



整合多组学分析稻瘟病中的铁死亡

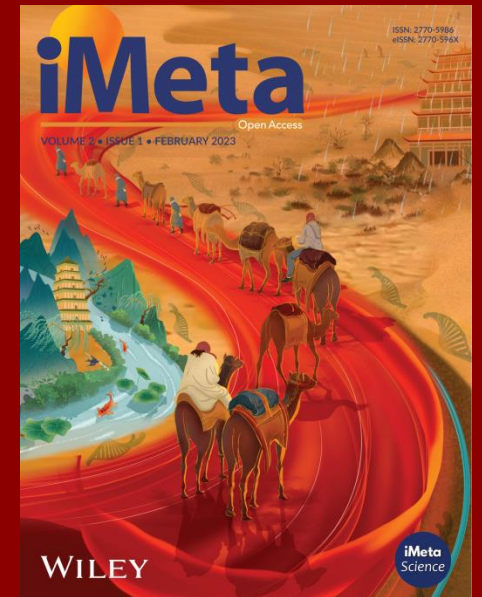
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Zhibin Liang, Qing Shen, Nam Khoa Nguyen, Ruhui Long, Yuming Ma, Qiao Liu, Naweed I. Naqvi, et al. 2026.
Decoding ferroptosis via integrated multi-omics analyses in rice blast. *iMeta* 5: e70178.

<https://doi.org/10.1002/imt2.70178>.



不同物种中的铁死亡

铁死亡

一种由过量脂质过氧化物积累驱动的、铁依赖性的程序性细胞死亡形式

哺乳动物细胞：

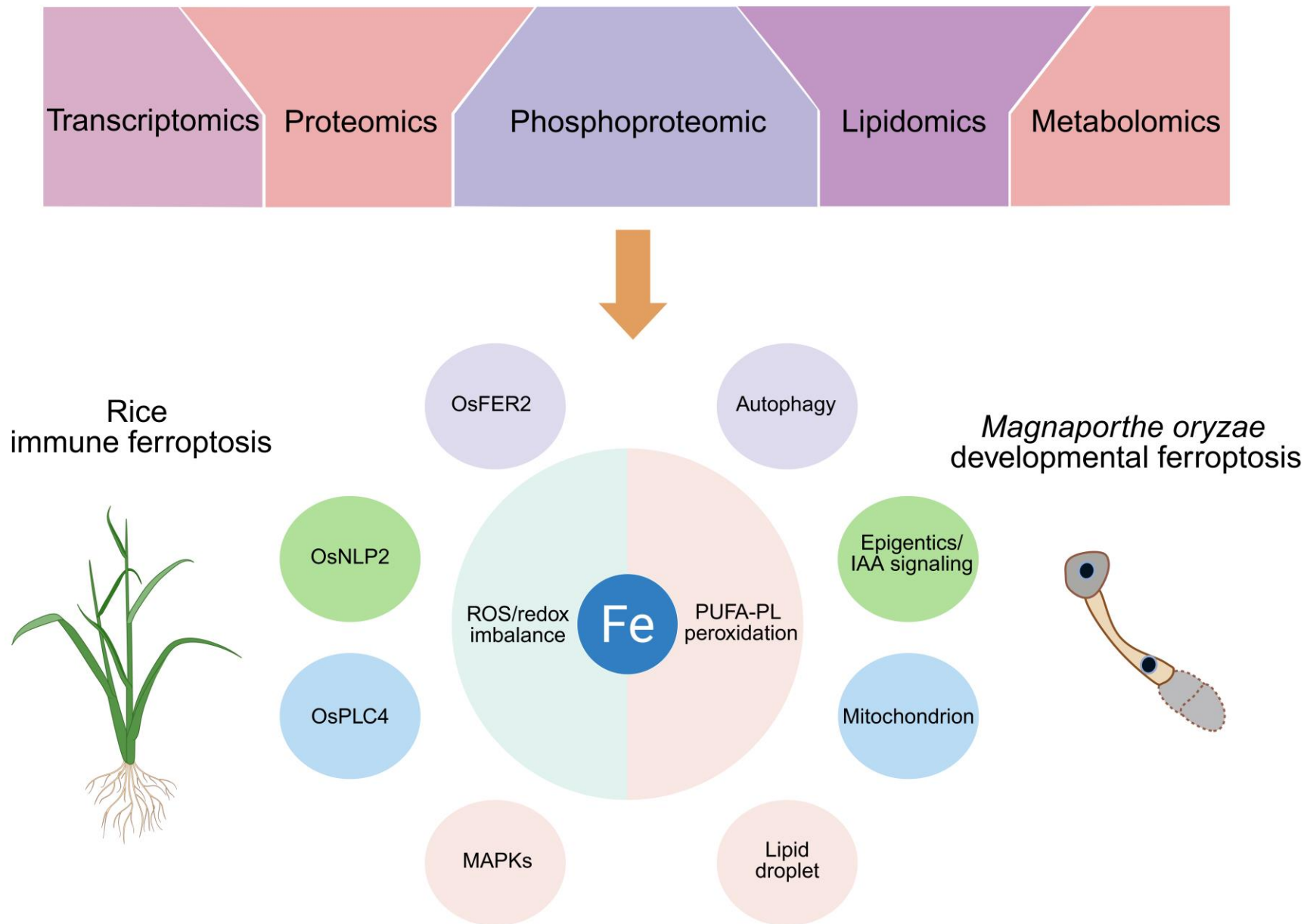
- 谷胱甘肽过氧化物酶4—谷胱甘肽轴 (GPX4-GSH)；
- 铁死亡抑制蛋白1—辅酶Q轴 (FSP1-CoQ)。

植物或真菌：

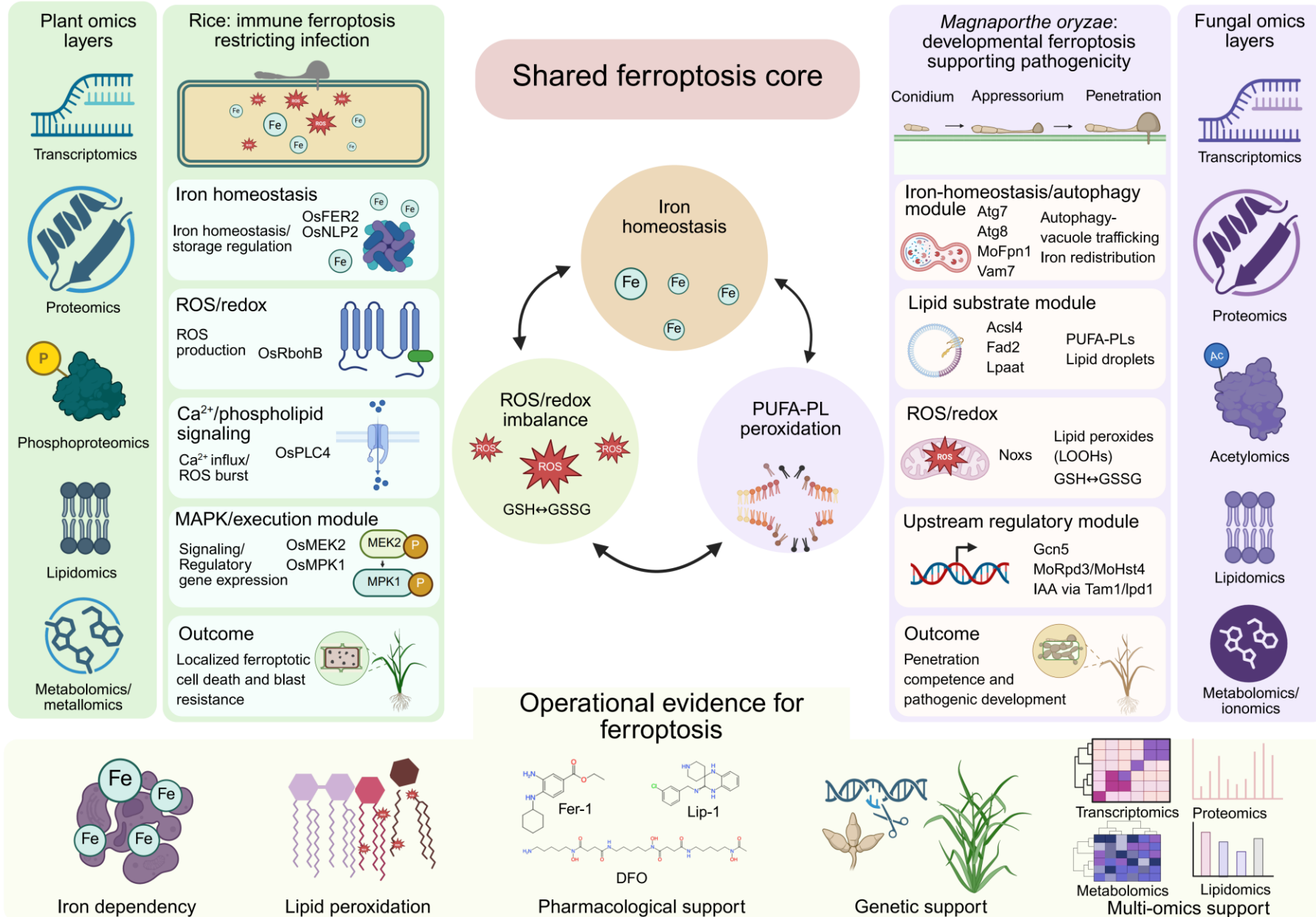
- **不存在：**以经典GPX4或FSP1为核心的铁死亡调控系统；
- **依赖：**趋同的生化、药理学、遗传学和组学证据。



水稻—稻瘟病菌互作中铁死亡介导的双重效应

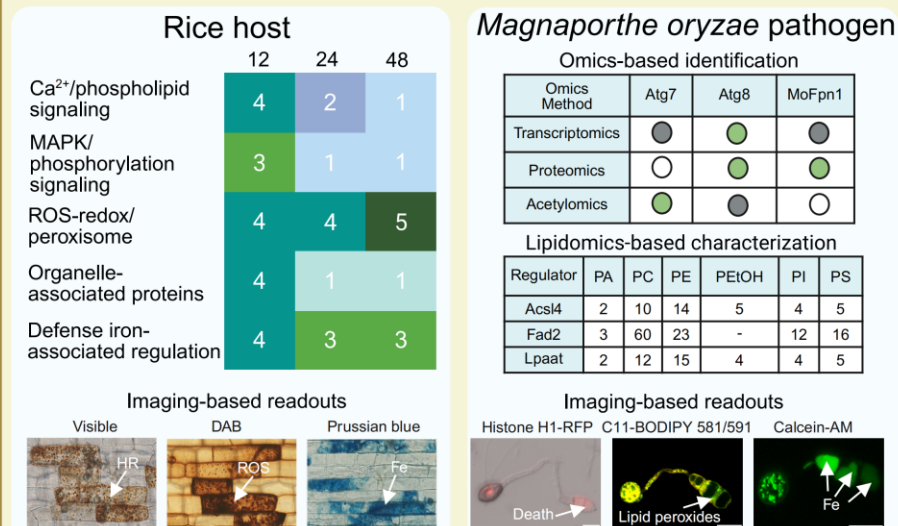


组学解析水稻—稻瘟病菌系统中的双重铁死亡

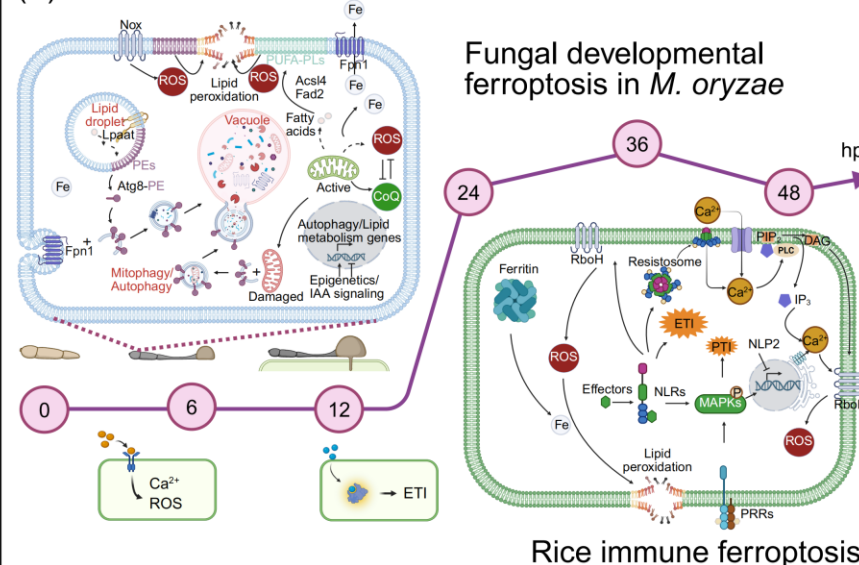


用于解析水稻—稻瘟病菌系统中铁死亡的多组学框架

(A) Representative data/evidence inputs



(B) Literature-based relative timeline

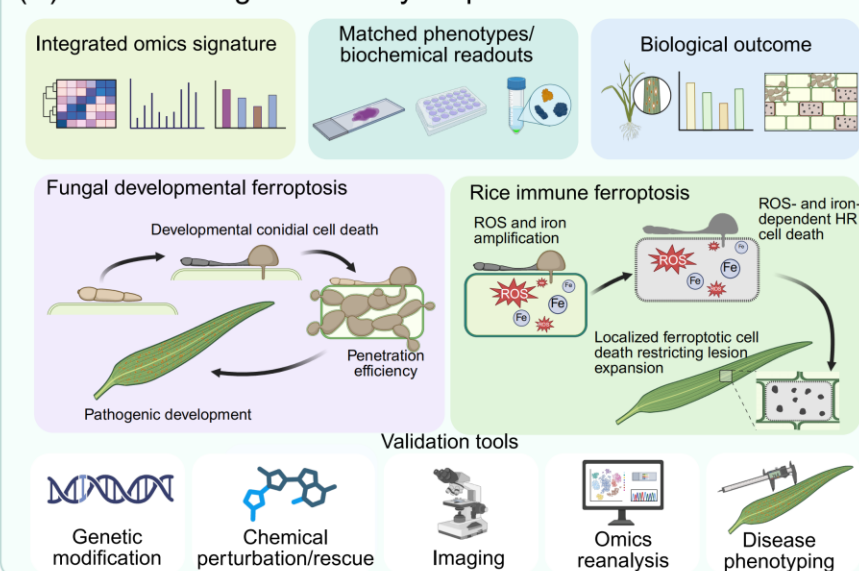


(C) Evidence matrix for ferroptosis-associated modules

	Transcriptome	Proteome	PTMome	Lipidome	Metabolome/ metallome	Genetics/ Pharmacology
Iron homeostasis	●	●	●	●	●	●
ROS/redox	●	●	●	●	●	●
Lipid-peroxidation substrate	●	●	●	●	●	●
Ca ²⁺ /MAPK/phospholipid signaling	●	●	●	●	●	●
Autophagy/organelle-associated remodeling	●	●	●	●	●	●

● Strong support ● Moderate support ● Limited support

(D) Validation logic and study outputs



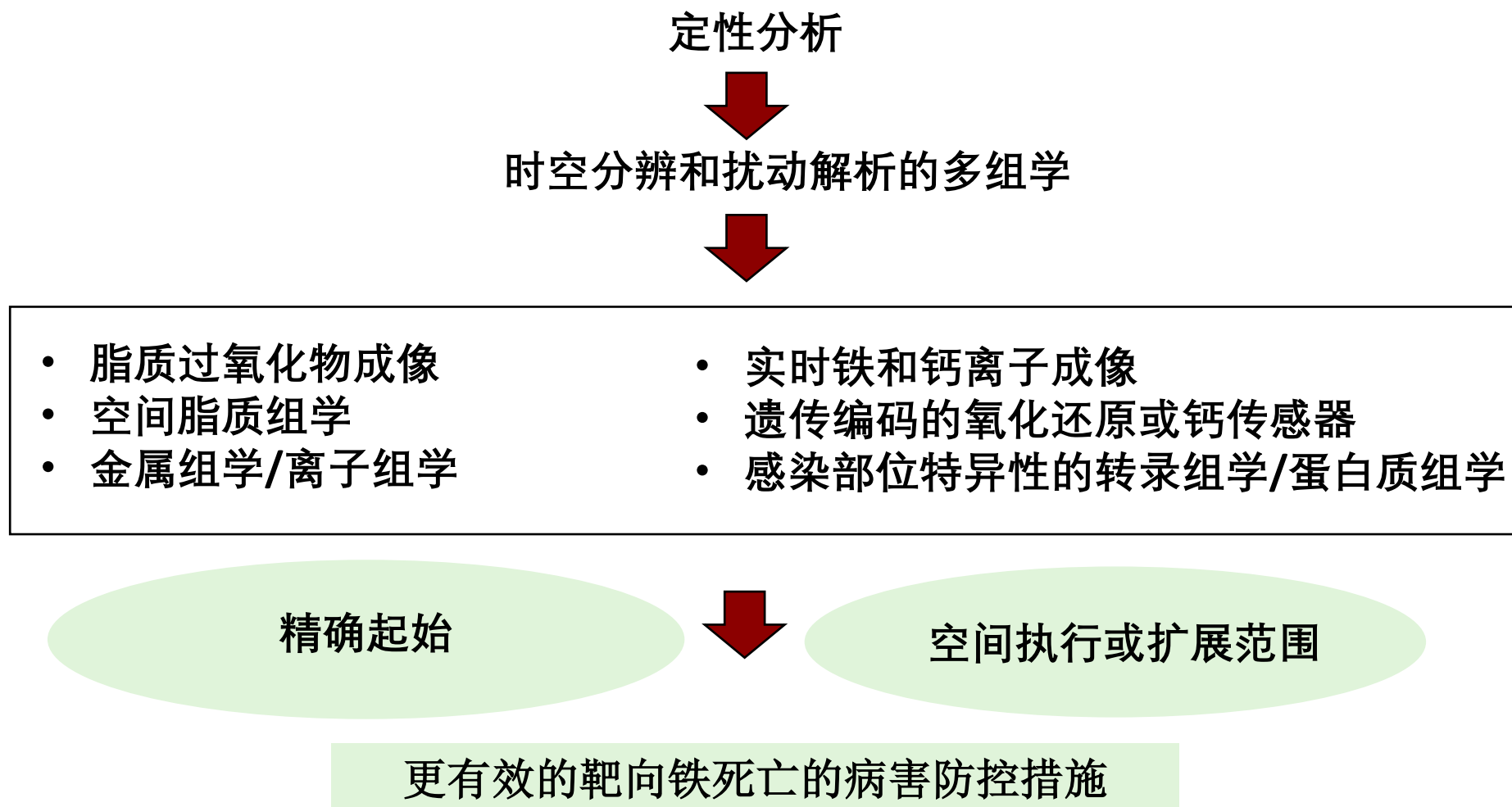


稻瘟病以外的比较边界设定

System	Biological context	Key omics readouts	Iron/lipid evidence	Genetic/pharmacological support	Suggested terminology	Key uncertainty
Rice–<i>Magnaporthe oryzae</i>	Incompatible interaction; immune ferroptosis during blast resistance	Transcriptomics, proteomics, phosphoproteomics, and metabolomics readouts	Fe accumulation, ROS burst, MDA accumulation, lipid peroxidation, and lipid ROS at infection sites	Strong; supported by ferroptosis inhibitors/inducers, iron chelation, ROS inhibition, and rice regulators including OsMEK2, OsMPK1, OsFER2, OsNLP2, and OsPLC4	Ferroptosis	Infection-site lipidomics and metallomics are still needed to define the oxidized lipid species and local iron pools
<i>Magnaporthe oryzae</i>	Conidial and appressorial developmental ferroptosis associated with pathogenic development	Transcriptomics, acetylomics, lipidomics, and metabolomics readouts	Iron dependency, PUFA-PLs, phosphatidylethanolamines, lipid peroxides, GSH/redox changes, IAA-associated metabolic regulation	Strong; supported by inhibitors such as CPX, Fer-1, and Lip-1, and by regulators including Atg7, Atg8, MoFpn1, Vam7, Acsl4, Fad2, Lpaat, Gcn5, MoRpd3, Tam1, and lpd1	Ferroptosis	Full <i>in planta</i> multi-omics integration is still needed to connect fungal developmental ferroptosis with host infection outcomes
<i>Arabidopsis thaliana</i>	Heat-shock-induced root cell death	Metabolic and redox readouts	GSH and ascorbate depletion, cytosolic ROS, lipid ROS, and iron dependency	Moderate; suppressed by iron chelators, Fer-1, and GSH supplementation, but broad pathway-level multi-omics remains limited	Ferroptosis-like cell death	Evidence is strong biochemically, but broader omics and genetic validation of plant-specific ferroptosis regulators remain limited
<i>Aspergillus flavus</i>	Fe ²⁺ -induced conidial death	Limited omics evidence; biochemical and genetic readouts	Iron-dependent ROS accumulation; Nox-associated oxidative response	Moderate; Fe ²⁺ -induced death is reduced by Nox inhibitors and deletion of <i>noxA</i>	Ferroptosis-like cell death	Lipidomics and broader omics validation are needed to confirm lipid-peroxidation-dependent execution



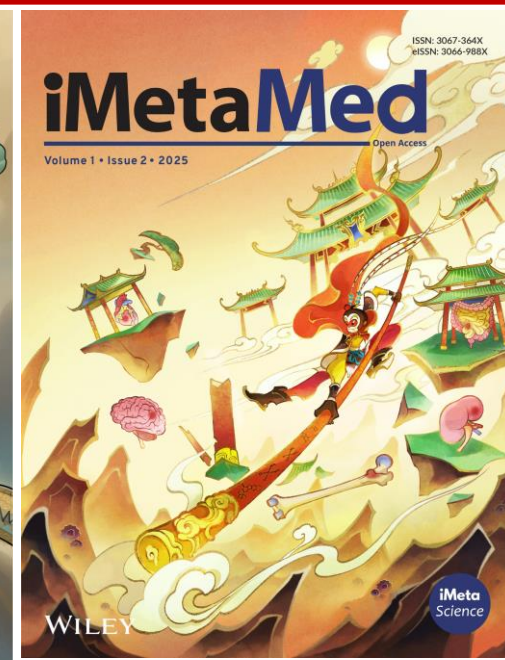
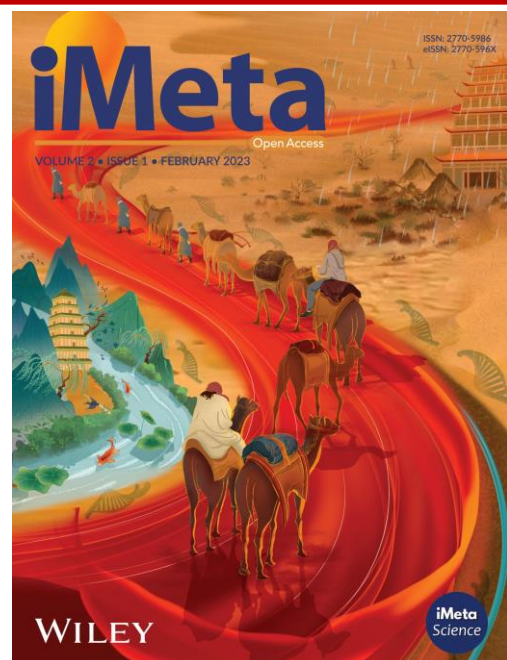
未来展望：从多组学特征到铁死亡引导的病害防控



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