

AB074. P046. Comprehensive analysis of links between diabetes and pancreatic cancer: a bioinformatical approach

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Background: Long standing type 2 diabetes (T2DM) is a risk factor for pancreatic cancer, whereas new-onset diabetes could be seen as a result of pancreatic ductal adenocarcinoma (PDAC). Additionally, diabetes has also been suggested to be an adverse prognostic factor for PDAC. This study was to investigate shared genetic etiology underlying diabetes and pancreatic cancer, and molecular alterations in PDAC with/without diabetes from the TCGA cohort.

Methods: By integrating data from disease related microarray analysis, we carried out a dynamic biological network analysis on the overlapping differential genes in diabetes (GSE28735) and pancreatic cancer (GSE25724) with expression profiles downloaded from GEO database.

Besides, differential molecular profiling for PDAC with/without diabetes from TCGA were performed by the differential gene expression and pathway analysis. Protein-protein interaction (PPI) and miRNA network were also constructed.

Results: The overlapping between diabetes and pancreatic cancer revealed 16 differentially expressed genes (DEGs). Gene ontology (GO) analyses revealed that most of the DEGs were significantly enriched in hormone metabolic process, organic hydroxy compound metabolic process and alcohol metabolic process. Highly relevant pathways include butanoate metabolism and propanoate metabolism. Comparative genomic analysis of TCGA data suggested that DEGs of PDAC with/without diabetes were mainly enriched in immunity-related pathways including T cell receptor signaling and natural killer cell mediated cytotoxicity.

Conclusions: The molecular connections between diabetes and pancreatic cancer are complicated. Glycometabolism and immunity alterations might hold the key to this riddle in terms of bioinformatics.

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