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Genomic landscape and differentiating genetic biomarkers of pancreatic and small bowel neuroendocrine tumors

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Neuroendocrine tumors can develop in multiple anatomic locations including the gastrointestinal tract, pancreas, lungs, cervix, thymus and thyroid. Approximately, 50% of these tumors are metastatic at the time of diagnosis, complicating identification of the primary tumor's site of origin. Determining the tumor site of origin is particularly important in NETs as both prognosis and treatment differ depending on where the tumor originated. Thus, the ability to determine NET site of origin can be of critical importance in the clinical management of these patients. To this end, we performed array-based comparative genomic hybridization to identify site of origin specific copy number variants in NETs of pancreatic (n=13) and ileal (n=10) origin. We used gene expression profiling to prioritize those genomic gains or losses that were associated with concordant differential gene expression. Four copy number variable regions exhibited statistically significant differential copy number status reflective of NET site of origin ($p<0.001$): 18q, 19q, 12p, and 9q. Several of these loci also exhibited differential gene expression changes suggesting they are functional copy number changes as opposed to by-stander genetic lesions. We tested the ability of fluorescence in situ hybridization (FISH) to identify these chromosomal lesions in formalin fixed paraffin embedded (FFPE) NET tissue. This work lays the foundation for the development of a clinical diagnostic assay that can be performed on all newly diagnosed NETs of unclear site of origin.

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The role and therapeutic potential EF2-kinase in pancreatic cancer

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Pancreatic cancer represents an unmet therapeutic challenge due to extremely poor mortality rates (1-5% 5 year survival rates). We recently discovered that eukaryotic elongation factor-2 kinase (EF2-Kinase) is highly up regulated in pancreatic cancer cell lines including those with KRAS mutations and promotes cell proliferation, survival, invasion/migration, drug resistance and tumorigenesis. We found that in-vivo therapeutic targeting of EF2K gene by si-RNA nano-therapeutics significantly inhibits tumor growth in pancreatic tumor xenografts in mice. Our data suggest that EF2K represent a novel therapeutic target in pancreatic cancer great promise as a therapeutic intervention for gene specific silencing therapy of oncogenes in cancer.

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