Title: Examining APOBEC3B in TCGA and ICGC Breast Cancer Datasets Reveals Increased Immune Infiltration

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Despite improvements in both screening and treatment, breast cancer mortality remains

high. Nearly a third of patients diagnosed with breast cancer will die of their disease worldwide. APOBECs are a large family of deaminase enzymes that act endogenously to restrict viral replication. Upregulation of APOBECs have been documented in many cancers, where its conversion of cytosine to uracil leads to a unique mutation signature dominated by C-to-T transitions. Within this deaminase family of enzymes, APOBEC3B (A3B) has been

demonstrated to have increased expression levels in breast cancer. It remains unclear if tumors with increased A3B mutational signature continue to display increased levels of expression. Moreover, the pathways that may be involved in modulating A3B levels is an area of increasing development and importance. Using DNA and RNA sequencing data from breast cancer patients in The Cancer Genome Atlas (TCGA) and the International Cancer Genome Consortium (ICGC), we found that tumors with increased A3B mutational signature enrichment continued to have relatively high levels of A3B expression. Using Kegg, Gene Ontology (GO), and Reactome pathway analysis between high and low A3B expressing tumors, we found an abundance of pathways related to immune signaling and recruitment. Furthermore, using TIMER 2.0 to estimate immune infiltration within TCGA samples, we found an increased abundance of CD8+ T-cells and Natural Killer (NK) cells. Together, this suggests that A3B expression levels may be a useful surrogate for A3B mutational burden, and the increased A3B expression may contribute to increased immune recruitment.