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论著

肺癌细胞A549中FAK通过下游PI3K/AKT途径抗失巢凋亡

傅松滨 刘改云 孟祥宁 王佳曦 赵育桢 王哲

哈医大生物教研室 哈医大遗传教研室

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摘 要 目的 研究FAK调节肺癌细胞A549抗失巢凋亡的功能和信号通路。方法 应用光镜观察，Hoechst33342染核、电镜、Western Blot、RNA干涉技术、PI3K的抑制剂，对FAK调节肺癌细胞A549抗失巢凋亡的功能和信号通路进行研究。结果 发现肺癌细胞A549具有抗失巢凋亡的能力；A549细胞悬浮培养时，FAKtyr-397/861/925的活性随时间的延长逐渐增加又降低，磷酸化的PI 3K和AKT表现出与其一致的活性变化；通过RNA干涉实验发现干涉组比对照组细胞出现明显的凋亡现象，同时PI3K/AKT的磷酸化水平下降；PI3K的抑制剂组比对照组出现更多的凋亡细胞。结论 FAK在肺癌细胞A549抗失巢凋亡中发挥了重要作用，其抗失巢凋亡的机制是通过激活下游PI3K/AKT通路来实现的。

关键词 失巢凋亡 肺癌 FAK PI 3K/AKT

分类号

FAK can resist anoikis through the downstream PI3K/AKT pathway in lung cancer cell, A549

Abstract Objective To study the function and signaling pathway on FAK resisting anoikis in lung cancer cell, A549. Methods Light microscope, Hoechst33342 stain, electron microscope, western blot, siRNA-FAK technique and the inhibitor of PI3K were employed to study the activity of FAK, PI3K and AKT in resisting anoikis. Results Lung cancer cell A549 can resist anoikis; When the cells were cultured by suspended, the activity of FAKtyr-397/861/925 increased gradually and decreased at last, and the level of phosphorylated PI3K and AKT show the same trend with FAK; Compared with the control group, more apoptotic cells were found in siRNA-FAK group, and meanwhile, phosphorylated PI3K and AKT decreased. More apoptotic cells were found in ly 294002 treated group than control. Conclusion FAK plays an important role of resisting anoikis in lung cancer cell, A549, which is caused by activity of PI3K/AKT kinases that were stimulated by FAK.

Key words anoikis lung cancer FAK PI3K/AKT

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通讯作者 傅松滨 fusb@ems.hrbmu.edu.cn