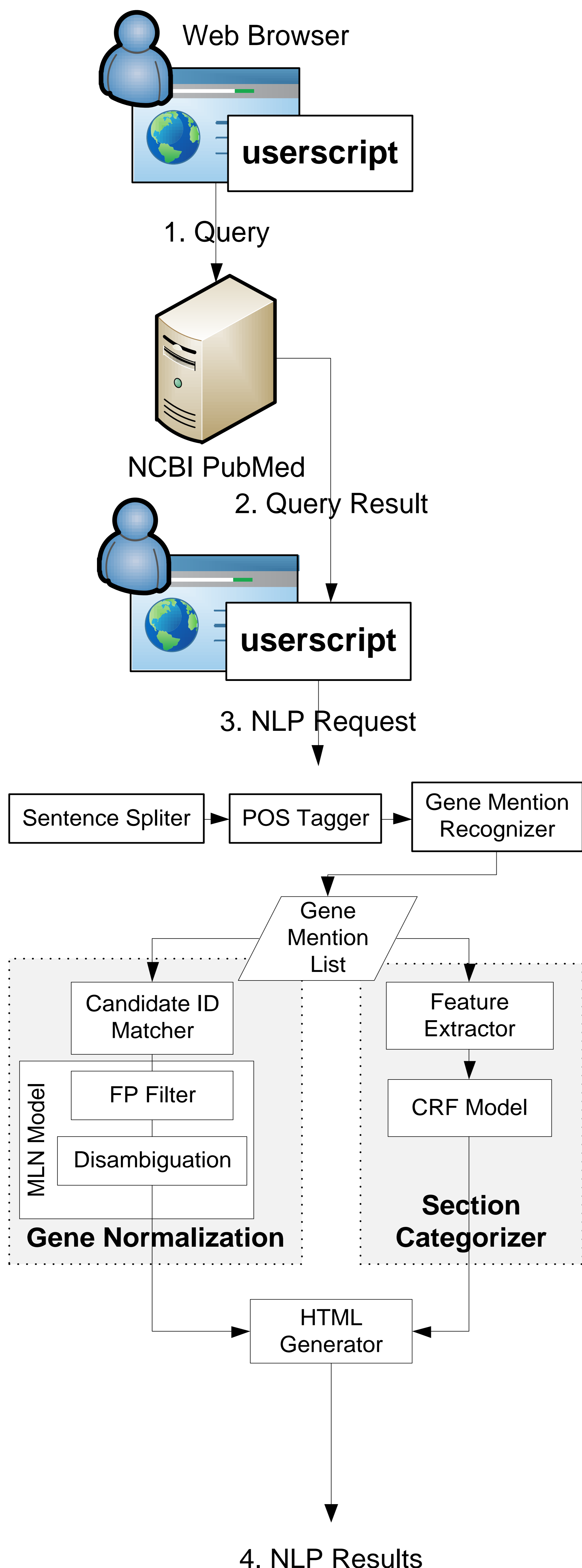




PubMed-EX: A Web Browser Extension to Enhance PubMed Search with Text Mining Features

We embedded our natural language system into the NCBI PubMed search website using augmented browsing technology. The system dynamically enriches the original search results with semantic annotations, including **section categorization**, **gene/disease name**, and **semantic relations**.



Cancer Epidemiol Biomarkers Prev. 2008 Dec;17(12):3536-42.
 3232. **Genetic and epigenetic alterations of familial pancreatic cancers.**
 Brune K, Hong SM, Li A, Yachida S, Abe T, Griffith M, Yang D, Omura N, Eshleman J, Canto M, Schulick R, Klein AP, Hruban RH, Jacobuzio-Donohue C, Goggins M.
 Department of Pathology, Medicine, Oncology, Johns Hopkins Medical Institutions, The Sol Goldman Pancreatic Cancer Research Center, 1550 Orleans Street, CRB2, Room 342, Baltimore, MD 21231, USA.

BACKGROUND: Little is known about the genetic and epigenetic changes that contribute to familial pancreatic cancers. The aim of this study was to compare the prevalence of common genetic and epigenetic alterations in sporadic and familial pancreatic ductal adenocarcinomas. METHODS: DNA was isolated from the microdissected cancers of 39 patients with familial and 36 patients with sporadic pancreatic adenocarcinoma. KRAS2 mutations were detected by BstN1 digestion and/or cycle sequencing. TP53 and SMAD4 status were determined by immunohistochemistry on tissue microarrays of 23 archival familial pancreatic adenocarcinomas and in selected cases by cycle sequencing to identify TP53 gene mutations. Methylation-specific PCR analysis of seven genes (FoxE1, NPTX2, CLDN5, P16, TFPI-2, SPARC, ppENK) was done on a subset of fresh-frozen familial pancreatic adenocarcinomas. RESULTS: KRAS2 mutations were identified in 31 of 39 (80%) of the familial versus 28 of 36 (78%) of the sporadic pancreatic cancers. Positive immunolabeling for p53 was observed in 57% of the familial pancreatic cancers and loss of SMAD4 labeling was observed in 61% of the familial pancreatic cancers, rates similar to those observed in sporadic pancreatic cancers. The mean prevalence of aberrant methylation in the familial pancreatic cancers was 68.4%, which was not significantly different from that observed in sporadic pancreatic cancers. CONCLUSION: The prevalence of mutant KRAS2, inactivation of TP53 and SMAD4, and aberrant DNA methylation of a seven-gene panel is similar in familial pancreatic adenocarcinomas as in sporadic pancreatic adenocarcinomas. These findings support the use of markers of sporadic pancreatic adenocarcinomas to detect familial pancreatic adenocarcinomas.

PMID: 19064568 [PubMed - indexed for MEDLINE]
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Information from MeSH
Pancreatic Neoplasms
 Tumors or cancer of the PANCREAS. Depending on the types of ISLET CELLS present in the tumors, various hormones can be secreted: GLUCAGON from PANCREATIC ALPHA CELLS; INSULIN from PANCREATIC BETA CELLS; and SOMATOSTATIN from the SOMATOSTATIN-SECRETING CELLS. Most are malignant except the insulin-producing tumors (INSULINOMA).

Information from Entrez Gene
Gene name: KRAS
Protein name: GTPase KRas
Taxonomy: *Homo sapiens (Human)*, Entrez, UniProt
Summary:
 This gene, a Kirsten ras oncogene homolog from the mammalian ras gene family, encodes a protein that is a member of the small GTPase superfamily. A single amino acid substitution is responsible for an activating mutation. The transforming protein that results is implicated in various malignancies, including lung adenocarcinoma, mucinous adenoma, ductal carcinoma of the pancreas and colorectal carcinoma. Alternative splicing leads to variants encoding two isoforms that differ in the C-terminal region. [provided by RefSeq]

RESULTS:
 KRAS2 mutations were identified in 31 of 39 (80%) of the familial observed in 57% of the familial pancreatic cancers and loss of sporadic pancreatic cancers. The mean prevalence of aberrant observed in sporadic pancreatic cancers.

CONCLUSION:
 The prevalence of mutant KRAS2, inactivation of TP53 and SMAD4, and aberrant DNA methylation of a seven-gene panel is similar in familial pancreatic adenocarcinomas as in sporadic pancreatic adenocarcinomas.

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Features

- Section categorization
- Gene names recognition
- Gene normalization
- Gene summary
- Disease recognition
- Disease summary
- Semantic relation analysis
- Cloud computing

Installation

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