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异硫氰酸苄酯对脑胶质瘤U-87 MG细胞活性氧的诱导作用及其机制研究

Effect and Mechanism of Benzyl Isothiocyanate on Active Oxygen Induced in Human Malignant Glioma U-87 MG Cell Line

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中文摘要:

目的 探讨异硫氰酸苄酯(benzyl isothiocyanate，BITC)对人脑胶质瘤细胞系U-87 MG的活性氧(ROS)的诱导作用及其机制。方法 应用MTS法检测BITC对肿瘤细胞生长的影响，2和5 μmol·L$^{?1}$ BITC作用U-87 MG细胞后，应用流式细胞术检测肿瘤细胞内活性氧(ROS)含量的变化，生化法检测GSH以及氧化应激相关的线粒体呼吸链复合体Ⅲ、过氧化物歧化酶(SOD)和醌还原酶(quinone reductase，QR)的活性变化，Western blotting法和报告基因技术检测p38-MAPK和相关转录因子ARE的转录活性变化。结果 BITC对脑胶质瘤细胞U-87 MG具有明显的抑制作用，其IC50值为15.2 μmol·L$^{?1}$，2和5 μmol·L$^{?1}$ BITC作用肿瘤细胞24 h后，ROS产生分别为对照组的376.3%和607.5%(P<0.05)，GSH水平分别为对照组的71.3%和44.9%(P<0.05)，SOD活性分别为对照组的63.5%和21.8%(P<0.05)，QR活性分别为对照组的55.2%和26.7%(P<0.05)，呼吸链复合体Ⅲ活性分别为对照组的48.5%和37.6%(P<0.05)，p38-MAPK的磷酸化水平显著上升，ARE的转录活性分别为对照组的141.1%和215.2%(P<0.05)。结论 BITC可诱导脑肿瘤细胞U-87 MG中ROS产生，可能与调节胞内的氧化应激相关基因表达有关。

英文摘要:

OBJECTIVE To investigate the effect and mechanism of benzyl isothiocyanate(BITC) on active oxygen induced in human malignant glioma cell line U-87 MG. METHODS U-87 MG was dealed with BITC, MTS assay was employed to determine the effect of BITC on the proliferation of cancer cells. After 2 and 5 μmol·L$^{?1}$ U-87 MG cells was treatment with BITC, the alteration of intracellular ROS was measured by flow cytometry, the

level of GSH, the activities change of complex III of the mitochondrial respiratory chain, the superoxide dismutase(SOD) and the quinine reductase(QR) was measured by biochemistry assay, the phosphorylation of p38-MAPK was measured by Western blotting assay and the transcriptional activities ARE was determined by reporter gene system. RESULTS BITC significantly inhibited the proliferation of U-87 MG with an IC50 of 15.2 μmol·L$^{?1}$. After 2 and 5 μmol·L$^{?1}$ BITC treatment for 24 h, intracellular ROS was 376.3% and 607.5%(P<0.05), while the level of GSH was 71.3% and 44.9%(P<0.05), the level of SOD was 63.5% and 21.8%(P<0.05), the level of QR was 55.2% and 26.7%(P<0.05) and level of complex III was 48.5% and 37.6%(P<0.05). Western blotting showed that the phosphorylation of p38-MAPK was upregulated and the transcriptional activities of ARE were 141.1% and 215.2%. CONCLUSION BITC can induce ROS elevation in the tumor cells and the mechanism may be the regulation of oxidative stress related gene expression.