# 植物系统获得抗性中的系统信号及其作用机制

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**摘要:** 系统获得抗性(systemic acquired resistance, SAR)是植物受到病菌侵染后诱导产生的一种防御机制, 表现为系统性广谱抗性。SAR中通常在侵染部位产生多种可移动的系统信号, 转运到植株其余部位, 并激活系统性防卫反应。本文综述了水杨酸和水杨酸甲酯、哌啶酸和*N*-羟基哌啶酸、壬二酸、甘油三磷酸、萜类物质(脱氢枞醛、松萜)等系统信号在SAR中的合成、功能、转运、作用机制及其相互关系。

**关键词:** 系统获得抗性; 系统信号; 防卫反应

在长期进化过程中, 植物形成了复杂且精确调控的免疫系统, 以应对环境中潜在病菌的侵染危害。植物的先天免疫系统由病原物模式分子诱发的免疫反应(PTI)和病菌效应子引发的免疫反应(ETI)组成。同时, 植物还进化形成一套诱导免疫机制, 包括系统获得抗性(systemic acquired resistance, SAR)。植物诱导免疫通常具有广谱抗性特征, 被认为是控制植物病害的一种新途径。SAR由病菌局部侵染激活, 在受侵部位产生系统信号, 转运到未受侵部位, 激活防卫反应, 从而产生对多种病菌的抗病反应。系统信号是SAR的关键因子, 目前已知的SAR系统信号包括水杨酸(salicylic acid, SA)及其衍生物水杨酸甲酯(methyl salicylate, MeSA)、哌啶酸(pipecolic acid, Pip)及*N*-羟基哌啶酸(*N*-hydroxypipecolic acid, NHP)、壬二酸(azelaic, AzA)、甘油三磷酸(glycerol-3-phosphate, G3P)、萜类物质[脱氢枞醛(dehydroabietinal, DA)和松萜]。本文综述了SAR系统信号的合成、功能、转运、作用机制及其相互关系。

## 1 植物SAR中的系统信号

### 1.1 SA与MeSA

SAR发生时植物体内SA浓度升高(Metraux等1990; Malamy等1990)。*NahG*烟草(*Nicotiana tabacum*)和拟南芥(*Arabidopsis thaliana*)植株不能积累SA和诱导SAR (Gaffney等1993; Delaney等1994), 证明SA是SAR的关键信号。

#### 1.1.1 SA的合成与调控

植物中存在两条不同SA合成途径, 即苯丙氨酸解氨酶(PAL)和异分支酸合成酶(ICS)途径, 但均从质体中的分支酸开始(图1-A)。在PAL途径中, 苯丙氨酸由PAL转化为反式肉桂酸, 再由羟酰基-CoA羟化酶AIM1介导的氧化作用形成苯甲酸(Bussell等2014), 最终经一个未知的苯甲酸羟化酶形成SA。ICS途径中, 质体中分支酸经异分支酸合成酶ICS1/SID2转化为异分支酸(Wildermuth等2001), 由MATE转运蛋白EDS5转运至胞质中(Serrano等2013), 再由氨基转移酶PBS3催化形成谷氨酸-9-异分支酸共轭物(Rekhter等2019a), 最后谷氨酸-9-异分支酸共轭物裂解或在BAHD酰基转移酶EPS1作用下形成SA (Rekhter等2019a; Torrens-Spence等2019)。在拟南芥中, 全部*PAL*基因突变后SA基础水平下降约75%、病菌诱导的SA积累水平下降约50% (Huang等2010), 而*ics1*中病菌诱导SA的积累水平仅为野生型的5%~10% (Wildermuth等2001)。在拟南芥中, 病菌诱导的SA中约10%来自于PAL途径, 而约90%来自于ICS途径(Garcion等2008)。在ICS途径中, UV处理后*ics1*中SA积累水平下降90%, 因此ICS1是ICS途径的关键酶(Garcion等2008)。但是, 大豆(*Glycine max*)中ICS途径和PAL途径在病菌诱导的SA合成中起同等作用(Shine等2016), 而烟草中PAL途径则主导了病菌诱导的SA合成(Ogawa等2006)。因此, ICS途径和PAL途径在病菌诱导SA合成中的贡献度具有物种专化性。

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植物中SA的活性形式是游离态SA, 其水平处于动态变化中。病菌侵染后激活防卫反应所需的SA水平受SA从头合成和代谢途径所调控。在ICS途径中, SA能抑制PBS3活性, 形成一个反馈抑制机制(Rekhter等2019a)。ICS途径贡献了绝大部分防卫相关SA, 且*ICS1*是ICS途径的关键基因, 因此*ICS1*的转录调控很大程度上反映了SA合成途径的调控机制。SARD1、CBP60g、WRKY8/28/46/48/75、TCP8/9、NTL9和CHE等转录因子正调控*ICS1*表达和SA合成(Zhang等2010; Wang等2009, 2011, 2015; van Verk等2011; Gao等2013; Zheng等2015; Guo等2017), 而ANAC019/055/072、EIN3/EIL1、CBP60a、WRKY18/40/54/70、DEL1等抑制*ICS1*表达, 并负调控SA的合成(Wang等2006; Chen等2009; Zheng等2012; Truman等2013; Chandran等2014; Birkenbihl等2017)。一些转录因子通过影响*SARD1*和*CBP60g*的表达来调控SA合成。病菌侵染后TGA1和WRKY70均能结合*SARD1*启动子, 分别激活和抑制*SARD1*表达, 从而影响SA合成(Sun等2018; Zhou等2018); GTL结合*CBP60g*启动子并诱导*CBP60g*表达(Volz等2018); CAMTA1/2/3通过直接或者间接方式抑制*SARD1*和*CBP60g*表达并负调控SA的合成(Kim等2013)。骨架蛋白PHB3与ICS1在叶绿体中形成复合体, 增强ICS1稳定性, 从而促进SA合成(Seguel等2018)。

SA可以代谢形成非活性态和储存态SA, 从而调控植物细胞中游离态SA水平。在拟南芥中, UDP-糖基转移酶催化SA转化成SA糖苷(SAG), 在液泡中储存(Lim等2002; Song 2006; Dean和Delaney 2008)。苯甲酸/SA甲基转移酶BSMT1催化SA生成水杨酸甲酯(MeSA) (Chen等2003; Koo等2007), 而SABP2催化MeSA转变为SA (Forouhar等2005) (图1-A)。乙酰氨基合成酶GH3.5共轭SA和天门冬氨酸形成SA-Asp共轭物(Chen等2013; Mackelprang等2017)。3-SA羟化酶S3H/DLO1和5-SA羟化酶S5H/DMR6分别把SA转化为2,3-羟基苯甲酸和2,5-羟基苯甲酸(Zhang等2013, 2017), 再由UDP-糖基转移酶UGT76D1形成SA-Glc和SA-Xyl共轭物(Huang等2018)。

### 1.1.2 SA不是SAR系统信号, 但SAR需要SA

在病菌侵染并诱导SAR后, 黄瓜(*Cucumis sativus*)植株韧皮部汁液中SA含量上升(Metraux等1990); 烟草受侵叶片中SA含量升高20倍、系统叶片中增加5倍(Malamy等1990)。同位素示踪试验发现, SA能从病菌侵染叶片转运到系统叶片中(Shulaev等1995; Molders等1996)。由此推测, SA是SAR的系统信号。但是, 受侵后4 h时黄瓜植株体内就已发生SA系统积累, 但在8 h时受侵叶片韧皮部渗出液中SA含量才达到可检测水平(Rasmussen等1991), 说明系统叶片中积累的SA并不是从受侵叶片中转运而来。*NahG*或*PAL*沉默烟草植株与野生型植株的嫁接试验证明: 受侵叶片中合成的SA并不是系统叶片中诱导SAR的关键因子; 虽然受侵叶片韧皮部渗出液中有SA积累, 但SA不是SAR系统信号; 系统叶片中诱导SAR需要SA积累(Vernooij等1994; Pallas等1996)。在拟南芥中, SAR需要系统叶片中*ICS1*表达和SA从头合成(Attaran等2009); 在*fld*和*fmo1*突变体中, 受侵叶片中有SA积累, 但系统叶片中没有SA积累, 也不能诱导或明显弱化SAR (Mishina和Zeier 2006; Singh等2013)。因此, SA本身不是SAR中的系统信号, 但系统叶片中诱导SAR需要SA从头合成与积累。

### 1.1.3 MeSA的系统信号功能

MeSA是一种挥发性SA衍生物, 无生物活性, 只有转化为游离态SA后才能起作用(Seskar等1998; Koo等2007)。TMV侵染后烟草植株会产生并挥发出气态MeSA, 引起受侵植株系统叶片和未受侵植株叶片中SA积累(Shulaev等1997)。因此, MeSA可能作为一种气传信号在未受侵叶片或植株间传递并激活防卫反应。同时, 受侵烟草植株叶片及其韧皮部渗出液中MeSA含量升高, 转运到系统叶片中, 并转化为SA, 从而诱导SAR (Park等2007), 表明MeSA也能作为一种在植物体内转运的SAR系统信号。不能形成MeSA的拟南芥*bsmt1*中SAR被明显弱化(Liu等2010)。据此建立MeSA作为系统信号的作用模型: 受侵叶片内SA大量积累抑制SABP2的酯酶活性, BSMT1催化SA形成MeSA; 积累的MeSA经韧皮部转运到系统叶片中, 由SABP2催化转变为SA, 从而诱导SAR。在拟南芥中, 敲除MeSA酯酶(烟草SABP2同源)的突变体中SAR明显弱化, 表明由MeSA酯酶催化MeSA形

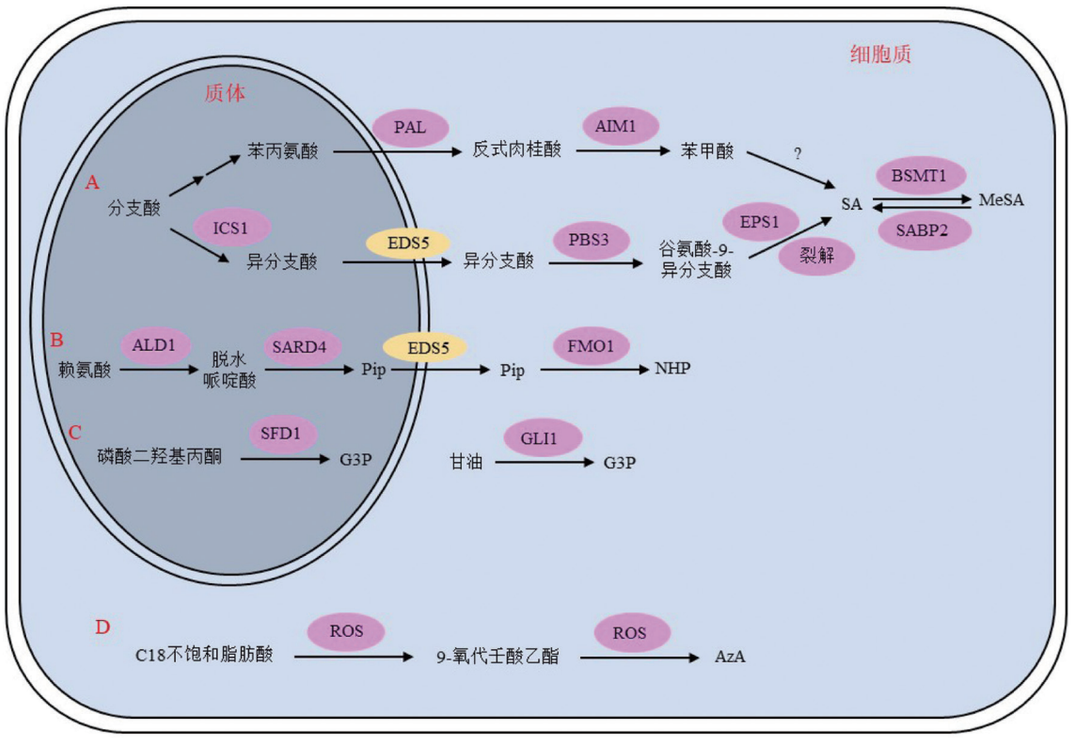


图1 SAR系统信号的合成途径

Fig.1 Biosynthetic pathways for SAR systemic signal compounds

PAL: 苯丙氨酸解氨酶; AIM1: 羟酰基-CoA羟化酶; BSMT1: 苯甲酸/SA甲基转移酶; ICS1: 异分支酸合成酶1; EDS5: MATE转运蛋白; PBS3: 氨基转移酶; EPS1: BAHD酰基转移酶; SABP2: SA结合蛋白2; SA: 水杨酸; MeSA: 水杨酸甲酯; ALD1: α-氨基转移酶; SARD4: 鸟氨酸环化脱氢酶; FMO1: 黄素单加氧酶; Pip: 哌啶酸; NHP: *N*-羟基哌啶酸; SFD1: 3-磷酸甘油脱氢酶; GLI1: 甘油激酶; G3P: 甘油三磷酸; ROS: 活性氧; AzA: 壬二酸。

成SA是SAR所必需的(Vlot等2008)。最近发现, 病菌侵染可诱导拟南芥UDP-糖基转移酶UGT71C3糖基化MeSA, 影响MeSA稳态, 从而负调控SAR (Chen等2019)。但是, MeSA作为SAR系统信号的作用仍有一些争议, 如拟南芥*bsmt1*仍能诱导产生SAR (Attaran等2009)。

### 1.2 Pip和NHP

Pip是一种非蛋白质氨基酸, 是动植物体内常见的赖氨酸代谢产物。早期发现罹病植物组织中有Pip积累(Pálfi和Dézsi 1968), 最近证明Pip及其代谢物NHP是SAR系统信号(Hartmann和Zeier 2018)。

#### 1.2.1 Pip和NHP的合成与调控

植物中Pip由甲基哌啶途径合成。拟南芥中Pip合成途径为(图1-B): α-氨基转移酶ALD1催化L-Lys上α-氨基基团的转氨作用, 形成开环ε-氨基-α-己酮酸(Ding等2016; Hartmann等2017), 脱水环化形成1,2-脱水哌啶酸, 异构化后形成2,3-脱水哌啶酸, 经鸟氨酸环化脱氢酶SARD4的作用形成L-Pip (Hartmann等2017), 最后由黄素单加氧酶FMO1 (属于甲基哌啶酸*N*-羟化酶)转化为NHP (Chen等2018; Hartmann等2018)。根据ALD1或SARD4蛋白的亚细胞定位推测, Pip在质体中合成(Sharma等2013; Cecchini等2015a; Jung等2016)。虽然目前不清楚FMO1的亚细胞定位, 但拟南芥单加氧酶家族分枝III中的成员$FMO_{GS\text{-}OX1}$已被证明定位于胞质(Li等2010)。在*eds5*中UV诱导的NHP积累显著下降, 表明MATE转运蛋白EDS5参与NHP合成(Rekhter等2019b), 推测EDS5可能促进Pip从质体向胞质转运。因此, Pip在质体中合成, 依靠EDS5转运到胞质, 经FMO1催化形成NHP (Rekhter

等2019b)。这个过程与ICS途径合成SA过程极为相似(Rekhter等2019a)。

NHP以游离态和糖基化形式存在, 而且病菌侵染后拟南芥中两种形式NHP均有显著积累(Chen等2018; Hartmann等2018)。病菌侵染后Pip/NHP合成途径基因*ALD1*、*SARD4*和*FMO1*上调表达(Mishina和Zeier 2006; Navarova等2012; Hartmann等2018)。Pip和NHP能诱导Pip/NHP合成途径基因*ALD1*、*SARD4*和*FMO1*的表达(Chen等2018; Hartmann等2018)。因此, NHP对Pip/NHP合成途径具有正反馈调节功能。Pip强烈诱导*SARD1*和*CBP60g*表达(Hartmann等2018), SARD1和CBP60g直接靶向Pip/NHP合成途径关键基因*ALD1*、*SARD4*和*FMO1*, 调控NHP合成(Sun等2015, 2018)。病菌侵染后激活MPK3/6, 增强WRKY33结合*ALD1*启动子的活性, 正调控其表达, 促进Pip和NHP合成, 形成MPK3/MPK6-WRKY33-ALD1-Pip/NHP的正调控链(Wang等2018)。TGA1能结合*SARD1*启动子, TGA1和TGA4激活*SARD1*和*CBP60g*表达, 从而影响Pip合成和积累(Sun等2018); CAMTA1/2/3突变后引起*ALD1*上调表达, 促进Pip合成(Kim等2020), 其中CAMTA3结合*CBP60g*启动子, *camta3*积累高水平SA、Pip和NHP, 说明CAMTA3通过影响*CBP60g*表达而负调控Pip/NHP合成(Sun等2020)。

#### 1.2.2 Pip和NHP合成途径是SAR必需的

正常情况下, 植物体内Pip和NHP含量极低甚至检测不到(Hartmann和Zeier 2018)。病菌侵染能激活植物中Pip和NHP合成, 如病菌侵染后, 水稻(*Oryza sativa*)、马铃薯(*Solanum lycopersicum*)、大豆、烟草、拟南芥叶片中有Pip积累(Palfi和Deszi 1968; Navarova等2012; Vogel-Adghough等2013; Abeysekara等2016), 烟草、番茄(*Solanum lycopersicum*)、芥菜(*Brassica juncea*)、拟南芥等植株中NHP含量和Pip/NHP合成前体物L-Lys含量均有增加(Navarova等2012; Holmes等2019)。*ald*和*fmo1*不能诱导系统叶片中SA积累、防卫基因表达和SAR (Song等2004a; Mishina和Zeier 2006; Liu等2011; Chaturvedi等2012; Navarova等2012)。*sard4*受侵叶片中Pip积累显著下降, 系统叶片中Pip积累无变化, 不能诱导SAR (Ding等2016)。外施Pip能诱导Pip合成缺陷型突变体产生SAR (Song等2004b), 但不能诱导*fmo1*产生SAR (Navarov等2012; Bernsdorff等2016)。过表达*FMO1*可以提高拟南芥植株的抗病性(Bartsch等2006; Koch等2006), 但是*SARD4*突变后过表达*FMO1*拟南芥失去抗病性(Ding等2016)。另外, 外施NHP能恢复*ald1*和*fmo1*的SAR表型(Hartmann等2018)。因此, Pip/NHP合成途径是SAR所必需的, FMO1在Pip下游起作用, 而NHP是SAR中的系统信号。

外施L-Pip能诱导SAR, 而D-Pip不能诱导SAR, 说明L-Pip是诱导SAR的活性形式(Navarova等2012)。外施Pip或NHP能诱导烟草、番茄和辣椒(*Capsicum annuum*)等作物产生SAR (Vogel-Adghough等2013; Holmes等2019)。过表达拟南芥*ALD1*同源基因的水稻植株能增强对稻瘟病的抗性(Jung等2016), 在本氏烟和番茄中瞬时表达*ALD1*或*FMO1*可以提高植株中NHP水平, 增强抗病性(Holmes等2019)。因此, 外施Pip/NHP或改造Pip/NHP合成途径提高植株内源NHP水平可以改良作物抗病性, 达到病害防治目的。

#### 1.2.3 NHP是SAR的系统信号

局部叶片受侵后, 系统叶片中Pip/NHP合成途径基因*ALD1*、*SARD4*和*FMO1*上调表达, 促进Pip和NHP合成和积累(Mishina和Zeier 2006; Navarova等2012; Hartmann等2018)。在拟南芥和大豆的受侵叶片韧皮部渗出液中Pip含量显著升高(Navarova等2012; Abeysekara等2016)。同位素示踪试验表明, $^{14}$C-Pip能从被注射叶片转运到系统叶片中, 并诱导SAR (Wang等2018)。在*sard4*的受侵叶片中Pip能积累到较高水平, 但系统叶片中Pip积累明显滞后(Ding等2016; Hartmann等2017)。因此, 在正常条件下Pip不可能大量地从受侵叶片转运到系统叶片中。病菌侵染后, 系统叶片中NHP积累早于SA和Pip积累(Hartmann等2018); NHP能诱导野生型和*fmo1*产生SAR, 且在系统叶片中检测到NHP-己糖共轭物(Chen等2018)。因此, NHP或其己糖共轭物可能作为SAR系统信号在植株体内转运。

### 1.3 G3P

#### 1.3.1 G3P的合成

G3P是糖代谢产物, 为脂类物质合成提供碳骨

架。G3P合成有2条途径(图1-C): 一是胞质中甘油在甘油激酶(GK/GLI1)作用下形成G3P; 二是质体中磷酸二羟基丙酮经磷酸二羟基丙酮还原酶(DHAPR)/3-磷酸甘油脱氢酶(G3Pdh) SFD1/GLY1的作用形成G3P (Chanda等2008)。

### 1.3.2 G3P在SAR中的作用

拟南芥的受侵叶片中G3P水平显著升高, 且系统叶片中G3P水平比受侵叶片中更高(Chanda等2008, 2011)。G3P处理拟南芥植株局部叶片后能激活系统叶片中基因表达重编程(Chanda等2011); 外施G3P可诱导拟南芥、大豆和小麦(*Triticum aestivum*)产生SAR (Chanda等2011; Yang等2013)。拟南芥中存在不同亚细胞定位的G3Pdh, 其中多个G3Pdh在SAR中起作用。SFD1/GLY1参与质体中G3P合成, 其G3Pdh酶活性是SAR所必需的(Lorenc-Kukula等2012)。*sfd1*受侵叶片中基础抗性、SA积累及防卫基因表达未受影响, 但是系统叶片中SA积累和防卫基因表达显著下降, 且不能诱导SAR (Nandi等2004; Chanda等2011)。另一个质体*g3pdh*和一个胞质*g3pdh*突变体也表现出SAR缺陷表型(Chanda等2011)。质体G3P合成途径中的脂肪酸去饱和酶基因突变体*sfd2*、*fad7*和单半乳糖基合酶基因突变体*mgd1*都表现出SAR缺陷表型(Chaturvedi等2008; Gao等2014)。另外, G3P代谢途径中G3P酰基转移酶(催化G3P形成甘油糖脂并引起G3P含量下降)基因突变体*act1*植株在病菌局部侵染后不能诱导SAR (Chanda等2011)。同时, 胞质中催化合成G3P的甘油激酶基因突变体*gli1*中G3P含量下降, 不能诱导SAR (Chanda等2011)。因此, 质体中DHAPR/G3Pdh途径和胞质中甘油激酶途径合成的G3P是SAR所必需的。

野生型拟南芥植株的受侵叶片韧皮部渗出液可诱导*sfd1*产生SAR, 但*sfd1*植株的受侵叶片韧皮部渗出液不能在野生型和*sfd1*中诱导SAR, 表明*sfd1*的SAR缺陷表型是因不能产生G3P或相关因子等系统信号所导致(Chaturvedi等2008)。用$^{14}$C-G3P处理受侵叶片后, $^{14}$C-G3P并不能转运到系统叶片中, 但在系统叶片中检测到一种未知G3P衍生物(Chanda等2011)。*dir1*和*azi1*在病菌侵染后不能积累G3P, 而且G3P不能诱导或部分诱导*dir1*和*azi1*产生SAR (Chanda等2011; Yu等2013)。因此, G3P诱导SAR时需要DIR1和AZI1, 且G3P、DIR1和AZI1互相依赖, 形成一个反馈调节通路(Yu等2013)。

## 1.4 AzA

### 1.4.1 AzA的合成

AzA是脂质氧化的一种C9二羧酸产物。拟南芥9-脂氧合酶(9-LOX)途径参与对真菌、细菌和病毒的抗病反应(Vicente等2012), 但在9-LOX的*lox1lox5*双突变体中病菌诱导的AzA积累未受影响(Zoeller等2012)。由于拟南芥不存在9-过氧化氢物裂解酶和13-过氧化氢物裂解酶, 因此AzA并非通过酶学途径合成, 而是通过活性氧(ROS)参与的非酶学途径合成(图1-D)。单半乳糖基甘油二酯(MGDG)和双半乳糖基甘油二酯(DGDG)等不饱和脂肪酸在ROS作用下裂解形成9-氧代壬酸乙酯, 最终形成AzA (Zoeller等2012; Wang等2014)。MGDG和DGDG合成缺陷突变体*dgd1*和*mgd1*植株在病菌侵染后不能产生AzA (Gao等2014), 从侧面说明AzA由ROS参与的非酶学途径合成。

### 1.4.2 AzA在SAR中的作用

无毒病菌侵染后, 拟南芥植株受侵叶片中AzA含量升高5~10倍, 而毒性病菌侵染后AzA含量仅有轻微增加(Jung等2009; Zoeller等2012)。AzA处理拟南芥植株下部叶片或根部, 能诱导系统叶片产生SAR (Jung等2009; Cecchini等2019); 用C18不饱和脂肪酸处理拟南芥植株, 病菌侵染后这些不饱和脂肪酸产生AzA, 并诱导SAR (Yu等2013); AzA前体也能诱导系统叶片中产生SAR (Wittek等2014)。但也有研究显示, 外施AzA并不能激活拟南芥和烟草植株系统叶片中的防卫反应(Zoeller等2012; Vicente等2012; Nagy等2017)。AzA不能诱导SA积累, 但病菌侵染后AzA能诱导系统叶片中SA积累并激活信号传导, 表明AzA可能作为一种敏化分子促使系统叶片中SA积累从而诱导SAR (Jung等2009)。AzA在SAR中的作用需要DIR1 (Jung等2009)、AZI1和EARLI1 (Jung等2009; Cecchini等2015b)、FMO1和ALD1 (Jung等2009)、MPK3/6 (Cecchini等2019)、FLD (Singh等2013)和LLP1 (Wenig等2019), 但不需要SFD1/GLY1 (Jung等2009)。

受无毒病菌侵染的拟南芥叶片韧皮部渗出液中AzA含量显著升高(Jung等2009); 表达细菌无毒蛋白的拟南芥植株叶片渗出液中检测到AzA及其前体物质, 且其积累依赖于EDS1 (Wittek等2014)。TMV侵染的烟草植株叶片渗出液中AzA含量也有显著增加(Nagy等2017)。用$^{2}$H-AzA处理拟南芥植株后, 在处理叶片的韧皮部汁液和系统叶片中均检测到$^{2}$H-AzA, 因此认为AzA是SAR系统信号(Jung等2009)。$^{14}$C-AzA能从处理叶片转运到系统叶片和根部中, 且大部分转运到根部(Cecchini等2015b), 但AzA并不能从根部转运到地上部分(Cecchini等2019)。AzA转运需要AZI1和EARLI1 (Jung等2009; Cecchini等2015b)。游离态AzA可以通过胞间连丝在共质体中转运(Lim等2016), 但在系统叶片中大多数AzA以衍生物形式存在(Yu等2013), 推测AzA转化成衍生物再转运。但是, 对于AzA作为SAR的系统信号也有一些争议, 如受侵叶片中的AzA只有约7%转运到系统叶片中(Yu等2013), 甚至在受侵叶片中根本没有AzA积累(Návarová等2012)。

### 1.5 萜类信号物质

#### 1.5.1 DA

DA是一种C20二萜类化合物。在针叶树中, 叶绿体中二萜合成酶催化香叶基二磷酸形成阿松香二烯, 氧化后形成脱氢阿松香二烯, 最后在胞质中由松香烷氧化酶(细胞色素P450单加氧酶)催化合成DA (Hamberger等2011)。拟南芥中存在松香烷氧化酶CYP720B4, 通过类似针叶树中的途径合成DA (Hamberger等2011)。在SAR中, 拟南芥植株叶片和韧皮部渗出液中DA含量没有变化, 但是外施DA能诱导拟南芥、番茄和烟草植株产生SAR (Chaturvedi等2012)。用$^{2}$H-DA处理拟南芥植株下部叶片后, DA能快速转运到系统叶片中(Chaturvedi等2012), 表明DA可能是SAR系统信号。DA处理局部叶片后能引起系统叶片中*ICS1*、*NPR1*和*FMO1*表达及SA积累, 表明DA诱导SAR需要SA合成及其信号途径(Chaturvedi等2012)。同时, DA诱导SAR时还需要DIR1和FLD (Chaturvedi等2012; Singh等2013), SFD1和AZI1介导的G3P和AzA信号可以促进DA诱导SAR (Chaturvedi等2012)。

#### 1.5.2 单萜类信号物质

挥发性有机物(VOC)在植物对害虫的直接和间接防御反应中起重要作用, 但其在植物SAR中的作用并不清楚。最近发现, SAR诱导后, 在*eds1*中至少五种VOC的挥发量显著下降, 包括由质体中类异戊二烯途径合成的α-松萜、β-松萜、莰烯和香松烯等四种萜类物质(Riedlmeier等2017)。α-松萜、β-松萜、莰烯能激活SA积累和防卫基因表达, 并诱导SAR; 单萜类合成酶香叶甲酰还原酶1突变体*ggr1*不能诱导SAR, 但不影响受侵叶片中SA诱导的防卫反应 (Riedlmeier等2017)。萜类合成酶突变体*tps24*中松萜挥发量显著下降, 也不能诱导SAR (Wenig等2019)。因此, 松萜能诱导SAR, 且需要SA合成, 但平行于SA。从SAR植株中挥发出来的松萜和莰烯能诱导相邻植株产生SAR, 因此这些萜类物质在植物-植物间防卫信号传递中起作用(Riedlmeier等2017)。松萜的合成与挥发需要LLP1、Pip和G3P, 而且LLP1、Pip和G3P调控松萜诱导的植物-植物间SAR, 其中Pip和G3P起协同促进作用(Wenig等2019)。

## 2 SAR系统信号的转运

在SAR中, 受侵叶片中产生的系统信号通过维管束空间转运到系统叶片, 并诱导SAR (Dempsey和Klessig 2012; Shah等2014)。一般认为, SAR系统信号通过植物韧皮部转运, 包括质外体和共质体途径(Singh等2017)。AzA和G3P通过共质体转运, 而SA和MeSA (或许包括Pip/NHP)在质外体中转运(图2), 但仅有极少量的AzA、G3P、SA能从受侵叶片转运到系统叶片中(Chanda等2011; Yu等2013; Lim等2016)。MeSA和松萜作为挥发性气传信号在植物-植物间传输防卫信号(图2)。目前对于SAR系统信号的转运机制所知甚少, 但一些蛋白因子参与植物体内SAR系统信号的转运(Chanda等2011; Chaturvedi等2012)。

### 2.1 DIR1

DIR1属于非特异性脂质转移蛋白(LTP)家族。拟南芥*dir1*在病菌侵染时能产生局部抗性, 但不能诱导SAR (Maldonado等2002)。病菌侵染局部叶片后, 野生型植株系统叶片韧皮部渗出液能诱

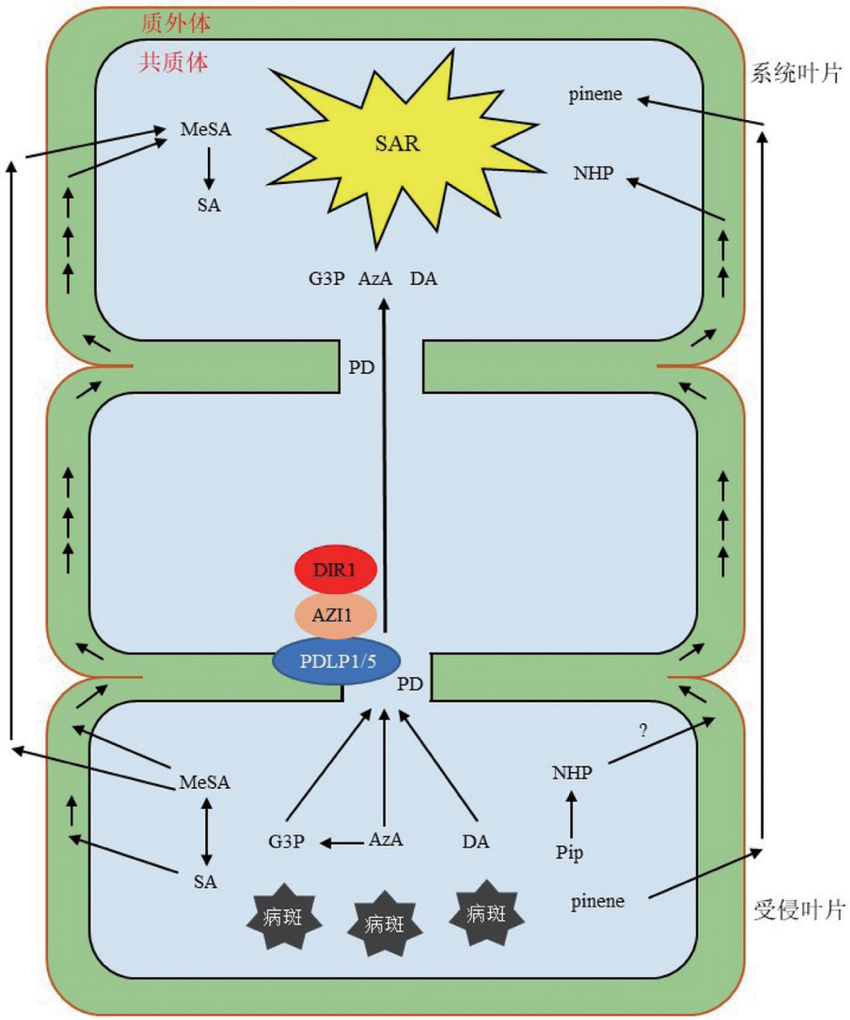


图2 SAR系统信号的转运途径

Fig.2 Translocation routes for SAR systemic signal compounds

SA; 水杨酸; MeSA: 水杨酸甲酯; Pip: 哌啶酸; NHP: *N*-羟基哌啶酸; G3P: 甘油三磷酸; AzA: 壬二酸; DA; 脱氢枞醛; pinene: 松萜; PD: 胞间连丝; PDLP1/5: 胞间连丝定位蛋白1/5; AZI1: 壬二酸蛋白1; DIR1: 诱导抗性缺陷蛋白1。

导*dir1*产生SAR, 但*dir1*植株系统叶片韧皮部渗出液不能诱导野生型植株产生SAR (Maldonado等2002; Chaturvedi等2008)。因此, *dir1*能感知SAR系统信号, 说明DIR1的功能是产生或转运SAR系统信号。拟南芥还存在一个DIR1类似蛋白, 但与SAR无关(Carella等2017)。

AzA、G3P和DA在诱导SAR时都需要DIR1 (Jung等2009; Chanda等2011; Chaturvedi等2012)。在受侵叶片韧皮部渗出物中检测到DIR1; 在*dir1*局部叶片中瞬时表达DIR1并诱导SAR后, 可以在系统叶片中检测到DIR1, 推测DIR1能从受侵叶片中转运到系统叶片中(Champigny等2011, 2013)。病菌侵染并诱导SAR的黄瓜叶片韧皮部渗出液能恢复拟南芥*dir1*的SAR缺陷表型, 而且可检测到类似拟南芥DIR1的蛋白(Isaacs等2016)。表达胞间连丝定位蛋白PDLP1或PDLP5的拟南芥植株不能诱导SAR, 在系统叶片韧皮部汁液中未能检测到DIR1, 表明DIR1可能通过胞间连丝在细胞间转运从而进

行系统性转运(Carella等2015)。在G3P介导的SAR中, G3P衍生物从受侵叶片到系统叶片的转运需要DIR1, 而DIR1从受侵叶片到系统叶片的转运则需要G3P, 而且G3P衍生物和DIR1的转运均发生在共质体中(Chanda等2011)。DIR1在系统叶片中可能与MeSA信号途径有关(Liu等2011), 但AzA转运并不需要DIR1 (Cecchini等2015b)。

### 2.2 AZI1及其同源蛋白

AZI1是一个类LTP蛋白, 隶属于植物杂合富含脯氨酸蛋白家族中的EARLI1亚家族。拟南芥EARLI1亚家族有7个成员, 其中AZI1和EARLI1定位于内质网、胞间连丝、叶绿体外膜及内膜系统接触位点(Fernandez-Calvino等2011; Cecchini等2015b), 推测AZI1和EARLI1协助SAR系统信号通过膜系统转运到维管束中。

拟南芥*azi1*和*earli1*中, 受侵叶片能激活防卫反应, 但系统叶片不能产生SAR, 且AzA不能恢复*azi1*的SAR缺陷表型(Jung等2009; Cecchini等2015b)。在*azi1*局部叶片中瞬时表达*AZI1*或*EARLI1*, 可恢复其SAR缺陷表型(Cecchini等2015b)。因此, AzA诱导SAR时需要AZI1和EARLI1。G3P和DA在诱导SAR时也需要AZI1 (Chanda等2011; Yu等2013; Riedlmeier等2017)。*azi1*和*earli1*叶片吸收$^{14}$C-AzA的能力减弱, $^{14}$C-AzA从处理叶片转运到系统叶片的水平显著下降(Cecchini等2015b)。因此, SAR中AzA转运需要AZI1及其同源蛋白。

### 2.3 PDLP1/PDLP5

胞间连丝是植物细胞间相互连接的通道, 调控共质体中的物质运输。胞间连丝定位蛋白(PDLPs)是定位在胞间连丝通道中的跨膜类受体蛋白, 参与植物免疫反应(Amari等2010; Lee等2011; Wang等2013; Caillaud等2014; Aung等2019; Liu等2020)。AzA和G3P通过共质体通路从受侵叶片转运到系统叶片中, 并受胞间连丝通道的调控(Lim等2016)。病菌侵染后, *pdlp1*、*pdlp5*和过表达*PDLP5*植株中受侵叶片能正常积累SA、AzA和G3P, 但不能诱导产生SAR; 外施SA、AzA和G3P不能诱导*pdlp1*、*pdlp5*和过表达*PDLP5*植株产生SAR (Lim等2016)。因此, PDLP1和PDLP5参与AzA和G3P在胞间连丝中的转运从而调控SAR。病菌侵染后, 过表达*PDLP5*植株中系统叶片韧皮部渗出液中检测不到DIR1, 推测DIR1也是通过胞间连丝通道从病菌侵染叶片转运到系统叶片中(Carella等2015)。PDLP1能与AZI1和PDLP5互作, 推测PDLP1/PDLP5与AZI1形成一个复合体, 介导SAR系统信号在共质体中的转运(Lim等2016)。

## 3 SAR系统物信号的互作

### 3.1 SA和Pip/NHP间的互作

SA和Pip/NHP在合成、信号放大和信号途径方面存在互作关系。Pip诱导SA合成途径基因*ICS1*、*EDS5*和*PBS3*表达, 强化病菌诱导的SA合成(Navarova等2012; Bernsdorff等2016; Hartmann等2018)。在*ald1*和*sard4*中, 受侵叶片中SA积累略有下降, 但系统叶片中SA积累完全被阻断(Song等2004a; Ding等2016), 表明系统叶片中SA合成需要Pip/NHP。SA诱导Pip合成途径基因*ALD1*和*SARD4*表达(Ding等2018), 且系统叶片中Pip积累需要SA (Wang等2018)。因此, SA和Pip/NHP间存在双向信号放大机制。在受侵叶片中, SA积累早于Pip/NHP积累, 但在系统叶片中NHP积累早于SA积累(Hartmann等2018)。因此, 系统叶片中NHP能强化SA合成和积累, 从而诱导SAR。SARD1和CBP60g是SA和Pip/NHP合成途径的共同正调控因子, 且被SA和Pip/NHP所诱导, 因此SARD1和CBP60g可能是SA和Pip/NHP间双向信号放大机制的主要节点。Pip/NHP诱导的SAR存在依赖SA和不依赖SA的不同调控机制, 而且SA起Pip/NHP信号放大器的功能(Bernsdorff等2016)。在*sid2*和*npr1*中, Pip和NHP积累水平增加, 特别在*sid2*中NHP有高水平积累(Hartmann等2018)。因此, SA对Pip转化为NHP的生化过程具有负调控作用。

EDS1和PAD4是SA信号途径中的关键因子。EDS1和PAD4促进Pip/NHP合成途径基因*ALD1*和*FMO1*表达(Song等2004a; Bartsch等2006), 在*eds1*和*pad4*中Pip和NHP积累水平显著下降(Hartmann等2018), 而且PAD4是Pip诱导SAR所必需的(Navarova等2012)。因此, EDS1和PAD4在Pip合成和羟基化反应两个层面上调控NHP合成, 位于NHP合成的上游。在*npr1*中, SA和Pip不能诱导SAR

(Navarova等2012; Bernsdorff等2016), 表明NPR1是SA和Pip/NHP信号途径的一个共享节点。MLO2是SA和Pip/NHP信号途径中的另一个共享节点, 因为SA和Pip均诱导*MLO2*表达, 而且SA和Pip诱导SAR时需要MLO2的作用(Gruner等2018)。

### 3.2 系统信号间及其与ROS的互作

AzA需要Pip/NHP合成途径基因*ALD1*和*FMO1* (Jung等2009), 系统叶片中Pip积累及Pip诱导SAR也需要LLP1、G3P和松萜(Wang等2018; Wenig等2019)。AzA可能通过促进G3P合成途径中关键基因*GLY1*和*GLI1*表达, 加快G3P合成, 从而诱导SAR (Yu等2013)。病菌侵染后, 拟南芥植株体内G3P含量上升早于AzA积累(Chanda等2011), 且AzA可诱导G3P合成途径突变体*sfd1*植株产生SAR (Jung等2009)。因此, AzA与G3P、NHP信号存在关联。G3P不能引起SA含量升高, 但能诱导系统叶片中*SABP2*上调表达和*BSMT1*下调表达, 促进MeSA向SA转变, 从而诱导SAR (Chanda等2011)。G3P不能诱导*sid2*植株产生SAR, SA也不能诱导*gly1*和*gli1*植株产生SAR (Chanda等2011), 因此SA和G3P存在互作关系。在松萜诱导SAR时, Pip和G3P在上游形成一个信号放大反馈调节环, 而AzA则在下游起作用(Wenig等2019)。

ROS在SAR中起作用, 且与一氧化氮(NO)形成反馈调节机制; ROS和NO在AzA-G3P信号链的上游起作用(Wang等2014)。与此相似, Pip促进ROS和NO的产生从而诱导SAR, 而且ROS和NO位于G3P上游; 不能合成NO、ROS、G3P或SA的突变体中, 系统叶片中Pip积累下降, 且系统叶片中Pip合成需要SA和G3P。由此认为, Pip在NO-ROS-AzA-G3P信号链的上游起作用, NO和ROS在受侵叶片和系统叶片中与不同SAR系统信号互作, 从而不断放大信号传导链(Wang等2018)。

## 4 小结与展望

系统信号是SAR的核心问题。解析SAR系统信号及其作用机制可以促进对植物诱导免疫的理论认识, 并以此为基础研发作物病害防控的全新绿色应用技术, 如根据SA结构特征研发的植物激活剂苯并噻二唑制剂在欧美国家推广应用, 改造NHP合成途径可以提高作物抗病性(Holmes等2019)。但是, 植物SAR系统信号领域仍有诸多问题有待解决: (1)新SAR系统信号。SAR系统信号的转运发生在病菌侵染后6 h内(Chanda等2011; Chaturvedi等2012), 明显早于系统叶片中SA、AzA和G3P积累, 说明在SA、AzA和G3P上游存在其他未知信号。(2) SAR系统信号的转运机制。SA、MeSA在质外体中转运, 而AzA和G3P在共质体中转运, 其中DIR1、AZI1、PDLP1/5可能参与AzA和G3P转运(Singh等2017)。但是, NHP或其衍生物是否为SAR系统信号有待确认, 且在正常生理条件下NHP能否进行系统性转运也有待嫁接试验证实。此外, SAR系统信号在韧皮部或共质体中转运的具体机制也不清楚。(3)系统叶片中SAR系统信号合成、积累、感受及其下游信号途径。病菌侵染后局部叶片合成的SA、AzA、G3P等SAR系统信号中仅有极少部分能转运到系统叶片(Chanda等2011; Chaturvedi等2012; Lim等2016), 诱导SAR所需的信号很大程度上来源于系统叶片中的重头合成。因此, 系统叶片中启动和调控SAR信号物质合成和积累的机制需要研究阐明。同时, NPR1/3/4作为受体感知SA信号并调控启动下游免疫信号途径(Zhang和Li 2019), 但是系统叶片如何感知NHP、AzA、G3P等SAR系统信号以及感知后如何启动并调控下游免疫信号途径等也尚不清楚。

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# Systemic signals and their mechanisms in plant systemic acquired resistance

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**Abstract:** Systemic acquired resistance (SAR) is a defense mechanism induced by local infection of pathogens, conferring broad-spectrum resistance within the whole plants. During SAR, a variety of systemic signals are generated at the primary infection site and transported to the distal parts of the plants, leading to the systemic activation of defense responses. This review summarizes the recent progress on the function, transport, mechanism and relationship of SAR systemic signals including salicylic acid and methyl salicylate, pipecolic acid and *N*-hydroxypipecolic acid, azelaic, glycerol-3-phosphate, and terpene compounds (dehydroabietinal and pinenes).

**Key words:** systemic acquired resistance; systemic signal; defense response

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