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Novel Slit/Robo and CXCL12/CXCR4-mediated signaling mechanisms that modulate small cell lung cancer progression and metastasis

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Small cell lung cancer (SCLC) represents 20% of lung cancers and is characterized by early dissemination, development of chemoresistance and a poor prognosis. Small cell lung cancer (SCLC) is a highly aggressive malignancy with a limited spectrum of therapeutic options. Therefore, identifying early biomarkers and targets may lead to the development of innovative therapies that will improve the survival of SCLC patients. Slit2, a secreted glycoprotein, has been shown to be suppressed in a number of cancers. Slit2 has recently emerged as an important tumor suppressor gene and acts through Roundabout Homolog1 (Robo1) receptor. Slit2/Robo1 signaling has been reported to inhibit the migration of a variety of cancer cells including non-small cell lung cancer (NSCLC). The chemokine receptor CXCR4 and its cognate chemotactic ligand CXCL12 play an important role in cell migration, cancer growth, angiogenesis, and metastasis. However, the molecular mechanism by which the Slit/Robo complex inhibits the migration of small cell lung cancer is not well defined. The aims of this study is to (1) Determine Slit2 and Robo1 expression in a wide range of pulmonary neuroendocrine carcinomas (NEC), including SCLC and in human SCLC patient samples (2) Analyze the role of Slit2 in tumor growth and metastasis in vivo using a Small cell lung cancer mouse model (3) Investigate the role of Slit2/Robo1 signaling pathway modulates the CXCL12/CXCR4-induced chemotaxis and metastasis in Small cell lung cancer.

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