木霉菌的生物防治机理*

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摘　要：木霉菌是重要的植物病害生物防治菌，本文从木霉菌通过对营养和空间的竞争来抑制病原真菌的生长和繁殖，木霉菌对病原真菌的重寄生机制，木霉菌产生抗生素抑制或杀死病原真菌的抗生机制，以及木霉菌促进植物生长诱导植物系统抗病性机制等综述木霉的生物防治机制。为全面理解木霉的对植物真菌病害生物防治机制提供理论指导。

关键词：木霉；植物病害；生物防治

The Mechanism of *Trichoderma spp.* Biological Control to Fungal plant phytopathogen

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Abstract: *Trichoderma spp*. is effective biocontrol agents for fungal plant phytopathogen. *Trichoderma* biocontrol mechanism includes competition for nutrients and space and mycoparasitism to fungal plant phytopathogen, production of antibiotics, promotion plant growth, and induction plant systemic resistance responses, etc. In the paper which we concluded will provide theoretical guidance for the comprehensive understanding the role of *Trichoderma spp.* in biological control mechanisms for plant fungus diseases.

Keywords: *Trichoderma*, fungal plant pathogens, biological control

植物真菌病害生物防治菌木霉（Trichoderma spp.）对多种植物真菌病害具有较好的防治效果。目前，世界已有商品化木霉生防菌剂（Biological Fungicide）投放市场。比如加拿大的“RootShield”杀菌剂（T. harzianum KRL-AG2）；美国的“F-Stop”杀菌剂（T. harzianum T-22G）；印度的“SARDAR ECO GREEN”（T. harzianum）以及中国的“木霉菌”（农药登记号LS20083122）等均为通过相关机构注册的商品化的杀真菌生物农药。木霉菌作为生物防治因子的优势首先在于对多种植物病原菌具有广谱的抗菌特性，比如可以防治可可黑荚病(Black-pod disease) (Tondje et al. , 2007；Hanada et al. , 2009)),葡萄孢疫病（Botrytis blight）(Olson et al.,2007)及西红柿和黄瓜枯萎病 (Fusarium wilt)(Chen et al. ,2010; Segarra et al. ,2010)，等多种真菌病害；不仅可以防治农作物及林木生长期病害还可防治采摘后的蔬菜

水果及园艺花卉的材料储存期病害（郭润芳等，2001）；同时还能刺激种子萌发、根的伸长、植物生长并且提早开花结实(Viterbo et al. 2010)。兼具杀真菌生物农药与促生长生物肥料(Chen L, et al.2011)及土壤改良剂等多种功效。

木霉菌的生防机制研究为木霉生物防治上的应用提供理论指导，木霉菌生物防治机制包括定植在植物根际的木霉菌通过对营养和空间的竞争来抑制病原真菌的生长和繁殖(Tondje et al. ,2007；Morán-Diez et al.,2009)，木霉菌对病原真菌的重寄生机制（Harman，2006），木霉菌产生抗生素抑制或杀死病原真菌的抗生机制(Anees et al.; Tijerino et al. 2010)，以及木霉菌促进植物生长诱导植物系统抗病性机制等（Benitez et al., 2004)等等。本文综述木霉的生物防治机制。

1、木霉与病原菌互作

木霉一般以重寄生或腐生重寄生形式与其他真菌的直接作用。从分子水平上对75种1100余株木霉菌调查显示，所有测试的木霉种对链格孢菌（Alternaria alternata），灰霉病菌（Botrytis cinerea）， 核盘菌（Sclerotinia sclerotiorum）这三种植物病原菌拥有潜在的重寄生能力。同时，木霉还能以死的真菌菌丝体为食，所以他们的生活方式包括腐生和活体营养两种营养方式（Harman ,2011）。

1.1识别寄主病原菌的存在

目前，3种生防木霉菌（深绿木霉，绿色木霉，T. jecorina）的基因组测序完成（Kubicek et al., 2011）以及木霉转录组学的应用（Lorito et al., 2010；Seidl et al., 2009）对木霉和病原菌的互用的分子生理学提供了一些重要的数据支持。许多编码蛋白酶和寡肽转运蛋白的基因在木霉菌和寄主病原菌接触以及接触之前就开始表达（Seidl et al.,2009；Suárez et al., 2007）。这些蛋白酶大多数属于类枯草杆菌蛋白酶组。例如：从不同培养条件下哈茨木霉CECT2413菌株的表达序列标签中可以发现编码这些酶的基因明显过表达（Suárez et al., 2007）。此外，从深绿木霉和寄主病原菌（立枯丝核菌，核盘菌）接触时所获得的表达序列标签可以发现大量的编码类枯草杆菌蛋白酶基因表达（Seidl et al.,2009）。过表达这些蛋白酶的（prb1基因编码）深绿木霉菌株表现出更强的重寄生能力（Flores et al.,1997）。深绿木霉表达的蛋白酶降解寄主病原菌菌丝表面的蛋白，使它释放寡肽类，之后寡肽类跟木霉感受器连在一起（Seidl et al.,2009）。这个机制会让人联想到食线虫真菌用线虫释放的寡肽类来诱捕（Dijksterhuis et al.,1994）（图1）。IV类G蛋白偶联受体（GPCRs）在深绿木霉（Seidl et al.,2009）中担当寡肽类的传感器（Kubicek et al., 2011）。深绿木霉，绿色木霉，T. jecorina基因组各有两个IV类GPCRs旁系同源物（Kubicek et al., 2011）。

GPCRs进一步参与对寄主病原菌的识别。比如，深绿木霉Gpr1蛋白是GPCRs类环磷酸腺苷的受体成员，是深绿木霉重寄生所必须的蛋白（Omann et al.,2009）。受体的进一步信号转导通过包含三个Gα亚基，一个Gβ亚基和一个Gγ亚基的保守G蛋白信号级联实现（图1）。缺失Gα亚基Tga1基因使深绿木霉完全失去了对三个寄主真菌的重寄生能力，而且几丁质酶的活力明显减小，抗真菌化合物6 - 戊基吡喃酮产量下降（Rocha-Ramirez et al. ,2002；Reithner et al. ,2004）；相反， tagA（tga1同系物）基因的缺失只是减少了绿色木霉对菌核菌重寄生能力（Mukherjee et al.,2004）。

促分裂素原活化蛋白激酶（MAPK）信号途径在真菌中最突出的信号转导系统之一（Schmoll et al.,2008）。木霉基因组保护基因harbour genes编码三个MAPKs:致病MAPK（ TmkA; 也称 Tvk1 和Tmk1 ），细胞完整性激酶(TmkB)，调节渗透作用的MAPK(Hog1)（Schmoll et al.,2008）。绿色木霉“P”链（“P”链产生gliovirin，它具有防治腐霉菌的效果）上缺失tmkA基因减弱其对菌核菌的拮抗作用但对核盘菌的作用不变(Mukherjee et al.,2003; Viterbo et al.,2005)。绿色木霉“Q”链上tmkA基因的缺失进一步提高了其对核盘菌和菌核菌的生物防治作用(Mendoza-Mendoza et al.,2007)。“P”和“Q”链的不同代谢模式可能是导致这种结果的原因，关于“P”链基因组的更多信息可以帮助验证这个假设。Tmk1同源基因的缺失可以引起深绿木霉对核盘菌的重寄生能力的减弱但增加几丁质酶和抗真菌化合物的产量(Reithner et al.,2007)。其他两个MAPKs，TmkB 和 Hog1的作用目前了解的不多。因为这些基因的突变体生长能力很弱，妨碍拮抗实验的成功。比如绿色木霉TmkB突变体（Kumar et al.,2010）和深绿木霉Hog1（有关耐渗透和氧化应激的蛋白）突变体（Delgado-Jaran et al.,2006）失去对菌核菌重寄生能力。

1.2 附着在寄主病原菌菌丝上

木霉重寄生需要环绕靶标病原真菌菌丝，并形成螺旋状菌丝（Harman et al.,2011；Harman et al.,2004），这种现象依赖于木霉识别寄主病原菌分泌的凝集素（Inbar et al.,1996）（图1）。此外，植物凝集素也能诱导类似程度的盘绕，表明在木霉附着寄主病原菌过程中凝集素并不是唯一的决定因素（Rocha-Ramirez et al.,2002）。此外，盘绕不是严格跟重寄生相关，没有猎物真菌时一些木霉盘绕自己的菌丝（Lu et al.,2004）。螺旋状生长是许多木霉种鉴别的特征。比如T.spirale 和T. helicum（见Trichoderma Online）。

木霉的重寄生初期，寄主菌丝上生长并形成乳头状小突起，并且乳头状小突起不受寄主真菌种类的影响（Rocha-Ramirez et al.,2002；Chacón et al.,2007）。之后，小突起开始降解细胞壁，侵入/穿透管腔（Harman et al.,2011；Harman et al.,2004；Chacón et al.,2007）。这

些结构和哈茨木霉诱导番茄时所形成的结构相似（Chacón et al.,2007），也与植物病原真菌的附着胞类似。稻瘟病病菌贮藏脂质产生丙三醇，使细胞肿胀，引起机械压力来侵入植物细胞壁（de Jong et al.,1997）。木霉乳头状小突起也为了相似的目的积累丙三醇，重寄生木霉接触阶段有关脂质分解代谢和渗透调节的基因在转录水平上增加（Seidl et al.,2009）。

不仅木霉的寄生菌丝可以接触和缠连潜在寄主病原菌，孢子也可以粘附病原菌菌丝，比如，深绿木霉孢子在萌发前可以粘附在腐霉菌菌丝上（Lu et al.,2004）。分生孢子与寄主菌丝亲和机制未知，但很有可能涉及小分子疏水蛋白，小分子疏水蛋白是含有八个半胱氨酸残基的两亲性蛋白。子囊菌类中木霉富含有这种蛋白（从基因组序列中推断所知）（Kubicek et al.,2008）。

1.3 木霉的防御响应

木霉属另一个特征是病原真菌能诱导其热休克反应，氧化应激和解毒过程的基因（如那些编码ABC转运蛋白和多效性和耐药性转运者）（Lorito et al.,2010；Seidl et al.,2009）（图1）。核盘菌形成菌核时分泌活性氧作为信号分子（Papapostolou et al.,2010），同时还分泌抗真菌的代谢物（Aliferis et al.,2010），活性氧和抗真菌代谢物都可能诱发木霉的防御反应。敲除深绿木霉编码ABC转运蛋白基因Abc2，导致其对核盘菌的生物防治能力减弱，证明该蛋白在重寄生过程中起解毒作用（Ruocco et al.,2009）。

1.4 杀死病原真菌

木霉分泌的抗真菌次生代谢物和细胞壁水解酶协同作用最终导致病原真菌的死亡。木霉基因组上富含编码合成次生代谢产物和细胞壁水解酶的基因，这反映出这些物质在重寄生过程中起重要作用（Kubicek et al.,2011）。比如绿色木霉和其他真菌相比有最多数量的（28个）非核糖体肽合成酶。此外，深绿木霉和绿色木霉同源基因（T. jecorina没有）似乎可以编码合成次生代谢产物的蛋白（Kubicek et al.,2011），从而代表未知抗菌化合物的合成机制。

细胞壁主要由几丁质，β-1,3-葡聚糖，α-1,3-葡聚糖和α-1,4-葡聚糖组成（Latgé et al.,2007），约占真菌细胞干重的30%和。深绿木霉和绿色木霉含有很多几丁质酶（分别为29个和36个）（Kubicek et al.,2008）。几丁质酶Chit33和Chit42中添加纤维素结合模块来提高几丁质酶的活动从而增加哈茨木霉的重寄生能力（Limón et al.,2004）。补加纤维素结合模块使这些几丁质酶更加紧密地结合不溶性甲壳素基质。一些来自木霉的几丁质酶在主动选择下进化的（Ihrmark et al.,2010），这是寄主和病原菌协同进化的典型特点。然而，由于基因重复，一些几丁质酶基因的缺失不会引起木霉重寄生能力或生物防治的能力的减弱（Benítez et al.,2004； Harman et al.,2004）。木霉还包含GH57家族的一套扩展壳聚糖酶，这些蛋白水解

壳聚糖，也能使几丁质（甲壳素）部分脱乙酰化（Kubicek et al.,2011）。

真菌细胞壁第二个富含的是有β-1,6分支的β-1,3-葡聚糖（Latgé et al.,2007），它可以被β-1,3-葡聚糖酶水解。和其他真菌基因组相比木霉基因组编码这种类型酶的基因过表达（Kubicek et al.,2011）。木霉与其寄主真菌相互作用的区域发现β-1,6-葡聚糖酶。哈茨木霉CECT 2413 β-1,6-葡聚糖酶Bgn16.3的过表达可以使它更有效的抑制灰霉病菌，核盘菌，柑桔褐腐疫霉菌的生长（Montero et al.,2007）。哈茨木霉和绿色木霉β-1,6-葡聚糖酶的高产表现出其对核盘菌，灰霉病菌，腐霉菌更有效的生物防治能力（Ihrmark et al.,2010；Djonovic et al.,2006）。

2．植物根际木霉菌的作用

根际是木霉属普遍的生态位，植物根分泌液为木霉生长提供活体营养和腐食营养。在根际木霉种丰富度最高（Mulaw et al.,2010），在非根际的土壤呈现差的物种多样性（Migheli et al.,2009）。木霉和根际的密切关系可以由其两种营养喜好来解释。首先，92％的陆生植物的根都被菌根真菌定殖，菌根真菌是菌根营养植物潜在的寄主。然而，木霉和菌根真菌之间的互作仍然知之甚少（Calvet et al.,1993； Datnoff et al.,1995；McAllister et al.,1994；Nemec et al.,1996；Siddiqui et al.,1996；Green et al.,1999），有一些研究表明这两种类型的真菌之间的协同作用，其他研究观察到木霉攻击丛枝菌根菌，抑制他们在植物根系的定殖。其二，植物的根，尤其是根尖被根冠最外层细胞分泌的由高度水化的多糖比如果胶，半纤维素（尤其是rhamnogalacturonans和阿拉伯木聚糖）组成的凝胶状黏胶囊（称作黏胶层）包围。木霉半纤维素酶很容易降解这些组分，这一作用可能进化为能利用腐木上的病原真菌释放的多糖。如，哈茨木霉CECT 2413定殖在番茄根际需要多聚半乳糖醛酸内切酶（Moran-Diez et al., 2009）。

植物根在根围分泌单糖和双糖类为菌根提供重要的碳源（Nehls et al.,2010）。绿色木霉定殖植物根时蔗糖也有类似的作用（Vargas et al.,2009）。绿色木霉，深绿木霉和T. jecorina基因组上包含编码细胞内（除细胞外）转化酶的基因，蔗糖水解之前被蔗糖透过酶吸收。绿色木霉定殖植物根的早期，其诱导特异性高的蔗糖转运蛋白，它的生化特性和植物蔗糖转运商相似（Vargas et al.,2011），这表明蔗糖主动从植物转移到真菌。另外，深绿木霉和绿色木霉基因组编码大量facilitator solute transporters（Kubicek et al.,2011），获得的其他根分泌液的作用仍然未知。总之，寄主真菌的存在和从根获得的营养素可能是吸引木霉属祖先定殖在根际并和植物互作的的主要原因。

2.1植物防御响应

植物激活其潜在的防御机制来对其他生物的存在做出反应。这是各种植物病原真菌，引起植物两个分支的先天免疫防御的最好解释（Jones et al.,2006）。第一阶段一般是病原体相关分子模式PAMPs或微生物相关的分子模式(MAMPs)的识别并对其作出反应---微生物普遍存在的分子模式一被称为PAMP触发免疫。第二阶段对病原体致病因素作出的响应称效应因子触发的免疫。木霉使有关茉莉酸和乙烯信号途径的组分（如过氧化氢酶，过氧化物酶和苯丙氨酸解氨酶等）积累量达到最高来诱导植物系统抗性（ISR）Shoresh, M., Harman, G. E. & Mastouri, F. 2010（图2）。例如木霉黏胶层的endopectinases释放糖醛酸来激活植物防御机制（Moran-Diez et al.,2009）。T. asperelloides定殖黄瓜根的早期可以发现，植物识别MAMPs或和他们相互作用早期，植物在细胞壁积累更多的纤维素和愈创葡聚糖并释放酚类化合物，阻止木霉进一步定殖（Yedidia et al.,1999；Segarra et al.,2007）。因为木霉不是植物病原真菌，所以他们预计不会引起第二阶段的植物固有免疫反应。然而，棘孢木霉以浓度依赖型方式与黄瓜根相互作用的早期诱导植物系统获得抗病性(SAR,通常存在于第二阶段的植物免疫反应) （Segarra et al., 2007）。必须指出，这些影响只在少数木霉品系中有研究，特别是在能有效刺激植物防御的菌株里,即木霉T.asperelloides T203，绿色木霉，以及原生质体融合杂种“ 哈茨”木霉T-22。

木霉属几类分子作为MAPKs，诱导植物防卫反应，如木聚糖，peptaibols，cerato-platanins（图2）。来自绿木霉T. viride ATCC 52438菌株的木聚糖内切酶Eix（Xny2）是木霉属中首次发现的能够刺激番茄和烟草中乙烯形成的蛋白（Dean et al.,1991），但此菌株的分类地位不确定。有效的生防菌绿色木霉也分泌一种相同于Eix的木聚糖内切酶（Hanson et al.,2004）。在木霉T. jecorina (在JGI T. reesei v2.0基因组上蛋白识别号 123818) 和绿色木霉(在JGI T. virens Gv29-8 v2.0基因组蛋白识别号为 72838) 基因组上发现编码同源酶的基因。

值得注意的是，Eix的催化作用在引起植物防御反应时非关键的（Enkerli et al.,1999；Sharon et al.,1993），因此，酶本身是MAMP而他的反应产物不是。事实上，引起植物响应，Eix与植物Eix受体2（Eix2;也被称为LeEix植物富含亮氨酸重复类受体蛋白超级家族的成员还还携带内吞作用的信号介导受体）结合，这是诱导防御反应所必需的（Ron et al.,2004；Bar et al.,2009）。此外，EIX与植物受体结合导致细胞膜的功能的改变，这也是刺激植物防卫反应所必须的（Bailey et al.,1992）。

通过扰乱编码peptaibol合成酶的基因来阻止peptaibols（一组的非核糖体多肽类）在绿色木霉中的合成导致该菌株不能引起/诱导黄瓜ISR,虽然这是可以添加peptaibol混合物来克服（Viterbo et al.,2007）。peptaibols诱导ISR的机制目前尚不清楚，这可能与这些多肽改变细

胞膜的功能的能力有关。

Swollenin是一种携带纤维素结合模块并能破坏植物细胞壁纤维素结晶结构的蛋白质（Saloheimo et al.,2002）。它在棘孢木霉在植物根根际定殖时起一定的作用并能诱导植物本地防御反应不是系统诱导反应（Brotman et al.,2008）。Swollenin与植物细胞壁扩展蛋白有相似性。植物细胞壁扩展蛋白是一类植物细胞壁蛋白，它能促进根和根毛中细胞壁的伸长（Guo et al.,2011）。木霉在植物根根际定殖时利用swollenin增加其在根际定殖区域。

Cerato-platanins是以四个半胱氨酸残基形成两个二硫键为特点的小分泌蛋白。绿色木霉Cerato-platanin Sm1(也称为Epl1)诱导玉米和棉花的系统诱导抗ISR（Djonovic et al.,2007）。SM1在深绿木霉的同源基因（Epl1）是真菌分泌的主要蛋白之一（Seidl et al.,2006）。SM1的糖基化使它保持单体形式从而诱导ISR（Vargas et al.,2008）。去糖基化导致形成SM1二聚体，其不能诱导ISR。研究表明植物通过去糖基化改变SM1的聚集状态，最终影响其诱导防御反应的能力。H. jecorina，绿色木霉和深绿木霉分别有三个sm1旁系同源物而大多数其他相关属真菌只有一个，这表明Cerato-platanin可能对木霉属很重要。木霉基因组编码的其他富含半胱氨酸的小分泌蛋白与担子菌类外生菌根双色蜡蘑小分泌蛋白所描述相似，累积在菌丝，并在根际定殖时有一定的作用（Martin et al.,2008）。

2.2 促进植物生长

木霉和植物根互作可以促进植物生长（图四）。比如绿色木霉增加拟南芥根系生物量和侧根的生长率。生长素介导的信号传递途径可能在木霉和植物相互作用上起一定的作用，植物途径缺陷型突变体与木霉相互作用的能力减弱（Contreras-Cornejo et al.,2009）。然而，植物激素乙烯的减少也促进植物生长（Wang et al.,2002），T. asperelloides T203 有一个编码抑制乙烯生物合成重要中间产物ACC的α-氨基环丙烷-1-羧酸脱氨酶（acc1）基因，该基因编在T. asperelloide和油菜根互作过程中表达。敲除这个基因时木霉促进根延长的作用减少，因为持续高浓度的乙烯会抑制根的伸长，所以Acc1酶在木霉促进植物生长过程中起非常重要的作用（Viterbo et al.,2010）。

另外，与其它子囊菌类相比，木霉基因组中包含许多编码腈水解酶的基因（Kubicek et al., 2011）。腈水解酶在水解β-cyano-l-alanine（由氰化物组成的代谢物，乙烯生物合成最后一步释放）或植物代谢产物吲哚-3 - 乙腈转换成吲哚-3 - 乙酸（IAA，促进植物根系生长的激素）有一定的作用（Piotrowski et al.,2006）。

3 内生菌

植物中普遍存在细菌和真菌等内生菌，内生菌发挥刺激植物生长、延迟干旱胁迫和阻止

病原体侵害等作用（Bae et al.,2009）。虽然其他许多种可以表现为兼内生真菌但只有少数木霉作为内生真菌分离出来（表1）。除了 T. koningiopsis, T. stilbohypoxyli 外几乎所有分离的内生真菌已被列为新种。T. stromaticum没有已知有性型。系统发育分析将他们放在他们分支的末端位置，这表明木霉属内生性菌近几年进化（Chaverri et al.,2011；Samuels et al.,2006；Samuels et al.,2009）。一些物种例如Trichoderma hamatum即作为植物内生菌也作为土壤和根际习居菌，这样的特点也是其他许多机会性真菌的属性（Rodriguez et al.,2009）。因此也不清楚木霉属是否存在专性内生菌。有趣的是，定殖在马铃薯根的丛枝菌根的菌丝体外侧被木霉用来进入植物根部（De Jaeger et al.,2010），这表明有关菌根营养的特性促进内生菌的进化。木霉属被分离的内生性木霉菌株基因组尚未被测序。

综上所述，木霉菌在植物真菌病害防治中较其他生防菌在生防效果上占优势。从木霉对寄主病原真菌的识别并附着在其菌丝上，木霉对寄主病原真菌存在的响应，最后杀死寄主病原真菌这四个方面对木霉和病原菌的相互作用机制在分子水平方面具有明显得优势；此外，木霉菌还能刺激植物防御响应和促进植物生长。上述两个方面说明了木霉菌在植物根际所发挥的重要作用。最后，木霉菌作为内生菌在植物体内发挥作用，不仅能够促进植物生长，还能延迟干旱胁迫和阻止病原体侵害等。

由于木霉菌生物防治上的绝对优势，美国能源部联合基因组研究所开展的大规模木霉属（如哈茨木霉，长枝木霉和棘孢木霉，深绿木霉，绿色木霉）基因组测序项目“JGI Fungal Genetics Program”，使我们能够在分子水平上更全面分析木霉的特性。这不仅将帮助我们了解木霉生物防治的分子生物学基础,同时也提高了木霉在生物技术，农业和其他领域的应用。

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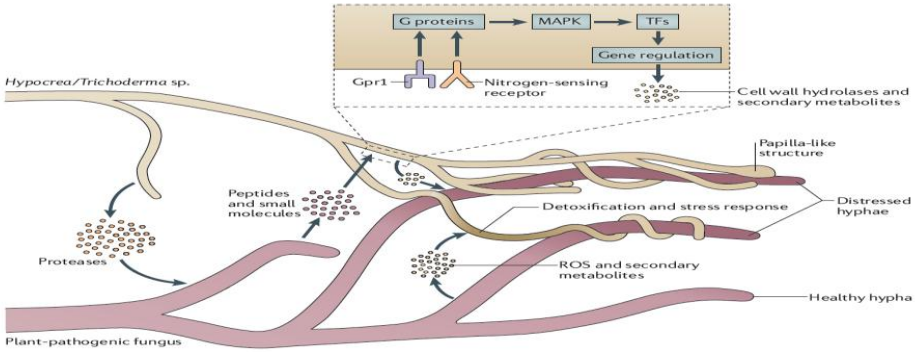
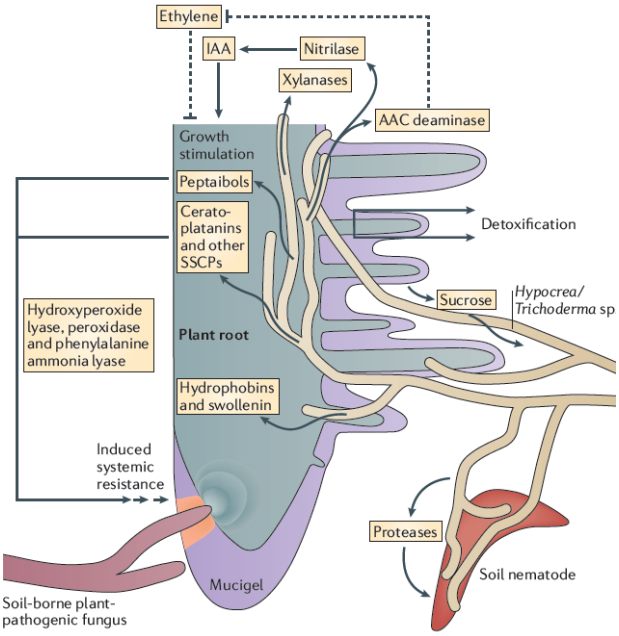


图一 土壤群落木霉属的重寄生。木霉通过病原菌释放的小分子物质来识别植物病原真菌。一些小分子物质可能是木霉与病原菌接触之前蛋白酶分泌的多肽类。这些小分子可能结合到木霉菌丝表面的 G 蛋白偶联受体或氮感应受体，从而引出包括 G 蛋白和和有丝分裂原活化蛋白激酶（MAPKs）的信号级联，这可能最终调节转录因子（TFS）的活动。这些因素，进而提高编码有关次生代谢产物的合成和细胞壁裂解的酶基因的组成型表达。来自致病真菌的凝集素和来自木霉菌丝的包含纤维素结合模块的蛋白在捕食者连接到被捕食者时起协作作用。同时，植物病原真菌形成次生代谢产物和活性氧（ROS）来引起木霉应激反应和解毒作用。

Figure 1 Mycoparasitism of *Hypocrea*/*Trichoderma* spp. within the soil community**.** *Hypocrea*/*Trichoderma* spp. recognize a plant-pathogenic fungus (a prey) via small molecules that are released by the pathogen; some of these molecules may be peptides that are released by the action of proteases secreted by the *Hypocrea*/*Trichoderma* sp. before contact. These molecules may bind to G protein-coupled receptors (such as Gpr1) or nitrogen-sensing receptors on the surface of the *Hypocrea*/*Trichoderma* sp. hyphae, thereby eliciting a signalling cascade comprising G proteins and mitogen-activated protein kinases (MAPKs), which may ultimately modulate the activities of as-yet-unknown transcription factors (TFs). These factors then enhance the constitutive expression of genes that encode enzymes for the biosynthesis of secondary metabolites and for cell wall lysis. Lectins from the pathogenic fungus and proteins harbouring cellulose-binding modules from hyphae of *Hypocrea*/*Trichoderma* spp. may collaborate in the attachment of the predator to the prey. At the same time, the plant-pathogenic prey responds by forming secondary metabolites and reactive oxygen species (ROS) that elicit a stress response and detoxification in *Hypocrea*/*Trichoderma* spp.



图二　木霉与根际其他生物体的相互作用。木霉菌丝释放一些物质来触发植物系统抗性。只显示了在根际发生的影响和由已知肉座菌/木霉引发的组分（有关更多正面效应如植物对非生物逆境产生抗性，提高光合效率和改善氮的使用的信息，见REF. 71）。Peptaibols 和the cerato-platanin Sm1（有些种内以Epl1所知）诱发植物系统抗性，最终在植物内合成过氧化氢酶，过氧化物酶和苯丙氨酸解氨酶（引起木质化）。此外，木聚糖酶EIX可能充当微生物相关的分子模式诱发植物防卫反应。α-氨基环丙烷-1-羧酸（ACC）脱氨酶抑制乙烯的形成从而促进根系的生长，腈水解酶的组成型分泌有利于生长素3 - 吲哚乙酸（IAA）的形成。木霉附着到植物根需要疏水蛋白和swollenin。最后，木霉从植物根系中吸收蔗糖作为碳源，从而实现其更快生长。木霉的nematophagy可能涉及几丁质酶，类枯草杆菌S8蛋白酶，SSCPs类，富含半胱氨酸的小分泌蛋白。

Figure 4 |Interactions of *Hypocrea*/*Trichoderma* spp. with other organisms in the rhizosphere**.** Hyphae of *Hypocrea*/*Trichoderma* spp. release several components that trigger systemic resistance in the plant. Only the effects that occur in the rhizosphere and are triggered by a known *Hypocrea*/*Trichoderma* spp. component are shown (for an update on further positive effects, such as resistance to abiotic plant stresses, enhancement of photosynthetic efficiency and improved nitrogen usage, see REF. 71). Peptaibols and the cerato-platanin Sm1 (also known as Epl1 in some species) induce a systemic resistance in the plants, culminating in the synthesis of plant hydroperoxide lyase, peroxidase and phenylalanine ammonia lyase (which induces lignification). Furthermore, the xylanase Eix elicits plant defence responses, probably acting as a microorganism-associated molecular pattern. The 1-aminocyclopropane-1-carboxylic-acid (AAC) deaminase inhibits ethylene formation by the plant, and this leads

to enhanced root growth; a constitutively secreted nitrilase might aid in the formation of the auxin 3-indole acetic acid (IAA). Attachment of *Hypocrea*/*Trichoderma* spp. to the plant roots requires hydrophobins and swollenin. Finally, *Hypocrea/Trichoderma* spp. benefit from the plant roots by receiving sucrose as a carbon source, enabling faster fungal growth. The nematophagy of *Hypocrea*/*Trichoderma* spp. probably involves chitinases and subtilisin-like S8 proteases. SSCPs, small secreted cysteine-rich proteins.

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