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香蕉枯萎病菌致病机理研究进展

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摘 要:香蕉是世界重要的水果作物和第四大粮食作物,也是世界贸易第一大宗水果,具有很高的经济价值,然而目前由尖孢镰刀菌古巴专化型(*Fusarium oxysporum* f. sp. *cubense*)引起的香蕉枯萎病正严重威胁着全世界的香蕉生产。由于病原菌通过土壤传播,迄今该病害还没有有效的防治措施,且其致病机理尚未完全明确。本文对香蕉枯萎病菌的遗传多样性及其致病机理研究进展系统进行概述,以期为香蕉枯萎病的综合防控提供理论依据。

关键词:香蕉枯萎病;尖孢镰刀菌古巴专化型;遗传多样性;致病机理

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Research progress in pathogenic mechanism of *Fusarium oxysporum* f. sp. *cubense*

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Abstract: Banana is the fourth largest food crop and the largest commercial fruit in the world. However, banana Fusarium wilt, caused by *Fusarium oxysporum* f. sp. *cubense* (*Foc*), has been seriously threatening banana industry worldwide. To date, three physiological races of *Foc* have been identified worldwide, of which tropical race 4 (TR4) is the most destructive pathogen and has significant impacts on banana production, and it is also the main pathogen of banana Fusarium wilt in China. The genetic diversity of *Foc* is different among different physiological races, and *Foc* has more genetic diversity within race 1 isolates than within race 4 isolates. All TR4 isolates belong to VCG01213/01216 and were identified with the same genetic lineage. As the typical representative of *F. oxysporum* species complex (FOSC) and the model fungus of soil-borne disease, *Foc* has been studied by scientists all over the world. However, the research on the pathogenic mechanism of *Foc* is relatively backward compared with that of *F. oxysporum* f. sp. *lycopersici*. Most of the current results on pathogenicity mechanism in *Foc* were sumarized from the results of studies on other form a specialis of *F. oxysporum*. However, because of the complex genetic background of FOSC, even different races and different VCG isolates possess distinct pathogenicity differentiation, the results of the other forma specialis of *F. oxysporum* are not completely applicable to *Foc*. So, it's necessary to conduct correlated research work on *Foc*. The process of infection by *F. oxysporum* can be divided into several steps: recognition between pathogen and host, attachment to root surface, penetration, colonization of the root cortex and finally presentation of diseased symptom on host plant. Based on the infection process, recent research results in the molecular mechanism of *Foc* were reviewed. 1. Recognition between pathogen and host plant, the first step in pathogenesis, is important for pathogen invading host plant. Researches on *Foc* showed that α-1,6- man-

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nosyltransferase Och1 and β-1,3-mannosyltransferase Gas1 were involved in fungal cell wall integrity, could affect the pathogen's attachment to the root of the host,then reduce the mycelia invasion and the virulence of *Foc*. Two mitogen-activated protein kinases (MAPK) signaling pathways represented by *Fmk1* and *Mpk1* were found to be highly correlated with cell wall integrity in *F. oxysporum*. Researches on *Foc* also confirmed that the gene (in the *Mpk1* signaling pathway) deletion mutants (ΔFoBck1, ΔFoMkk2 and ΔFoSlt2) showed more sensitive to cell wall inhibitors, Congo red (CR) and Fluorescent white (CFW), than that of wild type, indicating that the integrity of the mutants cell wall was destroyed. In addition, *FoSlt2* (homologue of *Mpk1*) knockout mutant also showed decreased virulence in Cavendish banana which further verified the relationship between cell wall integrity and pathogenicity. These results suggest that cell wall integrity is an important factor for the attachment and invasion of *Foc* during the recognition process. 2. Cell wall degrading enzymes (CWDEs), secreted by *F. oxysporum*, play an important role in invasion and colonization of pathogenesis. However, deletion of individual CWDE-encoding genes did not affect the virulence possibly because of functional redundancy. It is suggested that the pathogenicity is the result of the synergism of several synthesis genes of CWDEs. A protein kinase Snf1 isolated from *F. oxysporum* was identified as a transcriptional regulator and affects the activity of CWDEs. A transcriptional coactivator FoHfi1 in *Foc* was reported, and the encoding gene is located in the upstream of *Snf1* and affects the activity of CWDEs. 3. Fungal toxin plays an important role in colonization of pathogenesis. It was reported that fusaric acid (FA) was the main component of *Foc* crude toxin as well as beauverin and fumonisin. FA biosynthetic gene (FUB) cluster have been reported in *F. oxysporum*. The cluster was consisted of at least 12 genes. And among them nine genes (including two zinc finger transcription factors) were essential for the biosynthesis of FA. 4. Comparative genomics analysis has revealed that the genome of *F. oxysporum* is compartmentalized into two regions. One is the core genomic region which is responsible for essential functions such as basic metabolism and reproduction. And the other is accessory genomic region which is responsible for the host specialization and pathogen virulence. Genome compartmentalization provides new insight into how different strains adapted to different hosts which is also helpful for identification of new pathogenicity-related genes in *Foc*. 5. Some other pathogenecity factors have been reported in *Foc*, such as the G-protein subunits FGA1, FGA2 and FGB1 which may affect the virulence potentially via the cAMP-dependent protein kinase pathway. These results provide information for elucidating the role of G protein signaling pathway in the pathogenesis of *F. oxysporum*. In this paper, genetic diversity and molecular mechanism of pathogenesis in *F. oxysporum* f. sp. *cubense* were summarized. The deciphering of pathogenic mechanism will help us to identify new pathogenicity-related genes in *Foc*, and the identification of key pathogenic factors involved in pathogenesis of *Foc* will provide some targets for effectively controlling the banana Fusarium wilt.

Key words: Banana Fusarium wilt; *Fusarium oxysporum* f. sp. *cubense*; Genetic diversity; Pathogenic mechanism

香蕉，主要种植在热带地区，是世界贸易量最大宗的水果，也是联合国粮农组织认定的发展中国家继水稻、小麦、玉米之后的第四大粮食作物。我国是世界第二大香蕉生产国，也是全球香蕉的主要消费国，如今香蕉已经成为我国热带亚热带地区农业支柱性产业之一，为我国国民经济的增长做出了重要的贡献[1]。2011年香蕉和大蕉的全球总产量约为1.45亿t，总产值为441亿美元，大约15%的产品出口到国际市场，其余的85%只在当地市场销售[2]。据统计2017年全球香蕉出口总值为124亿美元[3]，如果按15%的出口份额来计算，则全球香蕉总产值达827亿美元，足见其在世界香蕉生产国经济发展中

的重要性。

然而，被人们称为“香蕉癌症”的香蕉枯萎病正严重威胁着世界香蕉产业的发展，造成的损失已超过4亿美元，制约着全球范围内的香蕉生产与贸易[4-5]。香蕉枯萎病又称香蕉黄叶病，起源于东南亚，1910年在中南美洲的巴拿马大流行，因此又被称为“巴拿马病”（Panama Disease），是一种侵染维管束的土传病害，蔓延快，根治难[6]。至今该病已在世界范围内的香蕉产区蔓延，少有地区能够幸免，并且还在不断蔓延至未发病的香蕉种植地区[5-12]。明确香蕉枯萎病菌遗传多样性及致病机理是有效防控香蕉枯萎病的重要基础，为此本文对香蕉枯萎病菌的遗传多样性及其致病机理研究进展进行了综述。

1　香蕉枯萎病菌的遗传多样性

香蕉枯萎病菌为尖孢镰刀菌古巴专化型(*F. oxysporum* f. sp. c*ubense*，Foc)，根据病原菌对不同香蕉品系的致病性可将其细分为3个小种（1号、2号和4号小种），其中4号小种可根据病害发生是否受冷害胁迫以及发生的地理区域进一步区分为热带4号小种（tropical race 4，TR4）和亚热带4号小种（subtropical race 4，STR4）[6]。1967年我国台湾首次发现由4号小种引起的“Cavendish”品系香蕉枯萎病[13]，后来有学者认为当时鉴定为4号小种的菌株和澳大利亚、加那利群岛以及南非发现的“Cavendish”品系香蕉枯萎病菌一样，应该属于STR4，都是由于所处亚热带地区，香蕉种植过程中易受冷害和冻害胁迫而感病[6,14]。目前，世界范围内蔓延最广、危害最重的香蕉枯萎病菌当属热带4号小种TR4[6]。近年来相继发现TR4引起的香蕉枯萎病在东南亚以外地中海沿岸的约旦和黎巴嫩爆发，在非洲莫桑比克以及亚洲巴基斯坦和宫古岛也发现该小种的危害[5,7-11]；随着“Cavendish”种植地区的转移，TR4也随之进入了大湄公河次区域国家扩大危害[12]。我国大陆自1996年广东省番禺万顷沙镇发现大面积香蕉枯萎现象以来，相继在海南、福建、广西和云南等地发现由TR4小种引起的香蕉枯萎病[15-20]。

为鉴定香蕉枯萎病菌的遗传多样性，Koenig等[21]用限制性片段长度多态性（Restriction Fragment Length Polymorphism，RFLP）分析方法研究了香蕉枯萎病菌13个营养体亲和群（Vegetative Compatibility Group，VCG）的165个菌株，鉴定了9个多态性RFLP位点和72个独特基因型，其中约一半的菌株归属于5个常见的基因型。O’Donnell等[22]通过比较核DNA和线粒体DNA的序列分析了香蕉枯萎病菌的遗传谱系，将其划分为5个无性系，且不同的无性系与其他专化型的尖孢镰刀菌间在分子水平上存在显著的差异，认为香蕉枯萎病菌的进化起源是相对独立的，是随着寄主香蕉的进化而演变。Fourie等[23]分析了20个VCGs共计70个香蕉枯萎病菌菌株，结果表明基因的水平转移和与宿主的协同进化在香蕉枯萎病菌进化过程中起到重要作用。最近对马来西亚香蕉枯萎病菌的遗传多样性研究发现，65%的供试菌株鉴定为TR4，认为该小种起源于马来西亚；而其他的菌株分别被划分成9个独立的遗传谱系[24]，说明除了TR4小种外，其他的香蕉枯萎病菌株间存在着丰富的遗传多样性。这与对来自我国台湾、广东及海南的香蕉枯萎病菌菌株进行扩增片段长度多态性（Amplified Fragment Length Polymorphism，AFLP）分析结果相似。在我国1号小种菌株间存在丰富的遗传多样性，而4号小种的菌株则紧密的聚在一起，说明这些菌株具有相同的遗传谱系[25]，PCR分子鉴定及VCG鉴定结果表明我国的4号小种多数为TR4，属于VCG01213/01216[26-27]，该结果为研究病原的传播提供了确凿的证据，也为病害的防控指明了方向。

小种的划分体现了病原菌与寄主之间的关系，但不能准确了解和区分不同菌株之间的亲缘关系。香蕉枯萎病菌遗传多样性的研究表明，即便是属于同一个小种其不同菌株间仍然存在着较丰富的遗传多样性[24-25]。在缺乏有性生殖的尖孢镰刀菌中，营养体亲和群的划分可以区分菌株之间的亲缘关系，不同的VCG可以代表遗传分离的群体[28]。据此香蕉枯萎病菌已经划分出24个VCGs[9]。然而，VCG的应用也有一定限制，有些菌株难以产生不利用硝酸盐的突变体，且菌株自身的非亲和性和弱异核反应或交叉亲和反应也容易产生VCG的误判[28]。

2　香蕉枯萎病菌的致病因子及其研究进展

香蕉枯萎病菌隶属于尖孢镰刀菌复合群（*F. oxysporum* species complex，FOSC），其致病过程也一样需要经历病原菌与寄主植物根的识别，病原菌到达并附着于寄主根表面，病原菌产生一系列的致

病因子(如抑制寄主产生抗病反应的效应子、产生致病相关的酶和毒素等)从而侵入寄主内部,并在寄主体内定殖最终在其外部表现发病症状的过程[29]。为了制定全面的香蕉枯萎病防治措施、开辟新的防治途径,本文拟从病原菌的致病过程出发,对尖孢镰刀菌致病机理及当前的研究进展进行综述。

2.1 细胞壁的完整性是影响尖孢镰刀菌与寄主识别、附着并顺利侵入寄主的重要因子

真菌的细胞壁由几丁质和葡聚糖为主的内层壁以及富含糖蛋白的外层壁构成,其中外层壁中糖蛋白的含量占尖孢镰刀菌细胞壁总量的50%~60%,是病原真菌与其寄主植物互作的第一线战场[30]。尽管真菌细胞壁本身不能感知外界的刺激,但是包裹在细胞壁外围的大量糖蛋白可能参与这一过程[31]。在香蕉枯萎病菌中,我们发现影响细胞壁完整性的致病因子Och1,该蛋白编码α-1,6-甘露糖转移酶,在蛋白的糖基化过程中负责在蛋白的N端添加甘露糖。敲除或T-DNA插入造成该基因的表达缺失严重影响菌丝的形态,使细胞壁外层的糖基化蛋白显著减少,对刚果红和荧光增白剂敏感性增强,在接种寄主香蕉时表现为对根部的附着力显著降低、不能穿透玻璃纸和根部组织,最终导致致病力明显降低[32]。同样通过影响蛋白的糖基化从而影响细胞壁的完整性的致病因子还有Gas1,在尖孢镰刀菌中编码β-1,3-甘露糖转移酶,*Gas1*敲除突变体对番茄的致病力和侵染能力显著下降[33]。

尖孢镰刀菌中共有3条相对保守的促分裂素原活化蛋白激酶(Mitogen-activated protein kinases,MAPK)途径(Fmk1,Mpk1和Hog1分别代表这3条途径中的关键酶),其中Fmk1和Mpk1与细胞壁的完整性高度相关[34]。*Fmk1*敲除突变体对细胞壁抑制剂(刚果红和荧光白)敏感,菌丝疏水性下降使其在气液交界处的生长受限,侵染寄主时影响菌丝在植物根部的附着和侵入生长[35]。对香蕉枯萎病菌的致病机理研究发现,敲除Mpk1通路中的基因*FoBck1*、*FoMkk2*和*FoSlt2*(*Mpk1*同源基因),导致菌丝呈畸形且在PDA平板上生长缓慢;相比野生型菌株,突变体表现为对细胞壁抑制剂刚果红(CR)和荧光增白剂(CFW)以及过氧化氢(H_2O_2)的敏感性显著增强;最终导致对寄主香蕉的致病力显著降低。通过对*FoMkk2*和*FoSlt2*基因互补,部分表型恢复到野生型水平。分析其致病力下降的原因,发现敲除这些基因会影响几丁质酶合成基因的表达,从而导致突变体的细胞壁完整性被破坏[36]。此外,尖孢镰刀菌细胞壁上还发现一个高度糖基化的跨膜黏液蛋白Msb2,是诱导MAPK通路关键蛋白Fmk1磷酸化所必需的,可促进病原菌侵染性生长并帮助顺利侵入寄主植物根部[37]。MAPK信号途径的上游基因*Rho1*,通过调控Mpk1通路中的基因表达参与细胞壁的形态建成并影响病原菌的致病力[38-39]。

2.2 病原菌分泌的细胞壁降解酶在侵入和定殖过程中发挥着重要作用

尖孢镰刀菌可以产生和分泌多种细胞壁降解酶(Cell wall degrading enzymes,CWDEs),如多聚半乳糖醛酸酶(果胶酶)、果胶酸盐裂解酶(果胶酶)、木聚糖酶(半纤维素酶)等,在侵入并定殖于寄主根部时,这些细胞壁降解酶发挥着重要的作用[29,40]。然而,研究表明敲除某一种细胞壁降解酶基因,并不影响病原菌的致病力[41]。Michielse等[42]认为,这可能与该病原菌中存在着其他冗余基因编码的细胞壁降解酶有关,突变一个基因,其他的冗余基因就会表达产生同样功能的酶,而表现为不影响病原菌的致病力。说明致病力不是单个基因决定的,而是多个基因协同作用的结果。因此,尽管多种细胞壁降解酶在病原真菌致病过程中发挥着重要的作用,但很难从基因水平证明单个细胞壁降解酶对致病力的影响。

从尖孢镰刀菌(*F. oxysporum*)和北美大豆猝死综合症病菌(*F. virguliforme*)上分离到一个蛋白激酶合成基因*Snf1*,可以调控多种细胞壁降解酶的表达。敲除该基因,多种细胞壁降解酶表达受到抑制,致病力显著下降[43-44]。从番茄枯萎病菌(*F. oxysporum* f. sp. *lycopersici*)上分离的F-box蛋白Frp1也属于这一类调控因子,敲除其编码基因后,突变体完全丧失对寄主番茄的致病力。对其致病机理的研究发现接种寄主后Δfrp1突变体中很多细胞壁降解酶基因不表达或是表达量显著降低,因此造成其不能穿透根部而顺利侵入寄主[45]。*Snf1*和*Frp1*基因功能的研究将从前单一研究一种细胞壁降解酶的致病作用转到研究一类细胞壁降解酶转录调节因子的研究上,开创了细胞壁降解酶致病机理研究的新思路。

通过对香蕉枯萎病菌T-DNA插入突变体库的筛选,我们也找到一个接种寄主时多种细胞壁降解酶基因表达下调的突变体L715[46]。克隆T-DNA插

入破坏的基因*Hfi1*，发现*Hfi1*位于*Snf1*的上游，编码一个转录激活因子。进一步通过基因敲除与互补进行功能分析，结果发现与野生型相比，*Hfi1*敲除突变体生长缓慢，基本丧失了产孢能力；多聚半乳糖醛酸酶（Peh）和果胶酸盐裂解酶（Pel）活性显著降低，荧光定量PCR检测发现细胞壁降解酶合成相关基因表达量在*Hfi1*敲除突变体中显著地下调；致病力检测结果发现，*Hfi1*敲除突变体的菌丝穿透能力减弱，接种12 d后仍不能侵入寄主香蕉的根部组织，对香蕉的致病力明显减弱。荧光定量PCR检测发现敲除*Hfi1*明显影响*Snf1*的正常表达。上述结果表明，香蕉枯萎病菌转录激活因子Hfi1除了影响病原菌的生长和产孢外，还可能直接或间接通过调控*Snf1*基因的表达影响多种细胞壁降解酶的酶活，从而影响病原菌侵入寄主根部皮层组织以及在寄主根部的定殖，并最终影响病原菌的致病力[47-48]。

2.3　毒素在病原菌定殖过程中的作用

毒素是植物病原真菌重要的致病因子，当病原菌侵入到寄主体内，毒素开始发挥作用，它可以引起植株外部形态和显微结构的变化，最终导致植株死亡。镰刀菌酸（Fusaric acid，FA）是镰刀菌产生的非专化性毒素[49]，能够抑制寄主植物防御酶系统的活性并快速引起寄主的生理反应，如改变细胞膜渗透性或膜电势，产生活性氧，影响细胞膜的完整性[50-51]。研究发现镰刀菌酸是香蕉枯萎病菌粗毒素的主要成份[52]，以粗毒素处理和病原菌孢子接种均可导致寄主香蕉的维管束薄壁细胞和分生组织细胞产生胶状物，并产生褐变等病理反应，且病变程度不存在小种间的差异，证明FA是导致香蕉枯萎的主要原因并且当其侵染香蕉时FA的浓度与发病程度呈正相关[53]。

镰刀菌酸生物合成所需的基因通常以基因簇的形式存在并共同调节基因表达[54]。通过比较基因组学及基因敲除证明了镰刀菌酸生物合成基因（FA biosynthetic gene，FUB）簇至少由12个基因组成，其中有9个基因是镰刀菌酸生物合成所必需的[55]。通过比较多个尖孢镰刀菌菌株、拟轮枝镰刀菌（*F. verticillioides*）、藤仓镰刀菌（*F. fujikuroi*）以及深绿木霉菌（*Trichoderma atroviride*）的FUB簇同源物，发现在基因簇中有一到两个位点具有非FUB簇基因的插入。虽然合成镰刀菌酸的能力与尖孢镰刀菌培养滤液的毒力紧密相关，但缺乏镰刀菌酸并不影响尖孢镰刀菌和拟轮枝镰刀菌对寄主植物的致病力。这些发现为解析镰刀菌酸生物合成的遗传和生化过程提供了新的见解。对香蕉枯萎病菌的研究发现镰刀菌酸的合成基因*FoFUB4*是产生镰刀菌酸必需的，敲除该基因突变体中检测不到镰刀菌酸，但是突变体的菌丝生长、孢子的产生以及对寄主的致病力并不受影响[56]。该研究还发现氮源在一定程度上可调节FA的生物合成，当培养基中加入$NaNO_3$可诱导镰刀菌酸的合成。

对香蕉枯萎病菌的毒素检测发现，除镰刀菌酸以外，还存在其他毒素类型。李春雨等[57]利用高效液相色谱—电喷雾串联质谱（HPLC-ESI-MS）法对所分离的4号小种菌株的次生代谢产物进行分析，发现了分子质量为179 ku的镰刀菌酸及分子质量约为783 ku的未知产物；对未知产物进行结构测定，结果表明该化合物为白僵菌素，而后又通过离体试验表明白僵菌素能够导致香蕉假茎腐烂，说明白僵菌素在侵染寄主的过程中的确有助于病原菌的致病力。此外，Portal等[58]研究了香蕉枯萎病菌1号小种培养滤液的有毒成分，最终鉴定这些毒性化合物为镰刀菌酸、白僵菌素和伏马菌素B1。

2.4　基因组和转录组水平对尖孢镰刀菌致病力分化的解析

根据对不同寄主的致病力尖孢镰刀菌可划分为不同的致病专化型，至今已经发现种下有120多个专化型[2,22]，而不同专化型的致病力分化原因也是近年来的研究热点。比较基因组学分析揭示尖孢镰刀菌的基因组分为负责基础代谢和繁殖的核心基因组以及负责对不同寄主专化型致病力的附属基因组两大部分。通过大片段的染色体转化，将番茄枯萎病菌一条富含致病基因的附属染色体转化到非致病尖孢镰刀菌FO47中，发现突变体菌株可以对番茄致病，表明尖孢镰刀菌对不同寄主的专化型致病基因大多集中在附属基因组上，基因水平转移是造成不同专化型致病力分化的主要原因，也进一步证实了尖孢镰刀菌的多系起源是基因水平转移的结果[59-60]。对尖孢镰刀菌5个不同专化型的45个菌株进行全基因组范围的效应子分布与种类研究发现，与寄主专化型相关的效应子大多分布在附属基因组上，相同的专化型具有相同的效应子分布谱，而且同一专化型不同的无性系菌株具有相同的效应子，这些全基因组比对基础上获得的分子标记为从分子水

平区分和鉴定尖孢镰刀菌专化型种类奠定了基础[61-62]。对香蕉枯萎病菌的基因组分析也发现致病基因在同一专化型的不同小种或不同菌株间都广泛的存在，但是决定致病力分化的致病相关基因与其他专化型的存在差异[61-63]。因此，通过比较基因组学分析寄主专化型水平的致病基因对研究尖孢镰刀菌致病机制的进化和多样性具有重要的意义。

此外，香蕉转录组的数据分析也为从不同的角度解析香蕉枯萎病菌致病力的分化奠定了基础。分别用1号小种和热带4号小种TR4接种巴西蕉（“Cavendish”品系香蕉），香蕉转录组数据显示TR4接种后的香蕉中果胶酯酶合成基因显著的高表达，而酶活检测也发现感病的香蕉组织中果胶酯酶大量积累，明确果胶甲酯酶参与诱导了香蕉枯萎病的发生[64-65]，为阐明香蕉枯萎病的发生机理提供了有力的论据。

2.5 sRNA及其他调控因子对尖孢镰刀菌致病力的影响

小分子RNA（small RNA，sRNA）在真核生物的生长发育过程中发挥重要的功能。MicroRNA（miRNA）是其中一类具有调控功能的非编码单链小分子RNA。其长度通常约为22 nt，通过与靶标信使RNA（messenger RNA，mRNA）特异性的碱基配对引起靶标mRNA的降解或者抑制其翻译，从而对基因表达进行转录后水平的调控[66]。在已报道的真菌sRNA中，发现有一些与动植物中的miRNA类似，被称为milRNA（miRNA-like RNA）[67]。真菌中有关milRNA功能的报道非常有限。对灰葡萄孢（*Botrytis cinerea*）sRNA的研究中发现，其产生的milRNA可跨界发挥功能，通过抑制寄主植物的免疫反应，帮助病原菌成功侵入寄主[68]。最近，在小麦条锈菌（*Puccinia striiformis* f. sp. *tritici*）中也发现一个milRNA（Pst-milR1），可以抑制寄主小麦的病程相关基因*PR2*的表达，从而抑制寄主小麦因病原菌侵染而产生的防卫反应，帮助病原菌侵入寄主[69]。这些结果表明病原真菌的milRNAs可以跨界行使其基因沉默的功能，为研究milRNA在病原菌致病过程中调控作用拓展了思路。2014年，在对番茄枯萎病菌的sRNA测序发现尖孢镰刀菌中存在milRNAs，但是与已经公布在miRBase数据库中的miRNAs没有交集[70]。我们最近的研究结果显示，以枯萎病菌接种香蕉根部24 h后，病原菌中与sRNA合成相关的Dicer和Argonaute编码基因显著高表达；sRNA测序结果也表明，接种后大量milRNA显著高表达，说明milRNA也参与了致病过程，但是具体的调控机制还有待进一步的研究。

在香蕉枯萎病菌中已报道G蛋白激活通路中负责G蛋白小亚基合成基因*FGA1*、*FGA2*和*FGB1*影响病原菌的致病力。其中*FGA1*调节生长、产孢、发育、致病性和耐热性；*FGA2*和*FGB1*可能通过cAMP-PKA信号传导通路调节真菌发育，进而影响其对寄主的致病力；*FGA3*只影响其孢子的耐热性而不影响对寄主植物的致病力[71-72]。这些研究结果为阐明G蛋白信号通路在尖孢镰刀菌致病过程中的作用积累了资料。

3 结语与展望

香蕉枯萎病大面积的发生与为害对全球的香蕉产业经济影响巨大，香蕉枯萎病菌已成为尖孢镰刀菌复合群的典型代表和植物土传性病害的模式菌被世界上越来越多的科学家重视和研究。香蕉枯萎病菌致病过程中关键致病因子的鉴定将为开发新且有效的防控策略提供作用靶标，致病机理的进一步解析将为制订香蕉枯萎病综合防控措施提供科学的依据，也有助于选育出抗性更高和更持久的抗枯萎病香蕉新品种。

由于香蕉种植上的地域限制，在我国香蕉枯萎病的研究大多集中在南方，而且针对其病原菌致病机理的研究也相对滞后。现有的大部分致病机基因的研究都借鉴于种内其他专化型的致病基因研究结果，然而由于尖孢镰刀菌复合群的遗传背景复杂，即使同一专化型不同小种、不同VCG、甚至不同的菌株致病力分化明显，这意味着其他专化型的致病机理研究结果并非完全适用于香蕉枯萎病菌，因此有必要开展香蕉枯萎病菌致病机理特别是致病力分化原因的研究；同时不能局限于致病基因以及转录因子的功能研究，比如致病过程中大量高表达的小分子RNA对致病的调控作用就是最近研究热点。相信这些致病因子的功能解析必将为香蕉枯萎病的防控提供新思路。

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