



Decoding ferroptosis via integrated multi-omics analyses in rice blast

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Ferroptosis across different species

Ferroptosis

Iron-dependent form of regulated cell death driven by the accumulation of lethal lipid peroxides

In mammalian cells:

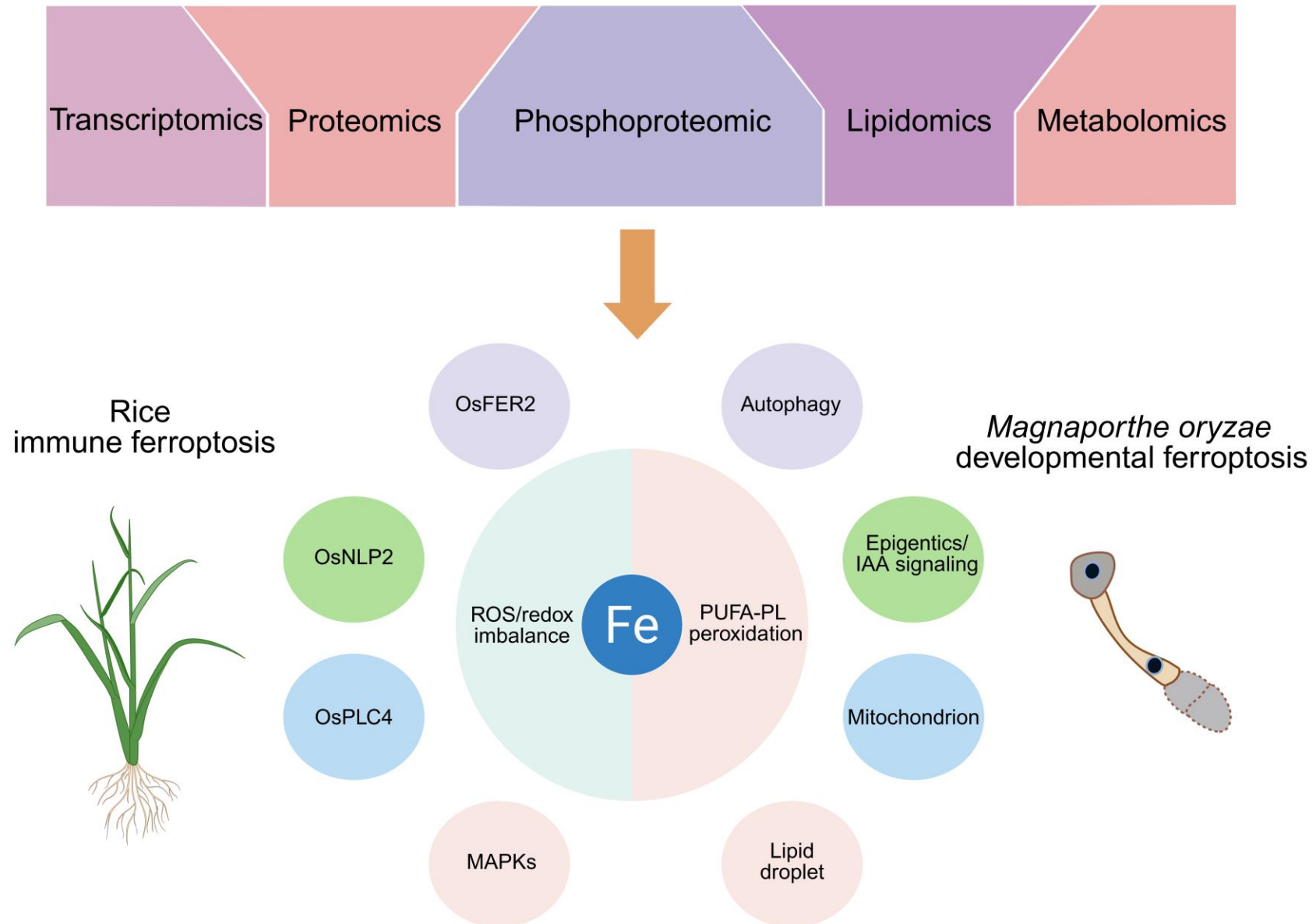
- the glutathione peroxidase 4-glutathione (GPX4-GSH) ;
- ferroptosis suppressor protein 1-coenzyme Q (FSP1-CoQ).

In plants or fungi:

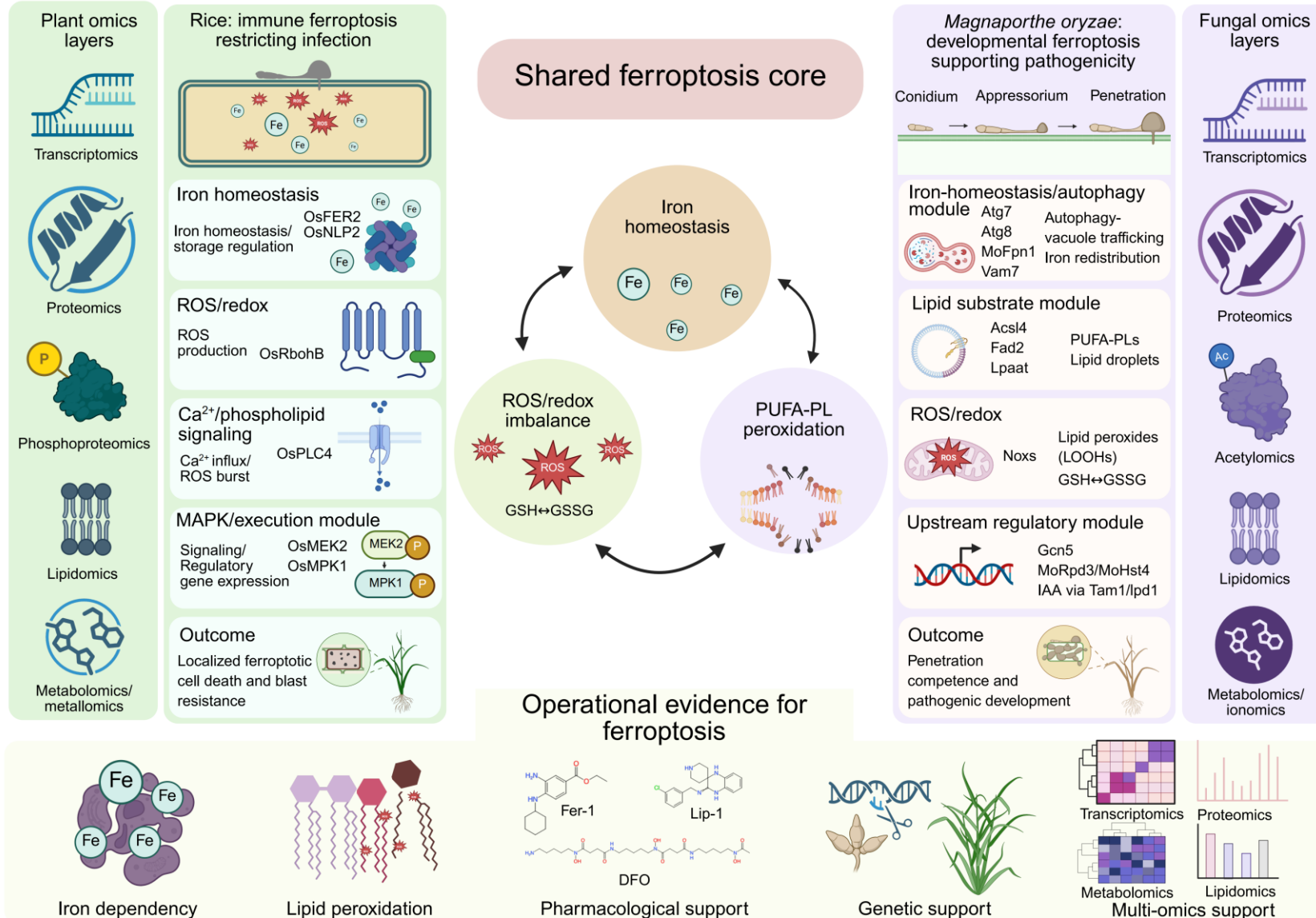
- **Absence:** counterpart canonical GPX4- or FSP1-centered systems;
- **Dependence:** convergent biochemical, pharmacological, genetic, and omics evidence.

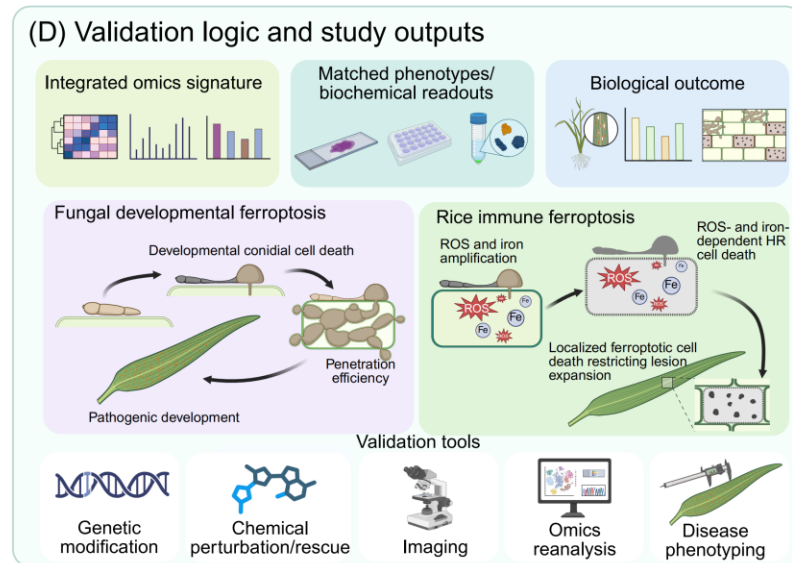
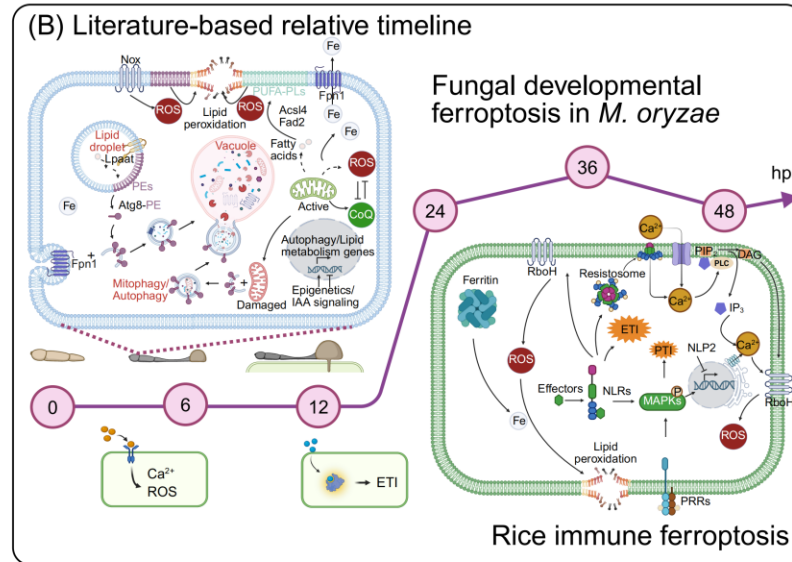


Dual ferroptosis in the rice–*M. oryzae* pathosystem



Omics-dissected dual ferroptosis in the rice-*M. oryzae* pathosystem







Comparative boundary-setting beyond rice blast

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



Future perspectives: From omics signatures to ferroptosis-guided disease control

Qualitative analyses



Spatiotemporal and perturbation-resolved omics



- Lipid-peroxide imaging
- Spatial lipidomics
- Metallomics/ionomics
- Real-time Fe and Ca^{2+} imaging
- Genetically encoded redox or calcium sensors
- Infection site-specific transcriptomics/proteomics

Precise ferroptosis initiation

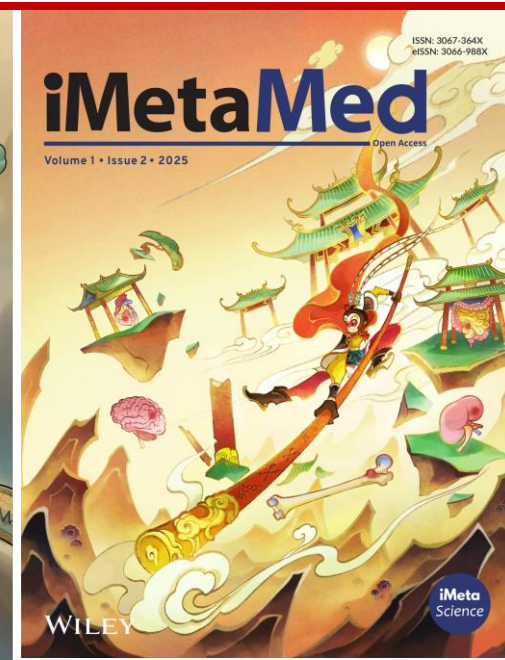
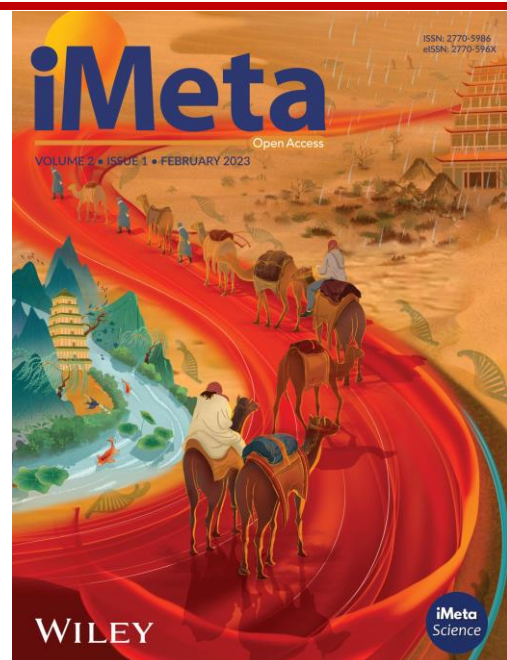


Spatial execution or
expansion

More effective management strategy by
targeting ferroptosis

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