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论著

热休克预处理可抑制N_甲基_N′_硝基_N_亚硝基胍对CHL细胞的遗传毒性

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摘要 背景与目的： 探讨热休克是否可诱导中国仓鼠肺细胞(CHL)中γH2AX焦点的形成，以及热休克对N-甲基-N′-硝基-N-亚硝基胍(MNNG)诱导γH2AX焦点形成的影响。 材料与方法： 用MTT实验检测热休克处理后细胞的生存率，用免疫荧光及流式细胞术检测细胞的γH2AX焦点的形成。 结果： MTT结果表明热休克对CHL细胞有细胞毒性作用；免疫荧光实验发现热休克处理后细胞中γH2AX焦点数量与对照组相比无显著差异(P>0.05)；流式细胞检测结果发现经过热休克预处理再经MNNG处理的细胞与只经过MNNG处理的细胞相比，γH2AX荧光强度由0.316±0.042下降为0.194±0.011，其差异有统计学意义(P<0.05)。 结论： 热休克可以使CHL细胞死亡，但是并不引起细胞的遗传毒性；热休克预处理可减少MNNG所诱导的γH2AX焦点的形成。

关键词 热休克； γH2AX； DNA损伤； 遗传毒性

Pre-heat Pre-heat Shock Protects CHL Cells from the Genotoxicity of N-methyl-N′-nitro-N-nitrosoguanidine

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Abstract BACKGROUND & AIM: To study whether heat shock could induce the formation of γH2AX foci in CHL cells, and the effect of heat shock on N-methyl-N′-nitro-N-nitrosoguanidine(MNNG)-induced γH2AX foci formation. MATERIALS AND METHODS: The cytotoxic effect of heat shock was evaluated by MTT test.The phosphorylation of γH2AX was assessed using immunofluorescent microscopy and flow cytometry. RESULTS: MTT test showed that heat shock decreased cell viability.There was no significant difference in the number of γH2AX foci between heat-shock treated group and control group as shown by immunofluorescent microscopy. Flow cytometry analysis revealed that pre-heat shock followed by MNNG treatment decreased the mean γH2AX fluorescent intensity from 0.316±0.042 in the MNNG-treatment alone group to 0.194±0.011. CONCLUSION: Heat shock did not induce γH2AX foci formation in CHL cells.In addition, heat shock could inhibit the phosphorylation of H2AX induced by MNNG.

Keywords heat shock γH2AX DNA damage genotoxicity

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