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Title: The ability of SDF-1/CXCR4 axis to proliferation adhesion and invasion of small lung cancer cell

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Body: Objective To investigate the effect on tumor invasion and the role of PI3K signaling pathway after the binding of SDF-1 and CXCR4 receptor. Methods Flow cytometry (FCM) and RT-PCR were utilized to detect the expression of CXCR4 in NCI-H446. CCK-8 assay was utilized to detect the proliferation of tumor cells treated by SDF-1,CXCR4 antagonist AMD3100 and the PI3K inhibitor LY294002.Chemotaxis and transwell invasion experiments were made in different groups to observe the changes of adhesion and invasion ability. Results NCI-H446 with high expression of CXCR4(91.6±2.1%). Compare with the control group,SDF-1,AMD3100 did not affect the proliferation of NCI-H446. LY294002(20μmol/L) could inhibit the proliferation of NCI-H446. 24h, 48h, 72h inhibition rates were 19.4%, 31.3%, 18.4%. Compared with the normal control group,100ng/ml of SDF-1 increased the adhesion ability of NCI-H446,OD values were 1.253±0.107 VS 0.783±0.071($P < 0.05$); it also increased invasion capacity, the penetrating number were 83.2±15.7 VS 30.8±6.5($P < 0.001$). Compared with 100ng/ml of SDF-1 treatment group, AMD3100 and LY294002 could inhibit the adhesion and invasion, OD values were 0.759 ± 0.088 VS 1.253 ± 0.107,0.652 ± 0.076 VS 1.253 ± 0.107 ($P<0.05$) respectively, the penetrating number were 37.8 ± 5.1 VS 83.2 ± 15.7,37.6 ± 7.2 VS 83.2 ± 15.7 ($P < 0.001$). Conclusion Small cell lung cancer cell line NCI-H446 has a high expression of the chemokine CXCR4, its ligand SDF-1 can promote the adhesion and invasion of the tumor cells. PI3K signaling pathway involved in the proliferation works in the adhesion and invasion of NCI-H446 by CXCR4 activation.