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病毒-植物-媒介昆虫跨界互作与病毒病害流行

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摘要 植物病毒引发的病害严重制约农作物的产量和品质, 在目前已报道的植物病毒中有约80%依赖媒介昆虫的传播. 阐明虫媒病毒病害循环中病毒-植物-昆虫这三种跨界生物间互作的分子信号及分子机制, 是更高效控制虫媒病毒病害的基础. 病毒病害的流行范围和强度主要取决于病毒的侵染和传播这两个关键环节, 得益于不断发展的分子生物学、细胞生物学和各种组学技术, 植物病毒如何成功侵染寄主植物以及如何高效地通过媒介昆虫传播被不断解析. 本文主要阐述了病毒逃逸植物免疫、突破媒介昆虫屏障和免疫、直接或间接地调控媒介昆虫传毒的具体机制, 展现了虫媒病毒操控寄主植物和媒介昆虫促进病害流行中的强大能力, 也揭示了虫媒病害防控中可供选择的分子靶标.

关键词 虫媒病毒, 病害流行, 植物免疫, 激素信号

植物病毒种类繁多, 目前已知的超过1600种, 严重制约着农作物的产量与品质, 有“植物癌症”之称. 据不完全统计, 全世界每年由植物病毒引起的作物经济损失高达4000亿元. 与动物病毒的寄主特性不同, 植物作为固着生物, 植物病毒的传播更多地依赖于媒介昆虫的传播, 这类虫媒病毒约占所有植物病毒的80%[1]. 媒介昆虫以刺吸式口器昆虫为主, 其取食后对植物细胞和组织损伤较小, 病毒侵染后更容易定殖, 并且媒介昆虫能主动为病毒寻找寄主植物, 因此, 刺吸式口器昆虫极大地加速了植物病毒的流行.

根据媒介昆虫获取病毒的时间、病毒在媒介昆虫体内存留的时间以及可被传播的时间可将虫媒传播植物病毒的方式分为持久型、非持久型和半持久型三类. 其中持久型病毒在媒介昆虫体内可以长期甚至终身存留, 且蜕皮后不会失去传播能力, 但其被媒介昆虫获取和传播的时间也会较长, 需要几小时甚至几天时间, 并且持久型病毒在昆虫肠道、血淋巴以及唾液腺组织均有分布, 与媒介昆虫之间具有复杂的互作; 而非持久型病毒, 媒介昆虫只需在带毒植物上取食几分钟甚至几秒钟即可获毒并传播, 但病毒在昆虫体内存留时间较短; 半持久型病毒则介于两者之间. 非持久型和半持久型的病毒仅存留于昆虫口针、食道或前肠, 随着下次昆虫取食植物时被重新分泌到植物中[2]. 因此, 虫媒病毒的流行与非介体传播病毒、真菌、细菌等病害存在较大区别, 不但受病原物与植物间的防御-反防御关系影响, 还受到病毒与媒介昆虫间直接互

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作和通过寄主植物介导间接互作的深刻影响, 这些复杂的多元互作关系造成了虫媒病毒的防控策略复杂、难度较大. 与其他农作物病害不同, 病毒病无法依靠农药从发病植物上完全清除, 且高抗病毒病的优良作物种质资源和品种又相对匮乏, 目前生产上主要依赖对媒介昆虫的化学防治来阻断病毒传播, 以实现虫媒病毒病害的防控, 但是化学杀虫药剂的不合理使用甚至滥用会导致环境污染、害虫抗药性、农产品农药残留等一系列问题, 严重威胁农产品安全和生态环境安全.

有鉴于此, 加强虫媒病毒与寄主植物、媒介昆虫间跨界互作的分子信号及分子机制研究, 有望为阻断虫媒病毒病的流行提供新的策略和思路, 并以此发展绿色、高效的虫传重大病害防控技术, 将有助于实现国家粮食安全和农业可持续发展. 本文从病毒-植物-昆虫间的跨界互作关系出发, 从病毒侵染和传播两个方面总结植物病毒通过跨界互作促进病害流行的分子机理, 以期为作物病毒病防控提供新策略和新靶标.

1 病毒对植物免疫的逃逸

植物病毒是细胞内寄生病原物, 它们几乎完全依赖寄主进行复制、运动等生命过程. 病毒和寄主植物之间在蛋白-蛋白、蛋白-核酸等维度进行着复杂的互作: 一方面, 植物通过互作诱导抗病毒免疫以消除或抑制病毒复制或运动; 另一方面, 病毒通过互作来抑制植物的抗病毒免疫并利用寄主资源完成复制增殖[3,4]. 植物在长期应对病毒的“进攻”中, 进化出了多种复杂的免疫系统及适应性机制进行“防御”, 如RNA沉默、蛋白质降解、植物先天免疫、植物激素等, 这些免疫信号精细复杂, 并通过交互作用形成了更为庞大复杂的调控网络, 植物可以激活一个或多个免疫反应抵御病毒的侵染, 并平衡防御和生长发育. 与此同时, 病毒也在与植物的长期共进化中, 形成了多种“反防御”的机制. 这种病毒与植物“进攻-防御-反防御”的军备竞赛关系, 最后的胜负决定了病毒能否高效地在植物细胞内复制增殖和通过运动系统侵染植株, 是病毒病害流行的基础.

1.1 RNA沉默

RNA沉默, 也被称为RNA干扰, 是一种在真核细胞中保守的天然防御机制, 通过特定的小分子RNA (small interfering RNA, siRNA)来调控内源基因表达或抑制外源核酸复制和转录, 被认为是植物抵抗病毒侵染中最主要的抗病毒防御反应. 病毒侵入寄主细胞后, 生成双链RNA(double-stranded RNA, dsRNA)或结构类似于dsRNA的RNA分子, 这些RNA可以被细胞内的核酸内切酶Dicer(植物中为Dicer-like, DCL)切割成长度为20~24个核苷酸的siRNA. siRNA与ARGONAUTE(AGO)蛋白结合形成RNA诱导的沉默复合体(RNA-induced silencing complex, RISC), 直接切割同源病毒RNA和/或抑制病毒蛋白翻译, 从而阻止病毒的复制和侵染[5]. 甚至有时候植物还能利用病毒来源的siRNA(virus-derived small interfering RNA, vsiRNA)靶向自身基因的表达抑制病毒侵染, 如小麦黄花叶病毒(Wheat yellow mosaic virus)的vsiRNA1能抑制小麦ROS清除基因的表达, 进而导致叶绿体ROS积累并激活防御反应, 抑制病毒入侵[6].

在病毒与植物的长期协同进化中, 病毒发展出了通过编码一种或几种功能多样的RNA沉默抑制子(viral suppressors of RNA silencing, VSR)抵抗植物抗病毒免疫的机制. VSR作为病毒成功侵染植物的关键蛋白, 在过去的20年间被广泛报道, 研究表明, 不同病毒的VSR通过作用于植物RNA沉默途径的不同阶段并抑制其功能, 从而帮助病毒突破植物的抗病毒免疫[7~9]. 例如, 水稻条纹病毒(Rice stripe virus, RSV)编码的NS3、番茄斑萎病毒(Tomato spotted wilt virus, TSWV)编码的NSs、绿萝潜隐病毒(Pothos latent aureusvirus)编码的P14均能直接结合dsRNA, 以减少siRNA的生成[10]; 花椰菜花叶病毒(Cauliflower mosaic virus, CaMV)编码的P6蛋白通过与DRB4互作影响DCLs的功能[11]; 小西葫芦黄花叶病毒(Zucchini yellow mosaic virus)编码的HC-Pro能够与RNA甲基转移酶Hua Enhancer 1(HEN1)互作, 抑制其活性[12]. 更有一些病毒能直接作用于RISC的核心AGO蛋白, 如黄瓜花叶病毒(Cucumber mosaic virus, CMV)编码的2b、芜菁皱缩病毒(Turnip crinkle virus, TCV)编码的P38、番茄环斑病毒(Tomato ringspot virus, ToRSV)编码的CP、甜菜西黄病毒(Beet western yellows virus, BWYV)编码的P0均能够靶向不同的AGO蛋白并通过调节其稳定性抑制抗病毒RNA沉默[13~16].

除了利用VSR对抗植物的抗病毒RNA沉默, 病毒还会主动利用寄主植物的RNA沉默系统, 通过vsiRNA

靶向寄主基因, 从而促进病毒的侵染和症状形成. 例如, CMV和大麦黄矮病毒(Barley yellow dwarf virus, BYDV)来源的vsiRNA均能靶向寄主植物的叶绿素合成基因, 促进黄化症状的形成[17,18]; RSV来源的vsiRNA靶向翻译起始因子, 导致烟草叶片扭曲和发育迟缓[19]; 而南方黑条矮缩病毒(Southern rice black streaked dwarf virus, SRBSDV)、中国小麦花叶病毒(Chinese wheat mosaic virus, CWMV)、烟草曲茎病毒(Tobacco curly shoot virus)来源的vsiRNA则分别靶向水稻转录因子ROC1、液泡质子泵基因、RNA解旋酶等抗性相关基因, 帮助病毒进一步侵染并加重症状[20~22].

1.2　蛋白质降解

蛋白质降解是细胞内一种重要的生物学过程, 参与细胞代谢、信号转导、免疫应答等多种生命活动. 在植物依赖蛋白质降解的抗病毒免疫中, 主要依赖泛素蛋白酶体和细胞自噬两种途径, 对病毒相关蛋白进行降解[23].

泛素蛋白酶体途径(ubiquitin-proteasome pathway, UPS)通过将特定的蛋白质泛素化标记为“废弃物”, 然后将其送入蛋白酶体降解, 这一过程主要依赖泛素激活酶(E1)、泛素结合酶(E2)和泛素蛋白连接酶(E3)进行调控, 且这种由泛素化介导的蛋白质降解的特异性主要由E3决定[24]. 越来越多的研究表明, UPS在植物抗病毒中发挥重要作用, 如甜菜严重曲顶病毒(Beet severe curly top virus)的C4蛋白[25]、中国番茄黄化曲叶病毒(Tomato yellow leaf curl China virus, TYLCCNV)卫星编码的βC1蛋白[26]、水稻矮缩病毒(Rice dwarf virus, RDV)的P2蛋白[27]、芜菁黄花叶病毒(Turnip yellow mosaic virus)的RNA聚合酶(RNA-dependent RNA polymerase, RdRP)[28]、大麦条纹花叶病毒(Barley stripe mosaic virus, BSMV)的γb蛋白[29]等均能被寄主植物特定的E3识别, 发生泛素化后被蛋白酶体降解, 抑制病毒复制和症状形成. 作为病毒的“反防御”, UPS也能为病毒所用, 调控其靶向植物防御信号, 促进病毒侵染, 如BWYV编码的P0蛋白具有F-box结构域, 参与组成E3泛素连接酶复合物, 通过靶向AGO1并将其泛素化后降解, 从而抑制RNA沉默促进病毒侵染[30]; 最近研究发现, 水稻草状矮化病毒(Rice grassy stunt virus)编码的P3蛋白能够诱导一个U-box类型的E3泛素连接酶P3IP1的表达, 后者能靶向水稻RNA介导的DNA甲基化关键因子RNA聚合酶Ⅳ并将其降解, 导致水稻矮化和分蘖增多等病毒症状的形成[31].

细胞自噬是真核生物将细胞内错误折叠的蛋白、受损的细胞器、有害病原物等通过双层膜结构的自噬小泡包裹, 送入溶酶体或液泡中进行降解的一种保守机制[23,32]. 早在2005年, 就有研究报道烟草花叶病毒(Tobacco mosaic virus, TMV)侵染能诱导烟草细胞自噬, 并通过过敏反应(hypersensitive response, HR)反应将TMV限制在侵染的局部叶片, 达到植物抗病毒的作用[33]. 此后, 自噬参与植物抗病毒的分子机制被广泛研究, 如拟南芥参与选择性自噬的蛋白NBR1能与CaMV的外壳蛋白或芜菁花叶病毒(Turnip mosaic virus, TuMV)编码的HC-Pro互作从而限制病毒的侵染[34,35]; 烟草或拟南芥的ATG8s和双生病毒的复制起始蛋白C1或毒力因子βC1互作介导对多种双生病毒的防御反应[36,37]; 烟草的Beclin1能够靶向多种正义单链RNA病毒的降解[38]. 最新研究发现, 水稻条纹花叶病毒(Rice stripe mosaic virus, RSMV)编码的糖蛋白能通过与寄主SnRK1互作并促进其磷酸化ATG6, 而ATG6则靶向糖蛋白的降解抑制病毒侵染, 这一机制也被其他弹状病毒广泛利用[39]. 有意思的是, RSMV糖蛋白的过度积累会导致细胞死亡, 而糖蛋白在细胞内的积累可以通过诱导自噬维持在一个合适的水平, 避免因寄主死亡而阻碍病毒的侵染和流行, 表明抗病毒细胞自噬有时也会被病毒利用来维持病毒的相容性侵染[39]. 作为病毒的“反防御”策略, 许多植物病毒被发现能通过编码的蛋白来破坏植物的抗病毒自噬反应. 如BSMV编码的γb和云南番茄曲叶病毒(Tomato leaf curl Yunnan virus, TLCYnV)编码的C2蛋白均能通过竞争性结合破坏植物自噬相关蛋白ATG8与ATG7的复合体形成, 从而抑制抗病毒细胞自噬[40,41]; 最近发现, BSMV还能通过编码的γa蛋白与液泡ATP酶的亚基互作, 阻断液泡腔的酸化并抑制自噬途径的降解以促进植物中的病毒感染[42]; 木尔坦棉花曲叶病毒(Cotton leaf curl Multan virus, CLCuMuV)编码的C4蛋白与真核细胞翻译起始因子eIF4A互作, 增强eIF4A与ATG5的互作来抑制自噬促进病毒的侵染[43]. 此外, 植物病毒还能劫持自噬途径降解寄主防御组分促进自身侵染. 如BWYV编码的P0蛋白除了能通过泛素化修饰外, 还能介导自噬途径降解寄主植物的AGO1蛋白[44];

TuMV编码的VPg和TYLCCNV编码的βC1则介导自噬靶向降解另一个RNA沉默途径的重要蛋白SGS3[45,46], 实现对植物抗病毒RNA沉默途径的攻击, 促进病毒的侵染; 而RSV编码的运动蛋白则通过抑制植物细胞膜脂筏的主要成分remorin蛋白的棕榈酰化修饰, 使其被自噬途径降解, 促进病毒在植株内的运动[47]. 最新研究发现, 茄果花叶病毒(Pepino mosaic virus)编码的RdRP作为桥梁连接番茄Beclin1与m^6A修饰系统的甲基转移酶辅助亚基HAKAI, 并利用自噬途径降解HAKAI, 从而抑制m^6A修饰介导的植物抗病毒防御[48].

1.3 先天免疫

植物的先天免疫(innate immunity)是抵御病原微生物的重要防御途径, 目前普遍认为植物的先天免疫系统包含两个层面, 即病原体相关分子模式(pathogen-associated molecular patterns, PAMP)诱导的免疫(PAMP-triggered immunity, PTI)和效应子诱导的免疫(effector-triggered immunity, ETI)[49]. 目前对植物PTI防御病毒侵染仍然存在一定的争议, 因为病毒普遍被认为不携带经典的PAMPs. 尽管如此, 越来越多的证据表明, 植物先天免疫系统在抵抗病毒侵染中也发挥关键作用. 如PRR核心受体激酶SERK3(BAK1)和SERK4(BKK1)负调控TCV[50]、油菜花叶病毒(Oilseed rape mosaic virus)、TMV[51]、CMV[52]和李痘病毒(Plum pox virus, PPV)[53]等RNA病毒的侵染, TMV和马铃薯X病毒(potato virus X, PVX)的CP蛋白也可以触发类似PTI的防御反应[54,55], 大豆中的MAPK4同源基因能够负调控水杨酸的积累, 进而防御大豆花叶病毒(Soybean mosaic virus, SMV)[56]. 此外, 植物也进化出一系列R蛋白作为细胞内受体, 识别病毒蛋白为效应物, 激活ETI反应[57]. 自1994年首个TMV的*R*基因*N*被鉴定后[58], 越来越多的植物抗病毒*R*基因被克隆, 如抗PVX的*Rx1*和*Rx2*[59,60]、抗TSWV的*Sw-5*和*Tsw*[61,62]、抗CMV的*RCY1*等[63]. 这类*R*基因编码的蛋白通常具有典型的R蛋白结构, 包括N端的白细胞介素受体(Toll/interleukin-1 receptor, TIR)同源结构域或螺旋卷曲(coiled-coil, CC)结构域、中间的核苷酸结合(nucleotide-binding, NB)结构域和C端富含亮氨酸重复序列(leucine-rich repeat, LRR)结构域[64]. 与PTI类似, R蛋白对效应物的成功感知将触发一系列下游防御反应, 通常以HR反应为主, 能有效地阻止病毒的侵染[65].

与之相对应地, 病毒也通过自身编码的蛋白调控植物的先天免疫, 以促进侵染. TLCYnV编码的C4蛋白能够与过敏性诱导反应蛋白(hypersensitive induced reaction 1, HIR1)互作破坏其二聚化, 单体化的HIR1则会被降解, 进而抑制植物的HR反应帮助病毒的侵染[66]. 已有多个病毒蛋白被报道能抑制ROS暴发和PTI相关基因的表达[52,53,67]. 而像TYLCCNV编码的βC1蛋白则能够抑制丝裂原活化蛋白激酶(mitogen-activated protein kinase, MAPK)级联信号, 帮助病毒复制[68]. 甜菜黑色焦枯病毒(Beet black scorch virus)侵染植物后, 会迅速激活MAPK级联通路, 进而调控防御基因的表达抵御病毒侵染, 但是病毒编码的外壳蛋白通过与植物生长调节蛋白14-3-3a互作, 竞争性地干扰MAPKKKα与14-3-3a的结合, 导致MAPKKKα被蛋白酶体降解, 从而削弱了MAPK介导的抗病毒反应[69].

1.4 植物激素

植物激素参与调节植物生长发育和对抗各种非生物和生物胁迫, 近年来病毒与植物激素相互作用的研究取得了诸多进展, 其中水杨酸(salicylic acid, SA)、茉莉酸(jasmonic acid, JA)、乙烯(ethylene, ET)作为三大防御相关激素, 被广泛报道参与了复杂的植物与病毒的“防御”和“反防御”[70].

SA是最先被发现参与抗病毒反应的植物激素, 早在1979年White[71]首次发现在烟草中注射SA或阿司匹林(乙酰水杨酸)可以明显增强烟草对TMV的抵抗力; 进一步研究发现, 当TMV侵染烟草时, 植物体内的SA含量显著提高, 抗病相关基因的表达也随之上升[72]. 有研究表明, SA抗植物病毒主要通过抑制病毒复制和抑制病毒的长距离运输和胞间运输[70]. 例如, PPV与ToRSV能够在寄主的接种叶片中进行复制, 但其系统性移动却受到抑制, 而在SA信号被抑制的转基因烟草中PPV与ToRSV的长距离运输显著增强, 系统叶片中病毒的积累量显著增加[73,74]. SA信号还能调控RNA沉默协同抵抗病毒, SA处理番茄后RNA沉默通路的DCL1, DCL2以及RNA依赖的RNA聚合酶RDR1和RDR2的表达量显著提高, 进而抑制番茄花叶病毒(Tomato mosaic virus)的侵染[75]. 与此同时, 植物病毒也进化出应对“反防御”SA抗病毒信号的策略, 如CMV编码的2b蛋白能够抑制SA的积累, 帮助病毒高效侵染[76], TMV的复制酶通过与转录因子ATAF2互作, 进而抑制

植物SA信号转导, 促进病毒复制[77]. 有研究表明, 在植物中存在一类保守的蛋白, 这类蛋白含有N端豆蔻酰化修饰或叶绿体转运肽, 当植物感受到病原物侵染时, 这类蛋白就会从质膜转运至叶绿体, 叶绿体则通过“逆行信号传递”将危险信号传递至细胞核, 激活SA防御信号, 增强抗病基因的表达, 以抵御包括病毒在内的病原微生物; 而TYLCV编码的C4蛋白能“模仿”这类植物蛋白, 在植物细胞感受到攻击时也从细胞膜转移至叶绿体, 进入叶绿体后却抑制叶绿体与细胞核之间的信号传递, 阻碍SA防御信号的激活, 最终为病毒的复制提供良好的环境[78]. 最近的研究发现, 水稻条纹病毒的P2蛋白能与SA信号途径重要调控因子NPR1(non-expressor of pathogenesis-related genes 1)互作并促进NPR1的降解, 从而削弱水稻NPR1介导的抗病毒能力[79], 而TuMV编码的NIb通过靶向拟南芥NPR1抑制其SUMO化和磷酸化, 而NPR1的这两种翻译后修饰对其在抗病毒免疫中至关重要[80]. 这种通过编码蛋白靶向NPR1并削弱NPR1介导的抗病毒作用, 促进病毒流行的机制在其他水稻病毒和马铃薯Y病毒属成员中也都存在, 表明这可能是病毒的一种保守的反防御机制[79,80].

JA信号在调控植物对病原微生物和昆虫的抗性反应中发挥重要作用. 研究表明, JA信号通路能与RNA沉默信号通路协同参与病毒防御. RSV侵染后会激活水稻JA信号, 导致其下游转录因子JAMYB的表达上调和转录激活活性释放. JAMYB能够结合并激活RNA沉默通路核心因子AGO18的启动子, 从而诱导AGO18表达, AGO18可以结合miR168以抑制其对AGO1的切割作用, 从而增强AGO1介导的水稻抗病毒免疫[81]. 在植物与病毒的长期进化中, 病毒也进化出一系列针对JA信号的反防御策略, 如TYLCCNV编码的βC1蛋白与甜菜曲顶病毒(Beet curly top virus)编码的C2蛋白均能抑制JA信号, 促进病毒复制[82,83]; 水稻黑条矮缩病毒(Rice black streaked dwarf virus, RBSDV)编码的P5-1通过与水稻CSN5A互作, 抑制由其介导的CUL1去羟基化, 破坏SCF^{COI1}复合物的完整性从而抑制JA信号, 促进病毒侵染[84]; SRBSDV编码的P8蛋白能够能与水稻JA信号转录因子MYC3互作, 干扰其与MED25的结合从而抑制转录激活活性, 同时P8也能与JAZ蛋白互作, 抑制MYCs的转录激活活性, 共同抑制JA信号途径, 促进SRBSDV侵染, 而RBSDV, RSV和RSMV等水稻病毒也都能利用编码的蛋白采取该策略对抗植物JA介导的抗病毒防御[85]. 最新研究发现, TSWV侵染辣椒后编码的NSs蛋白通过与TCP21互作, 进而同时靶向JA、脱落酸、独脚金内酯三种激素信号通路的受体蛋白, 抑制这些激素信号介导的病毒防御; 为了应对TSWV通过NSs的反防御, 辣椒的免疫受体Tsw则会模拟激素受体的结构域与TCP21结合, 并“诱骗”病毒NSs蛋白, 成功实现对植物病毒的“再防御”[86].

ET是一种重要的气态植物激素, 在植物生长发育及响应生物或非生物胁迫过程中起重要作用[87]. 虽然ET的生物合成和信号转导已经相对清楚地被解析[88], 但ET信号如何参与植物抗病毒, 尤其是ET下游通路参与抗病毒的具体机制尚不清楚. 有研究表明, ET信号能够负调控拟南芥对CaMV的抗性[89], 同时又正调控拟南芥对TMV的抗性[90]. 在水稻中, ET信号更多地表现为对病毒抗性的负调控. 如RDV编码的非结构蛋白Pns11蛋白能作用于水稻的ET生物合成途径, 与其中的SAMS1酶发生直接互作激活ET的生物合成, 从而帮助病毒的侵染[91]. SRBSDV侵染水稻早期, 其编码的P6蛋白能够与水稻ET信号的负调控因子RTH2互作, 抑制其活性从而增强ET信号, 促进SRBSDV在侵染早期的水稻植株中快速增殖[92]. 上述研究结果揭示了植物与病毒在ET信号通路中的“防御”与“反防御”之争, 而ET信号如何调控植物病毒抗性仍有待于进一步解析.

2　虫媒病毒突破昆虫屏障和免疫

由媒介昆虫以持久增殖型方式传播的植物病毒由于可以在虫体内长期复制增殖, 且一旦获得病毒可终身传毒, 因而在田间危害范围更广、传播速度更快、防治难度更大, 诸多农业生产上的重大病毒病害均以此类方式进行传播. 持久性的病毒往往在媒介昆虫体内循回, 通过昆虫口器随植物汁液进入媒介昆虫的消化系统后, 与中肠或后肠上皮细胞相互作用并被吸入, 随后穿过肠道释放到血淋巴并循回至唾液腺中, 最后在昆虫取食分泌唾液时水平传播至寄主植物体内, 有的甚至能通过生殖系统垂直传播至下一代[1]. 在此循回过程中, 病毒需要突破昆虫的多重屏障, 包括消化道屏障、唾液腺屏障和生殖腺屏障. 此外, 根据病毒在媒介昆虫体内循回时能否增殖, 可进一步细分为循

回增殖型和循回非增殖型，其中循回增殖型的病毒需要突破昆虫的免疫系统, 以在昆虫细胞内复制增殖.

2.1 虫媒病毒突破昆虫屏障

持久性病毒在虫体内侵染循回的过程中通常需要突破媒介昆虫的多重组织和膜屏障, 具体包括: (ⅰ) 消化道侵入屏障, 该屏障决定了病毒能否进入虫体, 是虫体获毒的关键; (ⅱ) 虫体内扩散屏障, 主要包括消化道释放屏障和唾液腺侵入屏障，该屏障是病毒进入虫体后再经过虫体内扩散，到达唾液腺的重要环节；(iii) 唾液腺释放屏障，这是病毒完成虫体内循环释放进入植物内的关键，决定昆虫能否传毒；(iv) 垂直传播屏障，部分植物病毒可突破媒介昆虫生殖系统的膜屏障, 实现病毒由亲代传递至子代的垂直传播，这对于病毒在田间长久维持和远距离扩散具有重要的流行学意义[1,93].

目前关于持久性传播的植物病毒如何突破昆虫消化道屏障已有诸多报道. 如RDV编码的非结构蛋白Pns10和SRBSDV编码的P7-1蛋白均能形成小管结构帮助病毒穿过中肠屏障[94,95]. 也有通过与昆虫肠道蛋白互作的方式, 如RSV的核衣壳蛋白(nucleocapsid protein, NP)和TYLCV的CP均能与媒介昆虫肠道上类似受体的蛋白互作突破中肠屏障[96~98]. 水稻瘤矮病毒(Rice gall dwarf virus, RGDV)和RDV进入其媒介电光叶蝉后, 能激活虫体内的自噬途径, 利用形成的自噬小体包裹病毒颗粒穿过中肠上皮细胞[99].

唾液腺是由基底膜包围而成的一种多细胞腺体器官, 细胞间紧密连接形成了阻止病毒侵入的屏障, 而持久性传播的植物病毒利用不同的机制突破此屏障[93]. RGDV通过编码非结构蛋白Pns11形成囊泡结构包裹病毒，当接近唾液腺后再以胞吐的形式侵入到唾液腺内[100]；RSV编码的NP能够与灰飞虱硫酸乙酰肝糖蛋白(heparan sulfate proteoglycan, HSPG)相互结合调控RSV进入唾液腺细胞[101]；TYLCV编码的复制相关蛋白(Rep)与烟粉虱唾液腺中增殖细胞核抗原蛋白PCNA和DNA聚合酶δ相互作用后，帮助病毒进入到唾液腺主腺的中间区域并进行复制[102].

部分植物病毒能通过媒介昆虫的生殖系统将病毒传递至子代，病毒突破垂直传播屏障的方式主要以病毒进入雌虫卵巢或雄虫精巢来实现，具体突破垂直传播屏障的机制因病毒的不同而存在差异. RDV附着在媒介黑尾叶蝉的共生菌*Sulcia*上，由共生菌携带病毒通过上皮组织进入卵母细胞，从而帮助病毒经卵垂直传播给叶蝉后代[103]；RSV编码的NP与灰飞虱的卵黄原蛋白互作并形成复合体，介导病毒侵染灰飞虱的卵巢管生殖区，并通过运输卵黄原蛋白的营养丝一同进入卵母细胞，介导RSV完成垂直传播[104]；RGDV编码的非结构蛋白Pns11可以形成小管结构包裹RGDV病毒粒体在电光叶蝉卵巢细胞间扩散并从滤泡细胞进入到虫卵内, 实现垂直传播[105]. 同时, RGDV的外壳蛋白P8还能与电光叶蝉精子的硫酸乙酰肝糖蛋白HSPG互作, 使RGDV病毒粒体附着于电光叶蝉精子头部, 当雄虫与雌虫交配后带毒精子暂储存于雌虫受精囊中，卵巢中成熟的虫卵从精巢排出后，病毒随着精子一同进入虫卵, 完成垂直传播[106].

2.2 虫媒病毒应对昆虫免疫

持久性传播的植物病毒可以在虫体内快速扩散大量复制，引起媒介昆虫行为、生理、生殖等多方面的变化, 也可被视为昆虫病毒. 因此, 要实现植物病毒在媒介昆虫体内的持续增殖和有效传毒，除了需要突破多道屏障完成循回，还需要制衡昆虫的免疫系统[107]. 昆虫与高等动物相比缺少获得性免疫系统，无法对入侵的病毒产生特异性抗体，因而在与病毒长期的“抗争”过程中逐步进化出独特的先天免疫系统, 包括消化道防御、细胞免疫、体液免疫、RNA干扰、Toll途径、Imd途径、JAK/STAT途径、细胞自噬和细胞凋亡等[108]. 昆虫的先天免疫屏障在维持虫体健康的同时，影响着媒介昆虫和病毒之间的亲和性，但是病毒在媒介昆虫体内循回过程中，也采用了不同的机制来对抗昆虫的免疫反应，使之得以在虫体内存活甚至增殖, 保障病毒的传播流行. 例如, RSMV侵染媒介电光叶蝉后, 通过编码NP与黑化反应途径的关键蛋白酚氧化酶原(prophenoloxidase)互作，阻断其向具有活性的酚氧化酶(phenoloxidase)转化, 抑制了血淋巴中的抗病毒黑化反应[109]. SRBSDV和RGDV均能通过不同的蛋白互作诱导在各自媒介昆虫体内的细胞自噬，但是有阻断形成的自噬体与溶酶体的融合，逃避了溶酶体对病毒的降解，而包裹病毒粒体的自噬体可突破中肠和唾液腺屏障进行扩散，通过这种诱导不完全自噬的方式实现病毒在媒介昆虫体内的持久侵染和高效增殖[110~112]. JAK/STAT信号通路可以抑制TYLCV在烟粉

虱体内的积累, 而TYLCV的外壳蛋白能通过与烟粉虱STAT结合来抑制其入核, 从而抑制STAT对下游基因的调控作用, 促进病毒增殖[113]. 此外, TYLCV的外壳蛋白还能在烟粉虱中肠和唾液腺细胞的质膜上通过“劫持”PEBP4, 同步激活细胞凋亡与自噬, 两者之间的相互拮抗形成了中度免疫平衡, 在保障烟粉虱生存的条件下确保病毒高效传播[114]. 同时也有报道称, TYLCV存在通过其核酸复制而不是编码的蛋白激活烟粉虱的细胞凋亡, 并以不通过抑制细胞自噬的方式提高病毒的积累和传播效率[115].

3　病毒与媒介昆虫直接互作促进传播

植物病毒在被媒介昆虫传播的过程中, 病毒通过直接互作改变媒介昆虫的免疫系统、神经系统、内分泌系统等, 影响着昆虫的寄主选择、取食、交配产卵等与传毒相关的行为, 继而调控媒介昆虫对环境的适应和传播病毒的效率. 阐明病毒对媒介昆虫行为操纵的规律与机制, 能够为阻断虫媒病毒传播提供新的靶标和策略.

媒介昆虫传播病毒的过程中, 首先需要在染病植株上取食获毒, 而在获取病毒并具备传毒能力后必须经过寄主转移的过程, 选择健康植株进行传毒, 实现病毒在寄主植物间的扩散. 昆虫对植物寄主的趋向性主要依赖嗅觉系统对植物气体挥发物的响应, 大量研究表明, 已获取病毒和未获取病毒的媒介昆虫对于健康植株和染病植株的趋向性存在差异, 常表现为无毒媒介昆虫趋向取食染病植株, 而带毒媒介昆虫则趋向取食无毒健康植株[116,117]. 如棉花卷叶病毒(Cotton leaf curl virus)的媒介烟粉虱[118]、马铃薯卷叶病毒(Potato leafroll virus, PLRV)的媒介蚜虫[119]、SRBSDV的媒介白背飞虱[120,121]等, 均表现为无毒状态时趋向染病植株, 获毒后转而趋向健康植株, 极大地促进了病毒的传播扩散. 媒介昆虫获毒前后趋向性的转换, 暗示着病毒通过直接互作调控媒介昆虫的嗅觉反应, 使其对植株的气体挥发物做出了不同的选择. 有研究发现, 病毒能够侵入媒介昆虫的神经系统, 影响昆虫的视觉和嗅觉感知能力, 如TYLCV在烟粉虱体内能通过一种炎症类似的免疫信号级联使烟粉虱脑部产生Caspase依赖的凋亡性神经退行, 削弱其嗅觉和视觉的辨识能力, 使烟粉虱更趋向健康植株[122]. 另有研究发现, 媒介昆虫在获毒后其气味结合蛋白(odorant binding protein, OBP)和化学感受蛋白(chemosensory protein, CSP)在转录水平上的表达发生显著变化, 暗示着病毒对媒介昆虫嗅觉系统的调控作用[123,124], 但是其中具体互作的分子机制还有待于进一步解析.

取食行为是媒介昆虫获毒和传毒的必需过程, 刺吸式口器昆虫在寄主植物上的取食活动能通过刺吸电位图谱(electrieal penetration graph, EPG)进行监测, 根据电位变化产生的相应波形区分昆虫对叶肉、木质部、韧皮部的刺探、吸食和唾液分泌等. 基于EPG技术发现了多种病毒操纵媒介昆虫取食行为的证据, 如携带TSWV的西花蓟马雄虫比无毒雄虫取食时间延长, 刺探频率也提高了三倍[125]; 桃蚜感染葫芦黄化病毒(Cucurbit aphid-borne yellows virus)后在植株韧皮部的取食时间延长[126]. 上述这些病毒对媒介昆虫趋势行为的直接调控, 都大大提高了昆虫的传毒效率, 体现了植物病毒通过直接操纵媒介取食行为促进病害的流行.

媒介昆虫的种群数量对病毒的传播流行具有重要影响, 尤其对于仅能通过虫媒传播的植物病毒, 媒介昆虫种群的快速增加更有利于病毒的流行. 植物病毒对媒介昆虫繁殖行为的影响主要体现在交配行为的影响和对产卵量的改变[116,127]. 例如, 西花蓟马雌虫携带TSWV后交配时间延长, 产卵量显著增多[128]; 烟粉虱在携带TYLCV后, 雌虫产卵量显著提高, 种群增长速度更快[129]. 卵黄原蛋白很有可能介导了上述病毒对媒介昆虫产卵的促进作用, 其作为幼虫胚胎生成和发育的重要营养来源, 直接影响雌虫的产卵数量和质量, 一些持久性病毒能通过与媒介昆虫的卵黄原蛋白原直接互作, 提高卵黄原蛋白原的表达, 由此促进雌虫产卵, 同时病毒也获得在媒介昆虫中母系垂直传播的机会[104,130,131]. 但是也有研究发现, 部分情况下病毒会抑制媒介昆虫的种群增长, 如电光叶蝉雌虫携带RGDV后, 产卵量较无毒雌虫显著减少[132], 白背飞虱携带SRBSDV后其繁殖能力也受到一定程度的抑制[133], 这或许与病毒控制媒介昆虫种群的过度增长, 防止它们共同的寄主植物被过度取食而死亡有关, 但相关规律和互作机制有待于深入解析.

昆虫是一种变温动物, 虫体温度会随着环境的改变而变化, 因此环境温度和昆虫温度耐受性在昆虫的种群动态和地理分布中发挥重要的影响[134~136]. 媒介

昆虫对温度的耐受性决定了其越冬越夏、种群维持和增长、迁飞和地理分布, 间接影响着其传播病毒的流行程度和范围. 已有相关研究表明, 媒介昆虫在携带病原物后会改变其对温度的敏感性[137,138]. 例如, 烟粉虱和香蕉交脉蚜分别携带TYLCV和香蕉束顶病毒(Banana bunchy top virus)后, 对低温和高温的耐受性均显著下降[139,140], 这或许是病毒限制媒介昆虫将病毒传播至非适温地区的原因, 也可能是病毒控制媒介昆虫种群数量的手段之一. 有意思的是, SRBSDV通过代谢调控降低了其媒介白背飞虱对低温的耐受性, 但显著提高了白背飞虱对高温的耐受能力[141,142], 这一机制使携带病毒的白背飞虱更适应在低纬度定殖, 且高温更有利于SRBSDV非结构蛋白P7-1形成的含有病毒的管状结构的组装, 有助于病毒的传播[143], 所以该重大病害主要在我国南方稻区流行. BYDV也能提高其媒介禾谷缢管蚜对于高温的耐受能力, 使禾谷缢管蚜可以承受小麦相对高温的上部叶面区域, 扩大生态位, 增强了与玉米蚜的种间竞争力, 帮助病毒更高效地传播[144]. 目前, 关于病毒影响媒介昆虫的温度敏感性的机制尚未完全揭示, 推测可能是受获毒媒介昆虫的营养、免疫力下降等因素, 从而改变了媒介昆虫的温度耐受性, 另一方面也可能与病毒操纵媒介昆虫应对极端高、低温环境, 使媒介昆虫向着有利于病毒流行的方向进化有关[145].

4 病毒调控寄主植物促进媒介传播

植物病毒除了直接互作调控媒介昆虫促进病毒传播外, 还能在营养状态、抗性水平、化学联系等方面对它们的共同寄主植物进行操控, 间接影响媒介昆虫的寄主选择、取食行为、生长发育等, 从而导致媒介在染病植物上的存活率、虫寿命、产卵量等发生改变, 最终导致病毒病害的暴发与流行[127,146,147].

早在2002年, 就有研究发现, 感染PLRV的马铃薯对媒介蚜虫的引诱能力增强[148], 随后在BYDV, RSV, CMV以及双生病毒等介导的寄主植物与媒介昆虫的互作中也发现了相似的染病植株更吸引媒介昆虫取食的现象[149~152]. 同时, 病毒还能够调控植物的营养物质水平或抗虫防御信号, 影响染病植株对媒介昆虫的适生性, 进而影响媒介昆虫的种群数量. 如番木瓜环斑病毒(Papaya ring spot virus)侵染南瓜后, 提高了南瓜的游离氨基酸、可溶性糖等营养物质的积累, 以及病株对棉蚜的适生性, 使棉蚜长时间取食获毒[153]. TuMV侵染寄主后, 通过定位到液泡的效应子NIa蛋白调控ET信号通路, 抑制寄主植物的胼胝质沉积, 进而引诱蚜虫取食, 并加速其繁殖速度, 促进病毒传播[154~156].

近年来, 随着分子生物学和组学研究的飞速发展, 以及微观生物学技术和理念对宏观生物学的快速渗透与交叉, 病毒调控寄主植物促进媒介传播的机制研究取得了诸多重要进展, 尤其是对非持久性传播的CMV和持久性传播的双生病毒与SRBSDV的研究, 揭示了它们通过分子互作对植物抗虫防御信号进行操控, 促进病害流行的分子机理(图1).

CMV作为一种由蚜虫传播的非持久性传播的病毒, 寄主范围很广, 造成非常严重的田间危害. 在西葫芦上的研究发现, CMV侵染后能通过增加气体挥发物的合成来增强对媒介蚜虫的引诱能力, 吸引蚜虫取食获毒; 同时, 感染CMV的西葫芦植株对蚜虫的适生性降低, 从而使取食获毒后的蚜虫尽快转移寄主, 在较短的持毒期内将病毒传播给健康植株[151], 类似的现象还在苜蓿花叶病毒(Alfalfa mosaic virus)或SMV侵染大豆后与蚜虫的互作中出现[157,158]. 烟草上的工作发现, CMV编码的沉默抑制子2b蛋白是调控植物与蚜虫互作的效应蛋白[159], 并且2b蛋白通过诱导烟草活性氧的产生, 引诱探针更短的蚜虫在病株上聚集, 并增加蚜虫的刺探频率, 刺探后的蚜虫活动能力增强, 能够更频繁地远离初始刺探地点, 最终促进病毒的高效传播[160]. 而在拟南芥上的研究清楚地解析了CMV调控植物JA信号的分子机制. CMV侵染拟南芥后, 其编码的2b蛋白能够与JA信号通路的阻遏蛋白JAZs发生直接互作, 削弱JAZs与JA信号受体COI1的结合从而抑制JAZs的泛素化降解, 导致JA信号通路被CMV侵染所抑制, 使植株更加吸引蚜虫取食并加快蚜虫繁殖, 促进病毒的传播[161]. 此外, CMV侵染植物后还能够释放卫星RNA Y-sat作为效应分子, 引起寄主植物变黄吸引蚜虫取食, 更有意思的是, 取食后Y-sat来源的小RNA随植物汁液进入蚜虫体内, 诱导蚜虫翅发育, 促进病毒传播[162].

双生病毒是一类侵染粮食、蔬菜等众多重要作物的毁灭性病害, 由烟粉虱以持久型方式传播. 早在2007年就有报道双生病毒在烟草上能提高媒介烟粉虱的存活率和产卵量[163]. 进一步以TYLCCNV为研究对象,

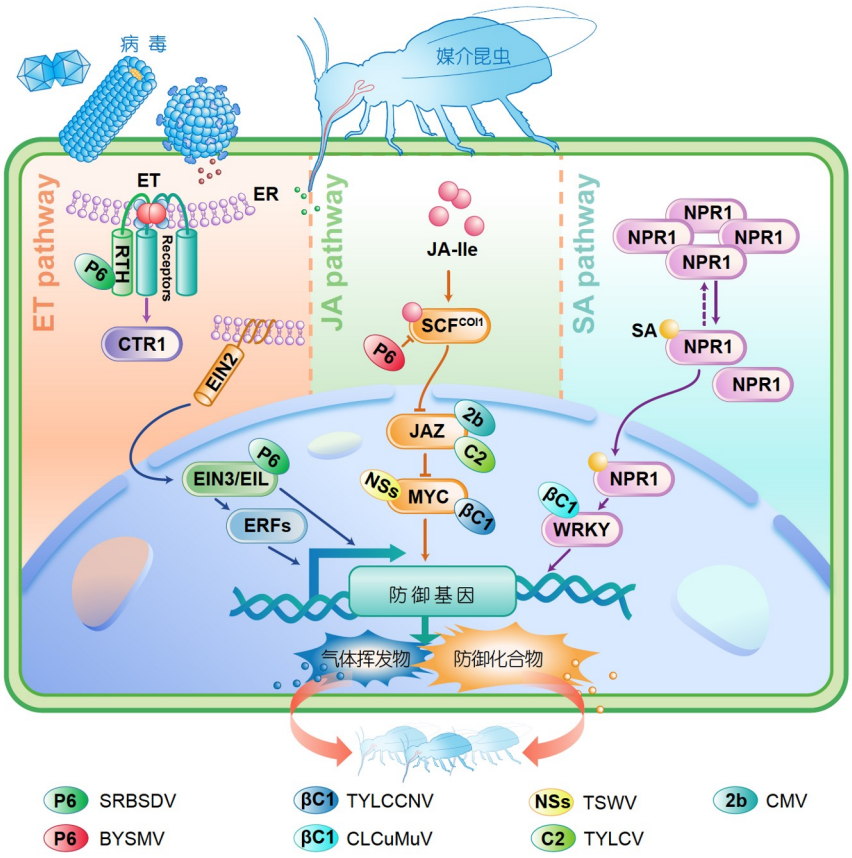

图 1　虫媒病毒操控寄主植物促进媒介昆虫传播的分子机制. 植物主要依赖乙烯、茉莉酸、水杨酸等激素信号通路抵御病毒侵染和昆虫取食, 这些激素通路调控下游气体挥发物和抗虫化合物等合成, 影响媒介昆虫的寄主选择、取食行为、生长发育等. 不同的病毒通过编码效应蛋白作用在这些激素信号转导的各个环节, 激活或抑制相应的激素信号, 操控媒介昆虫传毒促进病害流行

Figure 1　Molecular mechanisms by which arboviruses manipulate host plants to promote the transmission by vector insects. Plants mainly rely on ethylene, jasmonic acid, salicylic acid and other hormone signaling pathways to resist virus infection and insect feeding. These hormone pathways regulate the synthesis of volatiles and defense compounds to affect host selection, feeding behavior, growth and development of vector insects. Different viruses act on various stages of hormone signal transduction by encoding effectors, activating or inhibiting corresponding hormone signaling, and manipulating vector insects to promote the epidemics of viral diseases

研究其与寄主植物和媒介烟粉虱之间的三者互作关系, 发现TYLCCNV侵染植物后, 能增加韧皮部组织中游离氨基酸和糖的积累, 从而通过提高营养物质水平促进媒介烟粉虱的种群增长[164]; 同时还发现, TYLCCNV卫星分子编码的βC1蛋白通过负调控烟草JA信号抑制萜类化合物合成, 促进烟粉虱的种群增长, 从而实现病毒与昆虫的互惠共生[165,166]. 对βC1蛋白调控JA信号的分子机制开展研究发现, βC1蛋白可以靶向植物JA信号途径的转录因子MYC2, 通过干扰其二聚体形成抑制其转录活性, 进而破坏JA信号介导的抗虫反应, 如MYC2直接调控的萜烯类抗虫化合物的合成等, 从而引诱烟粉虱取食染病植株, 并促进烟粉虱种群增长[167]. 后续的研究发现, 双生病毒诱导的植株对媒介烟粉虱的引诱作用只有在红光或含有红光的白光条件下发生, 表明植物的光信号参与了双生病毒-植物-烟粉虱的互作; 深入探究后发现双生病毒卫星分子编码的βC1蛋白能与植物光信号通路中的关键转录因子光敏色素互作蛋白(phytochrome-interacting factors, PIFs)互作, PIFs和MYC2一样也能直接调控萜烯类化合物合成基因TPSs的转录, 因而βC1蛋白通过干扰PIFs二聚体形成抑制其对TPSs的转录激活, 促进烟粉虱的取食和病毒的传播流行[168]. 对于不含卫星的双生病毒, 如TYLCV编码的C2蛋白则采用另一种策略促进病毒传播, 即与核糖体蛋白S27a(ribosomal protein

S27a, RPS27a)互作, 抑制JA信号通路中的阻遏蛋白JAZ1降解, 同样实现了对寄主植物JA信号介导的抗虫反应, 最终促进病毒的高效传播食[169]. 除了靶向JA信号介导的抗虫免疫, 双生病毒还能靶向SA信号相关的抗虫反应. CLCuMuV是为害棉花的重要双生病毒, 其侵染棉花后不但能促进烟粉虱的生长, 还能大幅度提升植物对非媒介昆虫棉铃虫和蚜虫的抗性. 深入研究发现, CLCuMuV卫星分子编码的βC1蛋白通过与WRKY20互作, 干扰WRKY20的二聚化, 显著减少植物维管束组织中芥子油苷等抗虫化合物的积累, 最终引诱传毒媒介烟粉虱, 并促进其生长; 同时, 提高叶肉组织中脂肪族芥子油苷的水平, 激活植物体内SA的产生, 抑制棉花植株上烟粉虱的主要竞争对手棉铃虫和蚜虫的生长, 进一步促进烟粉虱的种群增长, 加速双生病毒的流行[170]. 在其他植物病毒中也存在类似的调控寄主植物促进媒介昆虫传毒的机制, 如TSWV通过其编码的非结构蛋白NSs通过与寄主植物MYC2互作抑制其转录活性, 进而抑制JA信号提高寄主植物对媒介西花蓟马的引诱力和适生性, 促进病毒流行[171]; 弹状病毒大麦黄条点花叶病毒(Barley yellow striate mosaic virus, BYSMV)采用了抑制JAZ蛋白降解的策略, 通过其编码的非结构蛋白P6与寄主植物的COP9复合体的第五亚基CSN5互作, 负调控E3泛素连接酶介导的JAZ蛋白的降解, 抑制JA信号响应, 进而提高对媒介灰飞虱的引诱能力, 促进病毒传播[172].

目前生产上为害水稻的病毒均由稻飞虱或叶蝉以持久型方式传播, 其中白背飞虱传的SRBSDV是当前为害最重的水稻病毒. SRBSDV侵染水稻后, 能增强病株对无毒白背飞虱的引诱能力, 从而有利于无毒媒介从病株上获毒, 促进病毒的传播和扩散[120,121]. 进一步系统研究发现, SRBSDV侵染后对媒介白背飞虱的引诱能力呈动态变化, 即病毒侵染早期, SRBSDV编码的效应蛋白P6主要定位在细胞质, 通过与水稻ET信号通路的负调控因子RTH2互作, 增强ET信号, 进而驱避媒介昆虫白背飞虱, 保护水稻植株免受昆虫过度取食死亡; 而随着病程进展, P6蛋白逐渐向细胞核积累, 并与水稻ET信号通路的关键转录因子EIL2互作, 破坏EIL2的二聚体形成, 抑制其DNA结合活性与转录激活活性, 最终抑制ET信号, 吸引媒介白背飞虱取食获毒, 促进病毒传播, 揭示了SRBSDV通过“推-拉”的方式双向调控水稻ET信号协调病毒侵染与媒介传播, 加速病毒流行的分子机制[92].

5 媒介昆虫调控寄主植物促进病毒流行

昆虫与其寄主植物之间也存在着“军备竞赛”, 即植物具有多种防御昆虫取食和定殖的策略, 包括物理和化学的组成型防御、直接或间接的诱导型防御等; 相应地, 昆虫为了能继续在植物上生存也进化出不同的反防御策略[173]. 唾液被认为是昆虫与植物进行信息交流的主要途径, 唾液中的蛋白能够与植物发生分子互作调节寄主免疫反应, 影响昆虫的生存和种群增长, 间接地促进病毒向寄主植物的水平传播[174].

昆虫唾液中能够减弱或抑制植物防御分泌蛋白被称为效应子. 最早的刺吸式口器昆虫的唾液效应子是从豌豆蚜取食后的蚕豆叶片中检测到的, 并命名为C002蛋白, 该蛋白具有促进豌豆蚜的取食植物的功能[175]; 桃蚜在取食植物时会分泌唾液蛋白Mp55来抑制植物中4-甲氧基吲哚-3-甲基硫代葡萄糖苷、胼胝质和H_2O_2的积累, 从而促进自身的繁殖[176]; 蚜虫唾液中还能分泌MIF蛋白, 通过削弱植株中SA通路的PR1和PR2以及JA通路的PR3的表达, 抑制植物对蚜虫的免疫[177]. 豌豆蚜唾液蛋白Armet和烟粉虱唾液蛋白Bt56, 均能激活植物的SA信号途径, 利用SA和JA信号的拮抗作用, 抑制JA信号介导的抗虫性, 从而提高自身在植株上的取食和生存[178,179]. 烟粉虱感染TYLCV后能诱导分泌蛋白Bsp9的表达, Bsp9进入植物后能破坏WRKY33的转录激活活性, 抑制MAPK免疫通路减弱拟南芥的抗虫反应, 促进病毒传播[180], 同时烟粉虱还能分泌铁蛋白BtFer1, 其具有铁离子结合和铁氧化酶活性, 可以减少植物中H_2O_2的含量来抑制ROS暴发以此促进烟粉虱在植物韧皮部吸食汁液[181]; 黑尾叶蝉唾液效应蛋白NcSP84以及电光叶蝉唾液中的钙结合蛋白(calcium-binding protein, CBP)在取食时分泌到植物中, 与流入筛管的Ca^{2+}结合从而抑制筛管堵塞, 帮助昆虫对植物的连续取食[182,183]; RGDV侵染电光叶蝉后, 通过抑制CBP分泌到植物中, 引起韧皮部胼胝质的沉积, 从而加大昆虫刺探频率, 并分泌更多的唾液进入植物, 最终促进病毒的传播[184]. 灰飞虱分泌的效应子卵黄原蛋白vitellogenin的C端多肽(vitellogenin C, VGC)到水稻植株中, 并与水稻转录因子WRKY71结合, 抑制WRKY71转录激活活性, 有效地削弱了H_2O_2

介导的植物防御[185]，同时灰飞虱唾液蛋白LsSP1能通过破坏水稻半胱氨酸蛋白酶与SA通路的正反馈调控回路进而抑制水稻的抗虫防御[186]. 此外，灰飞虱唾液腺中合成的脱氧核糖核酸酶Ⅱ能够与其他唾液成分一起分泌到植物组织中, 通过降解植物细胞间隙的DNA, 抑制过氧化氢水平和胼胝质积累，帮助灰飞虱取食[187]. 效应子BISP被褐飞虱分泌进入水稻后与水稻细胞质类受体激酶(receptor-like cytoplasmic kinase, RLCK)结合，降低RLCK的激酶活性从而抑制水稻基础防御反应，促进褐飞虱的取食[188]. 由此可见，传播病毒的媒介昆虫通过其唾液分泌的效应子，能通过多种不同的机制抑制植物抗虫免疫，帮助自身的取食和种群增长, 间接地促进所传播病毒的流行.

此外，部分媒介昆虫甚至还能通过唾液蛋白调控植物抗病毒免疫，直接帮助病毒在寄主植物中的侵染. 如由蚜虫唾液蛋白CathB能激活植物活性氧的产生, 虽然这会增强植物的抗虫反应[189], 但有可能会让植物病毒从中受益, 加速病毒的复制[190]. 最新的研究发现, 有翅蚜虫取食还会分泌大量唾液碳酸酐酶(carbonic anhydrase II, CA-II)到细胞质外体, 碳酸酐酶会降低植物质外体的pH值，进而加快囊泡运输，促进非持久性传播的病毒如CMV和TuMV的病毒颗粒从内膜系统向质外体的运输, 帮助病毒更好地侵染, 而无翅蚜虫唾液中CA-II的含量较低，不能很好地促进病毒侵染，这也是田间有翅膀蚜虫传播病毒效率更高的一个原因[191].

6　总结和展望

虫媒植物病毒具有传播快、分布广、防控难的特点，是严重制约农作物的产量与品质的重要因素. 因此，加强虫媒病毒病害循环中病毒-植物-昆虫这三种跨界生物间互作的通讯信号及分子机制研究，是科学绿色防控虫媒病毒病害的基础.

在病毒-植物-昆虫跨界互作体系中，病毒对植物的侵染和媒介昆虫的种群数量、对染病植株的选择、病毒在虫媒中的持留，以及获毒媒介昆虫向健株的转移，都对病毒的流行起了非常重要的作用. 21世纪以来，随着分子生物学的快速发展和应用，人们的研究从病毒结构、基因功能等“点”的研究，扩展到病毒-植物、病毒-昆虫互作“线”的研究，更进一步地到病毒-植物-昆虫多元互作的“面”的研究，极大丰富了人们对病毒、寄主植物、媒介昆虫之间互作关系的认识, 对病毒病害的发病和流行规律有了初步的解析. 但是自然界中，农作物并不是只受到单一病虫害的伤害，实际影响病害流行的还包括多种病原物、作物根际和叶际微生物、昆虫共生菌、非介体昆虫、天敌昆虫，以及包括温度、湿度、光照、养分等在内的非生物因素. 因此, 接下来的工作应该从平面到立体, 系统地探究多病原互作、微生物通讯、环境信号参与的病毒多元互作等方面的分子机制，寻找更贴合田间实际情况的病毒病害防控靶点，建立更高效的综合防控技术体系.

病毒受限于相对较小的基因组，往往编码较少的蛋白，但是利用这些蛋白病毒在与植物、昆虫复杂的互作中发挥了巨大的作用，往往能够操控寄主植物和媒介昆虫促进自身的流行. 本文的大部分研究工作聚焦的都是病毒通过蛋白与寄主植物或媒介昆虫的互作调控，对相关病毒蛋白和寄主因子的研究也为病害的防控提供了一些靶标，如利用基因编辑技术敲除病毒侵染必需的寄主因子，有助于抗病毒材料的培育[192~194]. 此外，利用媒介昆虫的抗性基因，在育种中加以利用, 也能有效阻止病毒病害的流行[195].

在病毒与寄主、媒介复杂的互作网络中，除了蛋白-蛋白互作这种机制, 还存在其他诸多调控机制参与病毒与寄主的博弈. 如非编码RNA，DNA和RNA的表观修饰、组蛋白修饰、昆虫嗅觉和神经系统等都值得深入挖掘，寻找新的病毒-植物-昆虫的互作关键位点. 相信未来随着对病毒-植物-昆虫跨界互作机制的深入了解, 虫媒病毒病害将会被更高效、绿色地防控, 农业生产和粮食安全将得到更坚实的保障.

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Trans-kingdom interactions between viruses-plants-vector insects and viral disease epidemics

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Plant viral diseases seriously restrict the yield and quality of crops. About 80% of the reported plant viruses are transmitted by vector insects. Elucidating the molecular signals and mechanisms of the trans-kingdom interactions between viruses, plants, and vector insects in the disease cycle of arbovirus is the foundation for more efficient control of arboviral diseases. The epidemic scope and intensity of viral diseases mainly depend on the two key steps of virus infection and transmission. With the continuous development of molecular biology, cell biology and various omics technologies, how plant viruses successfully infect host plants and how they efficiently spread through vector insects are constantly analyzed. This article mainly elaborates on the specific mechanisms by which viruses escape plant immunity, break through vector insect barriers and immunity, and directly or indirectly regulate their transmissions by vector insects. It demonstrates the powerful ability of arboviruses to manipulate host plants and vector insects to promote disease epidemics, and also reveals the molecular targets available for the prevention and control of arboviral diseases.

arbovirus, diease epidemics, plant immunity, hormone signaling

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张彤, 华南农业大学植物保护学院教授, 博士生导师. 分别于2007和2012年获浙江大学农学学士学位和理学博士学位, 2012~2015年在美国密苏里大学从事博士后研究. 2016年回国后入职华南农业大学植物病理学系, 2019年入选“广东特支计划”科技创新青年拔尖人才, 2022年获得国家自然科学基金优秀青年科学基金. 张彤教授长期围绕虫媒病毒致病成灾机制开展工作, 探索水稻病毒与寄主植物、媒介昆虫的多元互作分子机制, 研发水稻病毒病害的绿色防控新技术. 发表论文40余篇, 以第一/通讯作者发表在*Molecular Plant*, *Autophagy*, *Plant Biotechnology Journal*等期刊.