**Lentivirus-mediated Knockdown of ERK3 Inhibits Migration of Gastric Cancer Cells**

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**Introduction**
- Stomach cancer is the 5th most common cancer
- Stomach cancer often metastasizes, making it the 3rd leading cause of cancer-related deaths
- Clinical data has shown that ERK3 may play an inhibitory role in gastric cancer metastasis

**Objective**
To determine the role of ERK3 in vitro by knocking down its expression using short-hairpin RNAs (shRNAs).

**Methods**
- Design two shRNA targets
- Lentivirus plasmid construction
- Lentivirus packaging
- Selection of stable knockdown cell line
- Confirm knockdown efficiency by quantitative PCR (qPCR)
- Cell migration assay
- Cell proliferation assays
  - Colony formation
  - MTT

**Results**
- In the selection of a stable knockdown cell line, green fluorescence indicates successful infection by lentivirus.
- Following lentivirus infection, ERK3 mRNA is significantly downregulated.
- Knockdown of ERK3 inhibits cancer cell migration.
- Knockdown of ERK3 inhibits colony formation.

**Discussion**
- These experiments have demonstrated the effect of ERK3 on cells’ migratory and proliferative abilities in vitro
- An inconsistency arose in the results from the colony-formation and MTT assay; in vivo experiments could clarify the proliferative capacity
- In vivo experiments to test the tumorigenesis and metastatic potential of this cell line are needed to confirm the function of ERK3

**Questions**
- What contributed to the differing results of the colony-formation assay and MTT assay?
- Why does this experimental data, unlike clinical data, suggest that ERK3 contributes to metastasis?

**Conclusion**
ERK3 contributes to the metastasis and growth of gastric cancer cells in vitro.

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