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论著

ErbB2通过FAK-Src-MAPK信号通路诱导细胞转化和移动侵袭

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摘要 目的： 探讨表皮生长因子受体2（ErbB2）诱导肿瘤转化和侵袭的分子机制。
方法: 用表达ErbB2逆病毒颗粒感染FAK+/+细胞，Western印迹检测ErbB2在FAK+/+细胞中的表达，免疫沉淀检测ErbB2的功能。用Src阻断剂-PP2阻断Src，用MAPK阻断剂-UO126阻断MAPK，观察Src或MAPK被阻断后对ErbB2诱导的细胞移动和细胞转化的影响。
结果: 感染后ErbB2在FAK+/+细胞中稳定表达和激活。PP2抑制ErbB2诱导的FAK磷酸化以及ErbB2诱导的细胞移动。UO126阻断ErbB2诱导的MAPK磷酸化以及ErbB2诱导的细胞锚定依赖性生存-细胞转化。
结论: ErbB2通过FAK-Src-MAPK信号转导通路诱导FAK+/+细胞转化和移动。

关键词 受体,表皮生长因子 FAK-Src-MAPK通路 肿瘤侵润

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Oncogenic transformation,migration and invasiveness of cells induced by ErbB2 are mediated via FAK-Src-MAPK signaling pathway

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Abstract

<FONT face=Verdana>AIM: To explore the possibility that ErbB2-induced oncogenic transformation and invasion involve FAK-Src-MAPK signaling pathway.
METHODS: Parental FAK+/+ cells were infected by retro-vector particles expressing ErbB2.Expression of ErbB2 and its function were assayed by Western blotting and immunopreciptation,respectively.Src inhibitor PP2 or MAPK inhibitor UO126 was used to detect Src or MAPK function on ErbB2-induced cell oncogenic transformation and migration.
RESULTS: ErbB2 was overexpressed and functionally activated in FAK+/+ cells.The phosphorylation of FAK induced by ErbB2 was inhibited by PP2,and the inhibition of FAK by PP2 was associated with impaired cell migration and invasion.UO126 blocked phosphorylation of MAPK induced by ErbB2,and was responsible to impaired anchorage-dependent cell survival in soft agar.
CONCLUSION: Cell oncogenic transformation,migration,and invasion induced by ErbB2 are mediated via FAK-Src-MAPK signaling pathway.</FONT>

Key words Receptor epidermal growth factor FAK-Src-MAPK pathway Neoplasm invasiveness

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