Molecular pathways involved in epithelial-myoepithelial carcinomas and myoepithelial carcinomas. Implication of the HRAS activating mutations and identification of different tumor profiles

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Background & aims

Few data are available about the molecular description of salivary glands carcinomas. In a first study we have shown that some rare mutations (KRAS, PI3K, CKIT…) could be found in some specific histological subtypes. Epithelial-myoepithelial carcinoma (EMC) and myoepithelial carcinomas (MC) seemed to be particularly concerned.

The aim of this study was to address the molecular pathways involved in EMC and MC carcinogenesis.

Results

Immunohistochemical comparison of EMC and MC

- Fig. 1: Immunohistochemical comparison of EMC and MC. A) Proliferation index (Ki-67) B) Expression of p63 by IHC (% of positive tumor cells) C) Expression of C-KIT by IHC (% of positive tumor cells)

Genomic comparison of EMC and MC

- Table displaying the mutational profile of EMC and MC

Transcriptomic comparison of EMC and MC

- Unsupervised analyses of EMC and MC based on their transcriptome and pathways enriched in EMC vs. MC (GSEA)

Conclusions

- EMC and MC display different mutational and transcriptomic patterns. EMC have high mutational burden and MYC activation gene signatures. MAPK activation through HRAS/PI3K mutations defines a subtype of EMC with increased proliferation.