植物学报 *Chinese Bulletin of Botany* 2025, **60** (5): 722–733, www.chinbullbotany.com
doi: 10.11983/CBB25088 cstr: 32102.14.CBB25088

·特邀综述·

植物系统获得性抗性的转录调控

粟思琳[1, 2], 唐先宇[1, 2], 陈祎[1, 2], 王婷[1, 2], 夏石头[1, 2*]

[1]湖南农业大学, 植物激素与生长发育湖南省重点实验室, 长沙 410128; [2]湖南农业大学生物科学技术学院, 长沙 410128

摘要 系统获得性抗性(SAR)是植物重要的防御机制, 可显著增强植株对病原微生物的抗性, 且具有系统性、持久性和广谱性特征, 其转录调控为抗性激活的核心环节。该文从水杨酸(SA)和Pip/NHP的合成, NPR1、NPR3/NPR4受体和Pip/NHP移动信号等的转录调控, 以及TGA和WRKY转录因子家族调控等方面综述了SAR的转录调控研究进展, 为深入理解植物免疫调控网络以及系统探究植物在复杂环境中平衡生长与防御的机制提供参考。

关键词 系统获得性抗性(SAR), 转录调控, 水杨酸, 哌啶酸, N-羟基哌啶酸

粟思琳, 唐先宇, 陈祎, 王婷, 夏石头 (2025). 植物系统获得性抗性的转录调控. 植物学报 **60**, 722–733.

系统获得性抗性(systemic acquired resistance, SAR)是植物在局部感染病原体后, 通过信号传递激活全株范围广谱抗性的一种重要防御机制(Jones and Dangl, 2006)。SAR的激活依赖于局部感染组织中信号分子的合成与传递, 这些信号分子通过韧皮部运输至未受感染的系统叶片, 触发全株范围的防御基因表达(Ryals et al., 1996)。转录调控是SAR激活的核心环节, 涉及多个信号通路和转录因子家族的协同作用(Khan et al., 2022)。例如, 水杨酸(salicylic acid, SA)通过与受体NPR1 (Non-Expressor of PR Genes 1)及其同源蛋白NPR3/NPR4相互作用, 调控下游防御基因的表达。近年来, 研究发现哌啶酸(pipecolic acid, Pip)和N-羟基哌啶酸(N-hydroxy-piperidine-2-carboxylic acid, NHP)在SAR信号传递中发挥关键作用, 通过调控相关基因的表达实现对植物免疫反应的精细调节(Mohnike et al., 2023; Kumari et al., 2025)。Pip/NHP还通过与SA协同作用, 进一步放大SAR信号传递(Zavaliev and Dong, 2024)。此外, TGA和WRKY等转录因子家族通过直接结合防御基因启动子区域的顺式作用元件调控SAR相关基因的表达, 从而在植物免疫反应中发挥重要作用(Han et al., 2022)。本文综述了SAR转录调控的最新研究进展, 重点探讨相关转录因子对SA和Pip/NHP合成与信号转导的调控及其在SAR激活中的作用, 旨在为进一步解析植物免疫调控网络提供参考。

1 SA与Pip/NHP的合成调控

1.1 SA的合成调控

1.1.1 SA的生物合成途径

在植物中, 水杨酸(SA)的合成主要通过2条途径实现: 莽草酸途径(shikimate pathway)和异分支酸途径(isochorismate pathway)。在拟南芥(*Arabidopsis thaliana*)中, 异分支酸途径是合成SA的主要途径, 合成的SA占总量的90% (Peng et al., 2021)。在该途径中, 异分支酸合酶(isochorismate synthase, ICS)首先催化分支酸(chorismic acid, CA)转化为异分支酸(isochorismate, IC), 随后IC通过一系列酶促反应进一步转化为SA (Boatwright and Pajerowska-Mukhtar, 2013; Su et al., 2024)。其中, 异分支酸合酶1 (ICS1)催化反应为SA合成的第一步, 也是SA合成途径的限速步骤(Lefevere et al., 2020)。拟南芥*ics1*突变体在受到病原体侵染时, SA合成受到显著抑制, 导致植株对病原体的抗性降低(Kim et al., 2022)。除*ICS1*外, 拟南芥基因组还存在另一个*ICS*基因*ICS2*, 也参与SA的合成, 但其贡献相对较小, 仅在*ICS1*缺

收稿日期: 2025-05-19; 接受日期: 2025-07-08
基金项目: 国家自然科学基金(No.31971836)和湖南省研究生科研创新项目(No.CX20230686)
* 通讯作者。E-mail: xstone0505@hunau.edu.cn

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失时才能被明显检测到(Wu et al., 2023)。

而在莽草酸途径中，苯丙氨酸(phenylalanine, Phe)先经苯丙氨酸解氨酶(phenylalanine ammonia-lyase, PAL)转化为反式肉桂酸(*trans*-cinnamic acid)，随后反式肉桂酸可进一步转化为香豆酸(coumaric acid)或苯甲醛(benzaldehyde)；香豆酸可直接转化为水杨酸，而苯甲醛则需要先转化为苯甲酸(benzoic acid, BA)，并可在苯甲酸羟化酶(benzoic acid 2-hydroxylase, BA2H)的作用下最终生成SA。其中，苯丙氨酸解氨酶(PAL)是植物次生代谢的关键限速酶，在植株受到病原体侵染时，PAL途径能够迅速响应，合成SA及其衍生物，从而激活植物的防御机制(Mishra et al., 2024)。尽管PAL途径对拟南芥SA合成的贡献较小，但在某些植物如水稻(*Oryza sativa*)和大豆(*Glycine max*)中，PAL途径对SA合成的贡献与ICS途径相当甚至更大，是植物免疫反应的重要组成部分。而水稻PAL活性显著高于小麦(*Triticum aestivum*)，且在同一株植物中，不同组织部位PAL活性也不同，一般来说越幼嫩的部位PAL活性越高(Shine et al., 2016)。因此，莽草酸途径和异分支酸途径共同调控植物体内SA的合成(Ding and Ding, 2020)。

1.1.2 SA合成的转录调控

Wang等(2009)在研究拟南芥对细菌病原体的免疫反应时，发现CBP60g和SARD1的功能与SA合成和植物免疫反应密切相关。在*sard1*/*cbp60g*双突变体中，ICS1和SA的积累几乎被完全阻断，但二者在调控*ICS1*时发挥不同作用(Wang et al., 2009)。CBP60g为MAMP (microbe-associated molecular pattern)诱导SA积累的关键调控因子，其功能缺失导致植物对细菌病原体的抗性降低(Huang et al., 2021)。CBP60g是一个钙调蛋白结合蛋白，钙依赖性蛋白KINASE5 (CPK5)可直接磷酸化CBP60g，增强其转录因子活性，且TCH3 (TOUCH3)及其同源物CAM1/4/6和CPK4/5/6/11为PAMP (pathogen-associated molecular pattern)诱导CBP60g磷酸化所必需(Sun et al., 2022)。具体而言，CBP60g能响应病原体侵染后产生的钙信号，结合钙调蛋白CAM1/4/6，进而激活下游免疫相关基因的表达，包括*ICS1*的转录。这种钙信号依赖的调控机制使得植物能够迅速启动免疫反应，合成SA以抵御病原体的侵害(Sun et al., 2018b)。

SARD1与CBP60g在序列上高度相似，但不结合钙调蛋白，而是通过直接结合*ICS1*启动子上的GAAATTT基序来调控其表达，从而促进SA的积累(Li et al., 2021; Patil and Nandi, 2022)。SARD1的这种直接转录调控机制，使得其在植物免疫反应，尤其是在SA信号通路下游发挥重要作用(Choudhary and Senthil-Kumar, 2022)。Wang等(2011)也发现CBP60g和SARD1在调控*ICS1*表达中具有不同的时间动态：CBP60g在感染早期通过钙信号调控SA的快速积累，SARD1则在感染后期通过直接结合启动子区维持SA的持续合成，这种分工协作的转录调控机制为植物提供了多层次免疫防御能力。Sun等(2020)则在研究另一类功能冗余的钙调蛋白结合转录因子CAMTA1 (CALMODULIN-BINDING TRANSCRIPTION FACTOR 1)、CAMTA2和CAMTA3时，发现CAMTA1/2/3负调控SA和NHP的合成。在*camta1*/*2*/*3*三突变体中，SA和NHP在未受病原体感染的情况下显著积累，且CAMTA3可通过直接结合*CBP60g*的启动子并间接影响*SARD1*表达来负调控SA的生物合成(Sun et al., 2020)。EIN3、EIL1、EDS1和PAD4也参与调控*ICS1*，这些转录因子通过直接或间接结合到*ICS1*启动子上的特定序列调控*ICS1*的表达，从而影响SA的合成和植物免疫反应。例如, EIN3和EIL1为参与植物乙烯信号转导途径的转录因子，可抑制*ICS1*的表达，从而减少SA的合成(Fu and Dong, 2013; Tian et al., 2025)。

此外，植物内源的乙烯和茉莉酸也参与SA的合成调控。乙烯信号转导途径的激活能促进植物合成SA (Dubois et al., 2018)。乙烯处理可激活植物体内乙烯信号途径，从而影响一系列防御相关基因的表达，如直接上调*PR-3*和*PR-4*等基因表达或间接激活*CHS*和*4CL*等基因，促进抗菌物质的积累等(Wang et al., 2025b)。乙烯响应因子(ERFs)能够结合到特定DNA序列上，调控相关基因转录。例如，番茄(*Solanum lycopersicum*) ERF11可激活*BT4*基因表达进而导致SA积累，MdERF11能通过促进苹果(*Malus pumila*)中SA合成从而正向调控其对真菌的防御响应(Ma et al., 2024)。而Kazan和Manners (2008)发现，茉莉酸信号转导途径的关键组分(如冠菌素不敏感蛋白1 (coronatine insensitive 1, COI1)和茉莉酸ZIM结构域蛋白(jasmonate ZIM-domain proteins, JAZs))影响SA的合成。其中，*COI1*编码一个F-box蛋白，为

SCF E3泛素连接酶复合体的一部分，参与茉莉酸信号转导；JAZ蛋白作为茉莉酸信号转导的负调控因子，通过与SCFCOI1 (SKP1-CULLIN-F-BOX PROTEIN COMPLEX COI1)复合体互作而被泛素化降解，从而释放JIN1/MYC2等转录因子，激活茉莉酸响应基因的表达(Kazan and Manners, 2008)。当COI1与JAZs互作释放MYC转录因子后，MYC2可直接结合*NPR1*启动子抑制其表达，从而削弱SA通路，影响植物的免疫反应(Nomoto et al., 2021)。这种复杂的转录调控网络使得植物能够在不同的环境条件下精细调节自身的免疫反应，以应对各种生物和非生物胁迫。

1.2 Pip/NHP的合成调控

1.2.1 Pip的生物合成及其调控

Pip (哌啶酸)是一种杂环非蛋白氨基酸，也是植物体内的一种重要信号分子(Cai et al., 2021)。Pip的生物合成起始于赖氨酸，首先由ALD1 (AGD2-Like Defense Response Protein 1)催化赖氨酸的α-氨基转移到氧乙酸上，生成中间产物ε-氨基-α-酮己酸(KAC)和丙氨酸；随后，KAC通过自发分子内环化和脱水反应，形成环脱氢丙烯酸(DP)和酮亚胺1,2-DP；最终异构化为更稳定的烯胺互变异构体2,3-DP，进而生成Pip (Hartmann et al., 2017)。除ALD1参与Pip合成外，Ding等(2016)鉴定到Pip合成途径中的关键基因之一*SARD4* (*SAR DEFICIENT 4*)，其编码蛋白SARD4作为还原酶将P2C (D1-piperideine-2-carboxylic acid, Pip的前体)还原为Pip。在*sard4*突变体中，Pip水平降低，而P2C在远端叶片中积累，表明SARD4是Pip合成最后一步的关键酶，主要介导各组织中Pip的合成与积累(Ding et al., 2016; 吴楠等, 2022)。Sun等(2015)进一步研究发现，用丁香假单胞菌*Psm.* ES4326处理拟南芥野生型植株后，其*ALD1*和*SARD4*的表达水平显著增高；而在*sard1-1*/*cbp60g-1*双突变体中，*ALD1*和*SARD4*的表达水平显著降低，表明SARD1和CBP60g对*ALD1*和*SARD4*的表达具有正向调节作用。ChIP-PCR结果进一步证实，SARD1-HA和CBP60g-HA能够结合到*ALD1*和*SARD4*的启动子区域。这一发现揭示了SARD1和CBP60g通过调控*ALD1*和*SARD4*的表达影响Pip的合成(Sun et al., 2015; Li et al., 2024; Wang et al., 2025c)。研究表明，在*ics1*、*npr1*和*pad4*突变体所有叶片中，Pip积累水平均有所下降，表明SA、NPR1和PAD4 (Phytoalexin-Deficient 4)能促进系统组织中Pip的生物合成(Návarová et al., 2012; Sun et al., 2015)。与此相反，CAMTA1/2/3不仅抑制植物中SA的生物合成，还抑制Pip的生物合成。当CAMTA1/2/3功能丧失时，*ALD1*被诱导表达，导致植物中Pip水平升高(Kim et al., 2020)。不仅如此，Pip还存在自我调控机制，即Pip可激活SA和Pip生物合成中正调控基因的表达，包括*CBP60g*、*SARD1*、*PAD4*和*EDS1*。这种自我调控机制可能有助于植物在面对病原体侵染时迅速启动免疫反应，合成更多的Pip和SA，从而增强植物的抗病能力(Wang et al., 2018; Zeier, 2021)。

1.2.2 NHP的生物合成及其调控

NHP (N-羟基哌啶酸)为Pip羟基化的产物，是一种植物代谢产物和系统获得性抗性调节剂(Cao et al., 2024)。NHP的合成起始于L-赖氨酸，首先由ALD1 (AGD2-LIKE DEFENSE RESPONSE PROTEIN 1)将赖氨酸转化为ε-氨基-α-酮己酸；然后该化合物自发环化为Δ1-哌啶-2-羧酸(P2C)，并通过SARD4还原为Pip；最后，FMO1 (FLAVIN-DEPENDENT MONOOXYGENASE 1)将Pip羟基化，形成NHP (Mohnike et al., 2023)。Hartmann等(2018)在发现NHP的同时，还报道了外源Pip能诱导拟南芥FMO1依赖性转录反应，与SAR反应高度重叠，表明NHP能激活多个与SAR相关基因的表达。此外，NHP也能进行自我调控，通过NPR1激活SAR，并加强自身的生物合成，这种自我放大的反馈机制确保了NHP在植物体内的快速积累(Hartmann and Zeier, 2018; Huang et al., 2020)。而CAMTA3可通过直接结合*CBP60g*的启动子，并间接影响*SARD1*的表达来负调控NHP的生物合成(Sun et al., 2020)。

值得一提的是，NAC转录因子在NHP的生物合成和转录调控中也扮演重要角色。NAC家族是一类具有高度保守N端NAC结构域(负责二聚化、核定位和DNA结合)和可变C端转录调控域的转录因子，可形成同源或异源二聚体，并通过其DNA结合域识别特定的DNA序列(Fuertes-Aguilar and Matilla, 2024; Chen and Xia, 2025)。其中，NAC90、NAC36和NAC61形成的NAC三联体可直接抑制*ALD1*、*FMO1*和*ICS1*等NHP和SA生物合成关键基因的表达，从而负向调控

NHP和SA的合成。*NAC90*作为SARD1的靶基因，受病原体、水杨酸(SA)和NHP诱导表达后形成NAC90-NAC36-NAC61异源三聚体，增强了对NHP和SA生物合成的抑制作用。在*nac90*突变体中，NHP和SA水平升高，植物出现组成型免疫激活表型；而在*NAC90*过表达株系中，NHP和SA水平降低，抗病性受损。由此可见，*NAC90*及其同源基因*NAC36*和*NAC61*通过转录水平的精细调控来平衡植物免疫与生长之间的关系，防止过度或持续激活SAR，从而确保植物在抵御病原体时既能激活足够的防御反应，又不过度消耗资源影响生长发育(Cai et al., 2024)。

2 SA与NHP信号调控

2.1 SA信号调控

2.1.1 SA受体NPR1的正调控作用

Cao等(1994)通过拟南芥诱变和筛选，获得了1个*PR*基因无法正常表达的突变体，其对多种病原菌敏感，该基因被克隆并命名为*npr1* (*nonexpressor of pathogenesis-related genes 1*)。NPR1是SA信号通路中的核心调控因子，通过调控*PR*基因的表达激活植物SAR (Zavaliev and Dong, 2024)。冷冻电镜分析显示，NPR1在未激活状态下以同源二聚体形式存在，整体形状类似一只展翅的鸟，由1个中央BTB (Broad-Complex, Tramtrack and Bric-à-brac)结构域、1个BHB (BTB and carboxyterminal Kelch helix bundle)结构域、4个ankyrin重复序列(ANK)和1个无序的水杨酸结合结构域(salicylic acid-binding domain, SBD)组成(Kumar et al., 2022)。NPR1的结构与其功能高度适应，当植物受到病原体感染时，SA在植物体内积累，通过与NPR1结合触发NPR1的构象变化。由于NPR家族蛋白不含DNA结合域，NPR1必须与转录因子TGAGC序列特异性结合，因此NPR1作为转录激活因子参与调控基因的表达(Dong, 2004; Zhou et al., 2023)。为建立有效的SAR以应对病原菌感染，NPR1需在细胞质和细胞核中被精细调控。正常情况下，NPR1以寡聚体的形式存在于细胞质中，SA的结合可还原NPR1的二硫键，导致其从寡聚体转变为单体，单体化的NPR1能够通过核定位信号(NLS)进入细胞核(Jiang et al., 2025)。在细胞核中，NPR1与TGA转录因子(TGA2、TGA5和TGA6等bZIP类蛋白)相互作用，形成NPR1-TGA转录复合体，这增强了TGA转录因子结合DNA的能力(Lu et al., 2024)。

同时，*PR*基因的启动子上含有特定的as-1顺式作用元件，NPR1-TGA复合体通过结合到顺式作用元件上激活*PR*基因的表达(Nair et al., 2021)。Zhang等(2003)发现，*PR1*启动子区域的正向调控元件需依赖该复合体的协同结合才能被激活，进而启动*PR*基因的转录。这种相互作用不仅涉及结构域的直接互作(如ANK结构域与TGA的bZIP结构域结合)，还伴随染色质构象改变，促进RNA聚合酶II及转录辅助因子的招募，最终实现防御相关基因的高效表达(Zhang et al., 2003)。此外，NPR1的活性还受到细胞内氧化还原状态调控。在感染病原体后，细胞内发生氧化还原变化，硫氧还蛋白(thioredoxin, TRX)等抗氧化剂活性增强，使得NPR1的二硫键被还原，还原后的NPR1从寡聚体转变为单体，并通过核定位信号进入细胞核，启动*PR*基因的表达，从而激活SAR。这种氧化还原调控机制确保NPR1在适当的时间和地点被激活，以应对病原体的威胁(Tada et al., 2008; Yang et al., 2025)。通过上述机制，NPR1作为转录共激活因子，以SA结合为起点，通过与TGA转录因子的协同作用，精准调控*PR*基因启动子上的顺式作用元件，最终激活SAR相关基因的表达。这一过程不仅体现了植物通过单一核心蛋白整合信号输入与转录输出的高效性，也揭示了氧化还原状态、蛋白质构象变化与转录因子网络在植物防御反应中的深度协同(Yildiz et al., 2021; Huang et al., 2023)。此外，NPR1的翻译后修饰对其功能和稳定性至关重要。NPR1的磷酸化和SUMO化等修饰不仅影响其与TGA3等转录因子的相互作用，还调控其在细胞内的定位和稳定性，从而精细调控植物免疫反应的强度和时机。NPR1的去磷酸化可增强其与TGA3等转录因子的互作，如去磷酸化的NPR1能够更有效结合TGA3，形成增强子复合体，从而促进*PR*基因的转录激活。这种增强效应有助于在病原体攻击时迅速启动*PR*基因表达，增强植物的免疫反应。SUMO化修饰也能增强NPR1与TGA3等转录因子的互作，并通过调节NPR1的稳定性和定位，维持免疫反应的适当强度和持续时间(Pieterse and Van Loon, 2004; Zavaliev and Dong, 2024)。

2.1.2 SA受体NPR3/NPR4的负调控

除NPR1外，已确定NPR3和NPR4也是SA高亲和受体，在植物免疫特别是SAR中起到负调控作用(Fu et al., 2012)。NPR3和NPR4的C末端含有1个保守的EAR (ethylene-responsive element binding factor-associated amphiphilic repression motif)基序，该基序对于其转录抑制活性至关重要，能够招募组蛋白修饰复合体或直接干扰转录起始复合物组装，从而抑制靶基因表达(Liu et al., 2020)。在转录因子互作网络中，NPR3/NPR4通过ANK重复序列与TGA转录因子(如TGA2、TGA5和TGA6)相互作用，形成的蛋白复合体可靶向结合*SARD1*和*WRKY70*的启动子区，进而抑制这些基因的表达(Sun et al., 2015; Choudhary and Senthil-Kumar, 2022)。例如, TGA2与NPR4结合后，可介导复合体定位到*SARD1*启动子的TGACG顺式作用元件，通过EAR基序的抑制功能阻遏转录激活因子募集，从而抑制该基因的表达；而SARD1作为NHP生物合成通路的核心调控因子，其转录沉默会直接削弱SAR信号的系统性转导(Tian and Zhang, 2019)。

类似地，*WRKY70*启动子区的W-box元件与NPR3/NPR4-TGA复合物结合后，在非诱导状态下维持低表达水平，进而抑制下游防御基因的级联反应(Arndt et al., 2022)。Ding等(2018)通过进一步研究NPR3和NPR4的功能，发现*npr4-4D*为1个功能获得型突变体，能导致NPR4无法结合SA，从而持续抑制SA诱导的免疫反应。在*npr4-4D*突变体中，免疫基因(如*SARD1*和*WRKY70*)的表达被显著抑制，植物对病原体的抗性也显著减弱。与此相对, SA能够直接结合NPR3/NPR4，抑制它们的转录抑制活性，从而解除对*SARD1*和*WRKY70*等免疫基因的抑制，且SA处理并不会破坏NPR3/NPR4与TGA因子或启动子的结合，而是通过改变NPR3/NPR4的构象或功能状态来解除抑制作用(Ding et al., 2018; Liu et al., 2020)。

总之，NPR1和NPR3/NPR4在SA诱导的免疫反应中发挥相反的作用：NPR1作为转录共激活因子，而NPR3/NPR4作为转录共抑制因子，二者协同精细调控SAR (Wang et al., 2020; Ding and Ding, 2020)。

2.2 移动信号Pip/NHP对SAR的调控

Návarová等(2012)研究发现，Pip在SAR激活过程中发挥关键作用。Pip从局部受感染的叶片运输到未受感染的系统叶片，从而激活全株的防御反应，成为SAR中的一个关键移动信号(Návarová et al., 2012; Zeier, 2021)。随后，Hartmann等(2018)发现NHP是拟南芥中天然积累的一种防御性代谢产物，在接种病原菌后NHP大量积累于局部叶片，并通过韧皮部运输到系统叶片，从而激活SAR。

此后，针对Pip/NHP移动信号如何调控SAR的研究逐渐深入，主要集中在以下几个方面。(1) Pip/NHP与SA的协同作用。病原体侵染植物后，Pip和NHP在局部叶片中合成，并通过韧皮部运输到系统叶片。SA也在局部叶片中积累，并通过韧皮部运输到系统叶片。Pip和NHP的积累优先于SA，能够通过间接上调SA的水平来激活SAR反应(Ding et al., 2016; Yildiz et al., 2023)。(2) NHP通过NPR1激活SAR。Yildiz等(2023)研究发现，在*npr1*突变体中，NHP无法诱导SAR，表明NHP的作用依赖于NPR1。进一步的免疫荧光实验中可观察到NPR1在细胞核中定位，发现NHP处理能够增强NPR1的核定位。通过染色质免疫沉淀(ChIP)实验，发现NPR1能够结合到*PR1*等防御基因的启动子区域，表明NPR1在NHP诱导的转录重编程中起关键作用。外源NHP处理能够显著增强*PR1*等防御基因的表达，并且这种增强效应在*npr1*突变体中消失。表明NHP能够增强NPR1的活性，从而放大SA信号，促进防御相关基因的表达，达到调控植物SAR的目的(Yildiz et al., 2021)。(3) NHP糖基化调控SAR。Bauer等(2021)通过LC-MS、GC-MS和体外酶活性测定实验，证实UGT76B1能够糖基化NHP生成NHP-H2。抗病性和SAR检测结果表明, UGT76B1通过糖基化NHP来调节植物的免疫反应，*ugt76b1*突变体中NHP的积累导致抗性增强，而*UGT76B1*过表达株系中NHP的糖基化导致抗性降低, *fmo1*/*ugt76b1*双突变体的抗性与野生型相似，表明NHP积累是*ugt76b1*抗性增强的原因(Bauer et al., 2021)。同时，NHP的糖基化状态影响其在植物体内的移动性和稳定性，从而调节SAR的激活程度(Holmes et al., 2021)。当NHP被糖基化时，其作为信号分子的活性可能被抑制，从而抑制SAR的激活。这种机制允许植物在需要时激活SAR，同时在不需要时保持较低的免疫反应，以节省能量用于生长和发育(Mohnike et al., 2021)。

2.3 下游转录因子家族对SAR的调控作用

TGA转录因子是一类在植物中广泛存在的bZIP (碱性亮氨酸拉链)转录因子家族成员，因能特异性结合DNA序列中的TGACG基序而得名，在植物生长发育，尤其是在植物免疫反应中扮演重要角色(Tomaž et al., 2022; Yildiz et al., 2023)。通过染色质免疫沉淀-聚合酶链式反应(ChIP-PCR)分析，发现TGA1能够直接结合到*SARD1*基因的启动子区域，增强其转录活性，上调其表达。而对*CBP60g*而言，虽然TGA1不直接结合到*CBP60g*的启动子区域，但TGA1和TGA4的缺失会导致*CBP60g*的表达显著降低。因此, TGA1和TGA4可通过调控*SARD1*和*CBP60g*的表达促进*ICS1*表达，从而增加SA的合成，激活下游防御基因的表达(Sun et al., 2018a)。另外, TGA转录因子还通过调控*SARD1*和*CBP60g*的转录，促进*ALD1*和*SARD4*的表达，从而增加Pip的合成，放大植物的防御反应(Tomaž et al., 2022; Chen et al., 2024)。

WRKY家族是一类植物特有的转录因子，因含有WRKY结构域而得名，其通过多种机制调控植物的系统获得性抗性(Hsin et al., 2022)。WRKY结构域包含1个保守的WRKYGQK基序和1个锌指结构，能够特异性地结合到防御基因启动子区域的W-box顺式作用元件，进而调控这些基因的表达(Goyal et al., 2023)。例如，一些WRKY转录因子可作为转录激活因子，与WRKYGQK基序结合后激活*PR1*和*PR2*等基因的表达，从而增强植物的防御反应(Javed and Gao, 2023)。用丁香假单胞菌番茄致病变种DC 3000 (*Pseudomonas syringae* pv. *tomato* DC3000)处理拟南芥后，发现*WRKY6*、*WRKY29*和*WRKY53*在系统叶片中被显著诱导表达，且与SAR的建立密切相关。此外，WRKY转录因子还可与TGA2、TGA5和TGA6等TGA家族成员互作，增强对防御基因启动子的结合能力；同时，SA也可与WRKY转录因子相互作用，调控其转录活性，从而激活下游防御基因的表达(Ueno et al., 2013; Hussain et al., 2018; Foret et al., 2024)。Powers等(2024)发现，在*wrky54*/*70*双突变体中, SA诱导表达基因数目显著减少，表明WRKY54和WRKY70在SA介导的转录重编程中具有重要的正向调控作用，并通过进一步研究明确了WRKY54与WRKY70是NPR1-TGA复合物下游的主要转录因子。在SA信号级联反应中，NPR1通过与TGA转录因子结合启动*WRKY54*/*70*的表达。这些WRKY转录因子随后激活下游免疫输出基因的表达，形成一个转录调控级联反应。除作为SA信号通路中的正向调控因子外，WRKY54/70在SA合成的负调控中同样发挥作用，这种双重角色使得WRKY54/70在植物免疫反应精细调控中具有关键效应(Powers et al., 2024)。

3 总结与展望

SAR的转录调控是一个多信号通路协同和多转录因子网络互作的复杂过程(Zhou et al., 2021)。从病原体侵染植物局部叶片并激活信号感知机制开始，到Pip/NHP被转运到远端叶片，系统叶片中的NHP和SA水平上升并激活NPR1依赖的SAR反应的整个过程中，转录调控在SAR调控中发挥关键作用(图1)。其中，SARD1和CBP60g直接或间接结合*ICS1*、*ALD1*和*SARD4*启动子区域的顺式作用元件，正向调控SA和Pip/NHP的生物合成(Tian et al., 2025); TGA1/4通过调控*SARD1*和*CBP60g*的转录，间接促进下游信号分子积累(Sun et al., 2018a); 而NAC90-NAC36-NAC61三联体则通过直接抑制*ALD1*、*FMO1*和*ICS1*的启动子活性，负调控SA和NHP的合成(Wang et al., 2025a)。此外，NPR3/NPR4作为SA的负调控受体，与TGA2/5/6结合后抑制*SARD1*和*WRKY70*等基因的转录，与NPR1的正调控形成动态平衡(Choudhary and Senthil-Kumar, 2022)。而作为NPR1-TGA复合物下游的主要转录因子，WRKY54/70在SA合成的正向和负向调控中均发挥重要作用。UGT76B1介导的NHP糖基化则降低游离NHP水平，通过负反馈调控终止SAR信号的持续激活，体现了转录后修饰对转录调控网络的重要补充(Bauer et al., 2021)。植物激素在SAR中也不可忽略，乙烯可通过激活信号转导途径促进SA合成，上调防御基因表达并积累抗菌物质；茉莉酸信号组分(如COI1和JAZs)则通过调控MYC转录因子抑制SA通路。这种激素间相互作用形成的转录调控网络，使得植物能够在环境变化中更精细地调节免疫反应(Nomoto et al., 2021; Wang et al., 2025b)。

尽管有关SAR转录调控网络研究已取得显著进展，但仍有多个问题有待进一步阐明。已有大量研究

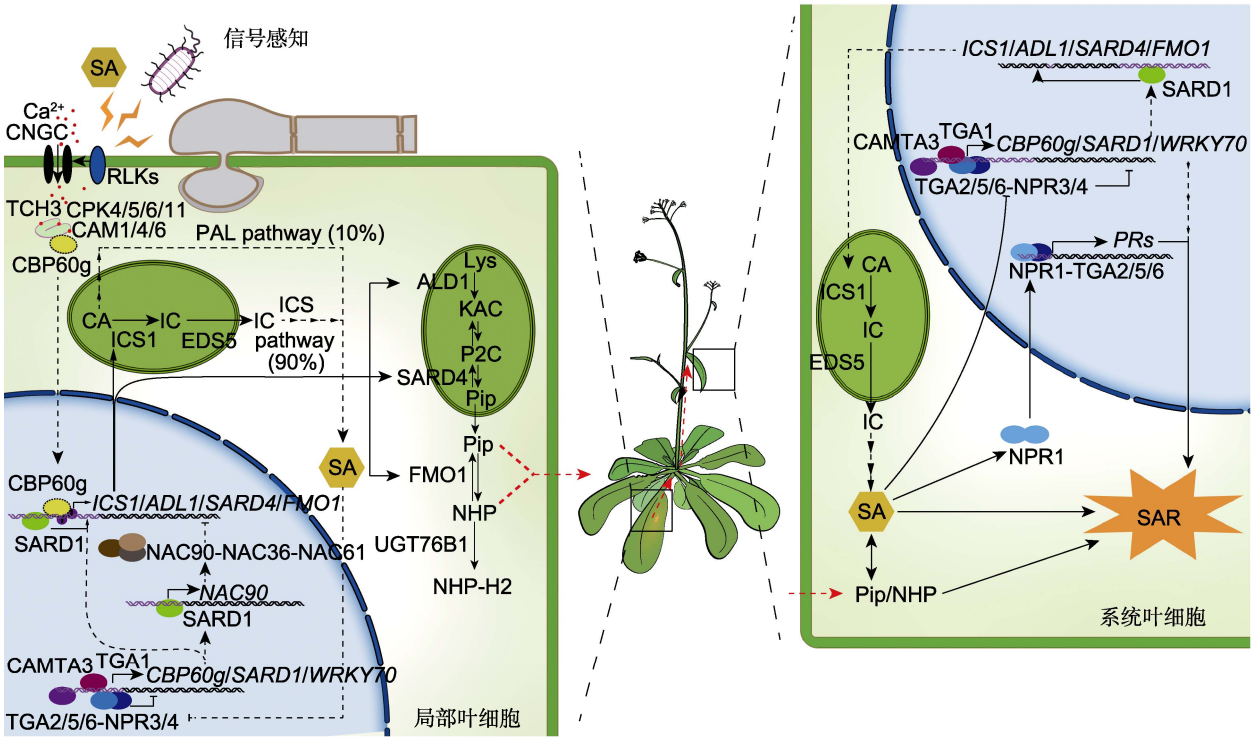


图1 植物系统获得性抗性(SAR)的转录调控

当病原体(细菌或真菌)侵染植物局部叶片细胞或施用水杨酸(SA)时，局部信号被感知后，受体样激酶(RLKs)激活导致钙离子内流，钙离子与钙调蛋白CAM1/4/6结合后与CBP60g蛋白互作，通过苯丙氨酸解氨酶(PAL)途径和异分支酸合酶1 (ICS1)途径促进SA的合成，引发局部抗性反应。而赖氨酸经ALD1和SARD4催化生成哌啶酸(Pip)，再由FMO1羟基化生成N-羟基哌啶酸(NHP)，而Pip/NHP可通过韧皮部转运至远端叶片。随着系统叶细胞中SA和Pip/NHP水平上升，SA与受体NPR1结合，触发NPR1构象变化并入核，进而与TGA2/5/6形成复合体，激活*PRs*基因表达和SAR反应。同时，SA也能直接结合NPR3/NPR4，抑制它们的转录抑制活性，从而解除对*SARD1*和*WRKY70*等的抑制，达到精细调控SAR的目的。实线箭头表示直接调控；虚线箭头表示间接调控；T型线表示抑制作用。

Figure 1 Transcriptional regulation of systemic acquired resistance (SAR) in plants

When local leaf cells of plants are infected by pathogens (bacteria or fungi) or salicylic acid (SA), is applied receptor like kinases (RLKs) are activated after local signal perception, leading to the influx of calcium ions. The calmodulin CAM1/4/6 then combines with calcium ions and interacts with CBP60g, triggeringa local resistance response through the phenylalanine ammonia-lyase (PAL) and isochorismate synthase 1 (ICS1) pathways to increase SA synthesis. Lysine, whereas, is catalyzed by ALD1 and SARD4 to form piperidine-2-carboxylic acid (Pip), which is then hydroxylated to N-hydroxy-piperidine-2-carboxylic acid (NHP) by FMO1. Pip/NHP can be transported to distal leaves through phloem transportation. After the levels of SA and Pip/NHP increase in systemic leaf cells, SA binds to the receptor NPR1, and triggers the conformational changes in NPR1, which enter into the nucleus, where NPR1 forms a complex with TGA2/5/6, and activates the expression of *PRs* genes and SAR. Moreover, SA can also directly bind NPR3/NPR4 to inhibit their transcriptional inhibitory activity, thereby relieving the inhibition of genes such as *SARD1* and *WRKY70*, achieving the goal of fine tuning the SAR. The solid arrows indicate direct regulation; the dashed arrows indicate indirect regulation; the T-shaped line indicates inhibition.

表明SARD1可正向调控ICS1、ALD1和SARD4等，但是如何对其进行差异性正向调控，从而控制NHP和SA的生物合成尚不明确。在病原体侵染时，虽然已知正调控转录因子启动SA和NHP的生物合成，但负调控转录因子维持SAR信号稳态的时间和机制仍不清楚。同时，考虑到植物在自然环境中面临的多重胁迫，又该如何将SA和Pip/NHP免疫信号与其它环境响应机制进行整合？对这些问题的深入研究不仅有助于了解SAR复杂的转录调控过程，还有利于全面理解植物免疫的复杂性及其应用前景。目前，SAR在作物抗病育种方面已有一些实质性进展。例如，关键基因*NPR1*可通过调控SAR信号转导增强小麦对白粉病等

多种病害的广谱抗性(Zhao et al., 2024)。随着基因编辑(如CRISPR靶向编辑转录因子)技术的不断发展，为SAR调控网络改造提供了新的思路和手段。一方面，可利用CRISPR-Cas9技术增强SAR正调控转录因子的诱导表达，进而促进SA和Pip/NHP的生物合成；另一方面，也可对负调控SAR的转录因子基因进行敲除或敲低，解除其对SAR信号的抑制作用，达到适时诱导SAR而又不影响植株正常生长发育。此外，还可利用基因编辑技术增强或降低转录因子与信号分子之间的互作，以进一步解析SAR转录调控网络的分子机制并应用于抗性育种。

作者贡献声明

粟思琳: 撰写并修改论文; 唐先宇: 作图; 陈祎和王婷: 参与文献的收集整理及文章修改; 夏石头: 选题构思、指导撰写并修改论文。

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Transcriptional Regulation of Systemic Acquired Resistance in Plants

Silin Su[1, 2], Xianyu Tang[1, 2], Yi Chen[1, 2], Ting Wang[1, 2], Shitou Xia[1, 2*]

[1]Hunan Provincial Key Laboratory of Phytohormones and Growth Development, Hunan Agricultural University, Changsha 410128, China; [2]College of Bioscience and Biotechnology, Hunan Agricultural University, Changsha 410128, China

Abstract Systemic acquired resistance (SAR) is a crucial defense mechanism in plants, which can significantly enhance the plant's resistance to pathogenic microorganisms. SAR has systemic, persistent, and broad-spectrum characteristics, whose transcriptional regulation plays a central role in the process. Here, the research progress on transcriptional regulation of SAR from the synthesis of salicylic acid (SA), Pip/NHP, transcriptional regulation of NPR1, NPR3/NPR4 receptors and Pip/NHP mobile signals, as well as TGA, WRKY transcription factor family regulation was reviewed, providing a reference for a deeper understanding of plant immune regulatory networks, and systematic exploration of the mechanisms by which plants balanced growth and defense in complex environments.

Key words systemic acquired resistance, transcriptional regulation, salicylic acid, pipecolic acid, *N*-hydroxypipecolic acid

Su SL, Tang XY, Chen Y, Wang T, Xia ST (2025). Transcriptional regulation of systemic acquired resistance in plants. *Chin Bull Bot* **60**, 722–733.

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* Author for correspondence. E-mail: xstone0505@hunau.edu.cn

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通讯作者简介

夏石头, 湖南农业大学二级教授, 博士生导师。长期从事植物免疫和植物与微生物互作研究, 重点关注油菜等作物与黑盘菌等真菌的互作机制。主持完成多项国家自然科学基金和湖南省自然科学杰出青年基金等项目。在*eLife*、*Plant Physiology*、*Journal of Fungi*和《植物学报》等重要核心期刊上发表高水平研究论文100余篇。曾获“湖南省科技进步一等奖”、“湖南省第二届优秀科技工作者”和“湖南省高等教育省级教学成果一等奖”等奖项。

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