综 述 Reviews

水杨酸介导植物抗病的研究进展

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摘要: 水杨酸(salicylic acid, SA)是一种在植物免疫反应中起着重要作用的信号分子, 植物受到活体营养型病原侵染后, 体内SA合成急剧增加, 这是激活植物对病原产生抗性反应所必不可少的细胞信号响应, 因此SA又被称为植物抗病激素。研究表明植物体内具有多种与SA作用或结合的蛋白, 其中NPR1 (nonexpressor of pathogenesis-related genes 1)、NPR3和NPR4在SA介导的信号感受和传导过程中起着关键的调控作用。本文围绕SA在植物抗病反应中的作用; SA的生物合成及其调控; 以及植物细胞利用SA结合蛋白(SA-binding-proteins, SABPs), 如NPR1、NPR3和NPR4等, 感受SA并调节抗病信号传导途径等进行简单的介绍, 并对其研究前景进行展望。

关键词: 水杨酸(SA); 信号传导; 水杨酸结合蛋白; NPR1; NPR3; NPR4

水杨酸(salicylic acid, SA)是一种简单的酚类化合物, 最先在柳树皮的提取物中发现(Vlot等2009)。早在1979年, White (1979)发现, 经过SA或其衍生物阿司匹林处理后的烟草对烟草花叶病毒(tobacco mosaic virus, TMV)的抗性提高。后来的研究发现, 在烟草和拟南芥中表达一种水杨酸羟化酶(salicylate hydroxylas, NahG)能阻止SA在体内的积累, 进而使植物无法启动效应因子激发的免疫反应(effector-triggered immunity, ETI)和系统获得性抗性(systemic acquired resistance, SAR)等抗病免疫反应(Gaffney等1993; Delaney等1994)。这些研究结果最先证明, SA是一种重要的植物防卫激素, 在植物防御反应尤其是SAR中起着显著的作用(Vlot等2009)。此外, SA还参与调节植物的生长发育和各种应激反应(stress response), 如植物的耐热性、耐旱性、种子萌发、气孔关闭、光合作用、开花及植物衰老等过程(Vicente和Plasencia 2011)。

1 植物的抗病免疫反应

植物在生长和发育过程中会受到多种病原的侵害, 为了应对外界的环境压力和病原菌的感染, 植物在漫长的进化过程中逐渐形成了一系列迅速而有效的防御机制。植物免疫活动通常被归纳为病原相关分子(pathogen-associated molecular patterns, PAMPs)激发的免疫反应(PAMPs-triggered immunity, PTI)和ETI。其中, PTI主要是指病原微生物表面的病原特征性分子(如细菌鞭毛蛋白、真菌细胞壁成分几丁质等)刺激诱导后, 宿主植物细胞产生的非特异性防卫反应(即基础防卫反应)(Boller和Felix 2009; Macho和Zipfel 2014)。植物细胞表面的模式识别受体(pattern recognition receptors, PRRs)识别微生物保守的PAMPs后, 其功能激活而诱发一序列的细胞响应, 例如活性氧(reactive oxygen species, ROS)的产生、外部Ca^{2+}涌入宿主细胞、促分裂素原活化蛋白激酶(mitogen-activated protein kinases, MAPKs)的瞬时激活和SA的产生, 最终导致PTI的建立(Tsuda等2008a, 2008b; Tsuda和Katagiri 2010); 在进化过程中, 病原菌也会躲避或者干扰植物的防御反应, 以确保它们能从宿主植物获取其生长所需要的营养(Bozkurt等2012)。一些病原菌通过产生一些毒性因子(virulent effectors)抑制或干扰植物的PTI, 使它们在与宿主植物交锋的过程中占有优势, 从而可以再度感染植物, 这种现象被称为效应因子触发的感病性(effector-triggered susceptibility, ETS) (Jones和Dangl 2006; Bozkurt等2012; Win等2012)。近年来, 研究者已经发现多种效应因子可以干扰SA信号传导途径(Uppalapati等2007; Djamei等2011; Caillaud等2013; Jiang等2013; Rabe等2013; Liu等2014)。例如, 丁香假单胞杆菌(*Pseudomonas syringae*)分泌毒性因子冠菌素(coronatine, COR), COR能抑制番茄中SA信

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号传导途径(Uppalapati等2007); 玉米黑粉菌(*Ustilago maydis*)菌株分泌的一种毒性因子Cmu1具有分支酸变位酶(chorismate mutase)活性, 通过催化分支酸(chorismate)变成预苯酸(prephenate)进而影响SA的合成, 从而干扰SA信号传导途径(Djamei等2011); 活体营养型卵菌(*Hyaloperonospora arabidopsidis*, *Hpa*)分泌的效应因子HaRxL44能抑制SA途径中抗性基因的表达(Caillaud等2013); 大豆疫霉菌(*Phytophthora sojae*)和黄萎病菌(*Verticillium dahliae*)分泌的异分支酸酶PsIsc1和VdIsc1, 作为效应蛋白通过抑制SA的前体破坏植物的SA代谢(Liu等2014)。

在与带有毒性因子的病原菌进行抗争的过程中, 宿主植物进化出抗性基因(Resistance genes), 也叫R基因(R-gene), 其编码的蛋白往往含有保守的亮氨酸富集重复结构域(leucine-rich-repeat, LRR), 其功能主要是检测病原菌编码的抑制宿主抗性反应的效应因子, 或效应因子对宿主细胞中靶蛋白功能的干扰活动, 然后启动植物抗病免疫反应, 即宿主植物的ETI (Jones和Dangl 2006; Lewis等2009)。ETI的激活也会导致SA更多的积累和MAPK的激活, 而SA的积累和MAPK的激活又对ETI中植物的抗病性具有重要促进作用(Tsuda等2013)。通常R蛋白激活ETI后, 植物所产生抗性具有典型的表现形式, 被侵染部位以局部组织迅速坏死的方式来阻止病菌的扩散, 即发生超敏反应(hypersensitive response, HR) (Mur等2008)。随后产生并释放系统性信号分子, 并引起植物对同一病原或其它病原产生抗性, 即产生SAR (Fu和Dong 2013)。系统信号分子的本质, 目前还不清楚, 但在这个过程中, SA起到非常关键的作用(Durrant和Dong 2004)。SA能诱导包括病原相关蛋白(pathogenesis-related protein, PR蛋白)在内的许多抗病相关蛋白的产生, 从而使植物产生SAR。人为施加SA足以诱导SAR的建立, 阻断SA合成或积累会导致植物无法产生SAR, 并且对病原菌更敏感(Delaney等1994; Wildermuth等2001)。

除SA外, 其他植物激素在植物抗病信号传导中也起着直接或间接作用, 如茉莉酸(jasmonic acid, JA)、乙烯(ethylene, ET)、脱落酸(abscisic acid, ABA)、细胞分裂素(cytokinin, CK)、生长素(auxin, AUX)、赤霉素(gibberellin, GA)、油菜素类酯(brassinosteroids, BRs)等(Pieterse等2012)。这些激素介导的信号传导途径存在交叉, 有的相互抑制, 有的相互促进(Mur等2006)。而SA介导的信号途径与Ca^{2+}介导的信号途径也存在交叉(Du等2009)。与SA相关的核心问题是, 抗病反应中SA的合成如何受到调控, SA介导的信号途径如何被激活, 以及SA信号如何激活植物的抗病性。

2 抗病反应中SA的生物合成与调控

2.1 SA的生物合成途径

已有研究显示, 植物中SA合成途径一般分为两种: 一种是异分支酸合酶 (isochorismate synthase, ICS)途径, 即分支酸经由ICS催化形成异分支酸(isochorismate, IC), 然后再由异分支酸丙酮酸裂解酶(isochorismate pyruvate lyase, IPL)催化转变为SA (Wildermuth等2001); 另一种是苯丙氨酸氨解酶(phenylalanine ammonia-lyase, PAL)途径, 即苯丙氨酸被PAL催化裂解为肉桂酸, 肉桂酸经由几个未知酶的催化转化成苯甲酸, 再由苯甲酸-2-羟化酶催化转化成SA (Lee等1995)。研究表明, 抗病反应中诱导产生的SA 90%以上是经由ICS途径合成的(Wildermuth等2001)。拟南芥、烟草、番茄、杨树、向日葵和辣椒等植物中都含有ICS和PAL同源基因(Wildermuth等2001; Cochrane等2004; Uppalapati等2007; Catinot等2008; Yuan等2009; Sadeghi等2013; Kim和Hwang 2014), 这表明在进化过程中, 这些SA合成途径对植物的生存有重要作用(Seyfferth和Tsuda 2014)。拟南芥中有2个*ICS*基因*ICS1* (也称作*SID2*)和*ICS2*。拟南芥*ics1*突变体在受到病原菌感染后, SA的积累量只有野生型植株的5%~10%。拟南芥包含4个*PAL*基因(*PAL1*~*PAL4*), 拟南芥*pal1 pal2 pal3 pal4*四突变体(PAL活性只有10%)在受到病原菌感染后, SA的积累量是野生型植株的50% (Huang等2010), 这些研究结果表明在植物产生免疫反应的过程中, SA主要是通过ICS途径来合成, PAL途径也参与SA的合成, 但其作用不明显。

在叶绿体中, ICS催化分支酸转变成IC (Wildermuth等2001; Strawn等2007; Garcion等2008), 进而转变成SA (Dempsey等2011)。在某些细菌中, IC变成SA是由IPL催化的。然而, 植物基因组中并没

有编码细菌IPL的同源基因。叶绿体中, ICS和细菌酶IPL的表达会导致SA的积累(Verberne等2000; Mauch等2001)。因此可以想象, 植物叶绿体中有某些尚未被发现的基因或基因家族的产物含有IPL活性(Seyfferth和Tsuda 2014)。然而, 一些代谢酶如酰基酰胺合成酶(acyl acid amido synthetase) (如GH3.12) (Nobuta等2007; Okrent等2009; Westfall等2010, 2012)以及酰基转移酶(如enhanced pseudomonas susceptibility 1, EPS1) (Zheng等2009)都与SA的积累有关, 这些酶可能为SA的生物合成提供前体来影响SA的积累。因此, SA在植物中的生物合成可能比在细菌中更复杂。SA在叶绿体中合成后通过MATE (multidrug and toxin extrusion)转运蛋白EDS5 (enhanced disease susceptibility 5) 转运至细胞质(Serrano等2013), 在*eds5*突变体中, SA的积累被抑制(Nawrath等2002; Ishihara等2008), 因此EDS5的转运对SA的积累及其在细胞中的分布至关重要。

2.2 抗病反应中SA的调控

对抗病过程中SA抗病信号途径的激活的认识, 最先是通过研究*R*基因介导的抗病反应获得的。大多数R蛋白结构中都含有核苷酸结合位点(nucleotide binding site, NBS)及LRR结构域。NBS-LRR类型的抗性基因主要分两类CC-NBS-LRR和TIR-NBS-LRR (Qian和Liu 2014)。CC-NBS-LRR类抗性基因介导的抗病性主要受依赖NDR1 (non-specific disease resistance 1)的信号通路调控。植物免疫反应建立过程中, NDR1在SA上游发挥功能(Century等1997), 功能缺失突变体*ndr1*不能正常诱导SA的合成, 功能互补实验表明, NDR1能恢复*ndr1*突变体SAR等方面的缺陷(Vlot等2009)。另一类抗性基因TIR-NBS-LRR介导的抗性主要受依赖EDS1功能的信号通路调控(Aarts等1998)。EDS1在抵抗活体病原菌的过程中, 通过与PAD4相互作用来激活TIR-NBS-LRR感知的ET1和植物基础免疫反应(Feys等2001)。

遗传学分析表明, 在SA相关信号传导途径中, PAD4、EDS1以及NDR1都位于SA合成基因*ICS1*的上游。拟南芥在没有受到胁迫或激素诱导情况下, 与SA合成相关的基因的表达维持在较低水平; 在植物受到生物或非生物胁迫时, *ICS1*基因的表达很可能通过去抑制作用来激活(Dempsey等2011)。因此, 在烟草和拟南芥等植物中, *ICS1*的转录调控对防御反应的激活具有重要作用。目前已知的多种转录因子能直接与*ICS1*的启动子结合并调控其基因表达。拟南芥钙调素结合蛋白(calmodulin-binding proteins, CBPs)中的CBP60基因家族中两个成员CBP60g和SARD1 (SAR defective1), 共同正向调节*ICS1*的表达(Zhang等2010b; Wang等2011), 影响PAMP诱导的SA积累(Wang等2009)。TCP 家族转录因子TCP8和TCP9也能正向调控病原菌侵染过程中*ICS1*的诱导表达和SA的积累(Zheng等2015)。通过酵母单杂交技术, Zheng等(2015)发现了激活*ICS1*表达的两个转录因子: NTL9 (NTM1-like 9)和CHE (CCA1 hiking expedition)。NTL9在形成气孔的保卫细胞中激活*ICS1*, 帮助气孔快速关闭, 阻止病原体进入。CHE是主要的生物钟响应因子, 介导SA水平的节律性变化。此外, SAR过程也需要通过CHE提高系统性的水杨酸合成(Zheng等2015)。此外研究表明, 外源SA能诱导SA信号途径中正调控基因的表达(包括ICS1、EDS5、PBS3、SGT1和UGT74F1), 而这些基因的表达又会导致SA的迅速积累, 从而形成一个SA信号的反馈放大回路。SA信号放大系统的存在, 暗示植物体内必然有SA信号的负调控机制来平衡SA信号。受钙/钙调素信号控制的AtSR1/CAMTA3转录因子可以抑制TIR-NB-LRR和CC-NB-LRR两类*R*基因激发的抗病信号正调控因子(*EDS1*和*NDR1*)的表达(Du等2009; Nie等2012), 进而防止SA的过度产生。EIN3 (Ethylene-insensitive 3)和ANAC019 (Arabidopsis NAC domain-containing protein 19)也可以对*ICS1*基因的表达和SA水平起负调控作用, 并影响SA与其他植物激素的相互作用(Chen等2009; Zheng等2012)。

另一方面, 其他激素, 如吲哚乙酸(indoleacetic acid, IAA)、JA和ET等也会影响SA信号途径。IAA、JA和ET通过影响SA代谢过程的调节因子(如BSMT1、GH3.5)来调控细胞内SA水平(Goda等2008), IAA、JA和ET通过这种方式能调控游离SA的积累, 反过来又抑制SA介导的防御反应的激活。此外, JA和ET能间接抑制ICS1介导的SA的合成(Chen等2009; Zheng等2012)。

3 SA结合蛋白

3.1 SABP

SA作为一种信号分子, 必须要与细胞中靶蛋白或受体蛋白结合才能激活下游信号元件。因此, 鉴定和分析与SA作用或结合的蛋白是一个很有意义的科学问题, 科学家们也为此做出了很多努力。SABP最先从烟草中分离纯化出来, 是植物中的具有过氧化氢酶(catalase, CAT)活性的蛋白, 其与SA亲和力较低(K_d=14 μmol·L^{-1})。研究人员设想SA与SABP结合并抑制其CAT活性, 导致ROS水平提高(如H_2O_2的积累), 进而诱导植物产生防御反应。然而Ruffer等(1995)发现, SA并不只与植物中的CAT结合, 也与真菌和动物中的CAT结合。进一步研究表明, SA也可以和其他含Fe^{2+}的氧化还原酶, 如顺乌头酸酶、脂肪氧化酶及过氧化物酶等结合(Ruffer等1995)。因此, 这些酶只是SA的普通的靶蛋白, 并不是特异的SA受体(Yan和Dong 2014)。

3.2 SABP2

与SABP相比, 烟草中SABP2与SA亲和力更强(K_d=90 nmol·L^{-1}) (Du和Klessig 1997)。通过结构和生化分析表明, SABP2具有水杨酸甲酯(methyl salicylate, MeSA)酯酶活性, 而SA是SABP2酶活性的强抑制剂(Kumar和Klessig 2003; Forouhar等2005)。拟南芥中至少有18个与SABP2同源的蛋白, 这些同源蛋白中, AtMES9 (*Arabidopsis thaliana* MeSA esterase 9)与SA亲和力最高, 约为烟草SABP2的2倍(K_d=45 nmol·L^{-1}) (Vlot等2008)。虽然SABP2可能参与诱导产生SAR, 但SABP2并不是作为SA受体起作用, 而是通过将没有生物活性的MeSA 转化成具有活性的 SA而发挥作用(Forouhar等2005)。

3.3 SABP3

SABP3是叶绿体中的一种碳酸酐酶, 其与SA结合能力在SABP和SABP2之间(K_d=3.7 μmol·L^{-1}) (Slaymaker等2002)。由于SA在叶绿体中合成后需要通过EDS5转运到细胞质中发挥作用, 而SA的受体并不存在于叶绿体, 因此SABP3不可能是真正的SA受体蛋白(Serrano等2013)。

3.4 其他与SA作用的蛋白(SABPs)

近年来, Tian等(2012)将光亲和标记技术和表面等离子共振技术相结合, 从拟南芥中分离出其他SABPs, 这些SABPs是戊二酸脱氢酶(α-ketoglutarate dehydrogenase, KGDH)的E2亚基和4种谷胱甘肽硫基转移酶(glutathione S-transferases, GSTs), 即GSTF2、GSTF8、GSTF10、GSTF11 (Tian等2012)。传统放射性标记SA的配体结合实验表明, 这些SABPs与SA结合能力很弱, 甚至根本不与SA结合。可见, 这些SABPs对SA的亲和力很弱, 或者只是瞬时与SA相互作用(Tian等2012)。这些SABPs在SA应答反应中的重要性有待进一步研究。

Moreau等(2013)利用蛋白质微阵列技术寻找拟南芥基因组所编码的SABPs, 发现了65个与AzSA (4-azido SA: SA类似物)作用的蛋白质。他们选择了其中的甲拌磷金属内肽酶(thimet metallo-endopeptidase, TOP)进一步分析, 结果证实SA可以与TOP结合并抑制该酶的活性。然而, 通过传统SA结合实验, 研究者发现在竞争实验中, 10 mmol·L^{-1}非放射性SA仅仅使300 nmol·L^{-1}放射性SA与TOP的结合信号下降了一半, 因此TOP是否与SA特异结合有待进一步核实(Moreau等2013)。

4 NPR1、NPR3及NPR4

4.1 NPR1是SA信号传导中的关键调节因子

NPR1作为植物SA信号传导过程中的关键调节因子, 位于SA的下游, PR蛋白基因表达的上游。在*npr1*突变体中, 编码PR蛋白的基因无法表达, 并且无法激活SAR, 不能产生抗病性, 这说明NPR1的缺乏会导致植株丧失SAR (Cao等1994, 1997)。在未受刺激的条件下, NPR1单体蛋白在合成后会通过半胱氨酸残基的分子间二硫键结合成低聚体复合物并在胞质中积累, 而细胞核中的NPR1维持在很低的水平(Mou等2003; 赵淑清和郭剑波2003; 彭金英和黄勇平2005)。

NPR1包含一个BTB/POZ结构域和一个锚蛋白重复序列结构域。酵母双杂交研究结果表明, 这些锚蛋白重复序列可以与TGA转录因子家族互作, 并可能调控TGA转录因子的活性; 虽然NPR1本身并非转录因子, 但在诱导SAR基因表达过程中, 它可以通过锚蛋白重复序列结构域与转录因子互作, 从而作为转录辅助因子调控很多抗病相关基因(*PR* genes, *PR*基因)的表达(Zhang等1999; Kinkema等2000; Zhou等2000)。*PR*基因编码的多肽大多是直接参与抵御微生物的效应蛋白。在受

到病原菌侵染或经SA处理后, 植物体内SA的积累会诱发植物细胞内氧化还原状态的改变, 从而使NPR1低聚体分子间的二硫键断开形成NPR1单体并转移到细胞核中。NPR1进入细胞核后被磷酸化, 随后被泛素化, 这些过程对植物SAR的激活具有重要意义(Spoel等2009)。任何NPR1单体进入细胞核后都会被逐渐降解, 但是只有磷酸化的NPR1在降解后才能激活SAR (Fu等2012)。在细胞核中NPR1与TGA等转录因子相互作用激活*PR*及其他调控植物免疫反应基因的表达(Kinkema等2000)。

除了与TGA转录因子一起调控*PR*基因的表达外, NPR1也参与PR蛋白翻译后加工并向质外体转运的过程。PR蛋白的加工成熟和向胞外的运输由包括BiP2在内的定位在内质网的分子伴侣(chaperone)参与完成。拟南芥*bip2*突变体受苯并噻二唑(benzothiadiazole, BTH)诱导后PR1在胞外的积累下降, 并且BTH不能诱导*bip2*产生SAR (Wang等2005)。研究表明, 很多参与PR蛋白成熟与分泌的分子伴侣基因的启动子区富含TL1顺式元件(Translocon 1 cis element, GAAGAAGAA)。通过鉴定识别TL1的转录因子时, 研究人员在拟南芥中发现热激转录因子TBF1 (TL1-binding factor 1, 也叫HSF4/HsfB1)可以识别并调控包括BIP2在内的内质网膜定位的分子伴侣蛋白(Wang等2005)。在*npr1*功能缺失突变体中, *TBF1*的表达水平明显下降, 并不再被外源SA诱导。从这些结果可以推测, NPR1至少间接调控PR蛋白的翻译后成熟加工并向细胞外运输这一过程(Pajerowska-Mukhtar等2012)。此外, NPR1也直接参与内源SA含量水平的调控。研究表明SA合成途径中ICS1的激活会促进SA的积累, 进而促使NPR1低聚体转变成NPR1单体并转运到细胞核中, 而细胞核中NPR1正常发挥功能反过来又抑制了*ICS1*基因的表达。当NPR1不能转运到细胞核时, *ICS1*基因的持续表达会导致SA过度积累从而产生毒害(Zhang等2010a)。

在拟南芥、胡萝卜、水稻、烟草、番茄、小麦和苹果等很多植物中都发现, *NPR1*基因的过表达会导致植物抗病性提高(Fitzgerald等2004; Lin等2004; Chern等2005; Makandar等2006; Malnoy等2007), 说明在高等植物中NPR1调控植物免疫反应是一种普遍现象。

4.2 NPR1及NPR3、NPR4近年来研究进展

*npr1*突变体中, SA诱导的抗病性出现缺陷, 这与NPR1作为转录辅助因子发挥作用的观点高度一致, 遗传学研究表明NPR1很可能是一种SA受体(Wang等2006)。但是Fu等(2012)通过传统受体-配体结合实验发现拟南芥NPR1并不能与SA结合, 而NPR1同源蛋白(NPR3、NPR4)与SA结合活性很高。而Wu等(2012)在平衡透析实验中发现, NPR1能与SA结合, 但是这种结合需要NPR1中两个半胱氨酸残基(Cys521及Cys529)和铜离子的协同作用。Fu等(2012)的结合实验中不能检测到NPR1和SA的结合活性, 其可能的原因是结合缓冲液中没有铜。但在缓冲液中添加铜后, 并没有检测到NPR1与SA的结合活性(Yan和Dong 2014)。如果那两个半胱氨酸残基对NPR1与SA结合至关重要, 那么在其他植物NPR1同源蛋白中, 这两个残基应该具有保守性, 但情况并非如此。因此, 如果说拟南芥NPR1是SA受体, 那么其他植物中NPR1可能通过不同的机制感受SA。另一项研究表明, 瞬时表达实验中报告基因的诱导需要Cys521及Cys529两个残基发生氧化(Rochon等2006), 而这两个Cys残基如何与铜结合还需要进一步的研究。大多数情况下, Cys残基只有在还原状态下才具有与金属结合的活性(Giles等2003)。Wu等(2012)的研究中, NPR1与SA结合能力很强(K_d=140 nmol·L^{-1}), 这比SABP及SABP3与SA的亲和力都要高。既然NPR1与SA亲和力很强, 那么Fu等(2012)传统配体实验应该能灵敏的检测出其结合能力, 有可能SA-NPR1复合体很不稳定而很难检测出来。因此, 拟南芥NPR1是否是SA受体仍有待进一步明确。

Spoel等(2009)的研究发现, NPR1的降解需要Cul3类型的泛素连接酶, 该连接酶往往利用含有BTB结构域的蛋白作为底物接头蛋白(Pintard等2004)。*npr1*突变体抗病性降低, 而*npr3 npr4*双突变体抗病性增强; 同时NPR1同源蛋白NPR3和NPR4都含有BTB结构域, 这说明NPR3和NPR4也可以作为Cul3类泛素连接酶底物接头蛋白(Zhang等2006)。Fu等(2012)也发现, NPR3和NPR4可以与NPR1相互作用, 它们可以作为Cullin3类型泛素连接酶底物接头蛋白介导NPR1的泛素化和降解。非常有意思的是, NPR3和NPR4与NPR1相互作用

受SA调控, SA抑制NPR4与NPR1相互作用, 但是促进NPR3与NPR1相互作用。NPR3和NPR4以不同的亲和度与SA结合(NPR3, K_d=980 nmol·L^{-1}; NPR4, K_d=46 nmol·L^{-1})。当SA量很低时, NPR4与NPR1结合, 降解NPR1, 使细胞中的NPR1维持在很低的水平; 当SA量很高时, NPR3与NPR1结合, 降解细胞核中的NPR1; 只有当SA含量适中时, SA抑制NPR4与NPR1相互作用, 但是又不足以促进NPR3与NPR1相互作用, 此时细胞核中的NPR1维持在较高的水平上, 从而调控下游基因的表达。植物受到病原菌等感染后, NPR3和NPR4作为感应SA的受体, 通过调节NPR1的含量, 决定细胞死亡或存活。在感染部位, SA含量升高促进NPR3与NPR1相互作用并降解NPR1, 导致细胞死亡并启动ETI; 而在感染组织周围, 较低水平的SA会抑制NPR4与NPR1相互作用, 但是不足以促进NPR3与NPR1相互作用, 此时NPR1蛋白大量积累, 促进SA介导的抗性反应和细胞存活(Fu等2012)。由此可见, SA的感受可以调控NPR3和NPR4介导的NPR1降解, 使其维持在合适的水平从而决定植物免疫反应的进程。

5 展望

SA在植物免疫反应中具有重要作用, 过去20年科学家对SA在抗病中的作用进行了大量研究。SA诱导抗性的机制对农作物保护具有重要意义。虽然现代生化和遗传学方法的结合有利于研究植物中SA的合成、激活、SA受体以及SA在生物和非生物胁迫中的作用, 但是现有的对SA的研究还远远不够, 很多问题有待解决: (1)植物中SA生物合成途径一般分为两种, 但其途径并不十分清楚, 比如植物中IC转变成SA到底是通过哪些酶来催化的? SA合成的ICS1途径是如何调控的? 参与PAL途径合成SA相关的酶又有哪些? PAL合成途径的时间节点并不清楚; 在拟南芥中, PAL途径在生物和非生物胁迫下诱导叶片SA合成的重要性并不明显, 但这并不能排除PAL途径合成SA的重要性(Dempsey等2011); (2)至今为止分离出的SABPs虽然并不是真正意义上的SA受体, 但他们在SA应答反应中的重要性有待研究; (3) NPR1作为重要的调节因子对SA信号传导途径具有重要意义, NPR3和NPR4是SA信号传导途径中的受体, 这一发现是SA信号传导研究中的一大进步。NPR3和NPR4能与SA结合, 但是NPR1是否直接与SA结合还有待于进一步研究; (4) SA与NPR3和NPR4的结合调控NPR1的含量, 进而调控SA介导的抗性反应和SAR的建立, 而为什么NPR3和NPR4与SA结合能力不同; (5) SA参与植物非生物胁迫响应过程, 但是SA介导植物非生物胁迫应答的生化机制仍有待研究。

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Recent advance of salicylic acid signaling in plant disease resistance

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Abstract: Salicylic acid (SA) plays a central role in plant innate immunity. Endogenous SA level increases drastically shortly after plants detect the attack of biotrophic pathogens, and plays an essential role in activating plant defenses. Hence it has also been accepted as a defense hormone in plants. Studies have revealed multiple SA-interacting/binding proteins, among which NPR1, together with NPR3 and NPR4 plays a master role in sense and conducting the SA-mediated signals during the establishment of plant defense responses. This review focuses on the role of SA in plant disease resistance, and gives an outline of how SA is synthesized and regulated, how it is perceived by SA interaction/binding proteins involving the actions of the NPR1, 3 and 4 as well as the subsequent signal transduction. Perspective on future research is also suggested on this topic.

Key words: salicylic acid (SA); signaling; salicylic acid-binding-proteins (SABPs); NPR1; NPR3; NPR4

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