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论著

Syk对三氧化二砷诱导脑瘤细胞周期阻滞的影响

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摘要 背景与目的： 探讨Syk(Spleen tyrosine kinase)的表达对As2O3在诱导脑瘤细胞周期阻滞过程中的作用。材料与方法： 将syk基因整合至逆转录病毒载体pIND，与载体pVgRXR协同转染携带突变P53基因的脑瘤细胞U373，诱导Syk表达。用Western blot法分析Syk诱导细胞周期负性调控因子P21的表达；用流式细胞仪分析细胞DNA含量，用共聚焦显微镜观察Syk的表达对As2O3在诱导脑瘤细胞周期阻滞过程中的调节作用。 结果： 转染syk基因的U373细胞(U373 Syk_ind)在诱导剂PonA的作用下表达Syk蛋白。As2O3可使U373细胞周期停滞于G2/M期，而表达Syk的U373细胞被As2O3阻滞于G2/M期的细胞比例下降。Syk的表达可上调P21的表达。 结论：As2O3可影响脑瘤细胞U373的细胞周期发展，将其阻滞于G2/M期，但在诱导癌细胞凋亡的同时也产生了致瘤性，而Syk蛋白的表达可以使被阻滞的细胞比例下降，减少了As2O3治疗肿瘤中的副作用。

关键词 Syk； As2O3； 细胞周期； 细胞凋亡

Syk Can Influence As2O3 Induced Brain Tumor Cell Cycle Defects

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Abstract BACKGROUND & AIM: To study the role of expression of Syk(Spleen tyrosine kinase) in the course of As2O3 induced brain tumor cell cycle defects. MATERIAL AND METHODS: To integrate syk gene into vector pIND and co_transfect tumor cell line U373 with mutated P53 gene together with regulator vector pVgRXR in order to induce the expression of Syk. The expression of cell cycle negative regulative factor P21 was measured by Western blot. The cell DNA content was assessed by FACS. The role of Syk's expression in As2 O3 inducing brain cell cycle defects was examined using the confocal laser scanning microscope system. RESULTS: U373 cells transfected with syk gene(U373 Syk_ind)could express Syk in the presence of inducing agent Pon A.As2O3 could make maintain U373 cells at G2/M stage,but the proportion was decreased when U373 expressed Syk.Syk could upregulate P21. CONCLUSION: As2O3 influenced brain tumor cell line U373 normal cycles,blocking them in G2/M stage.As2O3 could cause tumor cell apoptosis,while also inducing normal cells into tumor cells. The expression of Syk could decrease the proportion of blocked cells,so reducing the side_effects of As2O3 when used in treating cancer.

Keywords spleen tyrosine kinase； As2O3； cell cycle； cell apoptosis

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