

论著

GSH减少可加强MNNG对P38 MAPK磷酸化

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摘要 目的: 了解低浓度N-甲基-N'-硝基-N-亚硝基胍(MNNG)对P38MAPK的影响, 以及谷胱甘肽(GSH)在该信号通路中的调节作用。方法: 用L-丁硫氨酸-S,R-磺基(L-buthionine-S,R-sulfoximine)减少细胞谷胱甘肽含量后, 采用Western印迹法比较MNNG处理组和对照组P38MAPK磷酸化状态, 观察MNNG对P38磷酸化状态的影响。用光密度扫描仪测定各蛋白质条带吸光值。“P”为磷酸化P38MAPK的吸光值, “T”为总P38MAPK吸光值。在各时点以对照组磷酸化率P/T为1.0, 计算MNNG组的相对磷酸化率。结果: BSO预处理24 h后, MNNG处理2.5 h的相对磷酸化率为0.84, 2.5 h处理后换DMEM全培养基3 h的相对磷酸化率2.19。结论: 细胞内GSH含量降低可促进P38MAPK的活性。

关键词 [P38MAP激酶](#); [谷胱甘肽](#); [N-甲基-N'-硝基-N-亚硝基胍](#)

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GSH deletion increases the activity of p38MAPK in MNNG treatment

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Abstract

AIM: To explore the effect of low concentration of N-methyl-N-nitro-N-nitroguanidine (MNNG) on p38MAPK, and the function of glutathione (GSH) on p38MAPK. METHODS: Western blotting was applied to detect the p38MAPK phosphorylation in MNNG treatment group and control group. To study the effect of GSH on MNNG-regulated p38MAPK activity, the intracellular GSH level was reduced by pretreatment of L-buthionine-S, R-sulfoximine (BSO). Assuming the absorbance of band in control group as 1.0, the relative P/T values of the treatment groups were calculated, with the “P” served as absorbance values of phospho-p38MAPK and the “T” as absorbance values of total p38MAPK. RESULTS: In BSO pretreated groups, the relative P/T in samples treated with MNNG for 2.5 h was 0.84, and became 2.19 with 3 h more incubation in the fresh medium. CONCLUSION: GSH deletion increases the activity of p38MAPK in MNNG treatment.

Key words [P38MAP kinase](#) [Glutathione](#) [N-methyl-N-nitro-N-nitroguanidine](#)

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