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果蔬采后品质安全的分子基础及生物防控技术

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摘 要: 果蔬是膳食结构的重要组成部分, 与国民健康密切相关。然而, 果蔬采后品质劣变和病害不仅导致巨大损失, 而且病原真菌引起的腐烂和产生的毒素是诱发果蔬采后品质安全问题的重要原因。本文将基于团队多年的系统研究工作, 重点阐述果蔬采后品质安全分子基础研究进展, 展望果蔬采后品质维持与安全防控生物技术的应用前景, 为食品安全提供保障。

关键词: 果蔬; 病原菌; 致病基因; 分子基础; 生物防控

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Molecular basis of quality and safety and biological control technology of postharvest fruits and vegetables

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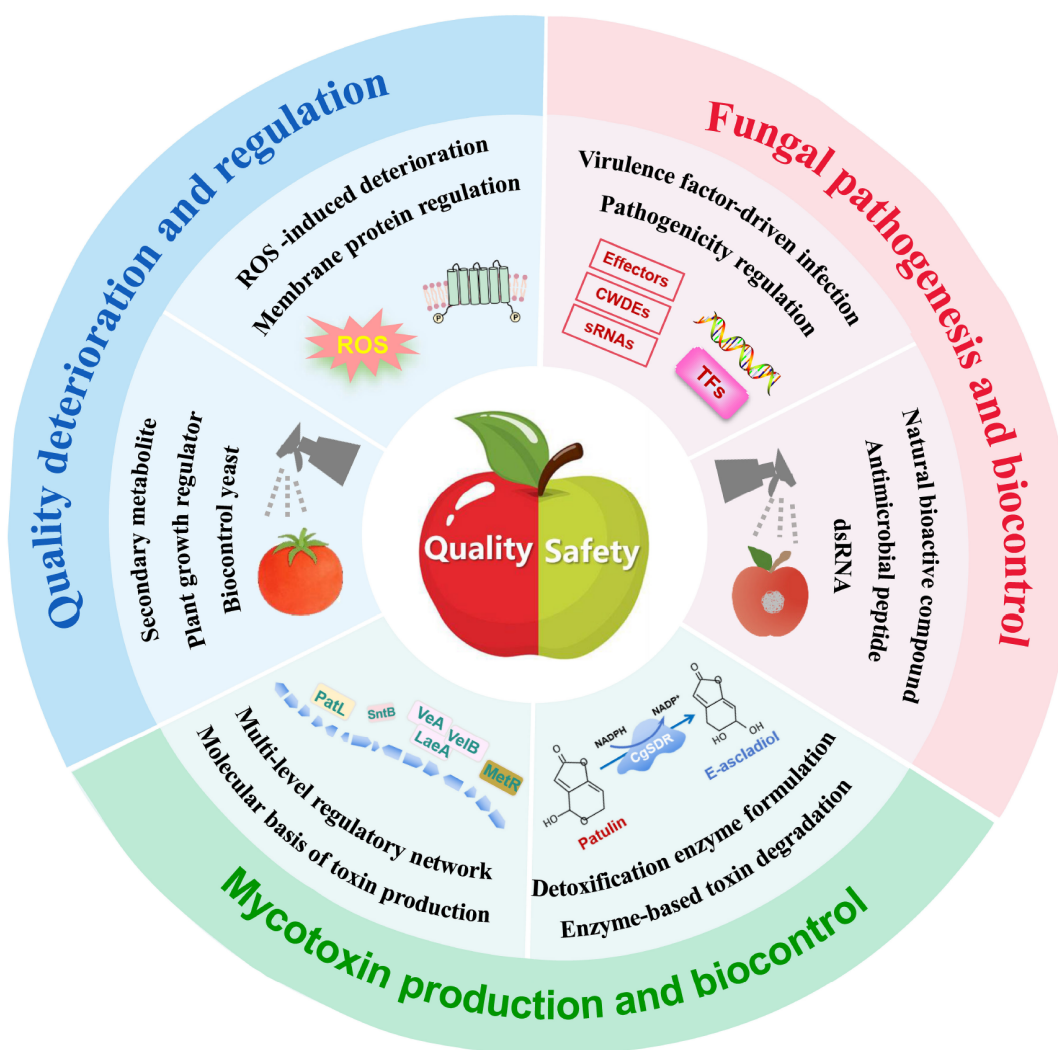
Abstract: Fruits and vegetables are vital for human nutrition and agricultural economies. However, significant postharvest losses occur annually due to physiological deterioration and pathogen infection. Fungal rot and the associated mycotoxin contamination further pose serious food safety risks. Addressing these issues requires a deep understanding of the underlying molecular mechanisms. This review synthesizes recent advances, with emphasis on our systematic research, in elucidating the molecular basis of postharvest quality and safety in produce. It also prospects the development of targeted, eco-friendly biocontrol technologies. The review is structured around three interconnected themes. First, we detail the molecular mechanisms driving postharvest quality deterioration in the host produce. A key focus is the disruption of reactive oxygen species (ROS)

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homeostasis, which accelerates senescence through oxidative damage to mitochondrial proteins, membrane lipid peroxidation, and the oxidation-mediated modification of key transcription factors. We also highlight the role of membrane proteins, such as those from the REM1 family, in stress sensing and in regulating ROS bursts and programmed cell death, processes pivotal for quality preservation. Second, we dissect the molecular basis of pathogenicity and toxigenicity in major postharvest fungal pathogens. This includes the function of virulence factors like cell death-inducing effector proteins and cell wall-degrading enzymes. We summarize how environmental cues are integrated via global regulators to modulate virulence. A highlighted breakthrough is the comprehensive deciphering of the patulin mycotoxin biosynthetic pathway in *Penicillium expansum*. Our work identified the complete *Pat* gene cluster, resolved its intracellular trafficking route, and elucidated its multi-layered regulation by specific transcription factors, global signaling pathways, and epigenetic mechanisms such as histone H3K4 methylation. Third, building on these molecular insights, we discuss the development of novel biocontrol strategies. For quality maintenance, we explore the use of plant-derived elicitors to prime the inherent defense responses of produce. For disease control, promising approaches such as spray-induced gene silencing (SIGS) are examined. For mycotoxin mitigation, we highlight a significant achievement: the identification and characterization of specific microbial short-chain dehydrogenases that efficiently degrade patulin into non-toxic compounds, offering a potent enzymatic decontamination strategy. In conclusion, this review underscores that integrating fundamental research on host physiology and pathogen biology is crucial for innovating postharvest management. Future perspectives include employing multi-omics and artificial intelligence to discover novel targets, advancing gene silencing technologies, and leveraging synthetic biology to engineer enhanced microbial biocontrol agents and horticultural crops, ultimately ensuring a more sustainable and safe food supply chain.



Key words: fruits and vegetables; pathogenic fungi; virulence gene; molecular basis; biological control

果蔬生产与消费是我国农业经济体系的核心组成部分,在国民经济中具有不可替代的战略地位。

据国家统计局数据,2024年全国水果种植面积1.98亿亩,产量达3.3亿吨;蔬菜播种面积3.35亿亩,产量

约7.9亿吨;二者总产值占种植业51%,占据第一产业首位。然而,果蔬采后腐烂损失严重,病原菌致病产毒是诱发食品安全问题的主要因素,新鲜果蔬的品质安全直接影响国民健康。因此,解析果蔬采后品质安全的分子基础,研发精准生物调控技术,对保障消费者膳食安全具有重要意义。本文聚焦近年来果蔬采后品质安全分子基础研究进展,系统阐述果蔬采后品质劣变和病原菌致病产毒的分子机制,并展望生物防控技术在保障食品安全和促进果蔬产业发展方面的应用前景。

1 果蔬采后的主要问题

1.1 衰老促发果蔬采后品质劣变

衰老作为果蔬发育的终末阶段,伴随生理机能的渐进性衰退,主要表现为呼吸作用增强、乙烯释放加速及膜透性增加。呼吸作用增强导致营养消耗加快,乙烯释放则促进果实成熟和衰老,而膜透性增加引发细胞内容物外渗,加速组织崩溃。活性氧(reactive oxygen species, ROS)积累和激素失衡进一步加剧衰老进程,导致果蔬软化、变色和风味丧失。该过程由细胞内源性的有序且不可逆的生物学事件触发,导致代谢模式从合成向分解转变,最终诱导程序性细胞死亡和组织解离^[1,2]。衰老是促发品质劣变的主要诱因,表现为外观色变、风味丧失及营养成分降低。衰老果蔬因酶促褐变和微生物侵染,品质快速下降,货架期大幅缩短。贮藏过程中水分流失和营养消耗导致果蔬失重、萎蔫,商品价值显著降低^[3]。揭示衰老和品质劣变发生的分子机制及确定其启动的关键调控事件,已成为采后生物学的核心方向。

1.2 病害导致果蔬采后巨大损失

病原菌侵染引起的腐烂是造成果蔬采后损失的最主要原因。据统计,我国果蔬腐烂每年的损失率近三分之一,造成严重的浪费和经济损失^[4]。引起果蔬采后病害的病原菌以真菌为主,包括青霉属(*Penicillium*)、灰葡萄孢属(*Botrytis*)、链核盘菌属(*Monilia*)、链格孢属(*Alternaria*)、刺盘孢属(*Colletotrichum*)和镰刀菌属(*Fusarium*)等^[5]。目前,植物病害的防控主要是使用化学农药,而长期使用化学农药防病容易导致病原菌产生抗药性,农残超标不仅污染环境,也诱发食品安全隐患和危害人类健康,成为制约产业发展的瓶颈和社会关注的热点。

1.3 真菌毒素诱发果蔬品质安全

真菌毒素(mycotoxin)是由真菌产生的次生代谢产物,对人和动物具有显著的毒性作用。一些采后病原真菌在侵染果蔬的过程中会产生多种类型的真菌毒素,污染果蔬及其加工产品,对消费者健康构成威胁。由青霉属和曲霉属真菌产生的棒曲霉素(patulin, PAT)和赭曲霉毒素(ochratoxins, OTs),由镰刀菌属等产生的单端孢霉烯族毒素(trichothecenes),以及由链格孢属产生的交链孢毒素(alternaria toxins)是污染果蔬及其加工制品的主要真菌毒素^[5]。真菌毒素具有急性毒性、亚急性毒性与慢性毒性,可导致机体组织与器官损伤,具有致畸、致癌及致突变作用。鉴于真菌毒素的危害性,全球主要国家都对食品中重要真菌毒素的含量制定了严格的限量标准。例如,我国强制性国家标准GB 2761—2017明确规定,以苹果、山楂为原料加工制成的水果制品、饮料及酒类中,棒曲霉素的最高限量标准均为50 μg/kg;欧盟对果蔬制品中棒曲霉素的限量要求更为严格,儿童及婴儿专用食品中最大限量为10 μg/kg。

2 果蔬品质劣变的分子机制及精准调控技术

果蔬衰老导致品质劣变,影响其营养品质和市场价值。果蔬品质劣变是涉及物质与能量代谢多水平变化的复杂生理生化过程,受植物激素、转录因子等内在因素,以及温度、气体成分等环境条件协同调控^[6,7]。深入研究果蔬衰老的诱因及其调控机制,对创制精准的保鲜技术至关重要(图1)。

2.1 活性氧代谢失衡促发果蔬品质劣变

活性氧(ROS)是细胞有氧代谢的副产物,常常受胁迫条件诱导产生,低浓度时作为信号分子调节气孔关闭、胁迫响应等生物学过程^[8]。“自由基”假说提出,胁迫条件下过量积累的ROS将损伤蛋白质、核酸和脂类等生物大分子,使其失去生物活性,表现为膜完整性受损、呼吸链功能紊乱和能量代谢失衡^[9]。果实采后主要以呼吸代谢为主,伴随超氧阴离子和H₂O₂累积。研究表明,ROS稳态的破坏将加速果蔬采后品质劣变,其机制主要涉及生物大分子损伤,如蛋白质、核酸和脂类的氧化降解^[3]。线粒体不仅主导细胞内ROS的生成,同时也是识别和响应氧化应激的重要亚细胞器。研究发现,桃和苹果果实的线粒体蛋白质在衰老过程中发生显著变化:过量累积的ROS诱发果实超氧化物歧化酶、外膜通

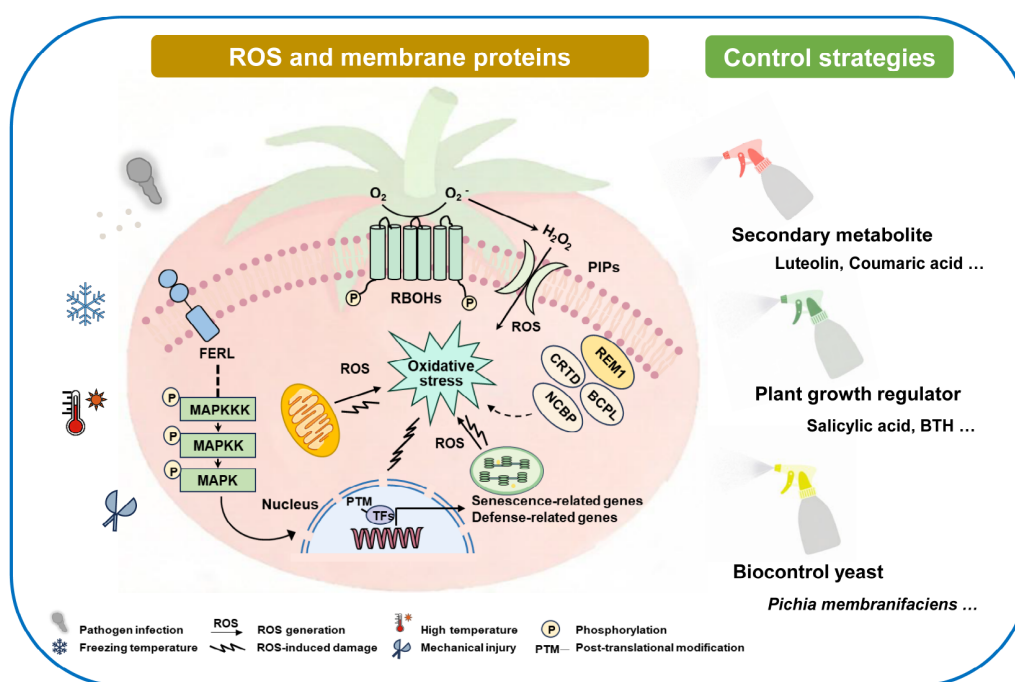


图1 活性氧与膜蛋白协同调控果蔬衰老和抗性的分子机制及精准调控技术

低温、高温、病原菌侵染或机械损伤等胁迫诱导活性氧(reactive oxygen species, ROS)大量积累,造成蛋白质、核酸和脂类等生物大分子氧化损伤,破坏膜完整性、损伤线粒体与叶绿体,并干扰核内基因表达。膜锚定蛋白REM1通过与富含半胱氨酸的跨膜蛋白(CRTD)、蓝铜蛋白类似物(BCPL)及核帽结合蛋白(NCBP)互作,触发ROS爆发;FERL则通过MAPK级联反应调控果实组织中的ROS水平与细胞死亡活性。外源施加木犀草素(luteolin)、香豆酸(*p*-coumaric acid)等次生代谢物,水杨酸(salicylic acid)等植物生长调节剂,以及膜醭毕赤酵母(*Pichia membranifaciens*)等生防酵母,可增强果蔬对氧化胁迫和衰老的抵抗力。

Figure 1 Molecular mechanisms and precise regulation of ROS-membrane protein synergistic regulation in fruit and vegetable senescence and resistance

Stresses (i.e. low temperature, high temperature, pathogen infection, and mechanical damage) induce the excessive accumulation of reactive oxygen species (ROS), which oxidatively damage biological macromolecules including proteins, nucleic acids, and lipids. These damages further disrupt membrane integrity, impair mitochondria and chloroplasts, and interfere with nuclear gene expression. The membrane-anchored protein REM1 (remorin 1) triggers ROS burst through interaction with CRTD (Cys-rich and transmembrane domain-containing protein), BCPL (blue copper protein-like), and NCBP (nuclear cap-binding protein). FERL modulates ROS levels and cell death in fruit tissues via the MAPK cascade pathway. Exogenous application of secondary metabolites such as luteolin and coumaric acid, plant growth regulators such as salicylic acid, and biocontrol yeasts such as *Pichia membranifaciens*, can enhance the resistance of fruits and vegetables to oxidative stress and senescence.

道蛋白等重要线粒体蛋白发生羧基化修饰,导致膜电位改变和外膜破损,引起线粒体功能受损^[9]。而线粒体DNA因无组蛋白保护,更易受ROS攻击,导致突变和编码蛋白功能异常,但相关机制尚需深入研究。这些研究结果明确了线粒体重要功能蛋白的差异表达与果蔬品质劣变密切相关。

此外,蛋白质翻译后修饰(如磷酸化、甲基化、泛素化等)可通过调控蛋白质活性、亚细胞定位及稳定性影响果实品质^[10]。ROS可通过对靶标蛋白中巯基的氧化,介导其发生次磺酸化、亚磺酸化或磺酸化修饰^[8]。灰霉菌侵染可诱导寄主ROS爆发,且ROS的积累水平与特定NADPH氧化酶成员的基因表达

呈正相关;此过程中发生次磺酸化修饰的蛋白质主要参与抗性应答、氧化还原稳态维持以及细胞代谢等功能类群^[10],表明ROS可在转录和翻译后修饰双重层面调控次磺酸化蛋白的表达与功能,进而参与寄主对逆境胁迫的防御响应。膜脂过氧化是ROS引发果实品质劣变的另一关键途径,其通过氧化不饱和脂肪酸启动自由基链式反应,生成丙二醛等中间产物,破坏生物膜的结构与功能^[11,12]。同时,ROS亦是转录因子调控网络中的重要组分,例如荔枝果实中LcNAC1与LcWRKY1通过拮抗作用调控交替氧化酶基因的表达,从而影响ROS的动态平衡^[13]。ROS还可介导番茄和香蕉果实中NAC类转录因子

的甲硫氨酸氧化修饰,进而调节果实衰老进程。其中,甲硫氨酸(Met)被氧化为甲硫氨酸亚砷(Met-SO)的过程可由甲硫氨酸亚砷还原酶(Msr)逆转;研究表明,番茄MsrA家族蛋白E4和MsrB家族蛋白2能够修复NAC类转录因子NOR的氧化损伤,并恢复其DNA结合能力^[14]。

2.2 膜蛋白对果蔬品质维持的重要作用

细胞质膜是植物与外界环境因子相互作用的重要场所^[15,16]。桃果实采后品质变化的多组学分析结果显示,0℃贮藏条件下,细胞膜中亚麻酸和磷脂酰乙醇胺的累积有助于增强桃果实的采后抗冷性,说明特定膜组分的含量构成也是一个关键抗逆因素^[12]。膜蛋白作为园艺作物识别并应答环境变化的关键因子^[17],在果蔬品质维持过程中发挥着重要作用。有研究表明:过表达膜蛋白REM1的番茄组织中ROS积累水平显著提升;而差异蛋白质组学分析显示,REM1能够与细胞死亡相关调控因子富含半胱氨酸的跨膜蛋白(CRTD)、蓝铜蛋白类似物(BCPL)和核帽结合蛋白(NCBP)相互作用,诱导细胞ROS爆发^[18]。在辣椒中,CaRIN4-12能够抑制由CaREM1.4引起的ROS爆发及细胞坏死,说明CaREM1.4是组织中氧化胁迫的正调控因子,而CaRIN4-12通过与CaREM1.4的结合发挥负调控作用^[19]。番茄类受体激酶FERL位于胞内的激酶结构域能够与MAP3K18相互作用并使之磷酸化,进而直接影响MAPK级联反应的活性,通过MAP2K2/MAP2K4精细调控叶片与果实组织中的ROS水平与细胞死亡活性^[20]。除了上述定位于细胞质膜的膜蛋白之外,细胞内膜系统蛋白在对环境信号的应答反应中也发挥关键作用。可以预见,蛋白质磷酸化、泛素化等翻译后修饰对膜蛋白活性及功能的调控机制将是今后果蔬采后品质维持机制研究的一个重要方向。

2.3 果蔬品质劣变的精准调控技术

诱导果蔬自身抗性是通过靶向激活其内在防御机制来延缓采后品质劣变的创新方法。这种方法利用生物源活性物质、生物激发子等外源处理,增强果蔬对氧化应激和衰老的抵抗力,从而提升果蔬的保鲜效果^[21]。当受到不利环境因素影响时,果蔬自身能够合成多酚、黄酮、萜类和生物碱等次生代谢产物,以增强其抵御环境胁迫的能力^[2,22]。生物胁迫和非生物胁迫均能诱导葡萄糖酚和类黄酮含量显著升高,而抗性苹果品种也能够通过调节酚类代谢增

强抗性。因此,利用具有诱导抗氧化能力和提高抗性水平的生物源物质可以有效延缓果蔬品质劣变进程^[17,23]。香豆酸(*p*-coumaric acid)能够延缓樱桃果实品质劣变,延长果实贮藏时间,主要归功于香豆酸能激活果实苯丙烷代谢途径,抑制细胞壁降解相关酶活性,并提高非酶促抗氧化剂含量和主要抗氧化酶活性^[24]。木犀草素(luteolin)则通过在转录水平和翻译水平影响苯丙烷代谢途径中的关键酶活性及关键基因表达水平诱导果实抗性,从而保持采后贮运过程中樱桃果实的品质,并通过调控抗氧化酶活性,提高总花青素和类黄酮的含量,进而提高果实抗氧化能力^[25]。

水杨酸(salicylic acid, SA)及其结构类似物能够激活采后果蔬防御反应,包括细胞壁增厚、抗性基因表达及代谢重编程等生物学过程^[26,27]。SA不仅作用于其合成部位,还能够在远端组织诱导系统获得性抗性。外源施加SA或其结构类似物,能有效诱导局部和远端的抗性响应。例如,膜黥毕赤酵母(*Pichia membranefaciens*)与0.5 mmol/L水杨酸协同处理采后甜樱桃果实,能显著降低果实品质劣变,其作用机制主要涉及提高抗氧化酶活性、改变过氧化物酶同工酶谱,以及上调抗氧化蛋白表达^[28]。苯并噻重氮(ASM)作为水杨酸的结构类似物,可诱导多种果蔬的抗性;此外,苯并咪唑啉啶(BTH)处理还能促进梨、桃、草莓和甜瓜等果实中H₂O₂的生成,增强超氧化物歧化酶活性,同时抑制过氧化氢酶及抗坏血酸过氧化物酶活性,并促进酚类、黄酮和木质素等非酶活性物质的积累,激活苯丙烷代谢途径^[29]。

3 病原菌致病的分子机制及生物防控技术

在果蔬采后病害发生过程中,病原菌通过分泌到胞外的致病因子破坏寄主的防御结构和免疫系统,进而在果蔬表面定殖并侵染,引起腐烂。病原菌的致病力受内源调控因子和外源环境因子的影响,因此解析病原菌致病过程中的关键基因及其分子调控网络,不仅为阐明病原菌复杂的侵染机制提供分子证据,也为研发病害生物防控技术提供关键靶点(图2)。

3.1 病原菌的致病因子

果蔬采后病原真菌多为坏死型病原菌,通过杀死寄主细胞并获取营养来完成定殖。病原菌在侵染早期可通过分泌效应因子(effectors)进入寄主质外

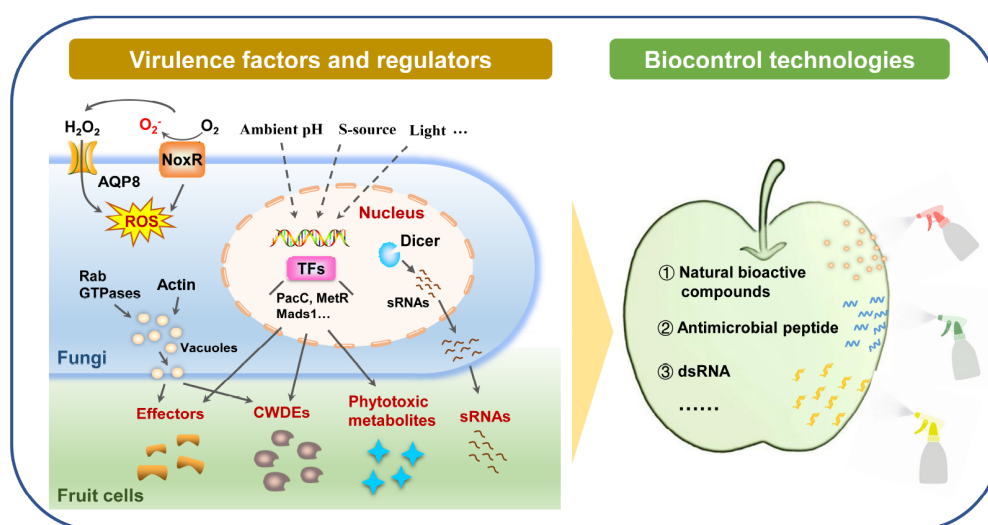


图2 果蔬采后病原菌致病的分子机制及生物防控技术

在果蔬采后病害发生过程中,病原菌通过分泌效应因子(effectors)、细胞壁降解酶(CWDEs)、小分子代谢物(phytotoxic metabolites)及小RNA(sRNAs)等多种致病因子,破坏寄主组织结构并抑制其免疫反应。病原菌的致病力不仅受囊泡运输介导的致病因子分泌、ROS的合成转运等内部精密调控机制的影响,还受到组织pH、寄主硫源和光信号等外部环境信号构成的复杂调控网络的调节。基于对上述致病机制的深入解析,发展出一系列新兴绿色防控策略,主要包括利用天然活性物质与抗菌肽实现多靶点抑制病原菌,以及应用喷施诱导基因沉默(spray-induced gene silencing, SIGS)等技术进行分子层面的精准干扰。

Figure 2 Molecular mechanisms and biocontrol technologies of postharvest pathogen infections in fruits and vegetables

During the occurrence of postharvest diseases in fruits and vegetables, pathogens disrupt host tissue structure and suppress immune responses by secreting various virulence factors, including effectors, cell wall-degrading enzymes (CWDEs), phytotoxic metabolites, and small RNAs (sRNAs). Pathogen virulence is regulated not only by intricate internal mechanisms, such as vesicle transport-mediated secretion of virulence factors and the synthesis and transport of reactive oxygen species (ROS), but also by a complex network of external environmental signals, including tissue pH, host sulfur sources, and light. Building on an in-depth understanding of these pathogenic mechanisms, a series of emerging green control strategies have been developed. These primarily involve the multi-target inhibition of pathogens using natural active substances and antimicrobial peptides, as well as molecular-level precision interference through technologies such as spray-induced gene silencing (SIGS).

体或细胞内,操纵寄主的免疫反应或代谢通路来促进侵染。细胞死亡诱导蛋白(cell death-inducing proteins, CDIPs)是采后病原真菌中鉴定到的主要效应蛋白类型。灰霉菌(*Botrytis cinerea*)分泌的BcSSP2能够进入植物细胞,诱发植物的坏死反应^[30],并通过其N端结构域与核孔复合体核心组分NUP62相互作用,抑制NUP62的相分离,进而影响MPK3的核质运输。指状青霉(*Penicillium digitatum*)分泌的PdCDIE1通过与柑橘热激蛋白CsHsp70互作,干扰钙调素(calmodulin, CaM)与靶蛋白的结合以及ROS平衡,进而导致细胞死亡^[31]。此外,采后病原真菌分泌的一些水解酶也具有诱导寄主细胞死亡的作用,这种诱导作用可以依赖(例如BcRAE和BcFAT)或不依赖(例如BcXYG1)其水解酶活性^[32,33]。

采后病原菌在侵染过程中还会分泌大量的胞外

酶,破坏寄主表面的角质层等抗性结构,分解细胞壁中的纤维素、半纤维素和果胶等组分,引起细胞崩解,产生腐烂症状^[34]。*B. cinerea*和扩展青霉(*Penicillium expansum*)基因组编码的胞外蛋白有1 000余种,其中主要为多糖水解酶、蛋白酶、氧化还原酶等胞外酶^[35]。其中,角质酶、纤维素酶、半纤维素酶、果胶酶等是采后病原真菌最主要的致病因子。例如,*B. cinerea*可以产生6种内切多聚半乳糖醛酸酶(polygalacturonase, PG),用于降解果胶的主要成分多聚半乳糖醛酸^[36]。其中,*BcPG1*和*BcPG2*的表达在侵染过程中被强烈诱导,两个基因的单独敲除均会导致*B. cinerea*致病力显著下降。

除了蛋白质类的致病因子外,病原真菌产生的一些小分子代谢物(毒素、有机酸、氨等)也在致病过程中发挥重要作用。*B. cinerea*产生的灰霉二醛

(botrydial)可以导致植物组织萎蔫、褪绿^[37]。与之不同,病原菌分泌的有机酸和氨不是通过细胞毒性行使功能,而是通过改变寄主组织pH、诱导细胞壁降解酶的表达和提高酶活性等方式发挥作用。不同病原菌采用的改变组织pH的策略不同,*B. cinerea*和核盘菌(*Sclerotinia sclerotiorum*)主要产生草酸,青霉菌主要产生葡萄糖酸和柠檬酸来酸化寄主组织,而炭疽菌(*Colletotrichum* spp.)和链格孢(*Alternaria* spp.)则通过分泌氨来碱化寄主组织^[36,38]。

小RNA(small RNAs, sRNAs)是一类具有调控基因表达功能的短链非编码RNA,是病原真菌中发现的新型致病因子。*B. cinerea*通过胞外囊泡分泌sRNAs,然后通过网格蛋白(clathrin)介导的内吞作用将sRNAs递送到寄主细胞中,并通过与AGO1蛋白结合劫持植物的RNAi系统,进而抑制寄主的防御反应^[39,40]。病原真菌与寄主互作过程中sRNAs的递送是双向的,即寄主来源的sRNAs也可以进入病原真菌的细胞中,降低病原菌的致病力^[41]。sRNAs跨界传递机制的发现不仅为理解病原菌与寄主之间复杂的互作机制提供了新的视角,也为提出病害防控新策略提供了理论依据。

3.2 病原菌致病的分子机制

病原菌分泌的蛋白类致病因子在内质网中折叠和修饰后被装载到囊泡中,经过分泌途径运输到胞外。Rab家族的小G蛋白对囊泡运输具有关键调控作用。*B. cinerea*的Rab家族蛋白有10个成员,在囊泡运输的不同阶段发挥作用。其中,Bcsas1/Rab8缺失后分泌囊泡不能与质膜融合,破坏了致病因子的胞外分泌^[42]。肌动蛋白(actin)也对囊泡分泌过程有重要影响。敲除*B. cinerea*中编码肌动蛋白的基因*BcactA*影响了40多个胞外蛋白的分泌,包括致病因子纤维二糖水解酶(BcCBH)和鼠李糖苷酶(BcRHA)^[43]。

病原菌产生的ROS主要在病原菌的生长发育和与寄主的互作过程中发挥作用。NADPH氧化酶复合物(NADPH oxidase complex, NOX)是真菌ROS产生的主要来源之一,它由催化亚基和多个调节亚基组成。*B. cinerea*中的NOX有两个催化亚基BcNoxA和BcNoxB,分别在侵染过程的定殖初期和扩展阶段发挥作用^[44]。BcNoxR是BcNoxA和BcNoxB的共同调节亚基,对两者催化产生ROS的功能具有重要影响。在*BcNoxR*敲除突变株中,ROS产量显著降低,ROS在菌丝中的极性分布状态也发生变化^[45]。同

时,BcNoxR还能通过调节下游的6-磷酸葡萄糖酸脱氢酶等影响*B. cinerea*的发育和致病力^[43]。除BcNoxR外,*B. cinerea*中已被鉴定的重要NOX复合体亚基还包括BcNoxD、BcPls1、BcBemA和BcCdc24等^[36]。转运途径也对ROS介导的病原菌致病力调控具有影响。细胞膜上的水通道蛋白Aquaporin可以跨膜转运H₂O₂。在灰霉菌中共鉴定到8个水通道蛋白,其中AQP8可以向细胞内转运H₂O₂,调控ROS在菌丝顶端的极性分布和侵染结构的形成^[46]。

环境信号对病原菌的致病力具有重要的调控作用。环境pH通过调控蛋白质的合成来影响*P. expansum*孢子的萌发^[47],同时影响*B. cinerea*分泌的胞外水解酶种类^[48]。在真菌中,环境pH信号转导途径的核心转录因子PacC可以增强*B. cinerea*中草酰乙酸乙酰水解酶和*P. expansum*中葡萄糖酸氧化酶编码基因和蛋白质的表达,促进草酸和葡萄糖酸的合成及在腐烂组织中的积累,进而增强病原菌的致病力^[49-51]。硫源是病原菌侵染寄主过程中的关键营养物质之一,参与多种细胞过程。在*P. expansum*中,bZIP转录因子PeMetR作为硫同化途径的正向调控因子,通过调控无机硫同化影响有机硫的合成,进而调控其生长与致病力^[52]。光信号对真菌生长发育、有性和无性繁殖以及次级代谢等方面都具有重要影响。在*B. cinerea*中,已经鉴定到的光信号途径重要调控因子包括VELVET复合体、BcWCL1/2、BcLTF1/2和Bcmads1等^[53]。最近研究发现,光信号途径对脂肪酸代谢和辣椒碱合成具有重要影响。环丙烷脂肪酸合成酶基因*BcCfaS*是受Bcmads1调控的靶基因,其表达受到光信号的强烈诱导,*BcCfaS*通过影响茉莉酸甲酯等重要脂肪酸的合成,调控*B. cinerea*的生长、发育和致病力^[54]。另一个受Bcmads1调控的靶基因*BcAMT1*是编码催化辣椒碱前体物质香兰素胺合成的关键酶,该基因以光依赖形式介导辣椒碱的生物合成,进而影响*B. cinerea*的光形态建成和致病力^[55]。

3.3 果蔬采后病害生物防控技术

传统化学农药防病容易导致农残超标和环境污染,诱发食品安全问题,因此,精准的靶向防控策略已成为当前的重要发展方向。研究表明,多种植物源和微生物源抑菌物质均能有效控制果蔬采后病害,如和厚朴酚、1-辛烯-3-醇、无患子皂苷及3-羟基香豆素等植物成分^[56-59],以及雷帕霉素、纳他霉素、

ϵ -聚赖氨酸和2,3-丁二酮等微生物代谢产物^[60-63]。这些化合物可通过诱导ROS积累、破坏病原菌细胞膜结构、干扰能量代谢和诱发细胞自噬等多靶点机制,高效抑制*B. cinerea*和*P. expansum*等多种采后病原菌^[64]。目前研究进一步聚焦于多种活性物质的复配协同增效,并借助微胶囊、缓释膜材等新型剂型技术,以提升其稳定性,延长作用时效。

抗菌肽作为一种新兴的靶向抑菌工具,近年来备受关注。这类来源于拮抗微生物和动植物的小分子肽,可通过破坏细胞膜完整性、抑制核酸或蛋白质合成以及干扰酶活性等机制,对*B. cinerea*、*P. expansum*、*P. digitatum*和*A. alternata*等病原菌具有显著的抑制作用^[65-68]。尽管大规模应用抗菌肽防病仍存在筛选难度大和生产成本高等主要瓶颈,但借助于合成生物学与基因编辑策略,有望实现规模化生产与功能优化,进而推动其商业化进程。

喷施诱导的基因沉默(spray-induced gene silencing, SIGS)技术代表了分子层面精准防病的前沿方向。该技术通过外源递送靶向病原菌关键致病基因的双链RNA(dsRNA),特异性沉默其基因表达,从而削弱病原菌的致病力。有研究表明, SIGS技术对灰霉病和镰刀菌枯萎病等多种采后病害具有显著的防控效果^[69,70]。该技术具备高度特异性且环境友好,然而其实际应用的关键在于开发出稳定且高效的RNA递送载体,并降低生产成本。同时,在应用前还需评估和克服对非靶标生物的潜在脱靶效应。

4 病原菌产毒的分子基础及生物防控技术

近年来,真菌毒素污染引起的食品安全问题越来越受到社会关注。棒曲霉素是造成果蔬及其加工制品污染的最重要的真菌毒素。通过十多年的系统研究,目前不仅在解析棒曲霉素生物合成的分子基础、合成转运的分子路径和降解机制等方面取得了重要进展,同时还研发了降解棒曲霉素的生物酶制剂(图3)。

4.1 棒曲霉素生物合成的分子基础

棒曲霉素,又称展青霉素,是一种结构简单、性质稳定的聚酮类化合物。棒曲霉素最早作为潜在的抗生素而被人们重视,其生物合成的生化途径在20世纪70~80年代被解析,但第四步反应是否存在分支途径长期以来仍存在争议^[71]。真菌毒素等次生代谢产物生物合成途径的相关基因一般在染色体上

以基因簇的形式存在^[72],该特征可用于在基因组水平预测和鉴定毒素合成基因簇。2015年,本课题组完成了产生棒曲霉素的主要病原菌*P. expansum*的全基因组测序^[35]。从预测到的55个次生代谢基因簇中鉴定到一个全长约41 kb的棒曲霉素合成基因簇,由15个基因(*PatA~PatO*)组成,包括11个编码催化酶的基因、1个编码转录因子的基因和3个编码转运蛋白的基因。转录组和单基因敲除分析表明,这15个基因均与棒曲霉素的合成密切相关,其中8个基因敲除后*P. expansum*完全不能产生棒曲霉素。绿色荧光蛋白(GFP)标签技术与细胞器特异性荧光探针共定位研究发现,棒曲霉素合成基因簇编码的15个蛋白分别定位于细胞质、细胞核、内质网、液泡、细胞膜和细胞壁等不同的亚细胞位点,行使催化、转运、调控等不同功能^[73]。由此,明确了棒曲霉素在细胞内的合成转运路径是细胞质-内质网-液泡-细胞质-细胞壁,并由跨膜蛋白转运到胞外,且合成途径的最后一步在细胞外完成,从无细胞毒性物质E-ascladiol转变为棒曲霉素。同时,基于底物饲喂、异源表达和体外催化三种方法,探明了所有催化酶基因在棒曲霉素合成途径中的定位及其催化产物,特别是明确了第4步反应产物为龙胆醇,而不是间羟基苯甲醛,解决了长期以来关于该分支途径的争议。

4.2 棒曲霉素生物合成的调控机制

棒曲霉素的生物合成受到特异性因子、全局性因子以及表观遗传因子等多层次的调控^[74]。棒曲霉素基因簇编码的C6型转录因子PePatL是棒曲霉素合成途径的特异性转录因子,其编码基因敲除可抑制基因簇所有基因的表达,导致*P. expansum*产毒能力丧失^[35]。一些响应环境信号的调控因子也在棒曲霉素的生物合成调控中起到关键作用。*P. expansum*中pH信号途径转录因子PePacC缺失可导致棒曲霉素基因簇所有基因表达下调,棒曲霉素产量下降^[49,75]。受PePacC直接调控的离子泵Ena家族蛋白也参与调控棒曲霉素生物合成途径^[76]。在真菌中,光信号转导途径调控因子Velvet复合体由LaeA、VeA和VelB组成,可在转录水平上调控棒曲霉素的生物合成^[35,77]。此外,碳源信号途径转录因子CreA和硫源信号途径转录因子MetR均对棒曲霉素合成具有正调控作用^[52,78]。

染色质状态在真菌次级代谢基因簇的表达调控中起到关键作用,研究发现组蛋白修饰对真菌毒素

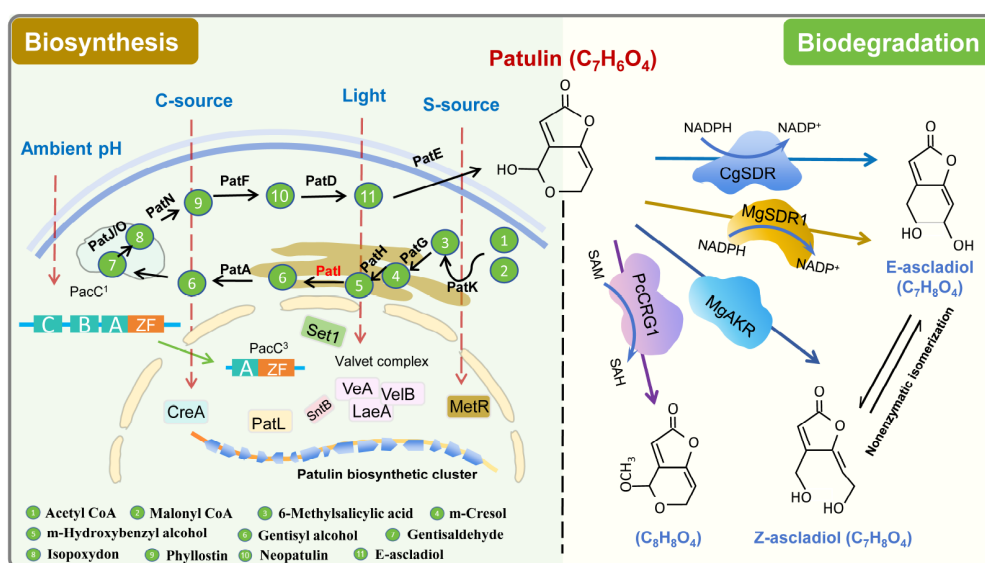


图3 扩展青霉中棒曲霉素的合成调控及生物降解技术

棒曲霉素生物合成涉及15个基因,经历至少10步酶促反应,其在细胞内的合成从细胞质开始,经内质网、液泡、细胞质、细胞壁后由跨膜蛋白转运到细胞外。棒曲霉素的生物合成受控于一套多层次的调控网络,合成途径特异性调控因子、环境信号-全局性调控因子以及一些表观遗传因子都参与其中。因具有高效、专一、不影响产品品质等优点,基于生物酶的毒素降解技术展现出良好的应用前景,一些微生物来源的棒曲霉素降解酶逐渐被分离鉴定,它们能够将棒曲霉素降解成低毒或无毒的降解产物,实现解毒目的。

Figure 3 Biosynthesis regulation of patulin in *Penicillium expansum* and its biodegradation technologies

The biosynthesis of patulin involves 15 genes and undergoes at least 10 enzymatic reactions. Its intracellular synthesis initiates in the cytoplasm, proceeds through the endoplasmic reticulum, vacuoles, cytoplasm, and cell wall, and is then transported to the extracellular space via transmembrane proteins. Patulin biosynthesis is governed by a multi-level regulatory network, in which pathway-specific regulators, environmental signal-responsive global regulators, and several epigenetic factors are all involved. Owing to the advantages of high efficiency, strong specificity, and no adverse effects on product quality, enzyme-based toxin degradation technology exhibits promising application prospects. Several patulin-degrading enzymes derived from microorganisms have been gradually isolated and identified, these enzymes can degrade patulin into low-toxic or non-toxic metabolites to achieve detoxification.

的生物合成具有重要影响^[79]。真核生物中保守的 Set1/COMPASS 复合体负责组蛋白 H3K4 甲基化^[80]。*P. expansum* 中 *PeSet1* 敲除后棒曲霉素基因簇中 *PatK*、*PatG* 和 *PatL* 等基因启动子区的组蛋白甲基化水平下降,基因表达水平降低,毒素产量仅为野生型菌株的 4%^[81]。进一步研究发现,Set1/COMPASS 复合体中其他六个组分的缺失也对棒曲霉素的合成具有重要影响^[82]。此外,与组蛋白乙酰化修饰相关的表观修饰识别蛋白 SntB 也参与棒曲霉素的合成调控,但具体机制仍需进一步分析^[83]。

4.3 棒曲霉素的生物防控技术

P. expansum 产生的棒曲霉素主要污染果蔬及其加工产品,因此,防控青霉病发生是从源头有效控制棒曲霉素污染的重要举措。如果含有棒曲霉素的果蔬原料进入加工环节,引起加工制品的污染,则需要

脱除技术来消减毒素的危害。近年来,棒曲霉素生物降解技术得到快速发展。与生产上使用的物理吸附技术相比,生物降解技术具有效率高、专一性强、不影响产品品质的优点。本课题组利用定量蛋白质组学技术,从能降解棒曲霉素的季也蒙假丝酵母 (*Candida guilliermondii*) 中发现一个受毒素强烈诱导的短链脱氢酶 CgSDR^[84]。体外表达并纯化的 CgSDR 可将棒曲霉素降解为无细胞毒性的产物 E-ascladiol。结构预测和点突变分析发现,CgSDR 氨基酸序列中的 Val¹⁸⁸ 和 Thr¹⁹⁰ 在酶与棒曲霉素的结合中起到关键作用,对毒素降解能力有重要影响^[85]。与游离酶相比,磁性 Fe₃O₄ 颗粒固定化技术可显著提升 CgSDR 的稳定性和重复使用性,使其能够高效降解苹果汁中的棒曲霉素,且对果汁品质没有影响^[86]。最近,从季也蒙迈耶氏酵母 (*Meyerozyma*

guilliermondii)中也鉴定了一个对棒曲霉素具有降解作用的短链脱氢酶MgSDR1^[87]。此外,从胶红酵母(*Rhodotorula mucilaginosa*)中分离到的乳清酸磷酸核糖转移酶^[88],从卡利比克毕赤酵母(*Pichia caribbica*)中分离到的S-腺苷蛋氨酸依赖的甲基转移酶^[89],以及从酿酒酵母(*Saccharomyces cerevisiae*)和季也蒙迈耶氏酵母中分离到的醛酮还原酶^[90,91],也被报道具有降解棒曲霉素的作用。

5 展望

对果蔬采后品质劣变和病原菌致病产毒的分子机制及调控网络的深入认知,为果蔬采后品质安全防控新技术的研发提供了新思路。绿色安全生物防控技术的应用,可以减少果蔬采后腐烂损失,降低农残和真菌毒素污染,为确保果蔬产品采后品质安全和推动果蔬产业发展奠定基础。当然,随着科技的快速发展和学科的融合交叉,未来的研究重点包括以下几个方面:

(1)基于多组学方法,阐明果蔬衰老与抗性应答的分子关联机制,揭示病原菌和果蔬寄主互作过程中sRNA跨界双向递送机制,通过寄主诱导的基因沉默(host-induced gene silencing, HIGS)和SIGS技术,发掘调控果蔬免疫与抗性应答的关键靶点。

(2)利用生物信息学技术,系统研究病原菌致病产毒关键基因涉及的代谢途径和分子调控网络,通过AlphaFold等人工智能技术和结构生物学,解析关键靶点的高精度三维结构,筛选高效特异性的小分子化合物和小肽抑菌剂,研发高效的生物防控技术。

(3)利用基因编辑技术和合成生物学方法,提出优良性水果蔬菜品种的改良策略,定向改良果蔬营养品质与抗性,培育多功能的新品种,实现果蔬优质与高产的协同发展。

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