小麦-禾谷镰孢菌互作机制的研究进展

苏培森^{1,2}, 葛文扬¹, 王宏伟¹, 孔令让^{1*}

1. 山东农业大学农学院/作物生物学国家重点实验室, 泰安 271018;

2. 聊城大学农学院, 聊城 252000

* 联系人: E-mail: lkong@sdau.edu.cn

收稿日期: 2020-12-06; 接受日期: 2021-06-21; 网络版发表日期: 2021-09-14

摘要 禾谷镰孢菌(俗称禾谷镰刀菌, *Fusarium graminearum*)引起的小麦赤霉病是小麦生产上一种极具毁灭性的真菌病害, 在世界各主要产麦国普遍发生, 我国是世界上受害面积最大的国家, 该病害严重威胁我国粮食生产和食品安全. 解析小麦-禾谷镰孢菌之间的互作机制, 挖掘有效抗病基因, 对于解决小麦赤霉病这一世界难题具有重要意义. 本文对禾谷镰孢菌致病机制, 小麦与禾谷镰孢菌致病因子DON毒素互作机制, 禾谷镰孢菌侵染与小麦识别互作机制以及寄主-病原菌基因水平转移等研究进展进行了综述.

关键词 小麦, 赤霉病, 禾谷镰孢菌, 小麦-禾谷镰孢菌互作, 呕吐毒素(DON)

小麦在生长发育过程中, 往往会遭受到外界各种病原菌的侵染. 禾谷镰孢菌(俗称禾谷镰刀菌, *Fusarium graminearum*)是引起小麦赤霉病的一种主要致病真菌. 赤霉病被称为小麦“癌症”, 会造成小麦大幅度减产. 有报道指出, 在全世界范围内独立病原菌以及昆虫等导致全球小麦平均减产21.5%, 其中因赤霉病引起的小麦产量损失仅次于位列第一的小麦叶锈病^[1]. 在世界上易发生赤霉病的小麦种植区域, 至少每4~5年就会发生一次严重的赤霉病流行^[2]. 我国是世界上小麦赤霉病受害面积最大的国家, 近年来, 受气候变暖、抗赤霉病品种缺乏以及以秸秆大量还田为主要模式的小麦-玉米轮作制度的推广普及等因素的影响, 小麦赤霉病愈发严重, 常发及重发区域也从长江中下游麦区扩展到黄淮麦区^[3]. 2010年后, 小麦赤霉病的发病率更加频繁, 每年年均赤霉病发病面积超过533.3万

公顷, 在2000~2018年间, 因赤霉病引起的小麦年均产量损失超过341万吨^[4,5].

小麦赤霉病除了造成小麦大面积减产外, 在其发病过程中, 禾谷镰孢菌等多个致病性镰孢菌种还会产生一类以脱氧雪腐镰刀菌烯醇(deoxynivalenol, DON)为典型代表的单端孢霉烯族毒素^[6]. 早在1973年, DON毒素已经在大麦和小麦中得以鉴定, 其能通过与核糖体结合进而抑制蛋白质的合成, 引起人畜腹泻、呕吐等急性中毒症状, 因此也被称为呕吐毒素^[7]. DON毒素几乎存在于所有的粮食作物中, 如小麦、玉米、大麦、黑麦和燕麦等, 是世界谷物中检出率最高的真菌毒素^[8~10]. 根据来自中国和中欧等地区的玉米、小麦样本的检测发现, DON毒素平均污染率为55%, 平均污染水平达到967 $\mu\text{g}/\text{kg}$, 其中最大检出量更是达到了50,289 $\mu\text{g}/\text{kg}$ ^[11]. 针对DON毒素的影响, 多

引用格式: 苏培森, 葛文扬, 王宏伟, 等. 小麦-禾谷镰孢菌互作机制的研究进展. 中国科学: 生命科学, 2021, 51: 1493–1507

Su P S, Ge W Y, Wang H W, et al. Advances in understanding the mechanisms of wheat-*Fusarium graminearum* interactions (in Chinese). Sci Sin Vitae, 2021, 51: 1493–1507, doi: 10.1360/SSV-2020-0355

个国家已经制定了其在食品和饲料中的限量标准, 我国规定在谷物及其制品中, DON含量不得超过1 mg/kg^[12].

1 禾谷镰孢菌致病机制研究

禾谷镰孢菌是引起小麦赤霉病最主要的致病菌. 禾谷镰孢菌侵染小麦并在寄主内繁殖和延展, 这与禾谷镰孢菌的生物学特性、侵染特点、寄主植物的生长特性以及病原菌与寄主之间的互作机制有关. 随着禾谷镰孢菌基因组序列的公布以及转录组等多组学技术的迅速发展, 人们对禾谷镰孢菌的致病机制进行了广泛而深入的探究, 大量的禾谷镰孢菌致病基因被克隆和研究. 迄今为止的研究表明, 禾谷镰孢菌致病性受其细胞外信号传导途径的调控, 主要包括有丝分裂原活化蛋白激酶(mitogen-activated protein kinase, MAPK)、环腺苷酸蛋白激酶A(cyclic adenosine monophosphate, cAMP)、Ca²⁺和雷帕霉素信号传导通路等. 禾谷镰孢菌在侵染过程中还会产生一系列有毒的次生代谢产物, 这些代谢产物对小麦具有强烈的毒害作用. 其中, 单端孢霉烯族毒素, 尤其是DON毒素在禾谷镰孢菌的致病过程中具有重要作用, 是病原菌的一个重要致病因子. 此外, 囊泡运输以及组蛋白翻译后修饰也会对禾谷镰孢菌致病性产生一定的影响. 基于此, 本文对禾谷镰孢菌致病性调控基因进行了总结, 具体见表1.

2 小麦与禾谷镰孢菌致病因子DON毒素互作机制研究

单端孢霉烯族化合物是由镰孢菌属霉菌产生的一类最重要、最广泛的镰孢菌毒素, 对禾谷镰孢菌的致病力具有重要作用. 迄今为止, 由镰孢菌等多种真菌产生的200余种该类毒素已被分离并鉴定^[11,70]. 基于其核心结构的差异, 单端孢霉烯族化合物被分为四种类型(A型、B型、C型和D型), DON属于其中的B型, C8位置含有酮(羰基)官能团, 可以在多种镰孢菌(尤其是禾谷镰孢菌)侵染过程中产生^[70,72]. 在镰孢菌中, 单端孢霉烯族化合物的生物合成路径是通过15个单端孢霉烯族化合物生物合成基因(trichothecene biosynthesis gene, *TRI*)调控的, 具体的生物合成路径详见图

1^[70,71,73].

禾谷镰孢菌在侵染小麦的过程中会合成DON毒素. 在侵染的早期, DON毒素被分泌并扩散到寄主组织内^[74]. 随着禾谷镰孢菌的继续侵染, 其营养方式由营寄生转为营腐生生长. 此时, DON毒素的合成加快, 阻碍植物细胞的蛋白质表达, 进而抑制了植物细胞防御物质的合成, 促进禾谷镰孢菌在小麦穗部的延展^[75~78]. 有报道指出, 当DON毒素关键合成酶基因*TRI5*突变后, 禾谷镰孢菌在侵染小麦穗部时丧失了合成DON毒素的能力, 进而抑制了其在小麦穗部的延展^[79,80]. 此外, 还有研究显示, DON能够诱导植物中活性氧(reactive oxygen species, ROS)的产生以及细胞程序性死亡^[81,82]. 上述研究结果表明, DON毒素是一种重要的致病因子, 并且在病原菌的侵染以及延展过程中发挥重要作用.

在与禾谷镰孢菌长期的互动斗争过程中, 植物也进化出了多种降低DON毒性的机制, 主要可以分为两类. 一种是植物通过化学修饰的方式降低DON毒素的毒性, 譬如DON毒素糖基化、去环氧化等. 第二种是植物通过内源性组分抑制DON毒素的合成, 从而降低DON毒素的富集, 譬如酚类、多肽类等抗氧化类物质^[83]. 这些过程依赖于植物体内抗性基因的调控. 迄今为止, 已报道大量DON毒素的脱毒基因, 除了众所周知的UDP-葡萄糖基转移酶外, 还涉及了谷胱甘肽转移酶、细胞色素P450、乙烯钝感、赤霉素敏感DEL-LA蛋白等. 谷胱甘肽转移酶(glutathione S-transferase, GSTs)是参与植物响应胁迫的重要酶, 它可以催化谷胱甘肽与有毒化合物或其氧化产物结合, 使有毒化合物丧失毒性, 进而保护植物免受毒素的胁迫. 近期, 本课题组^[73]克隆了来自长穗偃麦草的抗赤霉病基因*Fhb7*, 发现其编码一种谷胱甘肽巯基转移酶(glutathione S-transferase, GST), 该酶以呕吐毒素为底物, 催化谷胱甘肽结合在其C13, 从而破坏此处的环氧结构(环氧基为DON毒素的主要毒性基团), 导致DON的毒性大大降低, 进而提高小麦赤霉病抗性. 此外, 研究还发现, *Fhb7*除了可以降低DON毒素毒性外, 对其他具有相同环氧结构的A、B类单端孢霉烯族毒素, 如T-2毒素、NIV等也具有广谱的解毒作用^[73,84,85], 具体解毒路径见图1. 本文总结了植物参与DON毒素脱毒的基因(表2).

表 1 禾谷镰孢菌致病性调控基因

Table 1 Pathogenicity genes of *Fusarium graminearum*

分类	基因名称	基因描述	参考文献
孤儿蛋白	<i>OSP24</i>	孤儿分泌蛋白	[13]
	<i>FgBck1</i>	MAPK激酶激酶	[14]
有丝分裂原活化蛋白激酶	<i>FgMkk1</i>	MAPK激酶	[15]
	<i>FgMgv1</i>	有丝分裂原活化蛋白激酶	[16]
	<i>FgSte12</i>	编码渗透结构形成的关键因子	[14,17]
	<i>FgSte11</i>	有丝分裂原活化蛋白激酶	
	<i>FgSte7</i>	有丝分裂原活化蛋白激酶	
	<i>FgSte2</i>	信息素敏感G蛋白偶联受体	[18]
	<i>FgGpmk1</i>	有丝分裂原活化蛋白激酶	
	<i>FgOs1</i>	渗透传感器组氨酸激酶	[19]
	<i>FgRrg1</i>	响应调节蛋白	[20]
	<i>FgAtf1</i>	ATF/CREB激活因子	[21]
	<i>FgMap1</i>	有丝分裂原活化蛋白	[22]
	<i>FgHog1</i>	高渗透压甘油响应基因	[23]
	<i>FgSho1</i>	跨膜蛋白	[24]
G蛋白偶联受体	<i>Giv1</i>	G蛋白偶联受体基因	[25]
	<i>Giv2</i>		
	<i>Giv3</i>		
环腺苷酸蛋白激酶A	<i>CPK1</i>	cAMP依赖性蛋白激酶催化亚基	[26]
	<i>FAC1</i>	腺苷酸环化酶	[27]
	<i>FgCAP1</i>	环化酶相关蛋白	[28]
	<i>PDE2</i>	cAMP磷酸二酯酶	[29]
	<i>PKR</i>	PKA催化亚基	[30]
	<i>GzGpa1</i>	α 亚基	[31]
	<i>GzGpb1</i>	β 亚基	
	<i>Ras1</i>	GTP结合蛋白	[32]
	<i>Fgl1</i>	分泌脂酶	
钙离子信号通路	<i>Mid1</i>	机械敏感钙离子通道蛋白	[33]
	<i>Cch1</i>	L型钙离子通道蛋白	[34]
	<i>Fig1</i>	低亲和力和钙摄取系统的跨膜蛋白	[35]
雷帕霉素信号传导通路	<i>FgTOR</i>	雷帕霉素激酶	[36]
	<i>FgFkbp12</i>	肽基脯氨酰/反式异构酶	
	<i>FgTap42</i>	磷酸酶复合物	[36]
	<i>FgTip41</i>		
	<i>FgCak1</i>	激酶	[37]
	<i>FgNem1</i>	蛋白磷酸酶复合物	[37]
	<i>FgSpo7</i>		
	<i>FgPah1</i>	磷脂酸磷酸酶	[37]
代谢物与致病性	<i>Nps6</i>	非核糖体肽合成酶	[38]
	<i>Pks4</i>	聚酮合成酶	[39]
	<i>Pks13</i>		

(表1续1)

分类	基因名称	基因描述	参考文献
Fg3.54基因簇	<i>Fgm4</i>	bANK类调节基因	
	<i>NRPS5</i>	非核糖体肽合成酶	[40]
	<i>NRPS9</i>	非核糖体肽合成酶	
囊泡运输	<i>FgVam7</i>	可溶性N-乙基马来酰亚胺敏感性因子附着型蛋白受体	[41]
	<i>FgMon1</i>	鸟嘌呤核苷酸交换因子	[42]
	<i>FgRab7</i>	GTP结合蛋白	
	<i>FgAtg8</i>	自噬相关基因	[43]
组蛋白翻译后修饰	<i>Hep1</i>	异染色质蛋白	[44]
	<i>Kmt6</i>	甲基转移酶	[45]
	<i>FgSET1</i>	甲基转移酶	[46]
	<i>FgGcn5</i>	组蛋白乙酰转移酶	[47]
	<i>Sas3</i>	组蛋白乙酰转移酶	[48]
	<i>Elp3</i>	延伸子复合蛋白	[49]
	<i>HDF1</i>	组蛋白去乙酰化酶	[50]
	<i>FgGCN5</i>	赖氨酸乙酰转移酶	[51]
转录调控因子	<i>FgTFC7</i>	TFIIIC家族转录因子	[15]
	<i>FHS1</i>	Zn(II)2Cys6锌指转录因子	[52]
	<i>FgTeb</i>	锌指转录因子	[53]
	<i>FgNsf1</i>	锌指转录因子	[54]
	<i>FgCrz1A</i>	锌指转录因子	[55]
	<i>Fg01341</i>		
	<i>Fg01350</i>	钙调磷酸酶应答转录因子	[56]
	<i>Fgap1</i>	bZIP转录因子	[57]
	<i>FgSR</i>	调节甾醇生物合成转录因子	[58]
	<i>FgChy1</i>	CHY型锌指蛋白	[59]
	<i>FgHltf1</i>	组蛋白类转录因子	[60]
	<i>FgHtf1</i>	同源盒转录因子	[61]
	<i>FgStuA</i>	bHLH转录因子	[62]
	<i>MYT3</i>	MYB转录因子	[63]
	<i>Fgspt3</i>	Spt-Ada-Gcn5-乙酰基转移酶(SAGA)转录共激活因子	[64]
	<i>Fgspt8</i>	Spt-Ada-Gcn5-乙酰基转移酶(SAGA)转录共激活因子	[64]
	<i>ZIF1</i>	bZIP转录因子	[65]
磷酸酶	<i>FgPtc3</i>	磷酸酶	[66]
	<i>FgPP2AR</i>		
	<i>FgPP2AC</i>	磷酸酶	[67]
	<i>Fg06297</i>		
	<i>Fg03333</i>		
	<i>Fg03826</i>		
	<i>Fg07932</i>		
	<i>Fg10302</i>	磷酸酶	[15]
	<i>Fg10516</i>		
	<i>Fg03333</i>		
	<i>Fg12867</i>		
	<i>FgCdc14</i>		

(表1续2)

分类	基因名称	基因描述	参考文献
微管相关蛋白	<i>FgTUB1</i>	β-微管蛋白	[68]
	<i>FgTUB2</i>		
	<i>FgKar9</i>	微管相关蛋白	[69]
	<i>FgEB1</i>	微管末端结合蛋白	[53]
单端孢霉烯族化合物生物合成	<i>TRI5</i>	单端孢霉烯合成基因	[70,71]
	<i>TRI4</i>		
	<i>TRI6</i>		
	<i>TRI3</i>		
	<i>TRI11</i>		
	<i>TRI12</i>		
	<i>TRI17</i>		
	<i>TRI10</i>		
	<i>TRI13</i>		
	<i>TRI8</i>		
	<i>TRI9</i>		
	<i>TRI14</i>		
	<i>TRI1</i>		
	<i>TRI16</i>		
	<i>TRI101</i>		

3 禾谷镰孢菌侵染与小麦识别互作机制研究

小麦赤霉病主要是由禾谷镰孢菌引起, 该病原菌是一种寄生兼腐生生长的真菌病原体^[103]. 小麦扬花期遇到阴雨或大风天气, 禾谷镰孢菌的分生孢子或子囊孢子随风或雨水附着在小麦穗部的表面, 或直接进入到张开的颖壳, 随后, 孢子完成萌发, 形成菌丝. 菌丝开始侵染细胞, 并在细胞间和细胞内生长, 然后经花丝延展至子房和穗轴, 并经维管束传播至邻近小穗^[104~106]. 随着菌丝的生长, 寄主表现出病害症状^[107]. 康振生等人^[108]利用扫描电子显微镜和透射电子显微镜技术发现, 在接种禾谷镰孢菌36 h后, 其在寄主表面已经形成菌丝, 并以直接侵入的方式穿透表皮细胞进入寄主组织内. 本课题组也通过扫描电子显微镜分析了禾谷镰孢菌在抗、感不同小麦品种颖壳和叶片的侵染模式, 发现禾谷镰孢菌在接种12 h后已经在感病品种济麦22(JM22)颖壳表面形成菌丝; 接种24 h, 禾谷镰孢菌已经完成侵染, 而抗病品种苏麦3号(SM3)却表现出明显的抗性. 在整个侵染过程中, 与感病品种JM22相比, 抗病品种SM3的表面产生了很多类似于代谢物

的物质, 推测其可能与赤霉病抗性相关(图2). 随后, 本课题组^[109]通过代谢组学进一步证明了黄酮类代谢物山奈素和芹菜素可以明显提高小麦赤霉病的抗性. Zhang等人^[110]利用绿色荧光蛋白(green fluorescent protein, GFP)标记的禾谷镰孢菌接种不同抗性小麦品种, 发现相比感病品种Annong 8455, 抗病品种SM3接菌小穗基部可以明显抑制禾谷镰孢菌的延展. 本课题组也通过扫描电子显微镜比较了抗、感赤霉病品种的穗部结构, 结果发现, 与感病品种JM22相比, 抗病品种SM3接菌穗穗轴可以明显地抑制禾谷镰孢菌在穗轴内的延展(图3).

禾谷镰孢菌侵入植物组织内部时就受到植物细胞壁的防御. 细胞壁是一个高度动态的结构, 也是寄主抵御病原菌侵染的第一道防线. 当病原菌侵染时, 寄主往往会在病原菌侵染位点的细胞壁中沉淀胨胨质、酚类、木质素等化合物, 以此增加细胞壁的厚度和强度^[107,111,112]. 在植物主动应对病原菌的防御反应中, 细胞壁主要通过两种方式发挥作用, 一种是通过改变自身的结构和成分抑制病原菌降解酶对自身的降解, 进而延缓或阻止病原菌的侵染和延展; 另一种方式则是通过调节植物抗性调控通路的相关基因增强植物的系

图 1 DON毒素产生及解毒路径^[70,71,73]
Figure 1 DON biosynthesis and detoxification^[70,71,73]

统抗性^[113]。病原菌在侵染的早期, 会释放出多种水解酶, 其中多聚半乳糖醛酸酶(polygalacturonase, PG)可

以降解细胞壁的主要成分果胶, 破坏细胞壁结构, 进而导致病原菌的侵染^[114,115]。在与病原菌的对抗中, 寄主

表 2 抗毒素积累或脱毒相关基因

Table 2 Genes related to resistance to DON accumulation or detoxification

基因注释	基因名称	参考文献
细胞色素P450	<i>Ddna</i>	[86]
乙烯钝感	<i>EIN2</i>	[87]
赤霉素敏感DELLA蛋白	<i>TaRht-B1b TaRht-D1b</i>	[88]
甲硫氨酰tRNA合成酶	<i>TaMetRS</i>	[89]
ABC转运器	<i>ScPDR5</i>	[90]
	<i>TaABCC3.1</i>	[91]
UDP-葡萄糖基转移酶	<i>DOG1</i>	[92]
	<i>TaUGT3</i>	[93]
	<i>HvUGT13248</i>	[94,95]
	<i>Bradi5g02780</i>	[96]
	<i>Bradi5g03300</i>	[97]
	<i>TaUGT12887</i>	[98]
	<i>Ta-UGT3</i>	[99]
	<i>TaUGT5</i>	[100]
谷胱甘肽转移酶	<i>Traes_2BS_14CA35D5D</i>	[101]
	<i>Fhb7</i>	[73]
孤儿蛋白	<i>TaFROG</i>	[102]

图 2 禾谷镰孢菌在抗、感赤霉病小麦品种颖壳中的侵染(红色箭头指示扫描电子显微镜下, 在SM3颖壳表面观察到的物质)

Figure 2 The *F. graminearum* infection in glumes of susceptible (JM22) and resistant (SM3) plants. (The red arrows represent the substances observed on the surface of SM3 glume under a scanning electron microscope (SEM))

自身分泌的多聚半乳糖醛酸酶抑制蛋白(polygalacturonase inhibiting protein, PGIP)参与了植物自身的防御反应, 它可以特异性识别并结合病原菌产生的PG, 进而抑制其活性及分解细胞壁的能力。此外, PGIP还能促进多聚半乳糖醛酸的积累, 从而更有效地激活植物自身的防卫反应^[116~120]。康振生等人^[108,121]利用电子显微镜观察, 并结合细胞化学技术, 发现禾谷镰孢菌在侵染和延展的过程中, 分泌了大量的酶, 包括纤维素酶、

图 3 小麦抗、感赤霉病品种穗轴结构比较。A₁: 苏麦3号穗部赤霉病抗性; A₂: 苏麦3号穗轴赤霉病抗性; A₃: 明场下, 苏麦3号穗轴纵切结构观察图; A₄: 绿色荧光下, 苏麦3号穗轴纵切结构观察图; B₁: 济麦22穗部赤霉病抗性; B₂: 济麦22穗轴赤霉病抗性; B₃: 明场下, 济麦22穗轴纵切结构观察图; B₄: 绿色荧光下, 济麦22穗轴纵切结构观察图; C₁: 明场下, 济麦22接种后穗轴观察图; C₂: 绿色荧光下, 济麦22接种后穗轴观察图; D₁和D₂: 济麦22穗轴横切面扫描电子显微镜观察; E₁和E₂: 苏麦3号穗轴横切面扫描电子显微镜观察。红色箭头代表菌丝的延展

Figure 3 The difference in tissue structure of rachis between FHB-resistant (SM3) and susceptible (JM22) wheat. A₁–A₃: The spike, rachis and longitudinal section of floret and rachis of SM3; A₄: the rachis longitudinal section of SM3 under green fluorescence; B₁–B₃: the spike, rachis and longitudinal section of floret and rachis of JM22; B₄: The rachis longitudinal section of JM22 under green fluorescence; C₁ and C₂: the profiles of JM22 rachis after *F. graminearum* inoculation under bright light and green fluorescence, respectively; D₁ and D₂: the tissue structure of JM22 rachis after *F. graminearum* inoculation under a scanning electron microscope (SEM); E₁ and E₂: the tissue structure of SM3 rachis after *F. graminearum* inoculation under SEM. The red arrows represent the expansion of *F. graminearum* along the rachis

木聚糖酶和果胶酶等细胞壁降解酶, 在这些酶的作用下, 寄主细胞壁被分解, 使得寄主细胞坏死, 最终导致整个麦穗枯死。

Jiang等人^[13]独辟蹊径, 研究禾谷镰孢菌基因组中广泛存在但功能未知的孤儿蛋白, 发现了一个对禾谷镰孢菌致病力起关键作用的孤儿蛋白Osp24, 该蛋白被禾谷镰孢菌表达后, 分泌进入小麦细胞核, 进而靶向小麦中的保守关键激酶SnRK1。在小麦体内, 该蛋白通过招募泛素-26S蛋白酶体, 从而加速SnRK1的降解, 最终导致小麦感病。此外, 研究还进一步指出, Osp24可以通过半胱氨酸形成分子内二硫键保证自身在面对植物的防御反应时不被降解。在与禾谷镰孢菌的长期斗争过程中, 小麦体内也产生了一个孤儿蛋白

TaFROG, 该蛋白只有在感知到禾谷镰孢菌侵染产生的DON毒素之后才会被激活, 激活后的TaFROG也可以和SnRK1蛋白结合, 与Osp24蛋白形成竞争关系, 从而减弱Osp24对SnRK1的降解, 最终介导小麦对赤霉病的抗性. 该研究揭示了病原真菌和寄主的孤儿蛋白在病害发生过程中的功能, 阐明了其竞争互作机制, 建立了病原真菌和寄主植物互作的新模式, 为开发小麦赤霉病防控新策略打下重要基础. 然而, 禾谷镰孢菌和小麦之间互作机制的研究工作任重而道远, 仍需进一步探索.

4 寄主-病原菌基因水平转移

在自然界中, 植物与病原菌之间的相互作用对彼此的进化具有深远的影响. 伴随着植物与病原菌长期的共同进化, 偶尔也会出现基因之间的彼此交换, 这种不同种属间发生遗传物质交流的事件被称为基因水平转移(horizontal gene transfer, HGT). 在陆生植物中, 关于植物-真菌之间的HGT在苔藓植物中已被证明, 其中大部分的真菌基因涉及到植物的发育与胁迫响应^[122~127]. 然而, 有关从真菌直接到被子植物中的HGT现象鲜有报道. Shinozuka等人^[128]发现一个 β -1,6-葡聚糖酶基因从内生真菌转移到冷季草, 并且该基因被认为与抗真菌活性有关. 近期, 本课题组^[73]通过基因克隆、寄主基因组测序及比对分析发现, 来自偃麦草的抗赤霉病基因*Fhb7*可能来自于其内生真菌*Epichloë*, 从而首次提供了真核生物间核基因组DNA水平转移的功能性证据, 为进一步探索植物抗病基因和基因组进化机制开辟了一条新途径. 周俭民^[84]也对*Fhb7*的演化进程提出推测, 指出禾本科植物与内生真菌*Epichloë*共生获得对赤霉病的抗性, 在随后的演化中, 被水平转移到寄主植物中, 进而发挥其抗赤霉病作用.

5 总结与展望

小麦赤霉病是一种严重的小麦穗部病害, 其发生不仅会影响小麦产量, 而且在感病籽粒中产生多种真菌毒素, 严重危害人畜健康, 因此小麦赤霉病的防治显得尤其重要^[129]. 目前防治赤霉病的有效方式依然是化学防治为主, 然而, 长期大量的农药使用不仅会使病

原菌产生抗药性, 而且对生态环境具有深远的不良影响. 因此, 揭示病原菌与寄主植物的互作机制, 挖掘赤霉病抗性基因, 培育抗病品种, 同时开发绿色环保的生物农药等对小麦赤霉病防控具有重要的理论及实践意义. 目前, 人们已经对禾谷镰孢菌的致病机制有了一定的了解, 并发掘到了大量的相关致病基因. Jiang等人^[13]发现了对禾谷镰孢菌致病力起关键作用的孤儿蛋白Osp24, 并揭示了其靶向小麦中的保守关键激酶SnRK1, 最终导致小麦感病, 为人们找到了一个赤霉病菌与小麦分子互作的关键途径. 但是, 禾谷镰孢菌-小麦之间的互作比较复杂, 目前人们对于其互作机制的了解仍比较浅显, 未来这也将是小麦赤霉病抗性机制研究工作的重点.

发掘抗病基因, 培育赤霉病抗病品种是防治小麦赤霉病最有效、最环保的措施. 然而, 目前已经明确的小麦抗赤霉病基因只有7个(*Fhb1*~*Fhb7*)^[130], 其中仅有*Fhb1*和*Fhb7*被克隆, 而且*Fhb1*的抗病分子机制尚未明了^[73,129,131,132]. 历经20年, 我们成功克隆了来自于小麦近缘植物长穗偃麦草抗赤霉病基因*Fhb7*, 将*Fhb7*导入小麦后展现出较好的赤霉病抗性, 而且对产量没有明显不良影响. 这表明*Fhb7*在小麦抗病育种中具有重要应用潜力. DON毒素在小麦赤霉病发病过程中扮演着非常重要的角色, 揭示其合成机制, 挖掘其解毒基因对于抵抗小麦禾谷镰孢菌侵染具有重要意义. 已有大量研究揭示了植物多种解毒基因, 其中*Fhb7*已被证实对常见且毒性较大的A、B型单端孢霉烯族毒素具有广谱的解毒作用, 预测未来利用解毒基因的广谱解毒功能进一步开发单端孢霉烯解毒剂, 可以有效减少作物籽粒中真菌毒素的含量, 对维护粮食、食品和饲料安全具有重要意义. 因此, *Fhb7*无论是在未来种质创新、品种改良, 还是生物医药以及食品工业方面都具有广阔的应用前景.

在寄主和病原菌长期的互作过程中, 偶尔会发生基因水平转移事件. 本课题组^[73]研究发现, *Fhb7*可能来自于一种香柱菌属内生真菌, 通过基因水平转移的这种天然“转基因”的方式由真菌转入宿主植物. *Fhb7*基因的发现不仅丰富了作物的遗传多样性, 而且说明基因水平转移事件在作物的遗传改良中也蕴含着巨大的应用潜力^[85,133]. 然而, 关于基因水平转移机制及其在生产上的应用尚处于初步研究阶段, 未来仍有很多方面需要进一步研究. 譬如, 在偃麦草基因组中是否

还存在其他真菌基因水平转移, 基因水平转移机制以及评估这些真菌基因是否可被用于作物的基因改造等. 这些问题的揭示不仅会促进人们对病原菌和寄主

之间互作机制的了解, 而且可能为作物基因组编辑提供新思路, 进一步推动这些基因在作物育种及其他领域中的应用.

参考文献

- 1 Savary S, Willocquet L, Pethybridge S J, et al. The global burden of pathogens and pests on major food crops. *Nat Ecol Evol*, 2019, 3: 430–439
- 2 Figueroa M, Hammond-Kosack K E, Solomon P S. A review of wheat diseases: a field perspective. *Mol Plant Pathol*, 2018, 19: 1523–1536
- 3 Ma Z H, Chen Y, Yin Y N. Epidemiological analysis and management strategies of *Fusarium* head blight of wheat (in Chinese). *Bull Natl Nat Sci Found China*, 2020, 34: 464–469 [马忠华, 陈云, 尹燕妮. 小麦赤霉病流行成灾原因分析及防控对策探讨. 中国科学基金, 2020, 34: 464–469]
- 4 Ma Z H, Yin Y N, Chen Y. Research progress on occurrence regularity and integrated control technology of *Fusarium* head blight (in Chinese). In: China Society of Plant Protection. Chengdu. 2016 [马忠华, 尹燕妮, 陈云. 小麦赤霉病发生规律及其防控技术研究进展. 中国植物保护学会2016年学术年会. 成都. 2016]
- 5 Chen Y, Kistler H C, Ma Z. *Fusarium graminearum* trichothecene mycotoxins: biosynthesis, regulation, and management. *Annu Rev Phytopathol*, 2019, 57: 15–39
- 6 Kang Z, Buchenauer H. Immunocytochemical localization of *Fusarium* toxins in infected wheat spikes by *Fusarium culmorum*. *Physiol Mol Plant Pathol*, 1999, 55: 275–288
- 7 Vesonder R F, Ciegler A, Jensen A H. Isolation of the emetic principle from *Fusarium*-infected corn. *Appl Microbiol*, 1973, 26: 1008–1010
- 8 van Egmond H P, Schothorst R C, Jonker M A. Regulations relating to mycotoxins in food: perspectives in a global and European context. *Anal Bioanal Chem*, 2007, 389: 147–157
- 9 Goswami R S, Kistler H C. Heading for disaster: *Fusarium graminearum* on cereal crops. *Mol Plant Pathol*, 2004, 5: 515–525
- 10 Lee H J, Ryu D. Worldwide occurrence of mycotoxins in cereals and cereal-derived food products: public health perspectives of their co-occurrence. *J Agric Food Chem*, 2017, 65: 7034–7051
- 11 Streit E, Naehrer K, Rodrigues I, et al. Mycotoxin occurrence in feed and feed raw materials worldwide: long-term analysis with special focus on Europe and Asia. *J Sci Food Agric*, 2013, 93: 2892–2899
- 12 National Health and Family Planning Commission of the PRC, China Food and Drug Administration. GB 2761-2017, National Standard for Food Safety. Limit of Mycotoxins in Food (in Chinese). 2017 [国家卫生计生委, 食品药品监管总局. GB 2761-2017, 食品安全国家标准 食品中真菌毒素限量. 2017]
- 13 Jiang C, Hei R, Yang Y, et al. An orphan protein of *Fusarium graminearum* modulates host immunity by mediating proteasomal degradation of TaSnRK1 α . *Nat Commun*, 2020, 11: 4382
- 14 Wang C, Zhang S, Hou R, et al. Functional analysis of the kinome of the wheat scab fungus *Fusarium graminearum*. *PLoS Pathog*, 2011, 7: e1002460
- 15 Yun Y, Liu Z, Yin Y, et al. Functional analysis of the *Fusarium graminearum* phosphatome. *New Phytol*, 2015, 207: 119–134
- 16 Hou Z, Xue C, Peng Y, et al. A mitogen-activated protein kinase gene (*MGV1*) in *Fusarium graminearum* is required for female fertility, heterokaryon formation, and plant infection. *Mol Plant Microbe Interact*, 2002, 15: 1119–1127
- 17 Gu Q, Zhang C, Liu X, et al. A transcription factor FgSte12 is required for pathogenicity in *Fusarium graminearum*. *Mol Plant Pathol*, 2015, 16: 1–13
- 18 Jenczmionka N J, Maier F J, Lösch A P, et al. Mating, conidiation and pathogenicity of *Fusarium graminearum*, the main causal agent of the head-blight disease of wheat, are regulated by the MAP kinase gpmk1. *Curr Genet*, 2003, 43: 87–95
- 19 Ochiai N, Tokai T, Nishiuchi T, et al. Involvement of the osmosensor histidine kinase and osmotic stress-activated protein kinases in the regulation of secondary metabolism in *Fusarium graminearum*. *Biochem Biophys Res Commun*, 2007, 363: 639–644
- 20 Jiang J, Yun Y, Fu J, et al. Involvement of a putative response regulator FgRrg-1 in osmotic stress response, fungicide resistance and virulence in *Fusarium graminearum*. *Mol Plant Pathol*, 2011, 12: 425–436
- 21 Van Nguyen T, Kröger C, Bönninghausen J, et al. The ATF/CREB transcription factor Atf1 is essential for full virulence, deoxynivalenol

- production, and stress tolerance in the cereal pathogen *Fusarium graminearum*. *Mol Plant Microbe Interact*, 2013, 26: 1378–1394
- 22 Urban M, Mott E, Farley T, et al. The *Fusarium graminearum* *MAPI* gene is essential for pathogenicity and development of perithecia. *Mol Plant Pathol*, 2003, 4: 347–359
- 23 Ren J, Li C, Gao C, et al. Deletion of *FgHOG1* is suppressive to the *mgv1* mutant by stimulating *gpmk1* activation and avoiding intracellular turgor elevation in *Fusarium graminearum*. *Front Microbiol*, 2019, 10: 1073
- 24 Gu Q, Chen Y, Liu Y, et al. The transmembrane protein FgSho1 regulates fungal development and pathogenicity via the MAPK module Ste50-Ste11-Ste7 in *Fusarium graminearum*. *New Phytol*, 2015, 206: 315–328
- 25 Jiang C, Cao S, Wang Z, et al. An expanded subfamily of G-protein-coupled receptor genes in *Fusarium graminearum* required for wheat infection. *Nat Microbiol*, 2019, 4: 1582–1591
- 26 Hu S, Zhou X, Gu X, et al. The cAMP-PKA pathway regulates growth, sexual and asexual differentiation, and pathogenesis in *Fusarium graminearum*. *Mol Plant Microbe Interact*, 2014, 27: 557–566
- 27 Bormann J, Boenisch M J, Brückner E, et al. The adenylyl cyclase plays a regulatory role in the morphogenetic switch from vegetative to pathogenic lifestyle of *Fusarium graminearum* on wheat. *PLoS ONE*, 2014, 9: e91135
- 28 Yin T, Zhang Q, Wang J, et al. The cyclase-associated protein FgCap1 has both protein kinase A-dependent and -independent functions during deoxynivalenol production and plant infection in *Fusarium graminearum*. *Mol Plant Pathol*, 2018, 19: 552–563
- 29 Jiang C, Zhang C, Wu C, et al. *TRI6* and *TRI10* play different roles in the regulation of deoxynivalenol (DON) production by cAMP signalling in *Fusarium graminearum*. *Environ Microbiol*, 2016, 18: 3689–3701
- 30 Li C, Zhang Y, Wang H, et al. The PKR regulatory subunit of protein kinase A (PKA) is involved in the regulation of growth, sexual and asexual development, and pathogenesis in *Fusarium graminearum*. *Mol Plant Pathol*, 2018, 19: 909–921
- 31 Yu H Y, Seo J A, Kim J E, et al. Functional analyses of heterotrimeric G protein $G\alpha$ and $G\beta$ subunits in *Gibberella zeae*. *Microbiology*, 2008, 154: 392–401
- 32 Bluhm B H, Zhao X, Flaherty J E, et al. *RAS2* regulates growth and pathogenesis in *Fusarium graminearum*. *Mol Plant Microbe Interact*, 2007, 20: 627–636
- 33 Cavinder B, Hamam A, Lew R R, et al. Mid1, a mechanosensitive calcium ion channel, affects growth, development, and ascospore discharge in the filamentous fungus *Gibberella zeae*. *Eukaryot Cell*, 2011, 10: 832–841
- 34 Hallen H E, Trail F. The L-type calcium ion channel *ech1* affects ascospore discharge and mycelial growth in the filamentous fungus *Gibberella zeae* (anamorph *Fusarium graminearum*). *Eukaryot Cell*, 2008, 7: 415–424
- 35 Cavinder B, Trail F. Role of *Fig1*, a component of the low-affinity calcium uptake system, in growth and sexual development of filamentous fungi. *Eukaryot Cell*, 2012, 11: 978–988
- 36 Yu F, Gu Q, Yun Y, et al. The TOR signaling pathway regulates vegetative development and virulence in *Fusarium graminearum*. *New Phytol*, 2014, 203: 219–232
- 37 Liu N, Yun Y, Yin Y, et al. Lipid droplet biogenesis regulated by the FgNem1/Spo7-FgPah1 phosphatase cascade plays critical roles in fungal development and virulence in *Fusarium graminearum*. *New Phytol*, 2019, 223: 412–429
- 38 Oide S, Moeder W, Krasnoff S, et al. *NPS6*, encoding a nonribosomal peptide synthetase involved in siderophore-mediated iron metabolism, is a conserved virulence determinant of plant pathogenic ascomycetes. *Plant Cell*, 2006, 18: 2836–2853
- 39 Gaffoor I, Trail F. Characterization of two polyketide synthase genes involved in zearalenone biosynthesis in *Gibberella zeae*. *Appl Environ Microbiol*, 2006, 72: 1793–1799
- 40 Jia L J, Tang H Y, Wang W Q, et al. A linear nonribosomal octapeptide from *Fusarium graminearum* facilitates cell-to-cell invasion of wheat. *Nat Commun*, 2019, 10: 922
- 41 Zhang H, Li B, Fang Q, et al. SNARE protein FgVam7 controls growth, asexual and sexual development, and plant infection in *Fusarium graminearum*. *Mol Plant Pathol*, 2016, 17: 108–119
- 42 Li Y, Li B, Liu L, et al. FgMon1, a guanine nucleotide exchange factor of FgRab7, is important for vacuole fusion, autophagy and plant infection in *Fusarium graminearum*. *Sci Rep*, 2015, 5: 18101
- 43 Veneault-Fourrey C, Barooah M, Egan M, et al. Autophagic fungal cell death is necessary for infection by the rice blast fungus. *Science*, 2006, 312: 580–583
- 44 Reyes-Dominguez Y, Boedi S, Sulyok M, et al. Heterochromatin influences the secondary metabolite profile in the plant pathogen *Fusarium*

- graminearum*. *Fungal Genet Biol*, 2012, 49: 39–47
- 45 Studt L, Rösler S M, Burkhardt I, et al. Knock-down of the methyltransferase Kmt6 relieves H3K27me3 and results in induction of cryptic and otherwise silent secondary metabolite gene clusters in *Fusarium fujikuroi*. *Environ Microbiol*, 2016, 18: 4037–4054
 - 46 Liu Y, Liu N, Yin Y, et al. Histone H3K4 methylation regulates hyphal growth, secondary metabolism and multiple stress responses in *Fusarium graminearum*. *Environ Microbiol*, 2015, 17: 4615–4630
 - 47 Chen Y, Wang J, Yang N, et al. Wheat microbiome bacteria can reduce virulence of a plant pathogenic fungus by altering histone acetylation. *Nat Commun*, 2018, 9: 3429
 - 48 Kong X, van Diepeningen A D, van der Lee T A J, et al. The *Fusarium graminearum* histone acetyltransferases are important for morphogenesis, DON biosynthesis, and pathogenicity. *Front Microbiol*, 2018, 9: 654
 - 49 Lee Y, Min K, Son H, et al. ELP3 is involved in sexual and asexual development, virulence, and the oxidative stress response in *Fusarium graminearum*. *Mol Plant Microbe Interact*, 2014, 27: 1344–1355
 - 50 Li Y, Wang C, Liu W, et al. The *HDF1* histone deacetylase gene is important for conidiation, sexual reproduction, and pathogenesis in *Fusarium graminearum*. *Mol Plant Microbe Interact*, 2011, 24: 487–496
 - 51 Wang Q, Huang P, Zhou S. Functional characterization of the catalytic and bromodomain of FgGCN5 in development, DON production and virulence of *Fusarium graminearum*. *J Integr Agric*, 2020, 19: 2477–2487
 - 52 Son H, Fu M, Lee Y, et al. A novel transcription factor gene *FHS1* is involved in the DNA damage response in *Fusarium graminearum*. *Sci Rep*, 2016, 6: 21572
 - 53 Liu Z Y. The study of fungicide sensitivity regulated by transcription factor FgTeb and microtubule-associated protein FgEB1 in *Fusarium graminearum* (in Chinese). Dissertation for Doctoral Degree. Hangzhou: Zhejiang University, 2017 [刘尊勇. 转录因子FgTeb及微管末端结合蛋白FgEB1调控禾镰刀菌药剂敏感性机制研究. 博士学位论文. 杭州: 浙江大学, 2017]
 - 54 Shi D, Ren W, Jin W, et al. The transcription factor FgNsf1 regulates fungal development, virulence and stress responses in *Fusarium graminearum*. *J Integr Agric*, 2020, doi: 10.1016/S2095-3119(20)63339-1
 - 55 Chen L, Tong Q, Zhang C, et al. The transcription factor FgCrz1A is essential for fungal development, virulence, deoxynivalenol biosynthesis and stress responses in *Fusarium graminearum*. *Curr Genet*, 2019, 65: 153–166
 - 56 Zhang X, Cao S, Li W, et al. Functional characterization of calcineurin-responsive transcription factors Fg01341 and Fg01350 in *Fusarium graminearum*. *Front Microbiol*, 2020, 11: 597998
 - 57 Montibus M, Ducos C, Bonnin-Verdal M N, et al. The bZIP transcription factor Fgap1 mediates oxidative stress response and trichothecene biosynthesis but not virulence in *Fusarium graminearum*. *PLoS ONE*, 2013, 8: e83377
 - 58 Liu Z, Jian Y, Chen Y, et al. A phosphorylated transcription factor regulates sterol biosynthesis in *Fusarium graminearum*. *Nat Commun*, 2019, 10: 1228
 - 59 Cao S, Li W, Li C, et al. The CHY-type zinc finger protein FgChy1 regulates polarized growth, pathogenicity, and microtubule assembly in *Fusarium graminearum*. *Mol Plant Microbe Interact*, 2021, 34: 362–375
 - 60 Lv W, Wu J, Xu Z, et al. The putative histone-like transcription factor FgHltf1 is required for vegetative growth, sexual reproduction, and virulence in *Fusarium graminearum*. *Curr Genet*, 2019, 65: 981–994
 - 61 Fan G, Zheng H, Zhang K, et al. FgHtf1 regulates global gene expression towards aerial mycelium and conidiophore formation in the cereal fungal pathogen *Fusarium graminearum*. *Appl Environ Microbiol*, 2020, 86: e03024–19
 - 62 Lysøe E, Pasquali M, Breakspear A, et al. The transcription factor FgStuAp influences spore development, pathogenicity, and secondary metabolism in *Fusarium graminearum*. *Mol Plant Microbe Interact*, 2011, 24: 54–67
 - 63 Kim Y, Kim H, Son H, et al. MYT3, a myb-like transcription factor, affects fungal development and pathogenicity of *Fusarium graminearum*. *PLoS ONE*, 2014, 9: e94359
 - 64 Gao T, Zheng Z, Hou Y, et al. Transcription factors spt3 and spt8 are associated with conidiation, mycelium growth, and pathogenicity in *Fusarium graminearum*. *FEMS Microbiol Lett*, 2014, 351: 42–50
 - 65 Wang Y, Liu W, Hou Z, et al. A novel transcriptional factor important for pathogenesis and ascosporeogenesis in *Fusarium graminearum*. *Mol Plant Microbe Interact*, 2011, 24: 118–128
 - 66 Jiang J, Yun Y, Yang Q, et al. A type 2C protein phosphatase FgPtc3 is involved in cell wall integrity, lipid metabolism, and virulence in *Fusarium graminearum*. *PLoS ONE*, 2011, 6: e25311

- 67 Kim H K, Yun S H. Functional roles of a putative B δ regulatory subunit and a catalytic subunit of protein phosphatase 2A in the cereal pathogen *Fusarium graminearum*. *Plant Pathol J*, 2012, 28: 259–269
- 68 Liu S M, Duan Y B, Ge C Y, et al. Functional analysis of the β 2-tubulin gene of *Fusarium graminearum* and the β -tubulin gene of *Botrytis cinerea* by homologous replacement. *Pest Manag Sci*, 2013, 69: 582–588
- 69 Zhang J. Identification and functional analysis of FgKAR9 and its interaction protein in *Fusarium graminearum* (in Chinese). Dissertation for Master's Degree. Yangling: Northwestern A&F University, 2018 [张菊. 禾谷镰刀菌中FgKAR9及其互作蛋白的鉴定与功能分析. 硕士学位论文. 杨凌: 西北农林科技大学, 2018]
- 70 Alexander N J, Proctor R H, McCormick S P. Genes, gene clusters, and biosynthesis of trichothecenes and fumonisins in *Fusarium*. *Toxin Rev*, 2009, 28: 198–215
- 71 Gale L R, Bryant J D, Calvo S, et al. Chromosome complement of the fungal plant pathogen *Fusarium graminearum* based on genetic and physical mapping and cytological observations. *Genetics*, 2005, 171: 985–1001
- 72 McCormick S P, Stanley A M, Stover N A, et al. Trichothecenes: from simple to complex mycotoxins. *Toxins*, 2011, 3: 802–814
- 73 Wang H, Sun S, Ge W, et al. Horizontal gene transfer of *Fhb7* from fungus underlies *Fusarium* head blight resistance in wheat. *Science*, 2020, 368: eaba5435
- 74 Kang Z S, Huang L L, Buchenauer H, et al. Cytology of infection process of *Fusarium graminearum* on wheat spikes (in Chinese). *Acta Phytopathol Sin*, 2004, 34: 329–335 [康振生, 黄丽丽, Buchenauer H, 等. 禾谷镰刀菌在小麦穗部侵染过程的细胞学研究. 植物病理学报, 2004, 34: 329–335]
- 75 Parry D W, Jenkinson P, McLEOD L. *Fusarium* ear blight (scab) in small grain cereals—a review. *Plant Pathol*, 1995, 44: 207–238
- 76 Proctor R H, Hohn T M, McCormick S P. Reduced virulence of *Gibberella zeae* caused by disruption of a trichothecene toxin biosynthetic gene. *Mol Plant Microbe Interact*, 1995, 8: 593–601
- 77 Bai G H, Plattner R, Desjardins A, et al. Resistance to *Fusarium* head blight and deoxynivalenol accumulation in wheat. *Plant Breeding*, 2001, 120: 1–6
- 78 Bai G H, Desjardins A E, Plattner R D. Deoxynivalenol-nonproducing *Fusarium graminearum* causes initial infection, but does not cause disease spread in wheat spikes. *Mycopathologia*, 2002, 153: 91–98
- 79 Desjardins A E, Proctor R H, Bai G H, et al. Reduced virulence of trichothecene-nonproducing mutants of *Gibberella zeae* in wheat field tests. *Mol Plant Microbe Interact*, 1996, 9: 775–781
- 80 Langevin F, Eudes F, Comeau A. Effect of trichothecenes produced by *Fusarium graminearum* during *Fusarium* head blight development in six cereal species. *Eur J Plant Pathol*, 2004, 110: 735–746
- 81 Ponts N, Pinson-Gadais L, Barreau C, et al. Exogenous H₂O₂ and catalase treatments interfere with *Tri* genes expression in liquid cultures of *Fusarium graminearum*. *FEBS Lett*, 2007, 581: 443–447
- 82 Ponts N. Mycotoxins are a component of *Fusarium graminearum* stress-response system. *Front Microbiol*, 2015, 6: 1234
- 83 Boutigny A L, Richard-Forget F, Barreau C. Natural mechanisms for cereal resistance to the accumulation of *Fusarium trichothecenes*. *Eur J Plant Pathol*, 2008, 121: 411–423
- 84 Zhou J M. Fighting *Fusarium* head blight in wheat—a Remedy from Afar (in Chinese). *Chin Bull Bot*, 2020, 55: 1–3 [周俭民. 小麦抗赤霉病利器——他山之石. 植物学报, 2020, 55: 1–3]
- 85 Liu C M. The war between wheat and *Fusarium*: fighting with an alien weapon. *Sci China Life Sci*, 2020, 63: 1425–1427
- 86 Ito M, Sato I, Ishizaka M, et al. Bacterial cytochrome P450 system catabolizing the *Fusarium* toxin deoxynivalenol. *Appl Environ Microbiol*, 2013, 79: 1619–1628
- 87 Chen X, Steed A, Travella S, et al. *Fusarium graminearum* exploits ethylene signalling to colonize dicotyledonous and monocotyledonous plants. *New Phytol*, 2009, 182: 975–983
- 88 Saville R J, Gosman N, Burt C J, et al. The 'Green Revolution' dwarfing genes play a role in disease resistance in *Triticum aestivum* and *Hordeum vulgare*. *J Exp Bot*, 2012, 63: 1271–1283
- 89 Zuo D Y, Yi S Y, Liu R J, et al. A deoxynivalenol-activated methionyl-tRNA synthetase gene from wheat encodes a nuclear localized protein and protects plants against *Fusarium* pathogens and mycotoxins. *Phytopathology*, 2016, 106: 614–623
- 90 Mitterbauer R, Adam G. *Saccharomyces cerevisiae* and *Arabidopsis thaliana*: useful model systems for the identification of molecular mechanisms involved in resistance of plants to toxins. *Eur J Plant Pathol*, 2002, 108: 699–703

- 91 Walter S, Kahla A, Arunachalam C, et al. A wheat ABC transporter contributes to both grain formation and mycotoxin tolerance. *J Exp Bot*, 2015, 66: 2583–2593
- 92 Poppenberger B, Berthiller F, Lucyshyn D, et al. Detoxification of the *Fusarium* mycotoxin deoxynivalenol by a UDP-glucosyltransferase from *Arabidopsis thaliana*. *J Biol Chem*, 2003, 278: 47905–47914
- 93 Lulin M, Yi S, Aizhong C, et al. Molecular cloning and characterization of an up-regulated UDP-glucosyltransferase gene induced by DON from *Triticum aestivum* L. cv. Wangshuibai. *Mol Biol Rep*, 2010, 37: 785–795
- 94 Li X, Shin S, Heinen S, et al. Transgenic wheat expressing a barley UDP-glucosyltransferase detoxifies deoxynivalenol and provides high levels of resistance to *Fusarium graminearum*. *Mol Plant Microbe Interact*, 2015, 28: 1237–1246
- 95 Shin S, Torres-Acosta J A, Heinen S J, et al. Transgenic *Arabidopsis thaliana* expressing a barley UDP-glucosyltransferase exhibit resistance to the mycotoxin deoxynivalenol. *J Exp Bot*, 2012, 63: 4731–4740
- 96 Schweiger W, Pasquet J C, Nussbaumer T, et al. Functional characterization of two clusters of *Brachypodium distachyon* UDP-glucosyltransferases encoding putative deoxynivalenol detoxification genes. *Mol Plant Microbe Interact*, 2013, 26: 781–792
- 97 Pasquet J C, Changenet V, Macadré C, et al. A *Brachypodium* UDP-glucosyltransferase confers root tolerance to deoxynivalenol and resistance to *Fusarium* infection. *Plant Physiol*, 2016, 172: 559–574
- 98 Schweiger W, Steiner B, Ametz C, et al. Transcriptomic characterization of two major *Fusarium* resistance quantitative trait loci (QTLs), *Fhb1* and *Qfhs.ifa-5A*, identifies novel candidate genes. *Mol Plant Pathol*, 2013, 14: 772–785
- 99 Xing L, Gao L, Chen Q, et al. Over-expressing a UDP-glucosyltransferase gene (*Ta-UGT₃*) enhances *Fusarium* head blight resistance of wheat. *Plant Growth Regul*, 2018, 84: 561–571
- 100 Zhao L, Ma X, Su P, et al. Cloning and characterization of a specific UDP-glycosyltransferase gene induced by don and *Fusarium graminearum*. *Plant Cell Rep*, 2018, 37: 641–652
- 101 Gatti M, Choulet F, Macadré C, et al. Identification, molecular cloning, and functional characterization of a wheat UDP-glucosyltransferase involved in resistance to *Fusarium* head blight and to mycotoxin accumulation. *Front Plant Sci*, 2018, 9: 1853
- 102 Perochon A, Jianguang J, Kahla A, et al. *TaFROG* encodes a pooideae orphan protein that interacts with SnRK1 and enhances resistance to the mycotoxigenic fungus *Fusarium graminearum*. *Plant Physiol*, 2015, 169: 2895–2906
- 103 Ding L, Xu H, Yi H, et al. Resistance to hemi-biotrophic *F. graminearum* infection is associated with coordinated and ordered expression of diverse defense signaling pathways. *PLoS ONE*, 2011, 6: e19008
- 104 Boenisch M J, Schäfer W. *Fusarium graminearum* forms mycotoxin producing infection structures on wheat. *BMC Plant Biol*, 2011, 11: 110
- 105 Bushnell W M, Hazen B E, Pritsch C. Histology and physiology of *Fusarium* head blight. In: Leonard K J, Bushnell W R, eds. *Fusarium Head Blight of Wheat and Barley*. St. Paul: APS Press, 2003. 44–83
- 106 Reid L M, Mather D E, Bolton A T, et al. Evidence for a gene for silk resistance to *Fusarium graminearum* Schw. Ear rot of maize. *J Hered*, 1994, 85: 118–121
- 107 Jansen C, von Wettstein D, Schäfer W, et al. Infection patterns in barley and wheat spikes inoculated with wild-type and trichodiene synthase gene disrupted *Fusarium graminearum*. *Proc Natl Acad Sci USA*, 2005, 102: 16892–16897
- 108 Kang Z, Buchenauer H. Cytology and ultrastructure of the infection of wheat spikes by *Fusarium culmorum*. *Mycol Res*, 2000, 104: 1083–1093
- 109 Su P S. Isolation and functional study of wheat (*Triticum aestivum* L.) resistance genes for *Fusarium* head blight (in Chinese). Dissertation for Doctoral Degree. Tai'an: Shandong Agricultura University, 2020 [苏培森. 小麦抗赤霉病相关基因的分离和功能研究. 博士学位论文. 泰安: 山东农业大学, 2020]
- 110 Zhang X, Lee T, Dufresne M, et al. Infection of green fluorescence protein-tagged *Fusarium graminearum* on wheat and barley spikes. *Cereal Res Commun*, 2008, 36: 465–469
- 111 Zhang Y H, Ye H Z. A study on histology of resistance of wheat to wheat scab (*Fusarium graminearum*) (in Chinese). *J Sichuan Agric Univ*, 1993, 11: 444–450 [张永红, 叶华智. 小麦品种抗赤霉病的组织学研究. 四川农业大学学报, 1993, 11: 444–450]
- 112 Hükelhoven R. Cell wall-associated mechanisms of disease resistance and susceptibility. *Annu Rev Phytopathol*, 2007, 45: 101–127
- 113 Bellincampi D, Cervone F, Lionetti V. Plant cell wall dynamics and wall-related susceptibility in plant-pathogen interactions. *Front Plant Sci*, 2014, 5: 228
- 114 Hadfield K A, Bennett A B. Polygalacturonases: many genes in search of a function. *Plant Physiol*, 1998, 117: 337–343
- 115 Bézier A, Lambert B, Baillieux F. Cloning of a grapevine botrytis-responsive gene that has homology to the tobacco hypersensitivity-related

- hsr203J. *J Exp Bot*, 2002, 53: 2279–2280
- 116 Cervone F, Hahn M G, De Lorenzo G, et al. Host-Pathogen Interactions: XXXIII. A plant protein converts a fungal pathogenesis factor into an elicitor of plant defense responses. *Plant Physiol*, 1989, 90: 542–548
- 117 De Lorenzo G, D'Ovidio R, Cervone F. The role of polygalacturonase-inhibiting proteins (PGIPS) in defense against pathogenic fungi. *Annu Rev Phytopathol*, 2001, 39: 313–335
- 118 De Lorenzo G, Ferrari S. Polygalacturonase-inhibiting proteins in defense against phytopathogenic fungi. *Curr Opin Plant Biol*, 2002, 5: 295–299
- 119 D'Ovidio R, Raiola A, Capodicasa C, et al. Characterization of the complex locus of bean encoding polygalacturonase-inhibiting proteins reveals subfunctionalization for defense against fungi and insects. *Plant Physiol*, 2004, 135: 2424–2435
- 120 Federici L, Di Matteo A, Fernandez-Recio J, et al. Polygalacturonase inhibiting proteins: players in plant innate immunity? *Trends Plant Sci*, 2006, 11: 65–70
- 121 Kang Z S, Huang L L, Han Q M, et al. Cytochemistry of cell wall component alterations in wheat spikes infected by *Fusarium graminearum* (in Chinese). *Acta Phytopathol Sin*, 2007, 6: 623–628 [康振生, 黄丽丽, 韩青梅, 等. 禾谷镰刀菌侵染导致小麦穗组织细胞壁成分变化的细胞化学研究. *植物病理学报*, 2007, 6: 623–628]
- 122 Bowman J L, Kohchi T, Yamato K T, et al. Insights into land plant evolution garnered from the marchantia polymorpha genome. *Cell*, 2017, 171: 287–304.e15
- 123 Yue J, Hu X, Sun H, et al. Widespread impact of horizontal gene transfer on plant colonization of land. *Nat Commun*, 2012, 3: 1152
- 124 Guan Y, Liu L, Wang Q, et al. Gene refashioning through innovative shifting of reading frames in mosses. *Nat Commun*, 2018, 9: 1555
- 125 Zhang J, Fu X X, Li R Q, et al. The hornwort genome and early land plant evolution. *Nat Plants*, 2020, 6: 107–118
- 126 Wang S, Guan Y, Wang Q, et al. A mycorrhizae-like gene regulates stem cell and gametophore development in mosses. *Nat Commun*, 2020, 11: 2030
- 127 Sun G, Bai S, Guan Y, et al. Are fungi-derived genomic regions related to antagonism towards fungi in mosses? *New Phytol*, 2020, 228: 1169–1175
- 128 Shinozuka H, Hettiarachchige I K, Shinozuka M, et al. Horizontal transfer of a β -1,6-glucanase gene from an ancestral species of fungal endophyte to a cool-season grass host. *Sci Rep*, 2017, 7: 9024
- 129 Su Z, Bernardo A, Tian B, et al. A deletion mutation in *TaHRC* confers Fhb1 resistance to *Fusarium* head blight in wheat. *Nat Genet*, 2019, 51: 1099–1105
- 130 Ma Z, Xie Q, Li G, et al. Germplasms, genetics and genomics for better control of disastrous wheat *Fusarium* head blight. *Theor Appl Genet*, 2020, 133: 1541–1568
- 131 Rawat N, Pumphrey M O, Liu S, et al. Wheat *Fhb1* encodes a chimeric lectin with agglutinin domains and a pore-forming toxin-like domain conferring resistance to *Fusarium* head blight. *Nat Genet*, 2016, 48: 1576–1580
- 132 Li G, Zhou J, Jia H, et al. Mutation of a histidine-rich calcium-binding-protein gene in wheat confers resistance to *Fusarium* head blight. *Nat Genet*, 2019, 51: 1106–1112
- 133 Wang Q, Huang J. Fungal genes in plants: impact and potential applications. *Trends Plant Sci*, 2020, 25: 1064–1067

Advances in understanding the mechanisms of wheat-*Fusarium graminearum* interactions

SU PeiSen^{1,2}, GE WenYang¹, WANG HongWei¹ & KONG LingRang¹

1 State Key Laboratory of Crop Biology, Agronomy College of Shandong Agricultural University, Tai'an 271018, China;

2 College of Agronomy, Liaocheng University, Liaocheng 252000, China

Fusarium head blight (FHB) caused by *Fusarium graminearum* is one of the most serious and hazardous crop diseases which occurs on most wheat-growing regions worldwide, posing a serious threat to wheat production and food security. The control of FHB requires a deeper understanding of the mechanisms underlying host plant resistance to *F. graminearum* and discovery of the resistance genes. This review gave a systematic summary concerning the following four aspects: (i) pathogenesis of *F. graminearum*, (ii) the mechanism underlying the interaction between *F. graminearum* pathogenic factor DON and wheat, (iii) the mechanism underlying the interaction between *F. graminearum* infection and wheat recognition, and (iv) host-pathogen horizontal gene transfer.

wheat, fusarium head blight (FHB), *Fusarium graminearum*, interaction mechanisms, deoxynivalenol (DON)

doi: [10.1360/SSV-2020-0355](https://doi.org/10.1360/SSV-2020-0355)