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Abstract

Targeting Heme Oxygenase in Ferroptosis: A Novel Insight in Cancer Therapy †

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Abstract: The term ferroptosis refers to a peculiar type of programmed cell death (PCD) showing characteristic features that differentiate it from other historically well-known types of cellular death such as apoptosis, autophagy, necrosis and necroptosis. Ferroptosis is mainly characterized by extensive iron-dependent lipid peroxidation and mitochondrial dysfunction, together with the rounded morphology of the cell undergoing ferroptotic death. Recently, ferroptosis has been suggested as a potential new strategy for the treatment of several cancers, including breast cancer (BC). In particular, among the BC subtypes, triple negative breast cancer (TNBC) is considered the most aggressive, and conventional drugs fail to provide long-term efficacy. Our study's purpose was to investigate the mechanism of ferroptosis in breast cancer cell lines and reveal the significance of heme oxygenase (HO) modulation in the process. HO's effect on BC was evaluated by MTT tests, gene silencing, Western blot analysis, and measurement of reactive oxygen species (ROS), glutathione (GSH) and lipid hydroperoxide (LOOH) levels. In order to assess HO's implication, different approaches were exploited, using two distinct HO-1 inducers (hemin and curcumin), a well-known HO inhibitor (SnMP) and a selective HO-2 inhibitor. The data obtained showed HO's contribution to the onset of ferroptosis; in particular, HO-1 induction seemed to accelerate the process. Moreover, our results suggest a potential role of HO-2 in erastin-induced ferroptosis. In view of the above, HO modulation in ferroptosis can offer a novel approach for breast cancer treatment.

Keywords: HO-1; cell death; ferroptosis; lipid peroxidation; curcumin

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