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Time Series Expression Pattern of Key Genes Reveals the Molecular Process of Esophageal Cancer

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Introduction

Esophageal cancer is one of the most inadequately diagnosed and fatal cancers in the world. Although a series of studies on esophageal cancer have been reported, the molecular pathogenesis of the complaint is still fugitive. To probe the molecular process of esophageal cancer exhaustively and deeply. Differential expression analysis was performed to identify Differentially Expressed Genes (DEGs) in different stages of esophageal cancer. Also exacting gene commerce modules and mecca genes were linked in module commerce network. Further, though survival analysis, methylation analysis, pivot analysis, and enrichment analysis, some important motes and related function or pathway were linked to interpret implicit medium in esophageal cancer. A aggregate of 7457 DEGs and 14 gene commerce modules were linked. These module genes were significantly involved in the positive regulation of protein transport, gastric acid stashing, insulin-suchlike growth factor receptor list and other Natural Processes (BPs), as well as p53 signaling pathway, ERBB signaling pathway and Epidermal Growth Factor Receptor (EGFR) signaling pathway. Also, Recap Factors (TFs) (including HIF1A) and ncRNAs (including CRNDE and hsa-mir-330-3p) significantly regulate dysfunction modules were linked. Further, survival analysis showed that GNGT2 was nearly related to survival of esophageal cancer. And DEGs with strong methylation regulation capability were linked, including SST and SH3GL2. These workshop not only help us to reveal the implicit nonsupervisory factors in the development of complaint, but also consolidate our understanding of its deterioration medium.

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Esophageal cancer is one of the world's early cancers with poor opinion and high mortality. It has strong invasiveness and a fast growth rate. Dysphagia and unconscious weight loss are the most common clinical symptoms. Cases with esophageal cancer have further increased their difficulty in eating due to the staging and position of the excrescence and the poor adjuvant treatment. Studies have shown that there's a clear relationship between the development of esophageal cancer and helicobacter pylori infection, gastroesophageal influx complaint, smoking and severe alcohol use, as well as diet and other inheritable factors. From a remedial point of view, esophageal cancer can be divided into early esophageal cancer, locally advanced resectable esophageal cancer, locally advanced unresectable esophageal cancer and metastatic esophageal cancer. Because of the anatomical features of esophageal cancer, esophageal cancer is generally detected in the late stage, which vitally affects the treatment and prognostic of cases. Endoscopic remedy for early esophageal excrescences is effective and safe. Optimal results can be attained by using endoscopic mucosal resection, ablation remedy, and personalized styles combining both. Treatment for advanced esophageal cancer is limited and may be hampered by the presence of micrometastatic complaint.

In the development of esophageal cancer, the rs11473 polymorphism of the miR-483-5p binding point plays a vital part in the 3 ′-UTR of the basigin gene. Single nucleotide polymorphisms (SNPs) in TERT may be associated with vulnerability to esophageal cancer and contribute to the development of esophageal cancer. In terms of nonsupervisory motes, miR-502 regulates proliferation of esophageal cancer cells by promoting phosphorylation of AKT signaling. B7-H1 can be used as a prognostic factor for mortal esophageal cancer and may be an important remedial target for immunotherapy against this nasty excrescence. miR-20b may play an essential part in the tumorigenesis of esophageal cancer by regulating PTEN expression, which may be a implicit remedial target for the treatment of esophageal cancer. MicroRNA-506 inhibits proliferation of esophageal cancer cells by targeting CREB1. MiR-21 targets critical proteins in the PTEN/ PI3K/ AKT signal transduction to promote proliferation, cell migration, cell irruption, and cell cycle, as well as inhibition of cell apoptosis in mortal esophageal cancer cells. These findings have strengthened our understanding of the pathogenesis of esophageal cancer and guided us in the direction of farther exploration. Although the forerunners have reported a series of exploration results on esophageal cancer, the molecular pathogenesis of the complaint is still fugitive. To exhaustively and deeply explore the molecular processes of esophageal cancer progression and to explore implicit remedial targets for the progress of esophageal cancer, we conducted a methodical module analysis. Overall, our work details the part of multifactorial mediated dysfunction modules in the overall growth of esophageal cancer, relating essential genes and related Natural Processes (BPs), chancing implicit molecular mechanisms and remedial targets for esophageal cancer.