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Hans汉斯

病原微生物－植物宿主互作中茉莉酸的多重功能

张明磊，何亚文

上海交通大学生命科学技术学院，微生物代谢国家重点实验室，教育部代谢与发育科学国际合作联合实验室，上海

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摘　要

茉莉酸是一种重要的植物激素，在植物生长发育和防御响应过程中发挥重要的调节作用。最新研究表明，在病原微生物－植物宿主互作过程中，病原微生物的侵染可以激活植物宿主体内由茉莉酸介导的防御响应。同时病原微生物可以感应茉莉酸信号，通过多种机制操纵植物宿主茉莉酸信号传导途径，增强自身侵染能力，提高致病性。本文首先简述茉莉酸的生物合成途径与信号传导机制，随后介绍茉莉酸在病原细菌、真菌及病毒与植物宿主相互作用过程中的多重作用。这些研究结果有助于系统深入理解病原微生物与植物宿主互作中茉莉酸的多重功能。

关键词

茉莉酸，病原微生物，相互作用，防御响应

The Multiple Functions of Jasmonic Acid in the Interaction of Pathogenic Microorganisms and Plant Hosts

Minglei Zhang, Ya-Wen He

Joint International Research Laboratory of Metabolic and Developmental Sciences, State Key Laboratory of Microbial Metabolism, School of Life Sciences and Biotechnology, Shanghai Jiao Tong University (SJTU), Shanghai

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Abstract

Jasmonic acid is an important plant hormone that plays a crucial regulatory role in plant growth, development, and defence responses. Latest results indicate that the invasion of pathogenic microorganisms can activate the jasmonic acid defense mechanism of plant hosts. At the same time, pathogenic microorganisms can sense jasmonic acid and manipulate the jasmonic acid signal transduction pathways of plant hosts, thus enhancing their own pathogenicity. In this review, we first briefly describe the biosynthesis and signalling pathways of jasmonic acid, followed by an overview of the research progress on the multiple functions of jasmonic acid in the interaction of various pathogenic bacteria, fungi, and viruses with plant hosts. These findings will provide insights in understanding the roles of jasmonic acid in the interaction between pathogenic microorganisms and plant hosts.

Keywords

Jasmonic Acid (JA), Pathogenic Microorganism, Interaction, Defense Mechanism

1. 引言

茉莉酸(jasmonic acid, JA)是一类植物内源性的脂质类激素，参与植物生长发育调节与生物胁迫应对。JA 的发现可以追溯到 1962 年 Demole 等从植物香精油中发现了具有芳香味的茉莉酸甲酯(methyl-jasmonate acid, MeJA) [1]。自 20 世纪 80 年代起，JA 的生理功能和合成机制被逐渐揭示，随后的一系列研究发现 JA 在植物应对生物胁迫方面发挥着关键作用，被誉为植物应对病原体入侵的“瑞士军刀”[2]-[4]。本文首先简述 JA 的生物合成途径与信号传导机制，随后重点综述病原微生物－植物宿主相互作用过程中 JA 所发挥的多重功能。

2. JA 在植物与微生物中的生物合成途径与机制

2.1. 植物体内的十八烷和十六烷生物合成途径

JA 的生物合成途径在模式植物拟南芥中研究得相对成熟，包括从 α-亚麻酸(α-linolenic acid, ALA; 18:3)开始的十八烷途径和从十六碳三烯酸(16:3)开始的十六烷途径(图 1)，其中前者是更主要的合成途径[5]。两条途径均需要途经叶绿体、过氧化物酶体和细胞质基质三个反应区室。叶绿体是 JA 合成的第一个反应场所，在十八烷途径中这一阶段由 α-亚麻酸－甘油酯(α-linoleic acid glyceride)经磷脂酶(phospholipase, PLA)水解释放出 ALA 开始，随后 ALA 经脂氧合酶(lipoxygenase, LOX)、丙二烯氧化合酶(allene oxide synthase, AOS)、丙二烯氧化物环化酶(allene oxide cyclase, AOC)三个酶逐步催化的保守反应步骤生成 12-OPDA。12-OPDA 在叶绿体包膜定位转运蛋白 JASSY 和过氧化物酶体 ABC-转运蛋白 1 的作用下移位到过氧化物酶体。在过氧化物酶体中，12-OPDA 被 OPDA 还原酶(OPDA reductase, OPR)还原为 OPC-8:0，随后在羧基辅酶 A 连接酶(acyl-CoA synthetase, ACS)催化下合成 OPC-8:0-CoA。OPC-8:0-CoA 会历经 3 次重复的 β-氧化最终合成 JA，这一过程由 β-羟脂酰辅酶 A 脱氢酶(3-ketoacyl-CoA thiolase, KAT)、烯脂酰辅酶 A 水合酶(multifunctional protein, MFP)、酰基辅酶 A 氧化酶(acyl-CoA oxidase, ACX)共同催化完成。

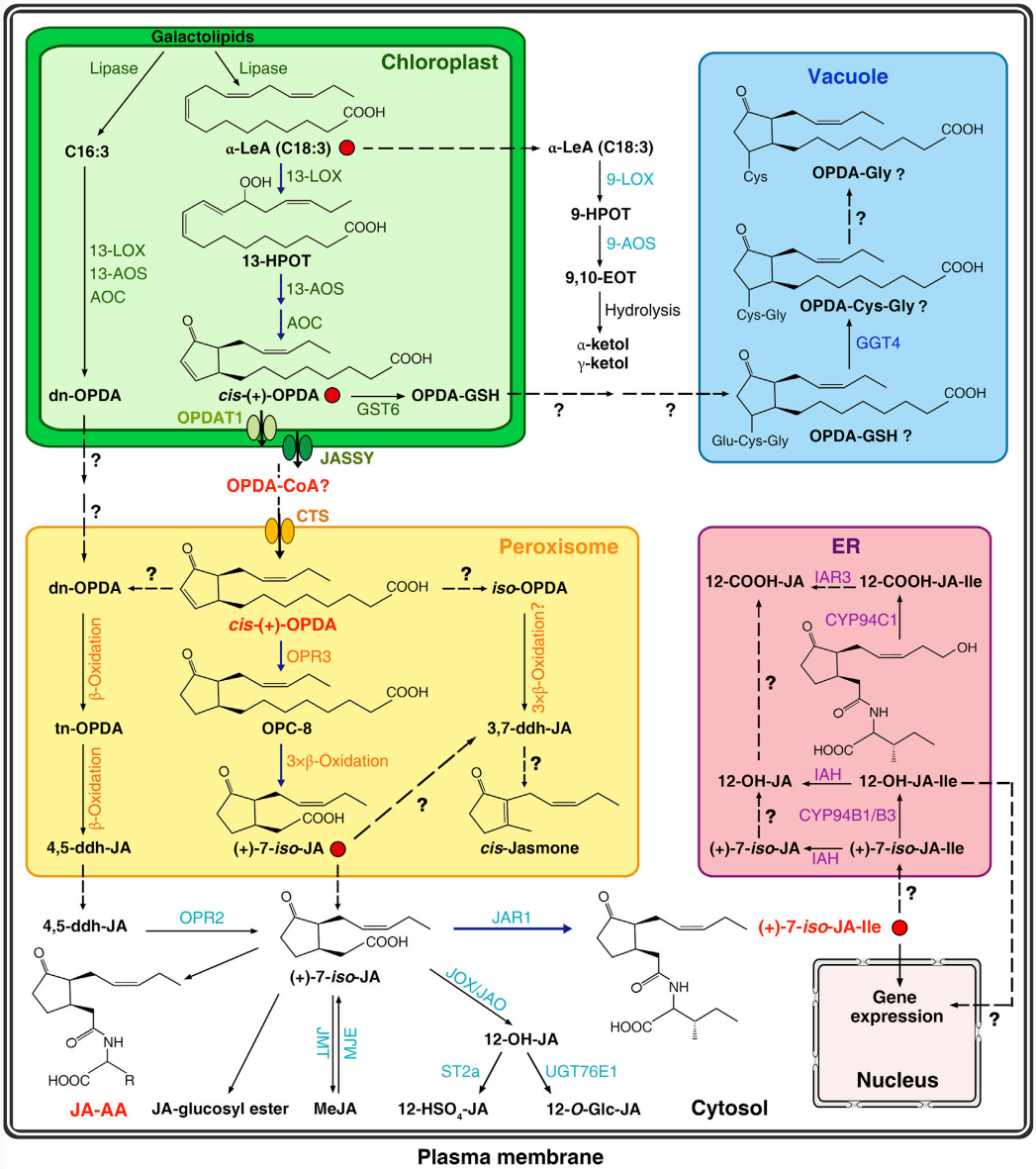

Figure 1. The biosynthesis of jasmonic acid [13]. The two synthetic pathways of JA are the octadecane pathway starting from ALA (18:3) and the hexadecane pathway starting from hexadecyltriaenoic acid (16:3). In the octadecane pathway, chloroplasts are the first reaction place for JA synthesis. In this stage, *α*-linoleic acid glyceride is hydrolyzed by PLA to release ALA, which is then subjected to a classical core reaction consisting of three enzymes: LOX, AOS, and AOC to generate 12-OPDA. 12-OPDA is translocated to the peroxisome by the action of chloroplast envelope localization transporter JASSY and peroxisome ABC transporter 1. In peroxisomes, 12-OPDA is reduced to OPC-8:0 by OPR, and then OPC-8:0-CoA is synthesized under the catalysis of ACS. OPC-8:0-CoA undergoes three repeated *β*-oxidation processes to ultimately synthesize JA, which is catalyzed by KAT, MFP, and ACX. In the hexadecane pathway, the reaction product of the chloroplast stage is dnOPDA, which can be catalyzed by OPR3 in peroxisomes to generate OPC-6:0 and join the octadecane pathway. In the cytoplasmic matrix, JA can be catalyzed by a member of the JAR to produce JA-Ile or by JMT to produce MeJA

图 1. 茉莉酸的生物合成[13]。JA 的两条合成途径分别为从 ALA (18:3)开始的十八烷途径和从十六碳三烯酸(16:3)开始的十六烷途径。在十八烷途径中第一步是在叶绿体中，*α*-亚麻酸–甘油酯经 PLA 水解释放 ALA，随后经 LOX、AOS、AOC 三个酶逐步催化生成 12-OPDA。12-OPDA 在叶绿体包膜定位转运蛋白 JASSY 和过氧化物酶体 ABC-转运蛋白 1 的作用下移位到过氧化物酶体。在过氧化物酶体中，12-OPDA 被 OPR 还原为 OPC-8:0，随后在 ACS 催化下合成 OPC-8:0-CoA。OPC-8:0-CoA 会历经 3 次 *β*-氧化最终合成 JA，这一过程由 KAT、MFP、ACX 共同催化完成。在十六烷途径中，叶绿体阶段的反应产物为 dnOPDA，在过氧化物酶体中可以由 OPR3 催化生成 OPC-6:0 汇入十八烷途径。在细胞质基质中，JA 可以被 JAR 催化生成 JA-Ile 或经 JMT 催化生成 MeJA 等衍生物形式

在十六烷途径中，叶绿体阶段的反应产物为脱氧甲基植物二烯酸(deoxymethylated phytodienoic acid, dnOPDA)，在过氧化物酶体中可以由 OPR3 催化生成 OPC-6:0 汇入十八烷途径。近些年来，非 OPR3 依赖性途径也被报道，即 dnOPDA 在过氧化物酶体中反应生成 4,5-ddh-JA，随后经 OPR2 催化生成 JA [6]。在细胞质基质中，JA 可以被加以多种修饰，最常见的为经荧光素酶超家族成员(jasmonic acid-amido synthetase, JAR)催化生成茉莉酸–异亮氨酸(jasmonic acid-isoleucine, JA-Ile)和经甲基化酶(jasmonic acid carboxyl methyltransferase, JMT)催化生成 MeJA，这两种形式是植物体内常见的活性形式。

2.2. 病原微生物中的 JA 合成与人工异源合成

研究显示，部分病原微生物也具备合成 JA 的能力。例如，在尖孢镰刀菌(*Fusarium oxysporum* f. sp. *matthiolae*)的培养滤液中检测到 22 种 JA 及 JA 相关化合物，其中主要是 JA 和 JA-Ile [7]。龙眼焦腐病菌(*Lasiodiplodia theobromae*)的培养物中同样可以提取出 JA、OPDA 等物质，Nabeta 等推测其内部具有 LOX、AOS、AOC 等酶类似活性的蛋白质，JA 的合成是来自脂肪酸合成途径的衍生反应[8]。在红蜡蘑(*Laccaria laccata*)和豆包菌(*Pisolithus tinctorius*)等物种中也可以观察到 JA 的生物合成和代谢[9]。侵染拟南芥的两种尖孢镰刀菌 *F. oxysporum* f. sp. *matthioli* (Fomt)和 *F. oxysporum* f. sp. *conglutinanas* (Focn)同样可以合成 JA、JA-Ile 和 JA-Leu [10]。有趣的是，JA 的合成能力仅在与植物存在相互作用的真菌中报道[11]。

此外，中国科学院深圳先进技术研究院罗小舟团队与美国加州大学伯克利分校 Keasling 团队利用合成生物学的思路，在酿酒酵母(*Saccharomyces cerevisiae*)中构建出第一条 JA 异源生物合成路线[12]。通过 LOX、AOS 等 15 种代谢酶类的引入、信号肽介导的代谢酶区室化分布设计和全局化代谢改造，工程化的酿酒酵母可以在葡萄糖为碳源的培养条件下完成滴度为 19.0 mg/L 的 JA 发酵[12]，这为 JA 的产业化应用提供了前提。

3. 植物体内 JA 信号传导途径

JA 的信号传导途径对于 JA 在病原微生物–植物宿主相互作用过程中的多重功能具有重要意义，是病原微生物与植物宿主“JA 攻防战”的焦点。然而，目前有关病原微生物 JA 应激响应通路的研究报道少之又少，因此，我们先阐述植物宿主 JA 信号传导途径的关键部分(图 2)。在植物宿主中，JA 信号传导途径的重要组分包括冠菌素不敏感蛋白 1 (coronatine insensitive 1, COI1)蛋白[14]、COI1 型 SCF 泛素连接酶复合体(the SCF^{COI1} ubiquitin E3 ligase, SCF^{COI1})复合体[15]、茉莉酮酸酯 ZIM 结构域蛋白(jasmonate ZIM-domain proteins, JAZ) [16]、髓细胞组织增生蛋白类转录因子(myelocytomatosis proteins, MYC) [17]等，其功能通路受到胞内外多种信号的精确调控[18] [19]。在拟南芥中，JA-lle 是 JA 的主要活性形式，当缺乏外界信号时，JA-Ile 的含量处于较低水平，JAZ 的含量积累并与 MYC 结合抑制 MYC 的功能。当存在外界信号刺激时，COI1 识别 JA-Ile 并与之组成的复合物与 JAZ 结合，随后通过泛素-26S 蛋白酶体途径降解 JAZ，被释放后的 MYC 与中介复合物(mediator 25, MED25)发挥互作进而驱动 JA 响应基因的表达[20]-[22]。

在 JA 信号传导过程中，JA 负调节因子 JAZ 被认为发挥核心作用[3]。除前述功能外，JAZ 还可通过 JAZ 接头蛋白(novel interactor of JAZ, NINJA)招募转录共抑制子(topless, TPL)来抑制 MYC 的转录和 JA 响应基因的表达。该过程中，乙酰转移酶(general control non-derepressible 5, GCN5)和去乙酰化酶(histone deacetylase 6, HDA6)充当了调控 JA 基因表达的分子开关：静息状态下，GCN5 对 TPL 进行乙酰化修饰，增强 TPL 与 NINJA 互作，MYC 的活性被抑制，JA 响应基因表达关闭；当植物体内 JA 积累时，HDA6 对 TPL 进行去乙酰化修饰，削弱 TPL 与 NINJA 互作，促进 TPL 与 MYC 解离，释放 JA 响应基因的表达[23]。

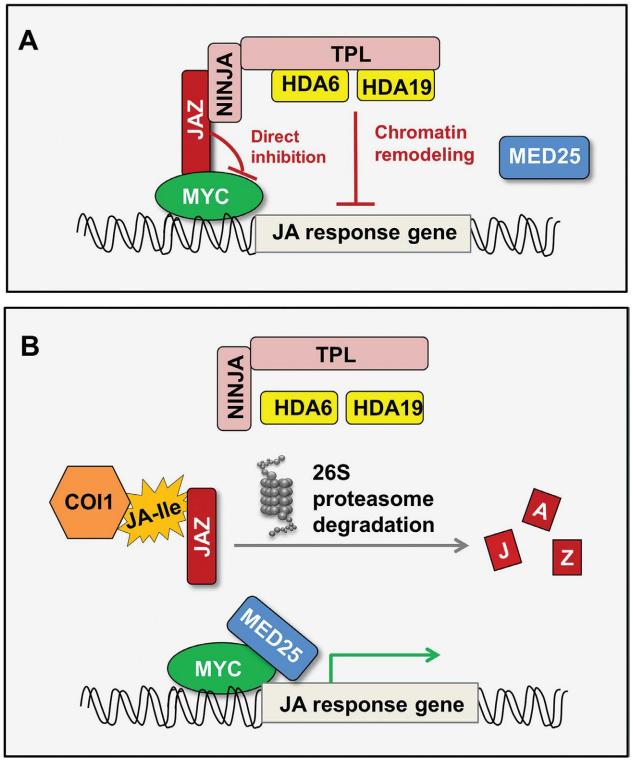

Figure 2. The signalling pathway of jasmonic acid [3]. When there is a lack of external signals, the content of JA-Ile is at a low level, and JAZ binds to MYC to inhibit its function. When there is external signal stimulation, the content of JA-Ile increases and is recognized by COI1. The complex composed of JA-Ile and COI1 binds to JAZ, which is then degraded through the ubiquitin-26S proteasome pathway. The released MYC interacts with MED25 complex, driving the expression of JA responsive genes

图 2. 茉莉酸的信号传导[3]。当缺乏外界信号时，JA-Ile 的含量处于较低水平，JAZ 与 MYC 结合抑制 MYC 的功能。当存在外界信号刺激时，JA-Ile 的含量提高并被 COI1 识别，JA-Ile 和 COI1 组成的复合物与 JAZ 结合，随后通过泛素-26S 蛋白酶体途径降解 JAZ，被释放后的 MYC 与 MED25 互作，进而驱动 JA 响应基因的表达

4. JA 在植物宿主抵抗病原微生物侵染过程中的双重作用

4.1. 病原微生物侵染激活或抑制植物宿主 JA 防御响应

病原细菌、真菌及病毒的侵染可以激活植物宿主的 JA 防御响应，进而激活免疫反应。水稻黄单胞菌(*Xanthomonas oryzae* pv. *oryzae*, Xoo)侵染过程中会抑制水稻中 JA 合成相关基因 OsAOS1 的表达，降低内源 JA 的含量，从而降低水稻抗性[24]。野油菜黄单胞菌(*Xanthomonas campestris* pv. *campestris*, Xcc)在侵染十字花科植物过程中则会引起 JA 含量和 JA 合成及响应基因表达量的显著上调[25]。值得注意的是，黄单胞菌典型的群体感应信号分子 DSF 的低浓度(0.1~10 μM)处理可以激活植物 JA 防御响应，使得抗氧化酶(peroxidase, POD)的活性增加和对 Xcc 的抵抗力提高，这为理解 JA 在植物病原细菌互作中的作用和防治十字花科植物黑腐病带来了新思路[25]。在小麦(*Triticum aestivum*)中，正常条件下 JAZ1、NINJA 和 TPL 形成复合物抑制 MYC4 的转录活性。而当 JA 响应白粉病菌(*Blumeria graminis* f. sp. *tritici*, Bgt)的侵染而积累时，TaJAZ2 降解以释放 TaMYC4 和其他转录因子，导致活性氧(reactive oxygen species, ROS)积累和发病机制相关基因(pathogenesis-related genes, PRs)的表达被抑制，从而增强植物对 Bgt 的抗性[26]。

水稻条纹病毒(rice stripe virus, RSV)的侵染可以提高宿主 JA 水平，JAZ6 降解释放出的茉莉酸响应转录因子 JAMYB 与 Argonaute 18 (AGO18)启动子结合，增强水稻的抗病毒响应机制[27]。然而，也有研究表明部分病毒感染抑制了 JA 介导的防御响应，增强了植物对病毒的敏感性，如水稻齿叶矮缩病毒(rice ragged stunt virus, RRSV)通过积累 miR319 抑制 JA 介导的防御，从而促进水稻病毒感染症状的发展[28]。

4.2. 植物宿主通过 JA 防御响应抵抗病原微生物侵染

JA 在多种植物对病原微生物的防御响应中具有不可忽视的重要作用。病原细菌的防御机制中 JA 的作用是不可或缺的。Brenya 等证明提前将拟南芥幼苗暴露在机械胁迫下，可增强 JA 介导的对坏死性病原菌的抗性[29]。在水稻中，介导组蛋白修饰抑制的 LHP1 蛋白通过抑制 NAC 转录因子家族及其靶标 BA/SA 羧基甲基转移酶 1 (brassinosteroids/salicylic acid-carboxylmethyltransferase-1, BSMT1)的表达，促进水杨酸(salicylic acid, SA)的积累，从而抑制 JA 和乙烯(ethylene, ET)免疫通路，增强对丁香假单胞菌的防御[30]。Loredana 等利用番茄 OPDA 缺失突变体 SiOPR3 证明 JA 合成前体 OPDA 的缺失可以增强番茄对丁香假单胞菌的抗性[31]。水稻通过多种调控方式增强 JA 防御响应抵抗 Xoo，OsVQ13 是 JA 应答性缬氨酸–谷氨酰胺(valine-glutamine, VQ)基序蛋白，通过激活水稻 OsMPK6-OsWRKY45 信号通路正向调节 JA 信号，从而调控水稻对白叶枯病的抗性[32]。OsEDS1 在调节 JA 介导的水稻抗白叶枯病中也起着积极的作用[33]。OsPHR2-OsMYC2 通路则通过增强 OsMYC2 的表达以提高 JA 含量从而增强 Xoo 抵抗力[34]。柑橘溃疡病菌(*Xanthomonas citri* subsp. *citri*)的侵染性与植物 JA 水平密切相关，壁相关受体样激酶(wall-associated kinase, WAKL08)过表达植株中 JA 含量、JA 生物合成和信号传导基因表达水平均显著升高，提高了柑橘对溃疡病的抗性[35]。越来越多的证据表明 JA 与植物应对病原真菌侵染有密切联系，这包括甘蓝链格孢菌(*Alternaria brassicicola*)、灰葡萄孢菌(*Botrytis cinerea*)、癣囊腔菌(*Plectosphaerella cucumerina*)、腐霉菌(*Pythium* spp.)等[3] [36] [37]。拟南芥 JA 生物合成突变体 *fad3fad7fad8* 和 *jar1* 对甘蓝链格孢菌和寡雄腐霉菌(*Pythium irregular*)的敏感性明显提高[38]-[40]。JA 信号传导突变体 *coi1-1* 则更容易被甘蓝链格孢菌，灰葡萄孢菌，癣囊腔菌侵染[38]。玉米的 *opr7opr8* 双突变体对腐霉菌表现出极度的敏感性[41]。番茄突变体 *jai1* 在根腐病实验中死亡率达到 100%，并且对灰葡萄孢菌和镰刀菌极度敏感[42] [43]。JA 合成途径的基因缺陷突变体 *aos*、*def1* 以及 *jar1* 证实了 JA 合成突变植株对番茄灰霉病菌、香蕉枯萎病菌和棉花黄萎病菌都表现出高易感性[44]。狭叶烟草(*Nicotiana attenuata*)的脂肪酶 GLA1 突变株表现出对烟草疫霉菌(*Phytophthora parasitica* var. *nicotianae*)和尖孢镰刀菌的易感性[31]。JA 在植物抗病毒防御过程中具有复杂的调节作用，如 JA 信号传导激活 RNA 沉默，并与其他植物激素协同促进水稻抗病毒防御，但仍需更多研究工作[27]。

4.3. 外源 JA 处理对植物宿主与病原微生物互作发挥调控作用

JA 类物质的外源添加可以极大增强植物的防御能力，这大多体现在真菌病害治理实践中。JA 外源处理极大激活了蔷薇(*Rosa chinensis*)对灰葡萄孢菌的防御[45]，并且可以诱导小麦中发病机制相关蛋白(pathogenesis-related proteins) PR4、PR5 和 PEROX 的显著上调，增强小麦对禾谷镰刀菌(*Fusarium graminearum*)的抗性[46]。外源施加 MeJA 可以增强梨果实对灰霉病的抗性，同时对梨果实抗青霉病和保鲜也有一定促进[47]，也能够促进葡萄果实中几丁质酶、β-1,3-葡聚糖酶和苯丙氨酸解氨酶等抗性物质的积累，并且可以抑制番茄灰霉病菌的孢子萌发、胚芽管生长和菌丝延伸[48]，同时可以通过显著上调 JA 合成和信号传导相关基因增强油菜(*Brassica napus*)对油菜菌核菌(*Sclerotinia sclerotionrum*)的抗性和三七(*Panax notoginseng*)对三七镰刀菌(*Fasarium solani*) 的抗性[49] [50]。对柑橘果实外源添加 MeJA 则显著增强 PEROX 和过氧化氢酶(polyphenol oxidase, PPO)的活性，从而有效抑制了绿霉菌(*Penicillium digitatum*)和蓝霉菌

(*Alternaria alternata*)的发生[51]。除此之外，本氏烟草(*Nicotiana benthamiana*)甲基化酶突变株系中 MeJA 的缺失和 MeSA 的缺失导致烟草花叶病毒(tobacco mosaic virus, TMV)的易感性显著提高[52]。

4.4. 病原微生物感应植物宿主的 JA 并做出响应

JA 在病原微生物与植物宿主相互作用的完整过程应包括四个环节，分别为病原微生物侵染激活或抑制植物宿主的 JA 防御响应，植物宿主通过 JA 防御响应抵抗病原微生物侵染，病原微生物对 JA 信号进行识别和响应及病原微生物通过操纵植物宿主 JA 防御响应增强自身侵染。然而，就目前的报道而言，其他三个环节均有较为深入的研究，而病原微生物对 JA 信号进行识别和响应这一环节的相关报道少之又少，仅有冠菌素(coronatine, COR)的合成调控过程中有些许涉及，Ullrich 等研究显示 COR 的合成受到双组分调节因子 CorRP 的调节且受到温度调节[53]。因此，有关病原微生物如何识别 JA 信号并做出代谢应激反应的研究是当前的一大空白。

5. 病原微生物通过操纵植物宿主 JA 防御响应增强自身侵染

5.1. 病原细菌通过多种机制操纵植物宿主的 JA 防御

多种病原细菌已经进化出劫持植物宿主 JA 信号通路的机制以增强自身的侵染性。丁香假单胞菌(*Pseudomonas syringae*)是研究这一问题的模式菌株。丁香假单胞菌通过 III 型分泌系统效应子(type III secretion system effector protein, T3SE)和冠菌素两条典型的途径应对植物的 JA 防御响应(图 3)。同时，多种病原细菌也被报道参与到植物 JA 防御响应的调控中。辣椒疮痂病菌(*Xanthomonas vesicatoria*)的效应子 XopH 则抑制 MYC2 的表达从而干扰 JA 信号[54]。在亚麻假单胞菌(*Pseudomonas cannabina* pv. *alisalensis*)、疥链霉菌(*Streptomyces scabies*)和新西兰亚麻的病原体 (*Xanthomonas campestris* pv. *phormiicola*)、柑橘溃疡病菌(*Xanthomonas axonopodis* pv. *citri*, Xac)，根癌农杆菌(*Agrobacterium tumefaciens*)等细菌中报道有 COR 样化合物的产生[55]-[57]。此外，在包括 *glycinea*、*alisalensis*、*atropurpurea*、*glycinea*、*maculicola*、*morsprunorum*、*porri* 和 *tomato* 等多种丁香假单胞菌亚种、青枯菌(*Ralstonia solanacearum*)、欧文氏菌(*Erwinia carotovora* subsp. *Atroseptica*, Eca)、胡萝卜软腐果胶杆菌(*Pectobacterium carotovorum* subsp. *carotovorum*)和玉米迪基氏菌(*Dickeya* sp.)中均鉴定出了参与 COR 生物合成的基因簇[58]-[61]。下面将着重介绍 T3SE 和 COR 两种代表性机制。

5.1.1. III 型分泌系统效应子

丁香假单胞菌所分泌用以调控宿主 JA 信号通路的 T3SE 主要有 3 种：1) HopZ1a 是由 *P. syringae* pv. *tomato* (Pst) DC3000 分泌的一种乙酰转移酶，可以直接与拟南芥 JAZ 蛋白相互作用并诱导其乙酰化，促进 JAZ 降解，从而激活 JA 信号传导[63] [64]；2) HopX1 是由 *P. syringae* pv. *tabaci* (Pta) strain 11528 分泌的一种半胱氨酸蛋白酶，以不依赖 COI1 的方式与 JAZ 蛋白相互作用并促进 JAZ 蛋白的降解，可以引发 JA 介导的对 SA 依赖性防御响应的抑制，并促进植物对这种丁香假单胞菌的易感性[24]；3) AvrB 是具有复杂效应的 T3SE。在拟南芥中，AvrB 以 COI1 依赖性方式增强 JA 信号传导[67]，这一过程需要拟南芥 RPM1 相互作用蛋白 4 (RPM1-interacting 4, RIN4)的参与[65] [66]。AvrB 与 RIN4 相互作用并以 RIN4 依赖性方式激活质膜定位的 H^+-ATP 酶(Arabidopsis plasma membrane H^+-ATPase, AHA1)。AHA1 和 AvrB 均增强 COI1-JAZ 相互作用和 JAZ 蛋白的降解，损害植物对丁香假单胞菌的防御[67]。除了靶向 JA 信号传导的核心成分外，AvrB 还与丝裂原活化蛋白激酶 4 (mitogen-activated protein kinase 4, MPK4)相互作用并诱导 MPK4 磷酸化，并通过热休克蛋白 90 (heat shock protein 90, HSP90)的共伴侣 RAR1 与 HSP90 结合，最终导致 JA 信号传导的激活，这一过程也可能通过 RIN4 但并未得到确认[66]。

5.1.2. 冠菌素

COR 则是多种丁香假单胞菌如 *glycinea*、*maculicola*、*tomato* 等均可以分泌产生的 JA-Ile 结构类似物[55]，包含冠烷酸(coronamic acid, CMA)和冠菌酸(coronafacic acid, CFA)两个部分，它们通过酰胺键共轭[68]。COR 以高亲和力直接与 COI1-JAZ 受体结合[69]-[72]。COR 同样引发 JA 信号通路激活，这可以抑制病原体应激免疫(pathogen-associated molecular patterns, PAMP)诱导的气孔闭合，抑制植物质体防御，最终抑制 SA 介导的植物防御来促进细菌感染[53] [73]-[77]。研究显示，COR 劫持植物 JA 信号模块 COI1-JAZ2-MYC2/3/4-ANAC19/55/72 [78]并通过 JAZ2 靶向的 MYC2/3/4 直接激活 SA 生物合成酶抑制剂 NACsTFs (ANAC19/55/72)的表达，从而劫持 JA 通路以抑制 SA 通路[78]。同时，COR 还可以激活拟南芥乙烯反应因子(ERFEthylene-responsive factor)基因 RAP2.6 的转录进一步调控 JA-SA-ET 的防御网络[47]。值得注意的是，冠菌素在 2021 年已经获得 98%冠菌素原药和 0.006%冠菌素可溶液剂两种农药登记证，根据冠菌素使用浓度的不同，可以达到诱导植物防御[79]、脱叶[80]、除草[80]、提高果品质量[81]、增强植物抗逆性[82]等不同效果。

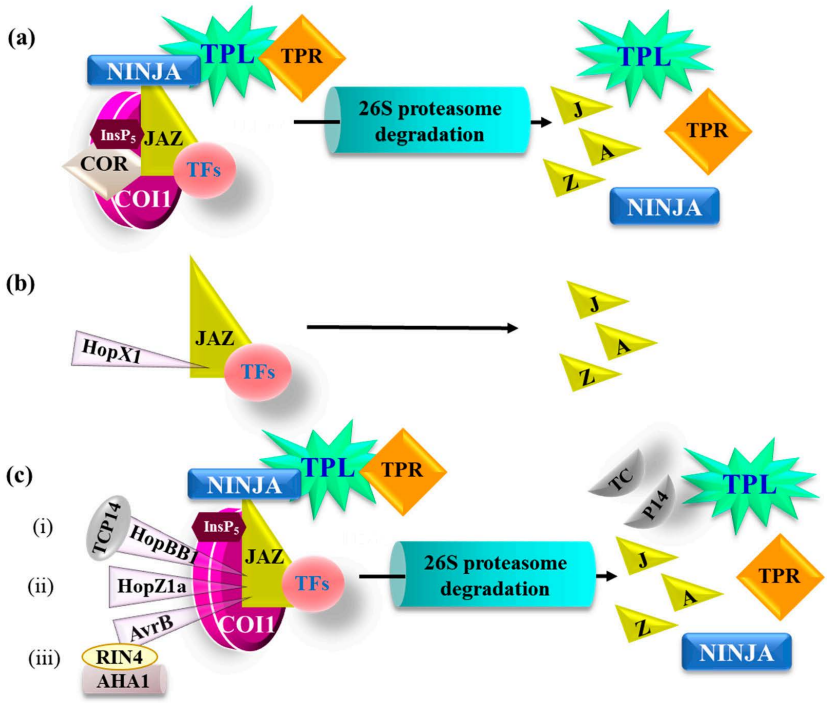

Figure 3. Jasmonic acid signaling pathway in the hijacking of plant hosts by *Pseudomonas syringae* [62]. HopZ1a interacts with JAZ and induces its acetylation, promoting JAZ degradation and activating JA signal transduction. HopX1 interacts with JAZ protein in a COI1 independent manner and promotes its degradation, which can trigger JA mediated inhibition of SA dependent defense response. AvrB is a T3SE with complex effects. In *Arabidopsis*, AvrB enhances JA signaling in a COI1 dependent manner, which requires the involvement of RPM1 interacting protein 4 (RIN4). AvrB interacts with RIN4 and activates plasma membrane H+-ATPase (AHA1) in a RIN4 dependent manner. AHA1 and AvrB both enhance COI1-JAZ interaction and degradation of JAZ protein. COR binds directly to the COI1-JAZ receptor with high affinity, which also triggers activation of the JA signaling pathway

图 3. 丁香假单胞菌劫持植物宿主的茉莉酸信号传导途径[62]。HopZ1a 与 JAZ 相互作用并诱导其乙酰化，促进 JAZ 降解，从而激活 JA 信号传导。HopX1 以不依赖 COI1 的方式与 JAZ 蛋白相互作用并促进 JAZ 蛋白的降解，可以引发 JA 介导的对 SA 依赖性防御响应的抑制。AvrB 是具有复杂效应的 T3SE。在拟南芥中，AvrB 以 COI1 依赖性方式增强 JA 信号传导，这一过程需要 RIN4 的参与。AvrB 与 RIN4 相互作用并以 AHA1。AHA1 和 AvrB 均增强 COI1-JAZ 相互作用和 JAZ 蛋白的降解。COR 以高亲和力直接与 COI1-JAZ 受体结合，同样引发 JA 信号通路激活

5.2. 病原真菌对植物宿主 JA 防御响应的调控

多种病原真菌也进化出应对植物 JA 防御的调控机制。尖孢镰刀菌可以劫持 JA 途径以促进植物易感性：*coi1*，*myc2* 和 *pft1/med25* 等拟南芥突变体表现出对这种病原体的抗性增加[83]-[85]。毛果杨(*Populus trichocarpa*)的共生真菌双色蜡蘑(*Laccaria bicolor*)可以分泌效应子 MiSSP7 蛋白稳定 JAZ 蛋白，抑制 JA 信号传导从而增强自身的定植能力[86]。HaRxL44 则是拟南芥霜霉病菌(*Hyaloperonospora arabidopsidis*)的核定位分泌效应子，通过与介质亚基 MED19a 相互作用和降解来将植物 SA 的防御响应逆转为 JA/ET 的防御响应从而增强疾病易感性[87]。大豆疫霉(*Phytophthora sojae*)通过分泌 RXLR 类效应子 Avh94 通过直接相互作用抑制 JAZ1/2 的降解以抑制 JA 信号传导[88]。来自稻瘟病菌(*Magnaporthe oryzae*)的单加氧酶 ABM 将真菌和植物来源的 JA 转化为 12-OH-JA，以减弱 JA 信号传导并促进宿主定植[89]。稻瘟病菌中 ABM 的缺失导致 MeJA 在真菌中的积累和植物防御的诱导[89]。

5.3. 病毒对植物宿主 JA 防御响应的调控

越来越多的证据表明病毒同样可以劫持 JA 信号传导，以促进植物的易感性使其自身受益。C2 蛋白和其同源的 L2 蛋白被鉴定为中国番茄黄化曲叶病毒(tomato yellow leaf curl virus, TYLCV)和甜菜曲顶病毒(beet curly top virus, BcTV)的病毒致病因子[90]，它们与拟南芥 CSN5 相互作用并干扰拟南芥 CSN5，导致 JA 介导的防御减弱[91] [92]。*β*C1 蛋白则是 TYLCV 的另一种病毒致病因子，直接与关键的 JA 信号转录因子 MYC2 相互作用并抑制 JA 信号从而调节的萜烯合酶基因的表达，降低宿主对其昆虫载体粉虱(*Trialeurodes vaporariorum*)的抗性并加速其自身的病毒传播[90] [93] [94]。花椰菜花叶病毒(cauliflower mosaic virus, CaMV)的 RNA 沉默抑制因子 2b 蛋白在 CaMV 感染时抑制 JA 反应基因的表达，这可能作为诱饵促进桃蚜(*Myzus persicae*)的活动以促进自身传播[95]-[97]。最近的一篇报道表明，OsNF-YAs 家族的基因表达产物通过破坏 OsMYCs 和 OsMED25 之间的相互作用，抑制 OsMYC2/3 的转录活性，从而损害 JA 介导的抗病毒防御[98]。水稻黑条矮缩病毒(rice black-streaked dwarf virus, RBSDV)编码的 P5-1 调节 SCF 泛素连接酶的泛素化活性，并抑制 JA 信号传导[99]。RSV 由褐飞虱(brown rice planthopper, SBPH)传播，是纤细病毒属的典型成员。MeJA 处理吸引了 SBPHs 以水稻植株为食，其中 JA 缺陷突变体的吸引力不如野生型水稻。这是因为由外壳蛋白诱导的 JA 通路激活了植物对 RSV 的防御，同时吸引 SBPH 进食，从而有利于病毒传播[100]。此外，斐济病毒属、纤细病毒属和弹状病毒属的水稻病毒都具有转录抑制因子，可直接解离 OsMED25-OsMYC3 复合物，抑制 OsMYC3 的转录激活，然后与 OsJAZ 蛋白结合，以有利于病毒感染及其载体摄食活性的方式协同克服 JA 通路[61]。

6. 总结展望

植物宿主利用 JA 信号介导的生理响应来优化应对病原微生物的防御，而病原微生物则分泌效应蛋白和植物毒素或产生 JA 类化合物用于调节植物宿主的防御响应。效应蛋白和植物毒素往往靶向宿主植物中的 JA 信号传导途径，以增强或减弱下游反应的形式提高自身的侵染性和定植能力。目前，有关病原微生物与植物宿主相互作用过程中 JA 扮演的角色已有部分研究成果，未来的研究在如下几个方面仍有深入探索的必要：

1) 在 JA 的合成和代谢方面，目前仅发现部分病原真菌具有 JA 的合成能力。然而，是否有更多的病原微生物可以合成 JA 或相关化合物仍需要检验。对于有 JA 合成能力的病原微生物，其具体代谢过程是怎样的？这些问题仍有待探索。

2) 病原微生物在侵染植物宿主过程中，JA 防御响应的相关调控效应蛋白或化合物合成酶系会相应的进行基因表达过程并发挥功能。病原微生物是如何感应植物所产生的 JA 信号的？是否存在相关的受

体蛋白或信号传导通路用于感应？以及在感应到信号后是如何做出分子响应的？是否有特异性的外排泵参与此过程？微生物的群体感应系统和双组分感应系统等是否参与？这些问题虽然在丁香假单胞菌中有部分研究[101] [102]，但仍有大量工作要做。

3) JA 与其他多种植物激素存在复杂的相互作用以共同应对病原微生物入侵。在作用网络中，哪些节点是关键的？不同信号通路之间是如何发挥协同或拮抗作用的？受到哪些胞内外信号的调节？这些问题值得研究解决。

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