Contribution of different types of -omic data on personalized diagnosis of cancer

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Cancer is a complex disease which is one of the main causes of death globally. Survival rate of cancer increases if early diagnosis and application of appropriate treatment are available [1,2]. Understanding differential response from one patient to another can aid in drug targeting, understanding disease progression and finding efficient treatment strategies. Tumour cells shown to have altered metabolism and the most commonly defined example is the Warburg effect in which tumour cells shown to use lactic acid fermentation instead of oxidative phosphorylation due to the increased energy demand caused by fast proliferation rate [3]. These alterations are controlled at genomic, transcriptomic, fluxomic and metabolomic levels causes the necessity of using systems biology approaches for investigations of cancer.

GSMMs became an important tool for simulation and analysis of such metabolic alterations. By integration of omic data to genome scale metabolic models, tissue/disease/patient specific flux distributions can be obtained and analyzed for personalized metabolic responses to diseases and treatments. By linking genotype to phenotype, they can allow identification of personalized treatment strategies for complex metabolic diseases. Additionally, providing cancer-specific and easily measurable markers makes risk group or population wide regular screening with less invasive methods possible.

In this study, contribution of genomic, transcriptomic and fluxomic data on classification of lung and pancreatic cancer, which is hard to diagnose at early stages, was performed by integrating genomic and transcriptomic data obtained from TCGA and NCBI-GEO databases and calculated personalized flux distributions from integration of transcriptome data to GSMMs. Using random forest classification of multi-omic data, individual effects of each omic level information as well as genes/reactions/metabolites that have potential to be used as biomarkers are demonstrated.

References

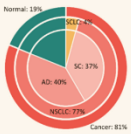
[1] Jemal A, Bray F, Center MM, Ferlay J, Ward E, Forman D. Global cancer statistics. CA Cancer J Clin. 2011;61(2):69–90

[2] Callejon-Leblic B, Garcia-Barrera T, Gravalos-Guzman J, Pereira-Vega A, Gómez-Ariza JL. Metabolic profiling of potential lung cancer biomarkers using bronchoalveolar lavage fluid and the integrated direct infusion/gas chromatography mass spectrometry platform. J Proteomics. 2016;145:197–206.

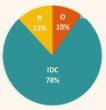
[3] Warburg, O. (1956) ‘On the origin of cancer cells’, Science, 123(3191), pp. 309–314.

DATA ACQUISITION

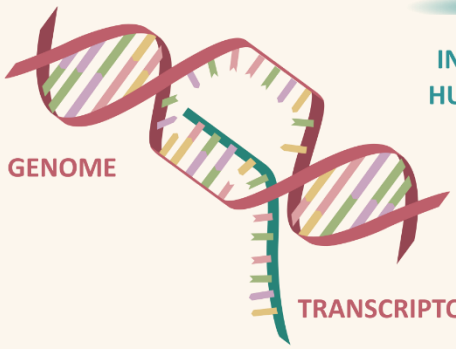
Lung Cancer



Pancreatic Cancer



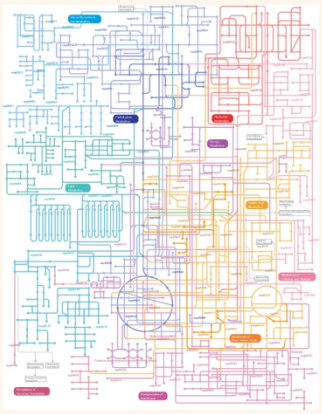
THE CANCER GENOME ATLAS
National Cancer Institute
National Human Genome Research Institute



GENOME

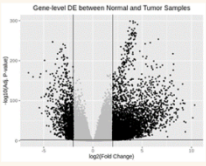
TRANSCRIPTOME

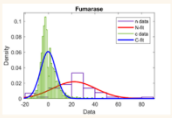
INTEGRATION TO HUMAN RECON3D

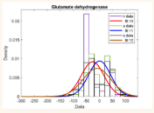


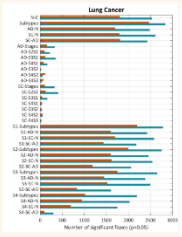
FLUXOME

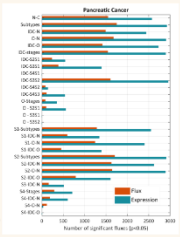
PATHWAY ENRICHMENT AND FEATURE SELECTION

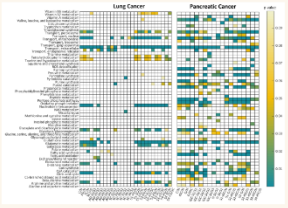






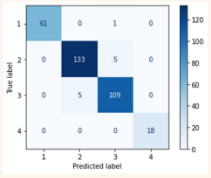


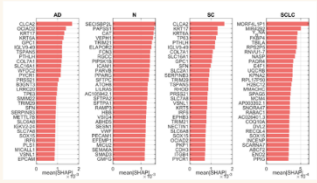




CLASSIFICATION

TRANSCRIPTOME





FLUXOME