)的结合位点(Zhang et al., 2013)。表明生物钟调控机制对PTI与ETI核心通路中基因表达周期性调控同样至关重要。

以往的研究主要关注植物免疫过程中基因表达的转录调控过程。然而在植物免疫过程中，mRNA的转录水平与其对应的蛋白质丰度并不完全一致，表明mRNA的转录后调控在植物免疫中发挥关键作用(Xu et al., 2017b)。越来越多的研究显示，植物免疫相关基因mRNA的功能受到可变剪接、m^6A (N^6 methyladenosine)修饰和亚细胞定位等转录后调控机制的影响。mRNA中的非编码序列(如上游开放阅读框(upstream open reading frames, uORF)和R-motif)在植物免疫中的功能逐渐被揭示。此外，非编码RNA，尤其是miRNA及siRNA对mRNA的靶向调控也备受关注。本文将从mRNA的非编码区结构、可变剪接及mRNA的修饰等多个方面，总结mRNA的转录后调控在植物免疫中的作用。

2 植物免疫中mRNA的调控机制

2.1 可变剪接

可变剪接(alternative splicing, AS)作为真核生物转录后调控的核心机制，通过选择性剪接前体mRNA

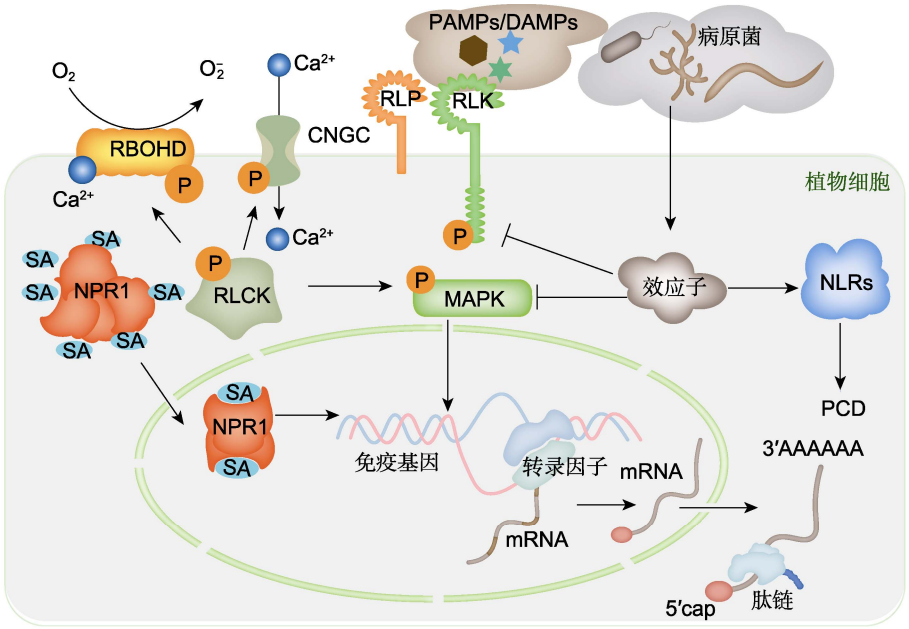

图1 植物免疫概述

植物细胞表面的PRRs识别病原菌侵染时释放的PAMPs或者产生的DAMPs后，激活下游RLCKs，进而激活MAPK级联激活途径，促进免疫基因的表达。同时，RLCKs可通过其激酶活性促进ROS产生及Ca^{2+}内流，协同抑制病原菌侵染。病原菌可通过分泌效应子抑制PRRs及MAPK的激活。当效应子进入植物细胞后，胞内NLR可特异性识别效应子进而诱发细胞的程序性死亡(PCD)，抑制病原菌扩散。此外，病原菌侵染会导致水杨酸(SA)的积累，SA结合NPR1后，NPR1由多聚体形式转换为二聚体形式入核，发挥转录共激活因子功能，促进免疫基因的表达。PRRs: 模式识别受体; PAMPs: 病原相关分子模式; DAMPs: 损伤相关分子模式; RLCK: 受体样细胞质激酶; ROS: 活性氧; RLP: 受体样蛋白; RLK: 受体激酶; MAPK: 丝裂原活化蛋白激酶; NLR: 核苷酸结合富含亮氨酸重复序列; RBOHD: 呼吸爆发氧化酶同源蛋白D

Figure 1 Overview of plant immunity

After recognition of PAMPs or DAMPs released during pathogen invasion, PRRs on plant cell surfaces activate downstream RLCKs, subsequently triggering the MAPK cascade to drive immune gene expression. Simultaneously, RLCKs enhance ROS production and Ca^{2+} influx through kinase activity, coordinately suppressing pathogen infection. Pathogens secrete effectors to inhibit PRRs signaling and MAPK activation. When effectors enter plant cells, intracellular NLR specifically detect these effectors, inducing Programmed Cell Death (PCD) to prevent pathogen spread. Furthermore, pathogen infection promotes salicylic acid (SA) accumulation. SA binding to NPR1 induces its transition from oligomeric to dimeric forms, enabling nuclear entry to function as a transcriptional coactivator, to potentiate the expression of immune-responsive genes. PRRs: Pattern recognition receptors; PAMPs: Pathogen-associated molecular patterns; DAMPs: Damage-associated molecular patterns; RLCK: Receptor-like cytoplasmic kinase; ROS: Reactive oxygen species; RLP: Receptor-like protein; RLK: Receptor-like kinase; MAPK: Mitogen-activated protein kinase; NLR: Nucleotide-binding leucine-rich repeat; RBOHD: Respiratory burst oxidase protein D

(pre-mRNA)的5′和3′剪接位点，能从单个基因模板中生成异构体mRNA (mRNA isoforms)，继而翻译出功能多样的蛋白质产物。根据剪接模式的特征差异，可将AS分为以下几种主要类型。(1) 外显子跳跃(exon skipping, ES): 某些外显子随内含子序列一起被剪切去除; (2) 内含子保留(intron retention, IR): 部分内含子在剪接过程中未被切除，这类剪接事件在植物基因组中具有显著富集特征(Reddy, 2007); (3) 选择性5′剪接位点(alternative 5′ splice site, A5SS)和选择性3′剪接位点(alternative 3′ splice site, A3SS)，分别体现在外显子5′或3′端存在多个可替代的剪接位点，导致外显子长度产生差异性改变; (4) 互斥外显子(mutually exclusive exons)，指2个或更多相邻外显子在单次剪接事件中仅保留其中1个; (5) 可变启动子使用(alternative promoter usage)，表现为基因启动区存在多个转录起始位点(transcription start site, TSS)，引发转录本5′端差异(Liu et al., 2022)。

转录组研究表明，拟南芥超过61%有内含子的基

因均存在可变剪接现象(Marquez et al., 2012)。值得注意的是, 除通过外显子拼接改变导致蛋白质序列及结构改变外, 这种剪接异构性还可通过其它途径调控基因的功能。例如, 引入提前终止密码子(premature termination codon, PTC)引发的无义介导的mRNA降解(nonsense-mediated mRNA decay, NMD), 或调控RNA二级结构影响转录后加工, 间接影响基因表达的时空特异性与功能输出(Filichkin et al., 2010)。

剪接体(splicesome)是一个复杂的RNA-蛋白质复合体, 可精确识别pre-mRNA的剪接位点并催化pre-mRNA的内含子切除, 从而产生成熟的mRNA (图2)。这一机制在植物免疫应答过程中受到精细调控, 其中拟南芥的SR (serine/arginine-rich)家族蛋白作为剪接体的重要组成部分, 大部分成员可通过改变其磷酸化状态, 影响与其它蛋白和mRNA的互作, 在植物免疫中扮演关键调控角色(dela Fuente et al., 2006; Kufel et al., 2022)。植物识别到PAMPs后会触发PTI信号通路, MAPK信号通路被激活。MPK4激酶可以通过磷酸化SR家族蛋白(如SCL30 (SC35-like splicing factor 30)), 诱导下游大量基因的剪接模式改变(Bazin et al., 2020)。此外, 其它剪接体组分也受到磷酸化调控。例如, 病原菌可通过分泌RALF (rapid alkalinization factor)小肽抑制植物的免疫(Masachis et al., 2016)。受体激酶FER (FERONIA)识别RALF1后, 通过调控剪接体组分GRP7的磷酸化状态, 增强其与另一剪接体组分U1-70K互作, 从而促进剪接位点的识别与内含子切除(Wang et al., 2020)。

可变剪接还可导致NLR基因的转录本种类和功能的多样性, 从而精细调控植物对病原体的识别和免疫应答。TNL类及CNL类基因的pre-mRNA均被证实存在可变剪接现象。烟草(*Nicotiana tabacum*)中的TNL类基因*N*基因是第1个被报道产生2个转录本的NLR基因。*N*基因通过可变剪接产生截短的转录本N_L与全长转录本N_S。仅表达截短转录本N_L或者将二者比例调整为1:1均不能实现抗病, 受侵染后迅速调整N_S与N_L的比例, 对于烟草抵抗烟草花叶病毒(TMV)的免疫反应至关重要(Dinesh-Kumar and Baker, 2000)。拟南芥的TNL类*R*基因*RPS4* (*RESISTANT TO PSEUDOMONAS SYRINGAE 4*)通过可变剪接可以产生不同的转录本, 在免疫过程中发挥重要作用。RPS4通过特异性识别丁香假单胞菌效应子AvrRps4, 激活植物的免疫反应。RPS4的抗病功能依赖于可变剪接产生的全长及截短形式的蛋白, 其中全长蛋白负责效应子的识别及信号转导, 截短蛋白则通过与全长蛋白互作形成异源二聚体以解除全长蛋白的自抑制状态, 从而激活下游免疫信号通路。仅表达其中1种转录本无法实现抗病, 双转录本共存是RPS4触发植物免疫的必要条件(Zhang et al., 2003)。马铃薯晚疫病抗性基因*RB*编码的CNL类免疫受体RB通过识别致病疫霉菌效应子IPI-O1 (Avrblb1), 触发ETI免疫反应(Champouret et al., 2009)。无病原菌侵染时, 内含子保留导致*RB_IR* mRNA产生提前终止密码子, 编码仅保留N端结构域的截短蛋白无法激活免疫。而致病疫霉菌侵染后, 效应子IPI-O1调控剪接体组分CWC15的亚细胞定位, 内含子被正常切除, 生成全长*RB_CDS* mRNA。值得注意的是, *RB_CDS*在非感染条件下过量表达会扰乱植物的正常生长发育, 而RB_IR通过与全长蛋白互作抑制其活性, 表明可变剪接可通过截短异构体维持生长与免疫间的动态平衡(Sun et al., 2024)。此外, 拟南芥的*SNC1* (*SUPPRESSOR OF NPR1-1, CONSTITUTIVE 1*)、*RPS6*和*RPP5* (*RECOGNITION OF PERONOSPORA PARASITICA* 5)等NLR基因均被证实可发生可变剪接(Parker et al., 1997; Kim et al., 2009; Yang et al., 2014)。

2.2 m^6A修饰

m^6A是一种重要的RNA表观遗传修饰, 由m^6A甲基转移酶复合物在腺嘌呤碱基的N6位点加入1个甲基基团(称为“写入”, writers)催化完成。该复合体主要由MTA (methyltransferase A)、MTB (methyltransferase B)、FIP37 (FKBP interacting protein 37)以及VIR (VIRILIZER)等组成。该修饰具动态可逆性, 可被m^6A去甲基化酶(如拟南芥中的ALKBH10B (alkylated DNA repair protein AlkB homolog 10B)和ALKBH9B (Duan et al., 2017, 2019))擦除(称为“擦除”, erasers)。作为真核生物mRNA中最普遍的内部化学修饰之一, m^6A主要集中于终止密码子周围及3'UTR内(Parker et al., 2020)。m^6A修饰的RNA可被特定的m^6A结合蛋白识别(被称为“读取”, readers) (图2)。目前, 拟南芥中只发现5种读取蛋白, 即ECT (evolutionarily conserved C-terminal region)家族的ECT1/2/3/4与CPSF30-L (cleavage and polyadenylation specificity

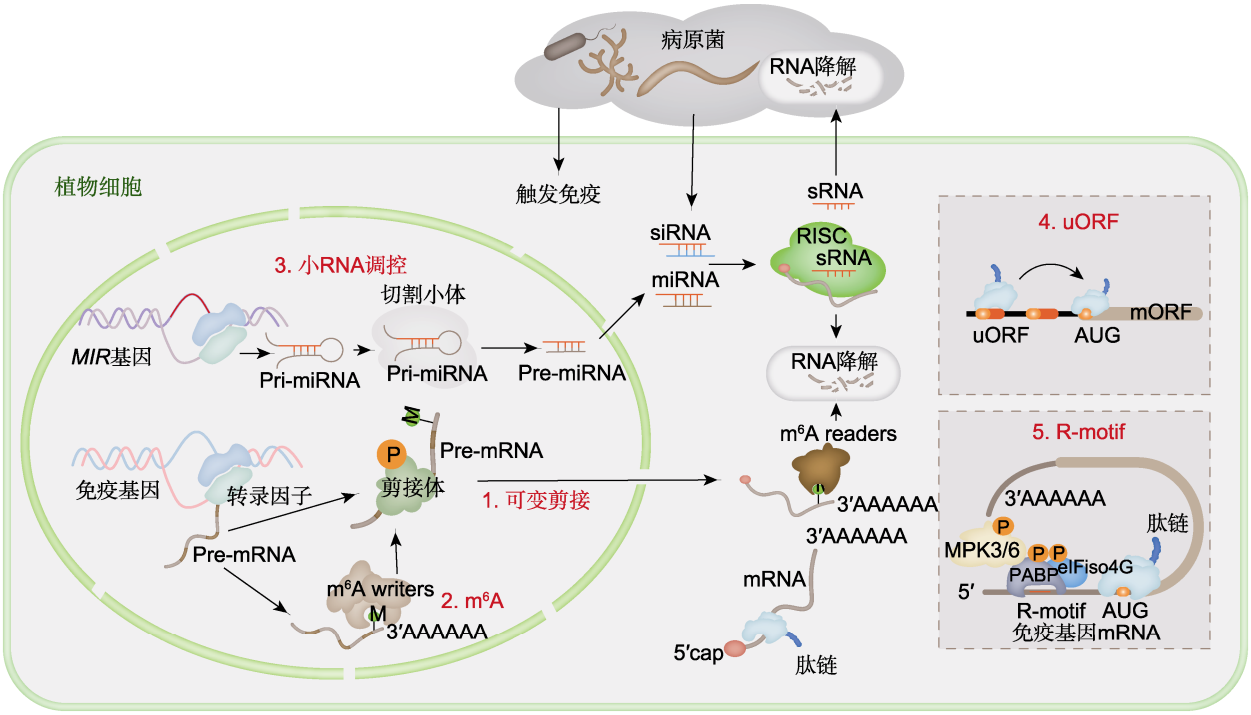

图2 植物免疫中RNA及其翻译的调控

植物免疫过程中，剪接体组分被磷酸化促进mRNA的剪接，此外可变剪接还可导致NLR基因转录本种类及功能多样性，各转录本协同调控植物免疫。mRNA的腺嘌呤N6位点可发生甲基化修饰(m^6A)，该修饰被特异性识别蛋白(readers)识别后调控mRNA的稳定性及核质转运，从而参与免疫信号的精细化调控。miRNA来源于*MIR*基因的转录，而siRNA来源于双链的dsRNA。二者在成熟后一条链被组装进入RISC，另一条链降解。RISC通过与目标mRNA结合进而切割mRNA或者抑制其翻译，部分产生的sRNA还可被运输进入病原体，抑制病原体关键致病基因，以抑制其侵染过程。此外，位于mRNA 5'端的uORF通常会抑制mORF的翻译，但是在受到胁迫时可解除uORF的翻译抑制，启动mORF的翻译。部分免疫基因的5'端具有可作为IRES元件的R-motifs，植物免疫激活后，R-motifs通过与PABPs结合，替代经典通路中eIF4E的功能，直接招募翻译起始因子启动非帽依赖翻译。此过程中，关键调控因子PABP的磷酸化修饰可增强其与R-motifs的亲和力，协同促进免疫蛋白的翻译。NLR: 富含亮氨酸重复序列; RISC: RNA诱导沉默复合体; uORF: 上游开放阅读框; mORF: 主开放阅读框; IRES: 内部核糖体进入位点; PABPs: 多聚腺苷酸结合蛋白; eIF4E: 真核翻译起始因子4E

Figure 2 Regulation of RNA and translational control in plant immunity

During plant immune responses, phosphorylation of spliceosome components enhances mRNA splicing efficiency, while alternative splicing diversifies NLR gene transcripts, enabling synergistic coordination among isoforms to regulate immunity. The m^6A methylation at mRNA adenine N6 positions is recognized by reader proteins, fine-tuning immune signaling through modulating transcript stability and nucleocytoplasmic transport. miRNA originates from *MIR* gene transcripts, whereas siRNA derives from dsRNA. After maturation, one strand is incorporated into the RISC, while the other strand is degraded. RISC suppresses target mRNA via cleavage or translational repression. Certain sRNAs may translocate into pathogens to silence virulence genes, inhibiting infection. Moreover, uORFs at mRNA 5′ termini typically repress mORF translation, but stress conditions alleviate this inhibition to activate mORF expression. Notably, R-motifs in the 5′ regions of select immune genes function as IRES elements. Upon immune activation, R-motifs recruit translation initiation factors by interacting with PABPs, bypassing the canonical cap-dependent pathway. Phosphorylation of PABPs strengthens their binding affinity to R-motifs, synergistically boosting the translation of immune-related proteins. NLR: Nucleotide-binding leucine-rich repeat; RISC: RNA-induced silencing complex; uORF: Upstream open reading frames; mORF: Main open reading frame; IRES: Internal ribosome entry site; PABPs: Poly(A)-binding proteins; eIF4E: Eukaryotic initiation factor 4E

factor30, CPSF30的同源蛋白)。这些m^6A结合蛋白可影响mRNA的多种生物学过程，包括pre-mRNA的剪接位点识别，及mRNA的核质运输、稳定性和翻译效率(Zhao et al., 2014; Wang et al., 2015b; Akhtar et

al., 2021; Tang et al., 2023b; Zhu et al., 2023)。通过调控这些生物学过程，m^{6}A修饰在植物的生长发育、生物及非生物胁迫等多个重要生理过程中发挥关键作用(Govindan et al., 2022; Li et al., 2025)。

m^{6}A修饰通过复杂的动态调控机制，在植物免疫反应中发挥关键作用。m^{6}A识别蛋白ECT家族蛋白具有无序区，能发生相分离。ECT1可将SA诱导的m^{6}A修饰的免疫相关mRNA招募到聚集体中促进其降解，抑制SA介导的免疫反应(Lee et al., 2024)。ECT2与ECT3则通过与poly(A)结合蛋白PAB2和PAB4相互作用，维持m^{6}A修饰的RNA稳定性，促进PTI过程中免疫基因的翻译(Wei et al., 2018; Song et al., 2023; Chen et al., 2024)。通过分析m^{6}A甲基转移酶复合体亚基FIP37的功能缺失突变体*fip37-4*，发现其对丁香假单胞菌*Psm* ES4326 (*P. syringae* pv. *maculicola* ES4326)及霜霉病卵菌(*Hpa*)表现出易感性。当突变体经elf18预处理后，其PTI防御反应显著受损，这直接证明了m^{6}A修饰对PTI建立的必要性。转录组分析进一步显示，在PTI响应过程中，包括防御正调控基因*EDS5*和*WRKY27*在内的免疫相关基因的mRNA转录本会出现特异性m^{6}A修饰，这为m^{6}A在PTI应答中对新生免疫转录本的动态调控提供了分子证据。值得注意的是，m^{6}A修饰对免疫相关mRNA表现出双重调控效应。在PTI的早期阶段，m^{6}A识别蛋白ECT2、ECT3和ECT4可识别被m^{6}A修饰的mRNA，提高免疫基因的翻译效率。而ECT1通过促进修饰mRNA的降解来实现免疫应答的精确终止(Chen et al., 2024)。但Prall等(2023)的研究得出了不同的结论，他们发现m^{6}A写入缺陷型拟南芥(如MTA突变体*mta*)对丁香假单胞菌的抗性更强，表明m^{6}A在免疫中的作用可能较为复杂。

2.3 sRNA

植物体内的小RNA (small RNA, sRNA)主要通过调控mRNA稳定性或翻译效率影响基因表达，在植物生长发育及应对生物胁迫中发挥至关重要的作用(Chand Jha et al., 2021)。sRNA主要分为两大类: miRNA (microRNA)和siRNA (small interference RNA)。这2类小RNA的大小相似，长度通常介于20–24个核苷酸之间，但在合成途径和前体结构上有所不同(Axtell, 2013)。

miRNA的生成起始于*MIR*基因(miRNA genes)的转录。*MIR*的初级转录产物为具有发夹状二级结构的pri-miRNA (primary miRNA)，随后被DCL1酶(dicer-like 1)及其辅助蛋白识别切割，生成pre-miRNA中间体。之后，pre-miRNA被转运至细胞质中进一步加工，最终形成由20–24个核苷酸组成的双链成熟miRNA (Rogers and Chen, 2013)。成熟miRNA的引导链会被组装入以AGO (ARGONAUTE)蛋白为核心的RNA诱导沉默复合体(RNA-induced silencing complex, RISC)，另一条链则被降解(Echevarría-Zomeño et al., 2013)。RISC通过miRNA与靶mRNA的序列互补性识别目标，进而切割mRNA或者抑制其翻译，调控基因表达。与miRNA不同，siRNA主要来源于互补的双链RNA前体dsRNA (double-stranded RNA)的加工。dsRNA前体通常由RNA依赖的RNA聚合酶(RNA-dependent RNA polymerase, RDR)以单链RNA为模板合成互补链形成，或者由外源病毒/病原菌导入。dsRNA前体会被DCL酶切割，产生长度均为20–24个核苷酸的短双链RNA分子。siRNA双链组合同样会被加工并组装入RISC复合体，进而靶向目标mRNA (图2) (Zhang et al., 2012; Qiao et al., 2021)。

作为RISC的核心成分，AGO蛋白在植物免疫中发挥重要作用。AGO通过识别不同类型的sRNA引导复合体靶向特定mRNA，导致目标mRNA的转录后基因沉默(post-transcriptional gene silencing, PTGS)。通过其核酸内切酶活性促进靶mRNA的切割，或者抑制靶mRNA的翻译。例如，拟南芥中*miRNA393*可被flg22诱导上调表达，在结合AGO1后靶向F-box生长素受体基因*TIR1* (*TRANSPORT INHIBITOR RESPONSE 1*)、*AFB2*与*AFB3* (*AUXIN SIGNALING F-BOX PROTEINS 2 AND 3*)的mRNA，抑制生长素信号通路，增强植物的抗病性(Navarro et al., 2006)。AGO2是唯一1种可被细菌诱导表达的AGO。AGO2可结合*miRNA393b**并导致定位于高尔基体的*MEMB12*基因沉默，使PR1的胞吐增加，增强对丁香假单胞菌的抗性(Zhang et al., 2011; Huang et al., 2019)。此外，miRNA可通过靶向植物免疫信号通路中关键基因的mRNA发挥作用。*mi472*靶向抗病基因*RPS5*及*RSG1* (*RESISTANCE SILENCED GENE 1*)的mRNA抑制其积累，负调控免疫。抗性基因*RPS2* (*RESISTANT TO P. SYRINGAE 2*)编码的蛋白可特异性识别丁香

假单胞菌效应子AvrRpt2, 进而诱导ETI发生。siRNA nat-siRNAATGB2靶向沉默*RPS2*负调控基因*PPRL* (*PENTATRICOPEPTIDE REPEATS-LIKE*)的mRNA, 在AvrRpt2诱导的ETI中充当正调控因子(Katiyar-Agarwal et al., 2006)。同一种miRNA在不同植物与病原体的互作中可能发挥不同作用。例如, 水稻(*Oryza sativa*)中*miR398b*通过促进ROS的产生, 在抵抗真菌病害方面起正调控作用, 拟南芥中则通过抑制PAMPs介导的胼胝质沉积, 在对细菌的抗性中起负调控作用(Li et al., 2010, 2019; Nadarajah and Abdul Rahman, 2022)。

除靶向植物内源mRNA, sRNA还可从植物中转移至病原菌体内, 诱导目标mRNA的降解, 达到抗病的目的。植物可产生与病原体毒性基因高度互补的sRNA, 被称为外泌体的一类细胞外囊泡运输至病原体细胞中, 通过RNA干扰途径沉默病原体的关键基因, 从而抑制病原体的致病性。例如, 拟南芥通过分泌*siR483*和*siR453*等sRNA靶向灰霉菌(*Botrytis cinerea*)参与囊泡运输、液泡蛋白分选及膜分泌运输等过程的相关基因, 干扰灰霉菌的囊泡运输过程, 抑制其侵染能力(Cai et al., 2018)。sRNA还可通过外源导入病原菌的*MIR*基因介导产生。例如, 在小麦中过表达靶向白粉病菌(*Blumeria graminis*)发育基因*GTF1/2* (*1,3-β-GLUCANOSYLTRANSFERASE 1 AND 2*)的siRNA可降低其致病性(Nowara et al., 2010)。该过程被称为宿主诱导的基因沉默(host-induced gene silencing, HIGS)。

2.4 uORF

uORF是位于mRNA 5'UTR区域, 以ATG起始的短的开放阅读框, 是一种常见的RNA调控元件, 在真核生物mRNA中普遍存在。uORF可精细调控主开放阅读框(major open reading frame, mORF)的翻译(von Arnim et al., 2014; Zhu et al., 2024)。根据uORF终止密码子位置的不同, 其主要分为3类: uORF终止密码子位于mORF前面(Type1); uORF终止密码子位于mORF内(Type2); uORF与mORF享用同一个终止密码子(Type 3) (Wang et al., 2024; Mou et al., 2025)。由于Type1 uORF终止密码子位于mORF前, 这种类型的uORF通常通过抑制核糖体的重新起始调控mORF的翻译。当核糖体完成uORF的翻译并在其终止密码子处停止时, 核糖体可能从mRNA上解离, 进而降低mORF的翻译效率(图2)。这种机制在许多基因的翻译调控中起重要作用。在遇到应激响应(如缺氧胁迫和免疫激活)时, uORF可以通过抑制mORF的翻译来调节关键蛋白的表达(Juntawong et al., 2014; Xiang et al., 2023)。而Type2 uORF终止密码子位于mORF的开放阅读框内, 与mORF存在部分折叠。在Type2 uORF翻译结束后, 核糖体无法再起始mORF的翻译。因此, mORF的翻译只能通过渗漏扫描发生, 即核糖体跳过uORF直接对mORF进行翻译(Hinnebusch et al., 2016)。与Type1 uORF相比, Type2 uORF对mORF翻译的抑制通常更加显著。Type3 uORF较为少见, 由于其共享同一个终止密码子, 通常mORF会被正常翻译出来, 但是其N端多余部分可能会影响蛋白的定位或者功能(Niu et al., 2020)。

uORF在植物免疫中扮演重要角色, 可响应病原菌侵染, 调控mORF的翻译, 实现调控免疫与生长的平衡。例如, 拟南芥中的转录因子TBF1 (TL1-BINDING FACTOR)可以通过结合顺式作用元件TL1 (TRANSLOCON 1; GAAGAAGAA)调控基因表达。TL1富集在受SA诱导激活的内质网驻留基因启动子上(Wang et al., 2005), 同时也富集在受elf18和flg22调控的基因启动子上(Pajerowska-Mukhtar et al., 2012)。因此, TBF1在SA以及elf18和flg22诱导的基因表达调控中起重要作用。TBF1的5′UTR包含2段Type 1类uORFs, 其序列特征为富含苯丙氨酸(Phe)密码子。这种多聚苯丙氨酸富集特性使该mRNA对苯丙氨酸的胞内可用性高度敏感。在病原菌侵染过程中, 宿主细胞内未与苯丙氨酸结合的$tRNA^{Phe}$浓度显著上升, 触发eIF2α的磷酸化修饰(Pajerowska-Mukhtar et al., 2012)。磷酸化的eIF2α通过募集核糖体结合至主开放阅读框(mORF)的起始密码子, 直接驱动TBF1蛋白的翻译, 并且解除uORF对mORF的翻译抑制作用。这一$uORF_{TBF1}$调控模块通过精准调控翻译起始事件, 实现了免疫相关蛋白在特定病理条件下的特异性表达, 从而最大限度地减少免疫激活过程对宿主正常生长发育造成的非必需代谢负担(Huot et al., 2014)。$uORFs_{TBF1}$已经被证明具有良好的应用价值, 能够实现对抗病基因的病原侵染诱导型表达。Xu等(2017a)在水稻中转入受$uORFs_{TBF1}$调控的拟南芥*AtNPR1*基因, 成功构建了广谱抗病且生长及产量不受影响的转

基因水稻。

2.5 R-motif

在大多数真核生物中, mRNA的翻译主要通过经典的帽依赖性机制(cap-dependent translation)完成。该过程起始于解旋酶eIF4A (eukaryotic initiation factor 4A)与支架蛋白eIF4G和帽子结合蛋白eIF4E组成的帽结合复合体(cap-binding complex), 该复合体通过识别mRNA 5′端的(m7G)“帽子结构”将由eIF2-GTP-Met-tRNA三元复合物与40S核糖体亚基构成的43S前体起始复合物招募至mRNA的起始位点。同时, mRNA 3′端的多聚腺苷酸结合蛋白(Poly(A)-binding proteins, PABPs)与eIF4G相互作用, 形成mRNA环化结构, 促进翻译起始复合物的组装(图2)。然而, 当细胞处于应激状态时, eIF2α亚基的磷酸化会阻碍三元复合物的再生, 导致整体翻译起始受阻(Echevarría-Zomeño et al., 2013)。

在植物免疫过程中, 部分免疫相关基因mRNA的翻译不依赖上述经典机制。最新研究显示, 在PTI激活期间, 翻译效率提升的mRNA在5'UTR区域高度富集由嘌呤组成的核心序列元件。其特征性重复基序包括GA重复、G(A)3重复、G(A)6重复及混合G(A)n重复, 这一结构被称为R-motif (Xu et al., 2017b)。作为新型的内部核糖体进入位点(internal ribosome entry site, IRES), R-motif通过与PABPs结合, 替代经典通路中eIF4E的功能, 直接招募翻译起始因子启动非帽依赖翻译。值得注意的是, R-motif介导的翻译调控受到PTI信号通路的调节。在PTI过程中, 关键调控因子MPK3/6磷酸化PABPs成员PAB8, 增强其与R-motif的亲和力, 进而促进免疫相关基因的表达(Wang et al., 2022)。这种不依赖帽子和5'UTR的方式促进核糖体募集及随后的翻译机制与病毒中已知的IRES作用模式相似, 但呈现出植物特有的免疫应答调控特征(Roberts and Wieden, 2018; Razumova et al., 2025)。

3 病原菌对宿主mRNA的调控

植物通过精确调控mRNA的剪接、稳定性和翻译, 确保免疫相关基因的时空表达。然而病原菌可通过分泌效应子操纵宿主mRNA的加工过程, 以抑制免疫反应。效应子能以多种方式影响植物宿主的可变剪接, 从而利于病原体的入侵和致病过程。例如, 大豆疫霉病菌(*Phytophthora sojae*)的效应子PSR1 (*Phytophthora* suppressor of RNA silencing 1)能够特异性与宿主植物中一个关键剪接因子PINP1 (PSR1-interacting protein 1)相互作用, 抑制其正常的RNA结合和解旋酶活性, 从而干扰宿主基因的pre-mRNA剪接过程, 影响植物miRNA的生物合成以及与免疫相关的mRNA剪接, 进而削弱植物的防御能力(Qiao et al., 2015)。番茄疫霉病菌(*Phytophthora infestans*)可分泌多种效应子(如SRE3 (splicing regulatory effector 3)、SRE6和SRE7)与剪接体核心组分U1-70K结合, 干扰其行使功能, 从而影响植物整体的可变剪接, 达到促进病原体侵染的目的(Huang et al., 2020)。效应子还可直接靶向mRNA, 影响其稳定性。例如, 亚麻锈菌(*Melampsora lini*)的效应子AvrM14是一种Nudix水解酶, 能在体外催化mRNA的脱帽反应, 进而降解mRNA。在植物体内, AvrM14同样发挥水解酶作用, 通过降解免疫相关基因的mRNA, 抑制植物的防御反应(Anderson et al., 2016; McCombe et al., 2023)。

此外, 病原菌还可分泌miRNA, 通过直接降解宿主抗病相关基因的mRNA或抑制其翻译, 削弱免疫反应。灰霉菌可以将其sRNA分泌至拟南芥细胞中靶向其中的AGO1, 进而劫持宿主的RNAi机制, 触发宿主免疫基因沉默。例如, 沉默植物免疫过程中MAPK信号通路的基因(*MPK1*和*MPK2*) (Weiberg et al., 2013)。小麦条锈菌(*Puccinia striiformis* f. sp. *tritici*, *Pst*)可以分泌miRNA *Pst*-milR1, 靶向小麦抗病基因*PR2* (*PATHOGENESIS-RELATED 2*), 抑制宿主的免疫反应(Wang et al., 2017)。

4 研究展望

调控植物免疫与生长的平衡是植物适应环境的关键策略。植物免疫系统的持续激活通常会抑制植物的正常生长。例如, 在水稻中过表达*NPR1*会增强水稻对细菌和真菌的抗性, 但同时也会导致植株矮小及对盐和干旱胁迫更加敏感(Quilis et al., 2008)。植物病原菌诱导型启动子可在植物被病原菌侵染时特异性诱导启动基因表达, 故可运用于植物的抗病育种中。然而由于启动子本身的复杂性, 病原菌诱导型启动子在

植物中通常存在自激活现象。例如，由病原体诱导型启动子*hsr203J*驱动病原效应子*popA*表达，popA会迅速在侵染部位诱导HR，增强对病原菌的抗性。但某些烟草品种由于*hsr203J*诱导的基因自激活而表现出细胞死亡失控，或者诱导的popA蛋白量不足以诱发HR反应(Belbahri et al., 2001)。如果对mRNA进行改造(例如，在mRNA的5'UTR非编码区引入uORF)，则可避免自激活所诱发的问题。现已发现响应不同信号的uORF，如可响应免疫信号的$uORF_{TBF1}$、可响应蔗糖信号的$uORF_{bZIP11}$和可感应到环境硼酸变化进而调控核糖体阻滞的拟南芥$uORF_{NIP5}$ (Pajerowska-Mukhtar et al., 2012; Yamashita et al., 2017; Tanaka et al., 2024)。利用uORF对作物进行抗病性改造效果显著，潜力巨大。例如，在水稻中使用$uORF_{TBF1}$调控*NPR1*表达后，既能增强水稻对细菌和真菌的抗性，同时也不影响其生长(Xu et al., 2017a; Mou et al., 2025)。

此外，寄主介导的基因沉默(HIGS)作为一种新的抗病机制，在转基因作物中引入沉默病原菌的致病基因，已广泛应用于多种作物的抗病研究中。小麦中表达小麦锈病菌关键致病基因*PtCYC1* (cyclophilin)的dsRNA可以显著抑制小麦锈病菌的生长(Panwar et al., 2013)。在苹果中表达mi159a可以增强对葡萄座腔菌(*Botryosphaeria dothidea*)的抗性(Yu et al., 2024)。在甘蔗中沉默甘蔗镰孢菌(*Fusarium sacchari*)合成细胞膜组分的*FsCYP51*基因，可显著增强对甘蔗镰孢菌的抗性(Yin et al., 2025)。然而，如何选择合适的靶基因和HIGS片段，以最大限度地抑制病原菌侵染，及如何确保siRNA在2个生物体间的有效运输，仍是HIGS技术应用于提高作物抗病性面临的挑战。

单细胞RNA测序(single-cell RNA sequencing)和翻译组测序(ribo-seq，亦称ribosome footprinting核糖体印记)作为近年新兴的技术，已应用于植物免疫的转录后调控研究。单细胞测序技术通过高通量分析单个细胞的转录组，揭示了植物免疫反应中的细胞类型特异性差异。例如，研究人员分析了拟南芥叶片在遭受半生物营养型真菌希金斯炭疽菌(*Colletotrichum higginsianum*)感染时的单细胞转录组变化，生成了1个包含95 040个单细胞转录组的拟南芥叶片细胞图谱，发现保卫细胞和维管细胞分别通过不同的基因通路响应病原体。在直接与病原体接触的保卫细胞中，ABA信号通路被激活，促进气孔关闭。维管细胞中则富集表达NLR基因(Tang et al., 2023a)。这些发现揭示了植物在真菌感染下的细胞类型特异性响应，为深入研究植物与病原体互作提供了宝贵的资源。除模式植物拟南芥，单细胞测序也应用于研究其它植物物种与病原菌的互作。例如，Bai等(2022)利用单细胞RNA测序技术分析了野草莓(*Fragaria vesca*)叶片在灰霉菌侵染过程中的基因表达图谱，发现在灰霉菌侵染下，表皮细胞和叶肉细胞从正常功能向防御反应转变的信号，及与病害抗性相关的基因在不同细胞类型中的表达模式不同。单细胞测序可为研究不同的宿主-病原体互作整体动态提供新见解，揭示植物免疫过程中不同细胞类型的异质性，并可指导抗性品种的基因鉴定和培育策略制定。

翻译组测序通过捕获核糖体结合的RNA片段，可直接反映翻译效率，填补了转录与蛋白表达间的空白。利用该技术并结合结构组学技术，研究人员首次揭示了植物免疫过程中mRNA的结构变化动态调节翻译起始密码子的选择，由此促进抗病相关mRNA的翻译效率提高，进而增强植物抗病性的具体机制。受免疫信号激活的mRNA5′端前导序列富集uAUG。在正常生长条件下，这些uAUG的下游具有稳定的RNA发夹结构uAUG-ds (double-stranded RNA structures downstream of uAUGs)，该结构可以有效减缓翻译起始复合物行进，从而促使其更好地识别uAUG并翻译uORF，进而抑制下游正常的开放阅读框翻译出免疫蛋白。当病菌侵染时，RNA解旋酶的表达量迅速提高，该酶结合在翻译起始复合物中，有效解开uAUG-ds，导致uAUG不再被识别，从而促进下游免疫蛋白的翻译(Xiang et al., 2023)。该研究开发的翻译起始位点预测模型也会在未来助力翻译起始位点的预测和uAUG-ds的设计，达到精准调控蛋白质翻译的目的。植物在生长发育过程中需要严格控制免疫基因的表达，从而使植物的生长和抗性维持平衡。植物避免免疫基因的过度翻译是其精细调控防御反应的有效策略。在植物ETI中，HEM1通过植物特有的低复杂度结构域(low-complexity domain, LCD)，与众多翻译因子互作形成凝聚体(condensate)。利用改良的翻译组测序技术，Zhou等(2023)发现HEM1凝聚体的形成有效抑制了免疫基因的过度翻译，以防止植物免疫过度激活引起组织损伤。

近期的研究通过构建单细胞多组学图谱，发现了

植物免疫反应中的一个罕见细胞状态——PRIMER (primary immune responder)细胞。其在植物免疫中起着关键作用, 可能是免疫反应的早期启动者, 它通过与周围细胞的通信传播免疫信号(Nobori et al., 2025)。综上, 随着单细胞测序、翻译组测序和空间转录组等新技术的兴起, 植物免疫研究正从组织水平迈向细胞与分子层面的精细解析, 深刻改变着领域的研究范式和应用前景, 并为开发新型植物病害防控策略提供了思路。

作者贡献声明

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Post-transcriptional Regulation in Plant Immunity

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Abstract Pathogen infection is a serious threat to plant growth and development, causing severe crop yield reduction. The plant immune system, which is mainly composed of PTI (pattern-triggered immunity) and ETI (effector-triggered immunity), plays an essential role in resistance against pathogen infection. A large amount of research focused on resolving the key immune receptors/co-receptors, the components and regulation mechanisms of the PTI and ETI signaling pathways, and the biosynthesis and signaling pathways of the plant immune hormones salicylic acid and jasmonic acid. The major events during plant immune responses include pathogen recognition, the outburst of reactive oxygen species, Ca^{2+} influx, MAPK cascade signaling, and the induced expression of downstream defense genes. Recent studies have revealed that the expression of plant immune-related genes is not only regulated at the transcriptional level. The stability, translation efficiency, and translation products of their mRNAs are affected by a variety of post-transcriptional regulatory mechanisms, including alternative splicing, m^{6}A modification, small RNAs, uORFs, and R-motifs. Here, we summarized the present understanding of the plant immune system and mainly introduced the latest studies of the post-transcriptional regulation of plant immunity. This review also covered some findings that showed how pathogen interferes with the host post-transcriptional regulatory machinery. Some post-transcriptional regulatory elements have been successfully applied in crops. This application provides new molecular tools for improving diseases resistance and contribution to food security, as well as useful components for molecular breeding.

Key words plant immunity, alternative splicing, m^{6}A modification, small RNA, uORF

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