with more samples, assessing expression of different proteins be done in the future.

TUNICAMYCIN-INDUCED ENDOPLASMIC RETICULUM STRESS UP-REGULATES TUMOUR-PROMOTING CYTOKINES IN ORAL SQUAMOUS CELL CARCINOMA.

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Objectives: Signal transducer and activator of transcription (STAT)-3 lies at the convergence point of key pathways involved in many malignancies including oral squamous cell carcinoma (OSCC). Endoplasmic reticulum stress (ERS) and the unfolded protein response promote either survival or apoptosis in different cancers. We investigated the expression of STAT3 pathway-related genes and proteins under ERS in OSCC.

Three normal oral keratinocyte (NOK) and three OSCC cell lines were subjected to tunicamycin to induce ERS for 24 hours or to the vehicle medium as control. A pathway-focussed array was used to analyse the modulation of STAT3 pathway gene expression under ERS using qPCR. The expression of key regulated proteins was investigated in the cell lines using immunocytochemistry and in 76 OSCC and 9 normal oral mucosa (NOM) tissue samples using tissue microarray technology and immunohistochemistry.

Findings: ERS resulted in up-regulation of interleukin-6 receptor (IL6R) gene in NOK cell lines ($p = 0.001$) and IL5 ($p = 0.005$) and IL22 ($p = 0.024$) in OSCC cell lines. Greater STAT3 ($p = 0.019$) and leukaemia inhibitory factor receptor ($p = 0.042$) protein expression was observed in treated than untreated NOK cell lines.

Conclusions: The gene and protein regulation patterns show that ERS plays a role in modifying the tumour microenvironment in OSCC by up-regulating tumour-promoting cytokines.

CLINICO-PATHOLOGICAL SIGNIFICANCE OF B-CATENIN AND E-CADHERIN EXPRESSION IN SALIVARY GLAND TUMOR AT UCH IBADAN.

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Objectives: β-catenin (B-Cat) is a cell adhesion molecule associated with the invasion and metastasis of carcinomas of the head and neck, esophagus while reduced expression of E-cadherin (E-cad), a transmembrane glycoprotein, is associated with loss of differentiation, acquisition of an invasive phenotype, and an unfavorable prognosis in carcinomas from several sites. B-Cat & E-Cad complex are involved in cell adhesion, signal transduction & motility. Aim is to identify the clinical / pathological significance of B-Cat & E-Cad expressions in salivary gland tumors (SGTs) presenting at the University College Hospital, Ibadan.

Findings: The expressions of β-cat & E-Cad were analyzed in 46 SGTs (10 pleomorphic adenomas PSA, 3 basal cell adenoma, 12 adenoid cystic carcinoma ADCC, 10 mucoepidermoid carcinoma MEC, 5 acinic cell carcinoma ACC, 4 polymorphous low grade adenocarcinoma PLGA & 2 papillary cystadenocarcinoma PCADCA) by immunohistochemistry in formalin-fixed, paraffin embedded specimens (Rabbit monoclonal; Satacruz biotechnology). Result shows immunostaining of B-cat & E-Cad were membranous & cytoplasmic without nuclear involvement, staining was more severe in the ductal areas especially in PSA, there was significant loss of membranous stain in ACC on multivariate analysis. E-Cad staining loss was significantly associated with tumour stage in ACC & MEC.

Conclusion: loss of β-catenin adhesion molecule may be involved in the development of ACC. E-cad expression is an independent indicator of clinical aggressiveness in patients with ACC.& MEC.

KRAS MUTATIONS DRIVE ADENOMATOID ODONTOGENIC TUMOR.

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Objective: KRAS is the most frequently mutated oncogene in human neoplasms and we have previously reported KRAS p.G12V mutations in adenomatoid odontogenic tumors (AOT). We aimed to expand this cohort of samples and to test the association of KRAS mutations with clinical and histopathological parameters. A convenience sample of 30 AOT cases was included in the study. The hotpot KRAS p.G12V mutation was assessed by TaqMan allele-specific qPCR and codon 12 was direct sequenced. Clinical information obtained included patients age, tumor site, association of the lesion with impacted teeth and clinical tumor size. In addition, tumor capsule thickness was evaluated by morphometric analysis. Statistical analysis was carried out to test the association of KRAS codon 12 mutations with clinico-pathological parameters.

Findings: Molecular results confirmed KRAS p.G12V mutation in 14/23 cases, and p.G12R in 1/23. Eight cases were wild-type and samples from 7 cases failed amplification. Codon 12 mutations were not associated with any of the clinicopathological parameters tested ($p>0.05$).

Conclusion: AOT show high frequency of KRAS codon 12 mutations (15/23, 65%), which occur irrespectively of patients' age, tumor location, association with impacted teeth, tumor clinical size or histopathological capsule thickness. Supported by FAPEMIG, CAPES and CNPq/Brazil.

CORRELATION OF HPV16 DETECTION AND P16 EXPRESSION IN ORAL SQUAMOUS CELL CARCINOMA.

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