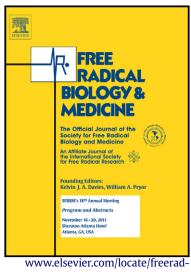
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The overexpression and nuclear translocation of Trx-1 during hypoxia confers HepG2 cells resistance to DDP and GL-V9 reverses the resistance by suppressing Trx-1/ref-1 axis

Li Zhao, Wei Li, Yuxin Zhou, Yi Zhang, Shaoliang Huang, Xuefen Xu, Zhiyu Li, Qinglong Guo



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1 The overexpression and nuclear translocation of Trx-1 during hypoxia confers HepG2 cells resistance to DDP and GL-V9 reverses the resistance by suppressing 2 Trx-1/Ref-1 axis 3 Li Zhao^{1a}, Wei Li^{1a}, Yuxin Zhou^a, Yi Zhang^a, Shaoliang Huang^a, Xuefen Xu^a, Zhiyu Li 4 ^b* and Oinglong Guo^a* 5 6 ¹Contributed equally to this article. 7 8 ^aState Key Laboratory of Natural Medicines, Jiangsu Key Laboratory of 9 Carcinogenesis and Intervention, Jiangsu Key Laboratory of Design and Optimization, 10 Key Laboratory of Drug Quality Control and Pharmacovigilance, Ministry of 11 12 Education, China Pharmaceutical University, 24 Tongjiaxiang, Nanjing 210009, 13 People's Republic of China 14 ^bSchool of Pharmacy, China Pharmaceutical University, Nanjing 210009, People's 15 Republic of China 16

17 Abstract

Microenvironmental hypoxia renders many tumor cells capacity for drug resistance. Thioredoxin (Trx) family members play critical roles in the regulation of cellular redox homeostasis under stressed environment. In this study, we reestablished hypoxia-drug resistance (hypoxia-DR) model using HepG2 cells and discovered that the overexpression and nuclear translocation of thioredoxin-1 (Trx-1) are closely associated with this resistance through regulating metabolism from the oxidative Download English Version:

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