植物病原菌效应蛋白与茉莉素信号途径互作的研究进展

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摘要: 茉莉素信号途径是植物抗病信号途径的重要组成部分, 病原菌的致病过程与茉莉素信号途径密切相关。近几年来, 植物病原菌效应蛋白在侵染植物过程中与茉莉素信号途径关键组分的互作研究取得了深入快速的发展。本文分别就植物细菌病原菌、真菌病原菌和卵菌病原菌在致病过程中与寄主茉莉素信号途径相互作用的最新研究进展加以概述, 以期对相关领域的研究起到一定的启迪作用。

关键词: 茉莉素信号途径; 病原菌; 效应蛋白; 互作

长久以来, 植物病原菌与寄主植物的互作一直是植物病理学研究的热点内容。茉莉素信号转导途径是植物免疫系统的重要组成部分, 在植物抵抗病原菌侵染过程中发挥重要作用。茉莉素类物质(jasmonates, JAs)包括茉莉酸及其挥发性衍生物茉莉酸甲酯(methyl jasmonate, MeJA)和氨基酸衍生物。茉莉素信号途径在植物生长发育如种子萌发、花粉发育、衰老等过程中发挥重要作用(Devoto等2002; Wang等2014), 此外也在植物的生物和非生物胁迫如干旱、高温、病虫害等抗性过程中起重要的调控作用(McConn等1997; Shan等2009; Song等2011)。近几年来, 茉莉素信号途径在植物抵抗病原菌侵染中的作用机制研究有深入和快速的发展, 本文将对该领域最新的发展现状进行综述。

1 茉莉素的生物合成

茉莉素是由亚麻酸经脂氧合酶途径合成的产物, 主要在叶绿体细胞器中累积, 在细胞质中很少。细胞膜在脂酶的催化下释放α-亚麻酸(α-linolenic acid, ALA), ALA在叶绿体的13-脂氧合酶(13-lipoxygenase, LOX)的作用下生成(13*S*)-氢过氧亚麻酸[(13*S*)-hydroperoxyoctadecatrienoic acid, 13-HPOT], 接着丙二烯氧化物合成酶(allene oxide synthase, AOS)和丙二烯氧化物环化酶(allene oxide cyclase, AOC)催化13-HPOT生成12-氧-植物二烯酸(12-oxo-phytodienoic acid, 12-OPDA), 以上3步都在叶绿体中进行。生成的12-OPDA再经12-氧-植物二烯酸还原酶 (12-oxo-phytodienoic acid reductase, OPR)作用和3次β氧化反应最后形成茉莉酸及其衍生物, 如MeJA (Cheong和Choi 2009)。在茉莉素生物合成过程中涉及的LOX、AOS、AOC等酶是茉莉素合成的关键酶。茉莉素合成之后与膜上的受体相结合, 会激活植物体内与防御相关的一系列基因得到表达, 从而使植物产生各种抗性。

2 茉莉素信号转导途径

茉莉素的受体是一个多成分组成的复合体, 具体由CORONATINE INSENSITIVE 1 (COI1)蛋白、茉莉素途径负调控因子jasmonate ZIM-domain (JAZ)蛋白和肌醇戊基磷酸盐(inositol pentakisphosphate)组成(Yan等2009; Sheard等2010)。在茉莉素信号传导途径中, 当受到伤害和病原菌侵染时, 有生物活性的茉莉酸衍生物jasmonoyl–isoleucine (JA–Ile)与茉莉素受体结合, 导致茉莉素途径负调控因子JAZ蛋白由26S蛋白酶体(即蛋白泛素化降解途径)降解, 并释放出茉莉素途径转录激活因子MYC2, 从而激活茉莉素信号途径下游与植物生长发育和抗逆相关的功能基因表达和生理变化(Chini等2007; Thines等2007; Zhang等2012; Chung和Howe 2009)。

COI1蛋白是茉莉素信号转导途径中的关键调控因子, 该蛋白的缺失突变能够导致植物丧失所有重要的茉莉素反应(Xie等1998)。2007年, Thines等(2007)和Chini等(2007)分别发现含有12个成员的JAZ蛋白家族是茉莉素信号途径中的一类抑制蛋白。JAZ蛋白是Skp1–Cul1–F-box (SCF)泛素连接酶复合体的一类直接底物(陈娟等2014)。茉莉素信号分子可以诱导SCF^{COI1}复合体结合JAZ蛋白,

收稿 2016-01-19 **修定** 2016-05-15

资助 中国烟草总公司科技重大专项项目[110201301009 (JY-09)]、河南省烟草公司科技项目(HNKJ200814)和中国烟草总公司重庆市公司项目(NY20140403030022)。

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使其泛素化并通过26S蛋白酶体降解, 最终解除JAZ蛋白对茉莉素信号通路的抑制作用, 引起下游茉莉素反应。JAZ蛋白含有中央的27个氨基酸组成的ZIM结构域和C末端Jas结构域。ZIM结构域同时也介导JAZ蛋白与其共抑制因子NINJA的相互作用, 从而共同抑制茉莉素信号通路。Jas结构域介导JAZ蛋白对茉莉素信号通路正调控转录因子MYC2的直接抑制作用(Thines等2007; Chini等2007)。

MYC2转录因子是从茉莉素不敏感突变体*jai1*/*jin1*分离到的茉莉素途径调控因子, 属于basic helix–loop–helix (bHLH)转录因子家族成员(Lorenzo等2004)。前期对MYC2转录因子的大量研究证明, MYC2是植物茉莉素应答调控因子, 在茉莉素途径调控植物对病害和虫害防御应答中发挥重要作用, 并与乙烯信号传导途径的ethylene response factor (ERF)转录因子拮抗性地调控下游虫害和病害防御基因的表达(Boter等2004; Lorenzo等2004)。JAV1是新近克隆的茉莉素信号途径的一个负反馈调节因子, 该蛋白可以通过茉莉素途径调控植物的抗性反应。当植物被害虫伤害或感染病原菌时, 植物会积累大量的茉莉素, 从而引发26S蛋白酶体通过降解JAV1蛋白激活相关抗性基因的表达来提升植物的抗性反应(Hu等2013)。

3 茉莉素信号途径在植物抗病途径中的作用

植物在长期进化过程中形成了一套严密的抵抗各类病原菌侵染的免疫系统。植物的免疫系统可以分为两个层次, 第一层次为pathogen-associated molecular patterns (PAMPs)激发的免疫反应(PAMPs-triggered immunity, PTI), 这是一种普遍存在但强度较弱的基础抗性, 可以抵抗多数病原菌的侵染; 第二层次是为了抵御寄主PTI, 病菌进一步分泌特异性的效应蛋白分子(effector), 而植物则进化出专一的抗病(resistance, R)蛋白直接或间接识别病原菌的效应蛋白, 从而产生效应蛋白激活的免疫反应(effector-triggered immunity, ETI), 表现为寄主植物在病菌侵染部位的少数细胞迅速死亡, 从而限制病菌扩散并进一步激发周围及整株植物细胞对病菌的抗性(Chisholm等2006; Jones和Dangl 2006; Dou和Zhou 2012)。病原菌效应基因(effector gene)与抗病基因之间的互作符合基因对基因假说(Chisholm等2006; Kazan和Lyons 2014)。茉莉素信号途径是植物体内重要的抗病信号途径, 近年来关于植物病原菌效应蛋白与茉莉素信号途径互作的研究取得了较大发展。

3.1 茉莉素信号途径与植物病原细菌的互作

植物病原细菌通过III型分泌系统(type III secretion system, T3SS)分泌III型效应分子进入寄主植物细胞抑制植物的免疫反应从而促进病原菌的侵染。植物病原细菌在效应蛋白的研究方面开展较早, 研究较深入, 取得了较大发展(Xin和He 2013)。番茄(*Solanum lycopersicum*)细菌性叶斑病丁香假单胞菌(*Pseudomonas syringae* pv. *tomato*, *Pst*) DC3000分泌一种命名为HopZ1a的半胱氨酰蛋白酶, 该效应蛋白与茉莉酸信号途径的关键因子JAZ蛋白互作, 从而影响植物对病原菌的抗性(Jiang等2013)。此外, 该菌株在侵染植物后分泌一种名为冠菌素(coronatine, COR)的毒素, COR可以刺激植物叶片气孔张开从而有利于细菌的进入(Melotto等2006, 2008)。同时COR也是一种茉莉素类似物, 它可以通过抑制水杨酸(salicylic acid, SA)介导的抗性来增强菌体在植物体内的繁殖能力, 该抑制过程则通过激活茉莉素信号途径来实现。茉莉素信号途径和水杨酸信号途径在调控植物抗性的机制中存在一种拮抗关系(Katsir等2008; Sheard等2010)。Cui等(2010)研究表明, *Pst*分泌的效应蛋白AvrB能够和MPK4、RAR1以及HSP90在植物体内形成一个复合物并激活MPK4, 然后MPK4直接与RIN4相互作用来促进茉莉素下游基因的表达。最新研究表明, AvrB可以通过另一种机制来影响植物抗性, 即AvrB-RIN4-AHA1, 该机制与茉莉素信号途径密切相关(Zhou等2015)。烟草野火病病原细菌(*Pseudomonas syringae* pv. *tabaci*) *Pta*11528分泌一种半胱氨酸蛋白酶效应因子, 命名为HopX1, 该效应蛋白与JAZ蛋白互作促进JAZ蛋白的降解, 使得茉莉酸信号途径抑制了由水杨酸介导的抗性反应, 从而使烟草对*Pta*11528表现感病(Gimenez-Ibanez等2014)。

3.2 茉莉素信号途径与植物病原真菌的互作

茉莉素信号途径在植物防御腐生性真菌芸薹链格孢菌(*Alternaria brassicicola*)、灰霉菌(*Botrytis cinerea*)、黄瓜织球壳菌(*Plectosphaerella cucumerina*)

和尖刀镰孢菌(*Fusarium oxysporum*)中发挥作用(Yan和Xie 2015)。如果茉莉素合成或信号转导途径受阻, 内源茉莉素不能积累, 将导致抗性基因无法转录表达, 抑制植物的应答反应。茉莉素信号转导突变体*coi1*表现为对这5种病原菌更强的感病性(Thatcher等2009)。拟南芥茉莉素信号合成突变体*jar1*、*aos*和*jin1*均容易感染灰霉菌等病原菌, 甚至致死(Thaler等2004)。番茄*jai1*突变体(拟南芥突变体*coi1*的同源突变体)表现为对灰霉菌和尖刀镰孢菌更强的感病性(Stintzi和Browse 2000)。茉莉素合成突变体*fad3*、*fad7*和*fad8*相对于野生型对照对尖刀镰孢菌更感病(Stintzi等2001)。真菌尖刀镰孢菌同样阻碍茉莉素信号转导途径使得植物更加感病, 表现为突变体*coi1*、*myc2*、*pft1*和*med25*表现得更为抗病(Kidd等2009; Thatcher等2009)。最新研究结果显示, 双色蜡蘑(*Laccaria bicolor*)分泌的效应蛋白MiSSP7可以与JAZ6蛋白互作来抑制茉莉素信号途径的反应(Plett等2014)。

核盘菌(*Sclerotinia sclerotiorum*)分泌类似效应分子SSITL (SS1G-14133), 在侵染早期阶段可以抑制茉莉素和乙烯(ethylene, ET)信号途径从而影响寄主抗性(Zhu等2013)。灰霉病菌(*Botrytis cinerea*)分泌一种表多糖(exopolysaccharide), 该种物质可以激发水杨酸信号途径, 由于水杨酸与茉莉素之间的拮抗关系, 使茉莉素信号途径受到抑制(El Oirdi等2011)。稻瘟病菌(*Magnaporthe oryzae*)体内的antibiotic biosynthesis monooxygenase (Abm)可以将茉莉素转化为茉莉素的羟基化物, 即12OH-JA, 而12OH-JA可以降低植物的抗病性。在稻瘟病菌侵染过程中, 分泌12OH-JA到寄主体内使寄主的抗性降低。而且, Abm在侵染中被直接分泌到寄主体内, 使寄主体内的自由茉莉素转化为12OH-JA, 从而促进病菌繁殖(Patkar等2015)。

3.3 茉莉素信号途径与植物病原卵菌的互作

卵菌与真菌在形态上类似但在进化关系上较远。引起马铃薯(*Solanum tuberosum*)晚疫病的致病疫霉菌(*Phytophthora infestans*)、侵染橡胶树(*Hevea brasiliensis*)的橡树疫霉菌(*Phytophthora ramorum*)、引起大豆(*Glycine max*)根腐病的大豆疫霉菌(*Phytophthora sojae*)和引起烟草(*Nicotiana*)黑胫病的寄生疫霉菌(*Phytophthora parasitica*)都属于卵菌门疫霉属。因为疫霉菌对农林作物的毁灭性危害, 一直以来都是研究的重点。随着卵菌许多病原菌全基因组测序工作的完成, 卵菌的分子遗传学研究进入一个快速发展时期(Tyler 2007)。作为卵菌生物学和病理学研究的模式种, 大豆疫霉菌的研究取得很大进步(Tyler 2007)。大豆疫霉菌的几个无毒基因已经得到克隆, 具体有*Avr1a* (Qutob等2009)、*Avr1b* (Shan等2004)、*Avr3a* (Dong等2011a; Qutob等2009)、*Avr3b* (Dong等2011b)、*Avr3c* (Dong等2009)、*Avr4/6* (Dou等2010)和*Avr5* (Dong等2011a)。此外, 大豆疫霉菌在效应蛋白鉴定和功能研究方面以及效应蛋白与寄主细胞组分的互作等方面都有深入的研究(Wang等2011; Bos等2006; Dou等2008a, b; Shan等2004; Rajput等2014; Song等2013; Yu等2012; Dong等2009, 2014)。

研究证明, 水杨酸、茉莉酸、乙烯3种植物激素是诱导防御信号途径的主要信号分子, 这些信号途径相互间存在协同作用或拮抗作用, 并以此为基础形成一套相当复杂的免疫网络来精确调控植物对病虫害等胁迫的防御反应(Laurie-Berry等2006)。Liu等(2014)报道, 从大豆疫霉菌和棉花黄萎菌(*Verticillium dahliae*)中鉴定了一组功能保守的效应因子, 发现他们在寄主细胞内可以降解植物水杨酸合成的前体, 从而抑制其积累, 降低植物的防卫反应, 促进病原菌的侵染。该研究组还发现, 在烟草上过表达大豆疫霉效应因子PsCRN115后, 可以引起水杨酸通路基因的高量表达, 提高烟草抗性, 从而限制烟草疫霉菌的侵染(刘廷利等2012)。

Mediator complex首先发现于酵母中, 是一个RNA聚合酶II的辅助因子, 在转录机制中起关键作用, 它是一个多蛋白的功能保守的复合体, 大约含有个20~30个亚基, 其中MED19a、MED21、MED25、MED16等9个亚基与植物对生物逆境的抗性密切相关(Conaway和Conaway 2011)。霜霉病菌(*Hyaloperonospora arabidopsidis, Hpa*)分泌的效应因子HaRxL44能与MED19a结合, 导致MED19a通过蛋白酶体途径降解, 改变信号途径防卫机制的平衡, 使水杨酸相关基因表达降低, 茉莉素/乙烯信号途径得到加强(Caillaud等2013)。图1为植物病原菌分泌效应因子和毒素与茉莉素信号途径互作的示意图。

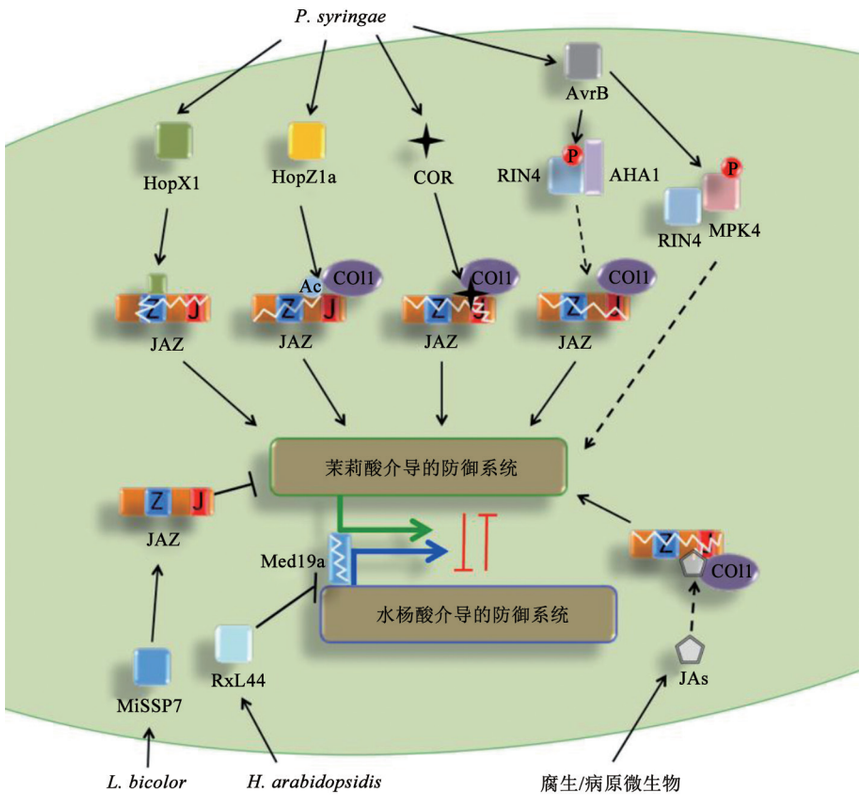

图1 植物毒素及病原菌效应蛋白与茉莉素信号途径互作的分子机制

Fig.1 Molecular mechanisms for phytotoxins and microbial effectors targeting the JA signaling components

MPK4: 促分裂原活化蛋白激酶4; AHA1: 编码细胞质膜H^+-ATPase的基因; 根据Gimenez-Ibanez等(2016)略作修改。

4 展望

Mukhtar等(2011)在*Science*发表研究结果称, 利用高通量的Y2H技术系统构建了拟南芥蛋白和来自细菌*Pst*和卵菌霜霉菌(*Hpa*)的效应蛋白之间的跨物种蛋白质互作网络, 他们称之为植物–病原体免疫网络(plant–pathogen immune network, PPIN)。该网络包含1 358个互作反应, 共有926个蛋白参与其中。研究结果表明这两种病原菌的效应蛋白与茉莉素途径关键因子JAZ蛋白有很强的互作作用(Mukhtar等2011)。Weßling等(2014)在此工作基础上, 分析了真菌白粉菌(*Golovinomyces orontii*)分泌的效应蛋白与拟南芥蛋白的互作关系, 表明白粉菌分泌的效应蛋白与茉莉素和水杨酸信号途径有较强的相互作用。由此, 可以预见, 在病原菌和茉莉素信号途径的互作机制研究方面有大量的工作需要去开展。

综上所述, 植物病原菌(包括细菌、真菌和卵菌)在致病过程中, 其效应蛋白与茉莉素信号途径有很强的相互作用, 这也证明了茉莉素信号途径在植物免疫系统中的重要作用。但是, 到目前为止, 病原菌效应蛋白与茉莉素信号途径主要组分的互作研究还比较有限, 在全基因组水平上分析病原菌侵入后茉莉素信号途径主要基因的表达变化还未见报道。因此, 以各病原菌的模式菌种为研究材料, 广泛深入地鉴定病原菌效应蛋白互作的寄主标靶蛋白, 以及在全基因组水平上分析抗性相关基因的表达水平变化仍将是该领域未来研究的重点内容。

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Advances in the interaction between pathogen effectors and jasmonate signaling pathway

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Abstract: Jasmonate (JA) plays a central role in plant disease resistance, and the process of pathogens infection is closely correlated with JA signaling pathway. In recent yeas, intensive studies have been conducted to investigate the interaction between pathogen effectors and the key components of JA signaling pathway during the microbial pathogenesis. In this review paper, the latest advances in the molecular interactions between the pathogens of bacteria, fungus and oomycete and the components of JA signaling pathway were summarized.

Key words: jasmonate signaling pathway; pathogens; effectors; interaction

Received 2016-01-19 Accepted 2016-05-15

This work was supported by the Key Special Program of China National Tobacco Corporation [Grant No. 110201301009 (JY-09)], the Science and Technology Project of Henan Tobacco Company (Grant No. HNKJ200814), and the Program of Chongqing Tobacco Company (Grant No. NY20140403030022).

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