



凝结魏茨曼氏菌XY2通过肠-脑轴调节色氨酸代谢与氧化应激-炎症互作通路缓解铜诱导的神经毒性

高誉芳, 郑晓冬, 阎芙洁*

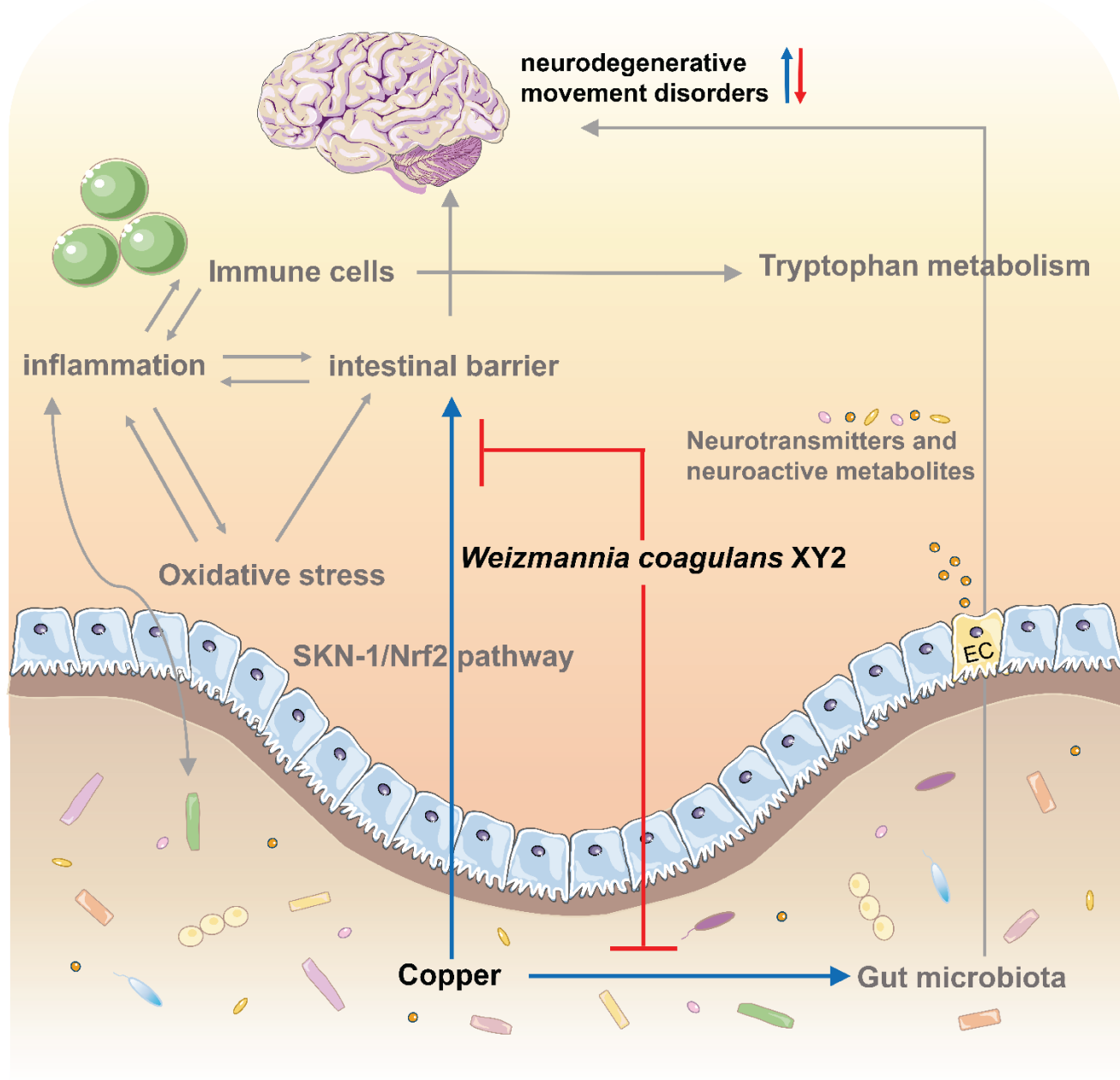
浙江大学生物系统工程与食品科学学院



Yufang Gao, Xiaodong Zheng, Fujie Yan. 2025. *Weizmannia coagulans* XY2 Mitigates Copper Neurotoxicity via Gut-Brain Axis Modulation of Tryptophan Metabolism and Oxidative-Inflammatory Crosstalk. *iMetaOmics* 2: e70066. <https://doi.org/10.1002/imo2.70066>



亮点

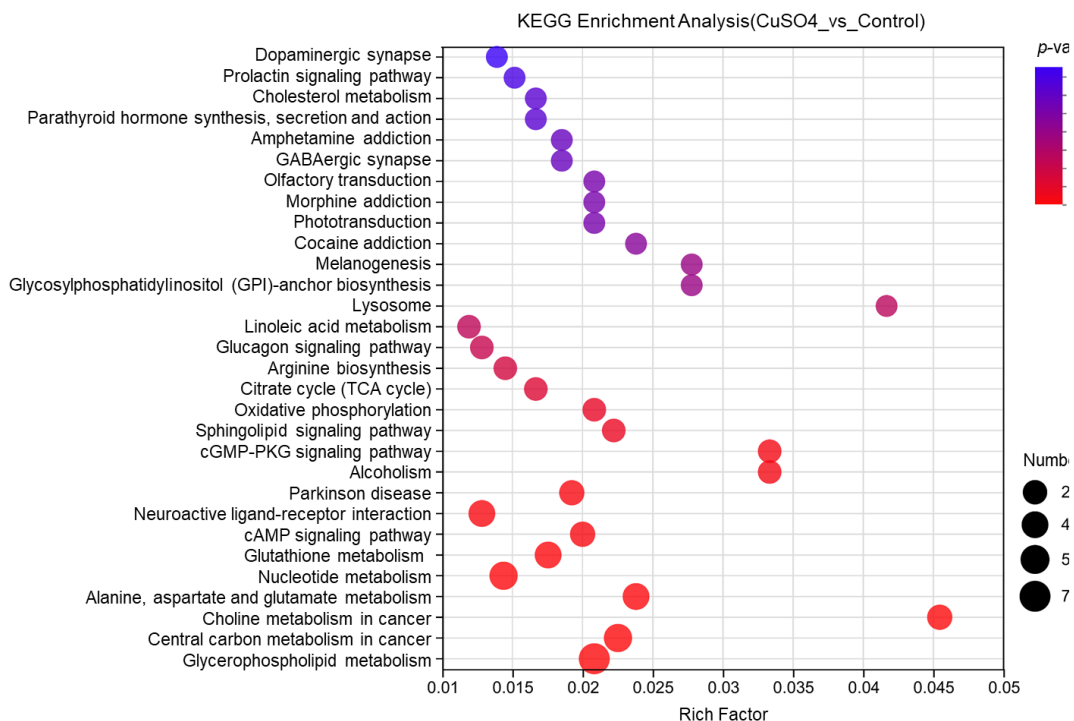


- 铜暴露通过调节肠道菌群干扰色氨酸代谢及5-羟色胺水平;
- 铜暴露诱发肠道屏障损伤与炎症反应, 进而导致神经损伤;
- 凝结魏茨曼菌XY2通过靶向“色氨酸代谢-抗氧化防御-肠脑轴”多维度调控网络缓解铜诱导的神经毒性。



结果

(A)



(B)

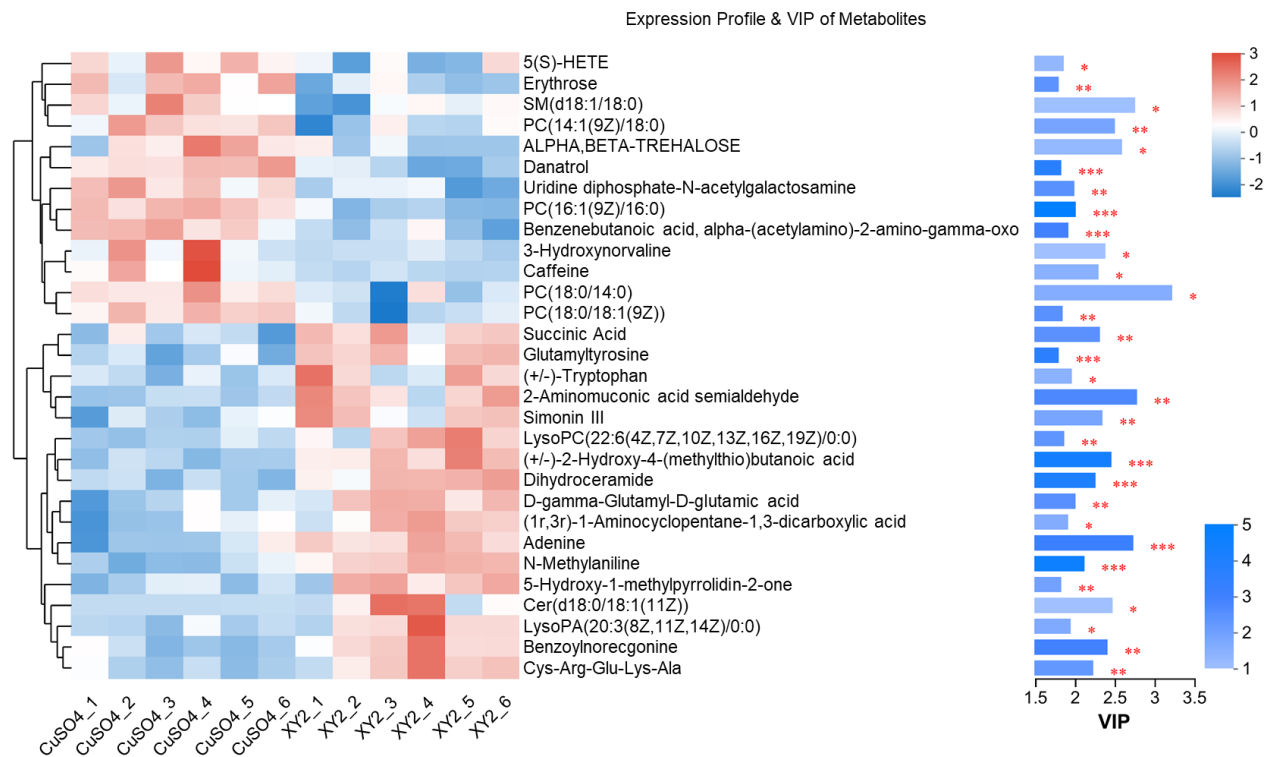
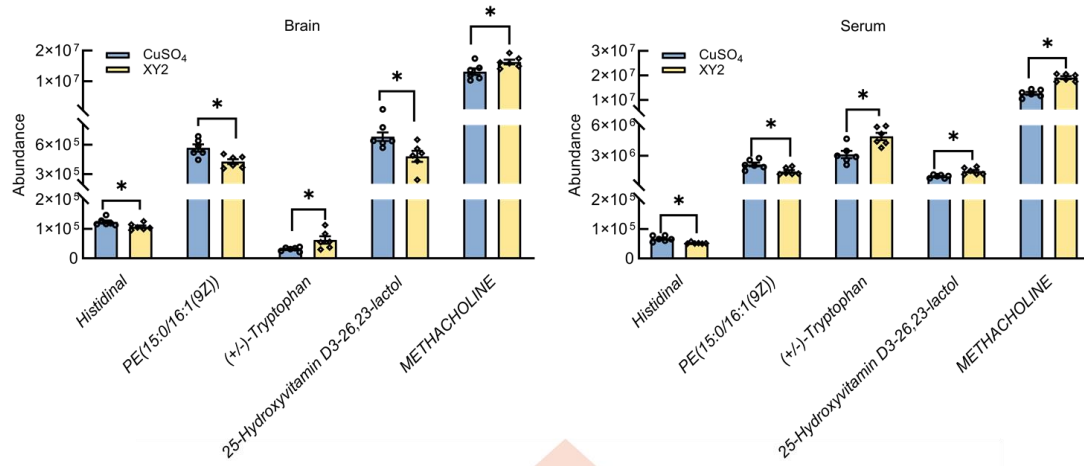


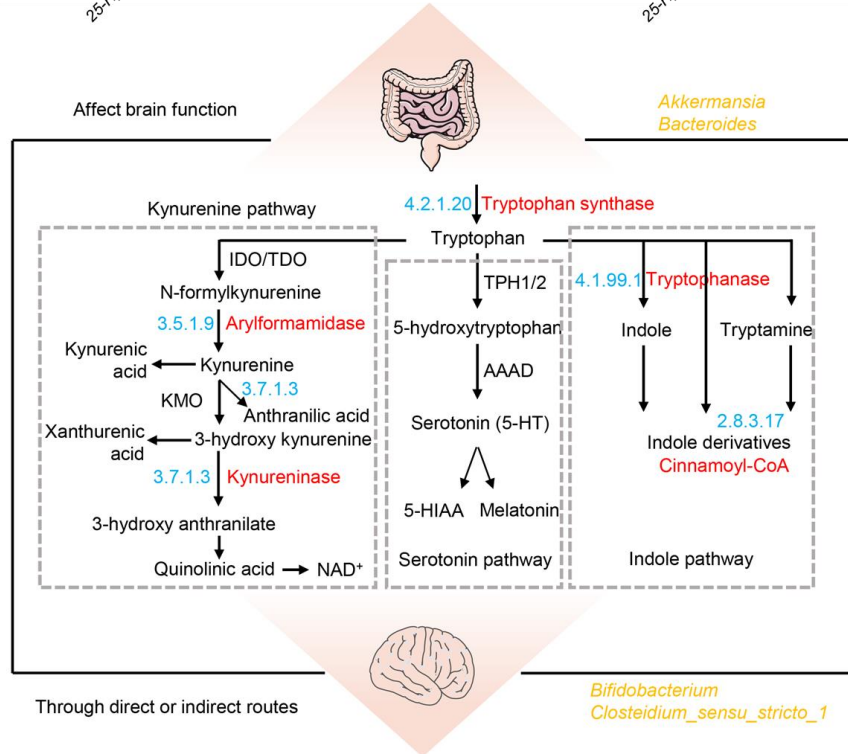
图1.色氨酸代谢是 *W. coagulans* XY2抑制铜毒性的关键通路

结果

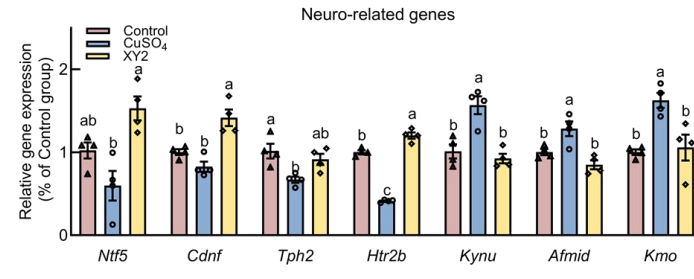
(C)



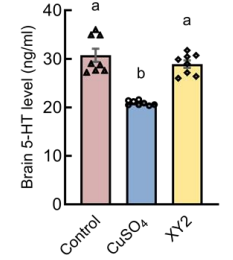
(D)



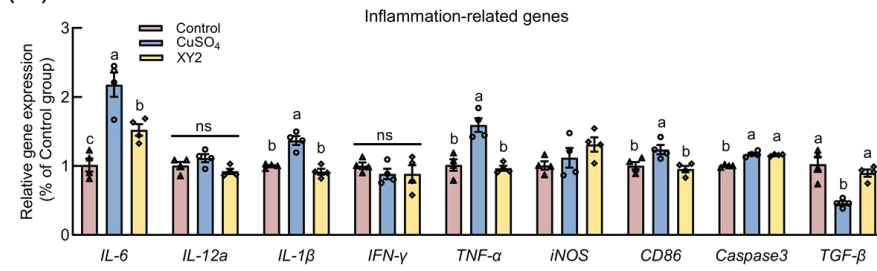
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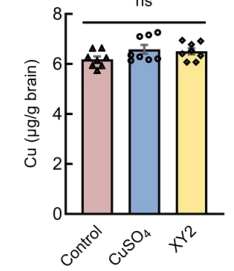
(F)



(G)



(I)



(H)

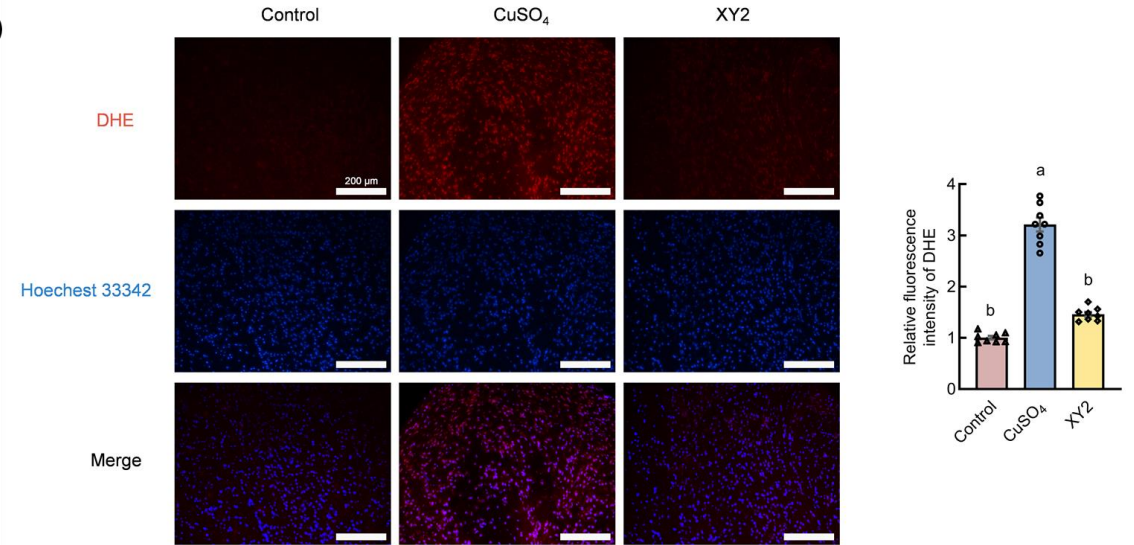
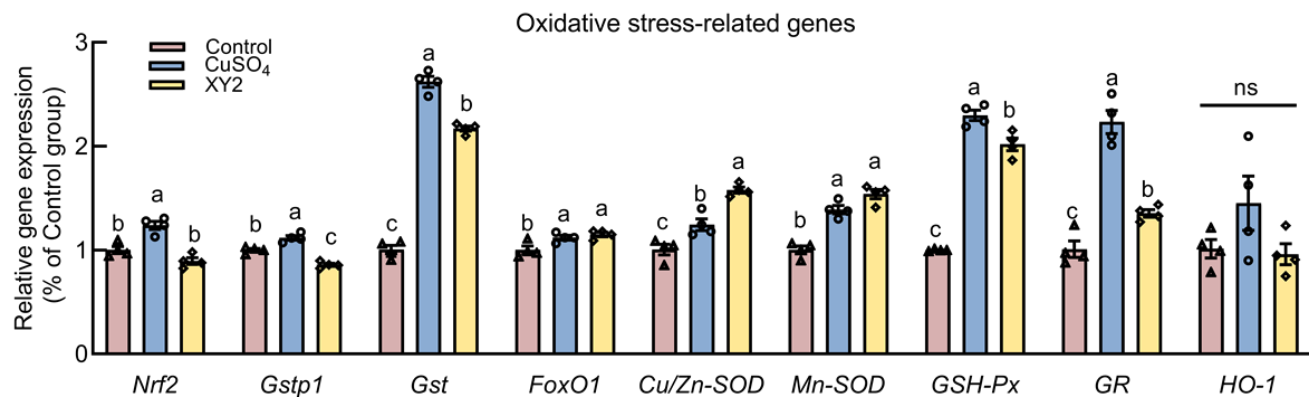


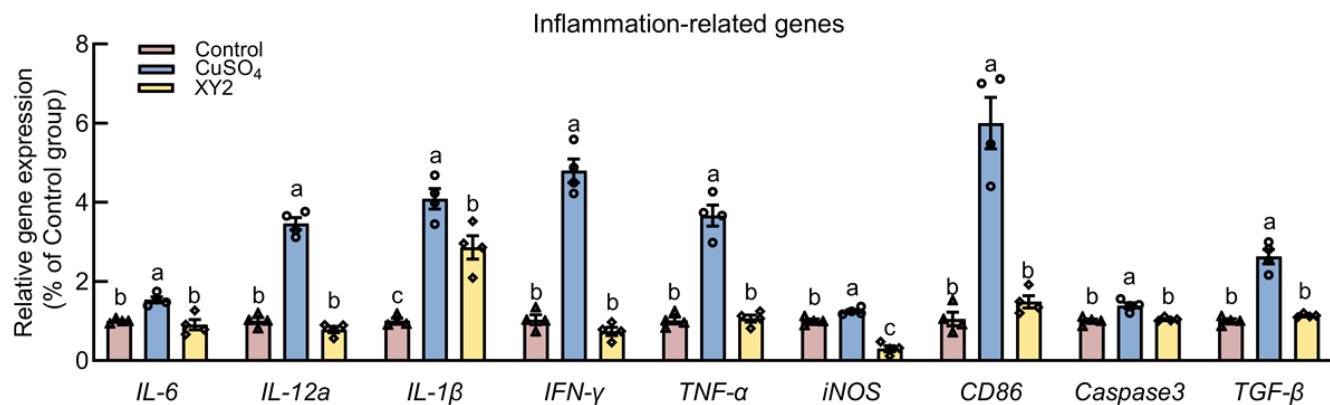
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结果

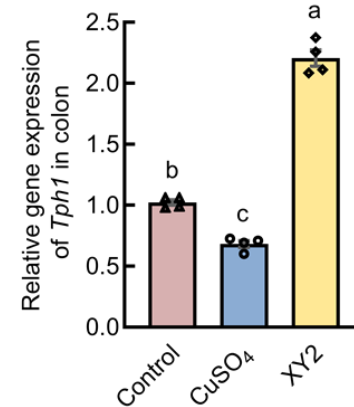
(A)



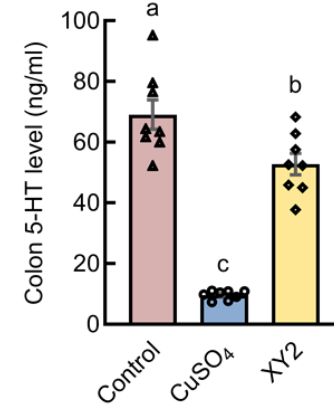
(B)



(C)



(D)



(E)

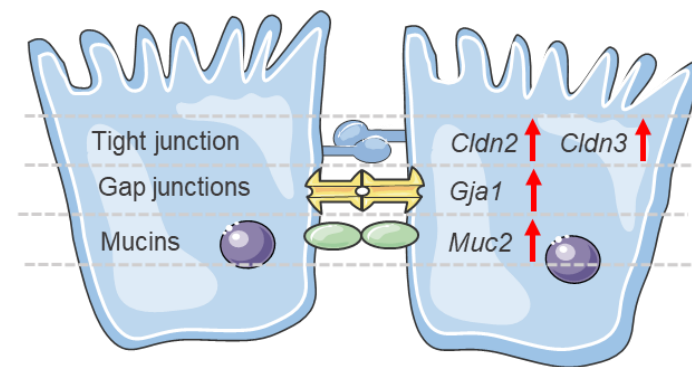
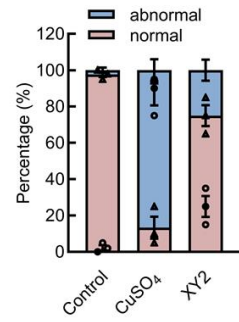
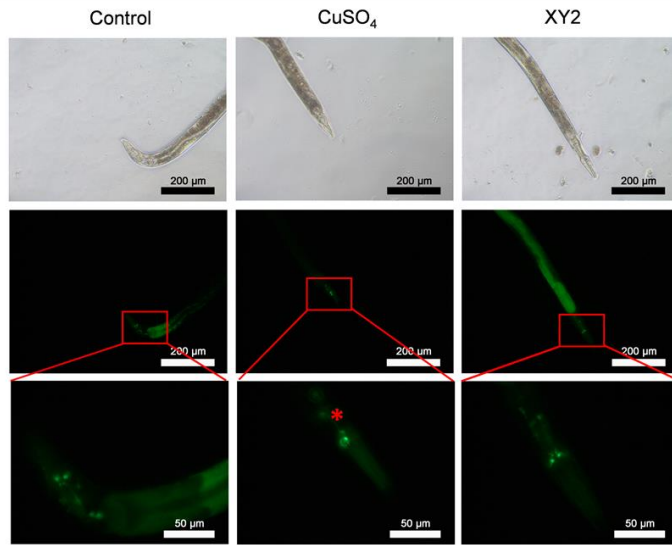


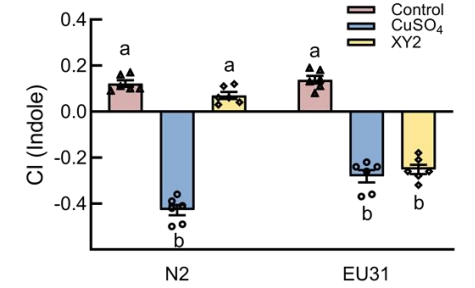
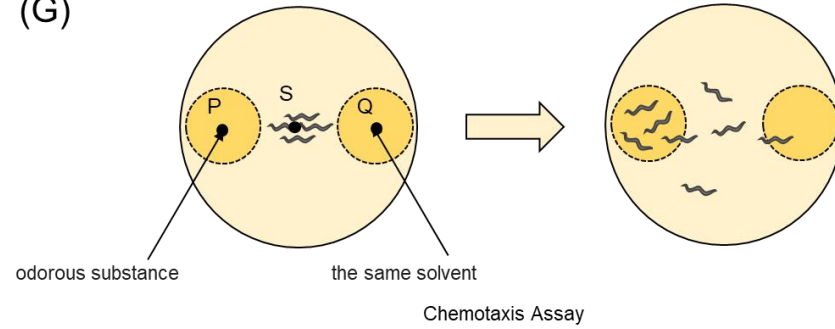
图2. *W. coagulans* XY2通过肠-脑轴抑制铜诱导的神经毒性

结果

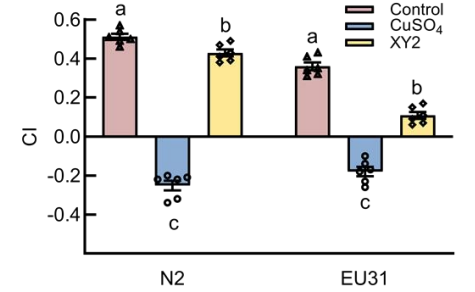
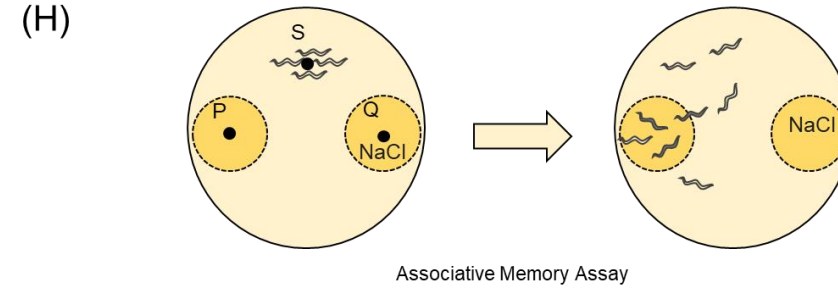
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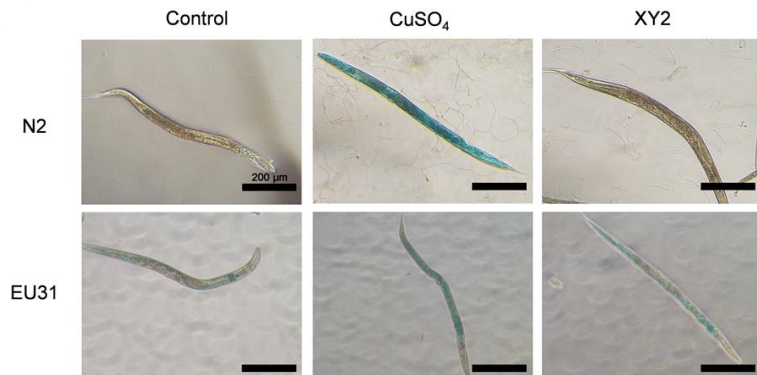
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(H)



(I)



(J)

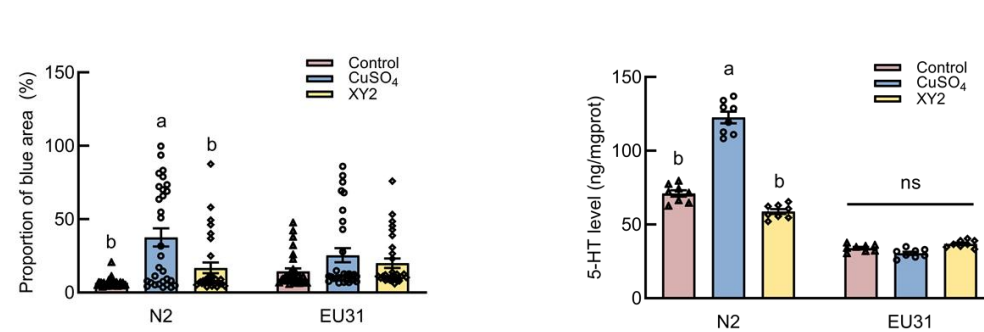


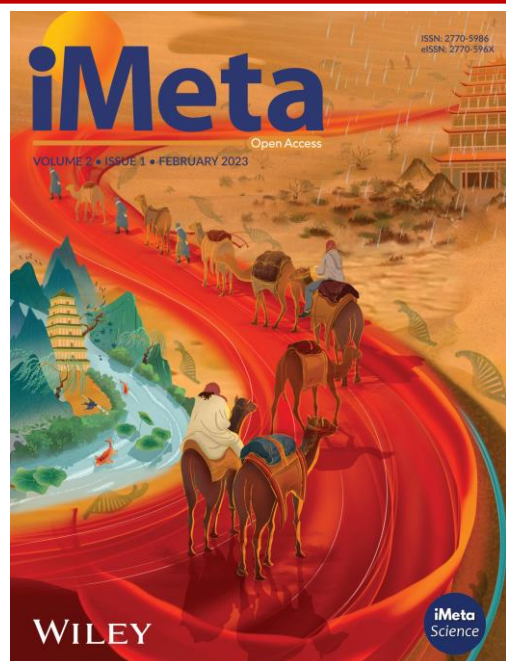
图2. *W. coagulans* XY2通过肠-脑轴抑制铜诱导的神经毒性



总结

- ❑ *W. coagulans* XY2通过肠-脑代谢轴发挥对铜毒性的神经保护作用，具体机制包括调控色氨酸代谢、增强抗氧化防御及维持肠屏障完整性；
- ❑ *W. coagulans* XY2促进5-HT生成、抑制神经毒性犬尿氨酸途径以及促进神经保护性吲哚生成来改善神经功能；
- ❑ *W. coagulans* XY2还激活SKN-1/Nrf2抗氧化通路并维持肠道屏障功能以预防氧化-炎症损伤；
- ❑ 这些发现为益生菌通过肠脑轴来减轻重金属毒性提供了实验证据，拓展了已报道的“铜诱导线粒体功能障碍”模型的研究范畴，为铜诱导神经退行性病变提供了安全、多靶点的干预策略，具有重要转化潜力。

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