

# 2024年中国林木病理学研究重要进展

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**摘要:** 2024年中国科研人员在林木病原菌分类及分子系统学、林木病害致病机制、林木与病原物相互作用和病害防控方面取得了诸多显著进展,明确了多种林木新病害的病原种类,建立了1个新科、4个新属以及200余个新种,阐明了重要林木病原物关键致病因子的作用机制及其调控网络,解析林木-病原物互作的分子机制,探索了多种林木病害综合防控技术。检索分析2024年有关中国林木病理学研究领域的论文发表情况,简要梳理林木病原菌分类、病害发生机制与防控等重要研究进展,以帮助读者了解中国林木病理学的发展态势,更好地面向国家重大战略需求,聚焦林木病害特色,开展科学研究和技术服务工作。

**关键词:** 病理; 机制; 防控; 林木; 中国

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## Achievements and advances of forest pathology researches in China in 2024

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**Abstract:** In 2024, Chinese researchers have made significant progress in the taxonomy and molecular systematics of forest pathogens, pathogenesis mechanisms of forest diseases, interactions between trees and pathogens, and integrated management of forest diseases. They are as follows: identifying causal agents of new forest diseases and established 1 new family, 4 new genera, and more than 200 new species; characterizing key virulence factors and their regulatory network of important forest pathogens; revealing the molecular interactions between forest trees and pathogens; providing new control measurements for forest tree diseases. This paper search and analyze the publications and briefly reviews the key research advances in taxonomy of forest pathogens, pathogenic mechanism and integrated management by Chinese researchers in 2024. This review will help us understand trends in forest pathology and think about how to better meet the major national strategic demands and enhance our capacity for original innovation in scientific researches and technical service.

**Key words:** pathology; pathogenic mechanism; disease management; forest; China

据统计,2024年中国科研人员在林木病原菌分类、病害发生机制与防控等方面发表研究论文303篇,其中SCI论文220篇。发文数量Top3的SCI期刊是*Plant Physiology*(14篇)、*Phytopathology*(11篇)和*Plant Cell & Environment*(10篇),见图1A;发文Top3的中文期刊是《菌物学报》(6篇)、《东北林业大学学报》(5篇)和《林业科学》(5篇),见图1B。英文关键词Top3是pathogenicity、virulence和plant immunity(图1C);中文关键词Top3是生物防治、致病力和生物学特性(图1D)。

文献检索数据来源 Web of Science <https://webofscience.clarivate.cn/wos/alldb/basic-search> 和中国学术期刊数据库 <https://c.wanfangdata.com.cn/periodical>, 时间为2024年1月1日至12月31日。

### 1 中国林木病原菌分类与分子系统学

林木病原菌分类与分子系统学是开展病害诊断、发生成灾机制研究和防治工作的基础。2024年在林木病原菌分类学的主要研究进展有:1)对特定区域

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图1 期刊和关键词的文字云图  
Fig. 1 Text nephogram of journals and keywords

注: A. 英文期刊; B. 中文期刊; C. keywords; D. 关键词。字体越大, 词条出现频率越大。

Notes: A. English periodicals; B. Chinese periodicals; C. keywords; D. keywords. The larger the font, the more frequently the entries appear.

林木病原中的子囊菌物种多样性进行了调查和种类鉴定。例如, 系统研究了北京、四川等地林木病原子囊菌的多样性 (Bai et al., 2024; Jia et al., 2024; Tian et al., 2024)。2) 重要子囊菌类群分类体系以及新分类单元的构建, 如齿小蠹伴生喙壳目 Ophiostomatales (Wang et al., 2024) 和林木腐烂病相关壳囊孢科 Cytosporaceae (Lin et al., 2024), 新建了新网孢菌科 Neodictyosporiaceae (Tian et al., 2024)、穴生菌属 *Cavernicola* (Razaghi et al., 2024)、长柄菌属 *Longistipes* (Li et al., 2024d)、*Neodictyocheirospora* 和 *Neogregarithecium* (Tian et al., 2024) 和 200 余新种, 主要涉及炭疽菌属 *Colletotrichum* (Sui et al., 2024)、壳孢属 *Cytospora* (林露和范鑫磊, 2024)、间座壳属 *Diaporthe* (Dissanayake et al., 2024)、番石榴澳柄锈菌 *Austropuccinia psidii* (G. Winter) Beenken 为云南省新记录种 (刘一帆等, 2024) 等。3) 通过分析担子菌门中的 488 个物种基因组序列, 构建了担子菌门系统演化基因组发育图谱, 将担子菌门划分为 4 亚门、20 纲、77 目、297 科、2 134 属, 厘清了担子菌门各级分类系统存在相互矛盾、部分名称不合法、分类系统和系统发育关系不吻合等问题 (He et al., 2024)。

## 2 林木病原致病机制及与林木互作机制

松材线虫病是中国最具危险性的林木病害, 威胁着中国生态安全和经济发展。2024 年涉及松材线虫病的重要研究进展有: 松材线虫效应子 BxPIE1 通过与黑松的 PtPrx3 互作, 抑制寄主过氧化物酶活性, 进而增强松材线虫的致病性 (Rui et al., 2024); 松材线虫类毒素过敏原蛋白 BxVAPs 可触发植物防御相关

程序性细胞死亡, 在调节马尾松 *Pinus massoniana* Lamb. 萜烯代谢过程中发挥重要作用 (Feng et al., 2024); 效应子 BxNMP1 与类甜蛋白 PtTLP-L2 和  $\beta$ -1,3 葡聚糖酶 PtGLU 互作, 可抑制松树水杨酸代谢途径, 导致寄主防御代谢紊乱而枯萎死亡 (Yang et al., 2024a)。松材线虫滞育期延长导致红松 *Pinus koraiensis* Siebold & Zucc. 越年枯死 (王佳楠等, 2024); 落叶松 *Larix olgensis* (Rupr.) Kuzen. 树体内携带的松材线虫受咖啡酸的诱导而导致发育迟缓, 进而使其越冬存活率降低 (Wang et al., 2024c)。

聚焦林木病原菌功能基因组学, 在毒性因子的答复及其信号传导、转录调控、表观遗传和效应子功能等方面取得很多重要进展。例如, 杉木炭疽病菌 (*Colletotrichum gloeosporioides*) 中 CgMkk1 磷酸化 CgCrzA 转录因子, 调控下游 2 个几丁质合成酶基因 *CHS5* 和 *CHS6* 的表达, 影响毒性和细胞壁完整性 (Yang et al., 2024b)。油茶炭疽病菌 (*Colletotrichum fructicola*) Cfb-ZIP1-CfMsg5 模块通过负调控 Slt2 信号通路的 Mpk1 影响氧化应答反应及致病过程 (Gao et al., 2024)。果生炭疽菌 (*Colletotrichum fructicola*) 效应子 CfRibo1 和 CfRibo2 能够直接靶向梨树的叶际微生物群落, 通过毒杀作用抑制叶际微生物群落的生长, 促进炭疽菌对梨树的侵染 (Wang et al., 2024a)。果生炭疽菌 CfEC12 与苹果 NPR1 共同竞争 NIMIN2 蛋白的 13-63 位氨基酸, 阻碍 NIMIN2 与 NPR1 结合, 抑制 NPR1 调控的下游 PRs 抗病基因表达和免疫反应 (Shang et al., 2024a)。果生炭疽菌分泌蛋白 CfEC28, 靶向莽草酸途径限速酶 3-脱氧-D-阿拉伯庚基磺酸 7-磷酸合成酶 (DAHPS),

并通过阻断锰离子与 DAHPS 结合抑制其酶活性,进而干扰莽草酸途径介导的寄主防卫次生代谢物的累积(Shang et al., 2024b)。

杨树腐烂病菌(*Cytospora chrysosperma*) CcSte11-CcSte7-CcPmk1 是致病核心调控模块,通过激活下游转录因子 Ste12 从而调控靶标毒性基因的表达(Yu et al., 2024); 苹果腐烂病菌(*Valsa mali*) CAP 家族效应子 VmPR1c 能够靶向苹果缬氨酸和谷氨酰胺蛋白 MdVQ29, 通过 MdVQ29-MdWRKY23-MdCOI1 的作用抑制苹果的茉莉酸信号通路,促进苹果腐烂病菌的侵染(Han et al., 2024b)。此外,苹果腐烂病菌还能分泌 siRNA, 进入植物细胞内干扰寄主的抗病基因表达,促进病原菌的侵染(Liang et al., 2024)。板栗疫病菌(*Cryphonectria parasitica*) 甲基转移酶 CpMTA1 能够对下游致病关键靶标基因 *CpAphA* 进行 m<sup>6</sup>A 修饰,并依赖于 m<sup>6</sup>A 修饰相关蛋白 CpYTHDF1, 从而稳定 *CpAphA* 的 mRNA(Zhao et al., 2024a) 以及 CpALKBH 通过对致病关键的转录因子 *CpZap1* 进行去 m<sup>6</sup>A 修饰保持其 mRNA 的稳定性(Zhao et al., 2024b)。

枣疯病致病植原体(*Candidatus Phytoplasma ziziphi*) 效应子 SJP3 和 PHYL1<sub>JWB</sub> 可以靶向枣树不同的靶标,实现干扰枣树生长发育的多功能效应,进而引起受害枣树出现丛枝和花器返祖的症状(Deng et al., 2024, Xue et al., 2024); 而效应子 SJP1 和 SJP2 可以靶向枣树 ZjTCP2 和 ZjTCP7, 促进其侵染致病过程(Ma et al., 2024a; Ma et al., 2024b)。荔枝霜疫霉(*Peronophythora litchii*) 果胶裂解酶 PIPeL1 和 PIPeL1-like, 不仅通过果胶裂解酶活性增强病原菌致病力,而且与 LcPIP1 互作增强植物的免疫功能(Li et al. 2024c)。柑橘黄龙病菌(*Candidatus Liberibacter asiaticus*) 不同 SEC 依赖的效应子能够作用于不同的下游靶标,从而抑制寄主免疫防卫反应的产生(Li et al., 2024d)。其中 SDE19 除了能够抑制柑橘的免疫防卫反应,还能靶向植物的囊泡运输相关组分 Sec12 并造成其降解,进而影响一些植物胞外抗病蛋白的分泌,从多个层次促进了病原细菌的侵染(Huang et al., 2024)。欧美杨细菌性溃疡病菌(*Lonsdalea populi*) 双组分系统 CpxA/CpxR, 通过磷酸化修饰激活并调控下游靶标,控制鞭毛形成、活性氧响应和毒性(Yang et al., 2024c)。

为抵御病原物侵染,植物进化出了病原相关分子模式引起的免疫(PAMP-triggered immunity, PTI)和效应子引起的免疫(effector-triggered immunity, ETI), PTI 和 ETI 涉及到植物转录组重编程。通过分析杨树接种胶孢炭疽菌后的转录组动态变化,构建了

PagWRKY18 及其潜在靶基因的分层基因调控网络,并验证了 PagWRKY18 可能参与调节 PTI 和 ETI 之间的交叉作用,激活强大的免疫反应并维持细胞内活性氧稳态(Chen et al., 2024)。苹果 *Malus pumila* Mill. 自噬相关蛋白 MdATG16 参与调控了苹果对腐烂病的抗性,而具有 E3 蛋白酶体降解活性的苹果腐烂病菌效应子 Vm\_04797 能够靶向降解苹果的抗病蛋白 MdAP-2 $\beta$ , 而 MdAP-2 $\beta$  又能与 MdATG16 相互作用,通过破坏寄主的细胞自噬过程,从而阻碍苹果发挥免疫抗病作用,最终促进苹果腐烂病菌的侵染(Sun et al., 2024c)。苹果腐烂病菌的木聚糖酶 VmXyl2 可诱导细胞坏死,其活性依赖 BAK1 信号传导组分,敲除该基因会显著降低该病菌的毒力与木聚糖酶活性(Cui et al., 2024)。

小蠹虫及其伴生长喙壳类真菌严重威胁针叶林健康,通过单独和联合接种多种长喙壳类真菌(*Leptographium qinlingense*, *Ophiostoma shennongense*, *Graphilbum parakesiyee*, *Ophiostoma* sp.), 结合 GC-MS(气相色谱-质谱联用)和转录组分析等方法,发现长喙壳类真菌弱毒菌株诱导华山松 *Pinus armandi* Franch. 苯丙烷、类黄酮等代谢通路的显著上调表述(Wang et al., 2024)。此外,相较于长白落叶松 *Larix olgensis* A. Henry, 落叶松八齿小蠹 *Ips subelongatus* Motschulsky 伴生长喙壳真菌 *Endoconidiophora fujiensis* 对日本落叶松 *Larix kaempferi* (Lamb.) Carrière 的致病性较强,病原菌诱导抗性树种中月桂烯、3-萜烯、柠檬烯等防御性单萜寄主挥发物的显著积累,并提早激活 CAT、PR 等防御基因的显著表达(Liu et al., 2024)。

### 3 林木病原防控

林木病害防治方面也取得了许多新进展。例如,通过制备纳米农药(AVM@EC@Pectin),在松树受到松材线虫和松墨天牛 *Monochamus alternatus* 侵染时,智能判断释放农药,切断松材线虫和媒介昆虫的侵染循环,实现松材线虫病的精准防控(Ma et al., 2024); 从 *Streptomyces ahysroscopicus* 中提取的四霉素 B3 能抑制松材线虫的取食和生长,有良好的防治效果(Sun et al., 2024b)。在增效剂复配和改进施药方式方面,以 N,N-二甲基甲酰胺和乙酸苄酯为溶剂、吐温-40 为有效的乳化制剂,在常温下作为树干注射剂药效很好(Li et al., 2024b)。将氟吡菌酰胺和甲维盐按照质量比 3:5 进行复配,共毒系数最高,松材线虫的产卵量和卵孵化率都低于单独药剂,对防治松材线虫病具有增效效果(张娇等, 2024)。在防治黄栌枯

萎病方面,主要采用灌根丙环唑和树干注射多菌灵与啞菌酯复配组合进行防治(郭瑞峰等,2024)。施用80%乙蒜素乳油能有效抑制苹果树腐烂病菌菌丝生长,田间施用后发病率显著下降,对苹果树腐烂病有良好的防治效果(苏晓州等,2024);施用400 g/L 氯氟醚菌唑·吡唑醚菌酯悬浮剂可有效防治苹果褐斑病(陈敏等,2024)。

利用微生物间的拮抗作用或微生物代谢产物能有效防治林木病害。松树内生菌(*Bacillus velezensis* Pt-RP9)具有极强的杀线虫能力,能抑制线虫繁殖,可作为防治松材线虫病的一种重要生物防治剂(Sun et al., 2024a)。从健康茶叶中分离的芽孢杆菌对茶炭疽病(tea anthracnose)具有较强的拮抗活性,可诱导植物产生抗性(Wu et al., 2024);利用芽孢杆菌也能显著提高银杏对叶枯病的抗性,并能促进银杏生长和改善根瘤菌群中的微生物群落(Ji et al., 2024)。在易感病害的土壤中添加芽孢杆菌菌剂可显著降低黄栌枯萎病的发病率,芽孢杆菌制剂与杀菌剂丙环唑的组合可有效防治黄栌枯萎病(Guo et al., 2024)。木霉菌不仅可以防治杨树叶枯病,而且能促进杨树生长(Han et al., 2024a)。从台湾杉木 *Cunninghamia konishii* Hayata 提取的雪松醇作为一种抗真菌活性物质,可通过改变真菌细胞膜通透性,有效抑制褐根病菌的菌丝生长和繁殖,对杉木褐根病的防治具有积极作用(Hsiao et al., 2024)。

综上所述,围绕中国重大林木病害及地区性严重危害林木健康的重要病害,在分类学、病理学及其防控方面在2024年取得了许多重要进展。今后应结合中国重大林业有害生物防控的基础理论和技术研发需求,系统研究林木病害发生成灾机制,挖掘关键致病基因及调控网络,解析病原菌与林木互作的分子基础及林木免疫系统的特异性,开展绿色精准防控技术的集成研发,为林木病害的可持续防控提供理论与技术支撑。

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