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Poster Submissions

Poster Title

Integrative genomic mining for markers of tumor initiation and progression in oral squamous cell carcinoma

Authors and their Affiliation

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Please describe the extent of your work in this research

I am responsible for all computational/statistical analyses utilizing the described gene expression signatures, including the processing and integration of genomic datasets, as well as the ongoing development of the described search algorithm.

Abstract Submission

- GSI_2015_Kartha.docx

Would you like your abstract to be considered for an oral presentation (students and post docs only)?

Yes

Title: Integrative genomic mining for markers of tumor initiation and progression in oral squamous cell carcinoma

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Abstract: Progression of oral squamous cell carcinoma (OSCC) to metastasis involves complex changes in epithelial cell growth, survival and migration. Prior studies have identified a co-dependent homeostatic pathway 'network' consisting of protein N-glycosylation, Wnt/β-catenin and Hippo pathways to be deregulated in OSCC, playing a vital role in its tumorigenesis. While their involvement in cancer has been independently highlighted, the interplay between these pathways in promoting tumor metastasis remains less understood. Furthermore, identifying exact mediators of these changes still remains a challenging task, crucial to the discovery of novel and lasting OSCC therapeutics. Here, we perform a multi-omic investigation using a combination of OSCC cell-line gene expression profiles and massive public genomic data to help determine potential mechanisms driving OSCC pathogenesis. Gene expression signatures pertaining to genetic knockdowns of DPAGT1 - a gene crucial to protein N-glycosylation, and TAZ and YAP - two transcriptional activators involved in the Hippo pathway, were first derived using SCC2 cells. Primary human OSCC high-throughput gene expression data from The Cancer Genome Atlas (TCGA) was then projected onto these signatures and analyzed for their association with clinical features, including tumor grade and stage. By scoring samples based on their level of pathway deregulation, and additionally leveraging Copy Number Alteration (CNA) and somatic mutation data using a novel heuristic search algorithm, we are able to identify potential regulators of human OSCC development in the context of the DPAGT1/β-catenin/YAP signaling network, paving the way to discovering targets for OSCC therapy.