Introduction: Formerly known as mammary analogue carcinoma (MASC), secretory carcinoma of the salivary glands (SCSG) is a low-grade malignancy harboring the ETV6-NTRK3 translocation. SCSG shares morphological and genetic features with secretory carcinoma of the breast. It shows a characteristic protean histological phenotype reminiscent of acinic cell carcinoma, mucoepidermoid carcinoma, and adenocarcinoma, NOS. We report a case of SCSG in a pediatric patient showing a pure zymogen-poor acinic cell carcinoma morphology.

Clinical presentation: A 9-year-old female patient presented to the ENT clinic with 8 months history of left facial swelling. Imaging studies showed a 2.7 cm lesion of the left parotid gland with no lymphadenopathy. Left lateral lobe parotidectomy with facial nerve dissection and preservation was performed. Gross examination of the tumor revealed a tan, hemorrhagic ill-defined mass, measuring $2.5 \times 2.4 \times 2$ cm. Microscopic examination showed lobulated growth pattern consisting of papillary architecture and microcystic spaces. Lobules of tumor cells were identified in the intraparotid lymph node. The tumor was negative for DOG-1 and strongly positive for Mammaglobin, S-100, GATA-3, CK 7, and Pan cytokeratin. The ETV6 gene translocation was subsequently detected by FISH.

Literature review: A total of 17 cases of pediatric SCSGs (age range: 8-18 years) have been reported in the literature. The parotid gland (88%) is the most common location. The mainstay of treatment is surgical resection. Based on the limited number of documented cases, SCSG appears to show an indolent biological behavior rarely occurring in pediatric patients.

Conclusion: Because of the spectrum of histological patterns of SCSG, immunohistochemical markers and genetic documentation of the ETV6-NTRK3 gene fusion are essential to correctly diagnose this tumor.

IMMUNOHISTOCHEMICAL DISTRIBUTION OF THE SIBLINGS IN AMELOBLASTOMA AND ODONTOGENIC KERATOCYST.

DR. CORNELIUS IYOGUN[A], DR. OLUFEMI OMITOLA[B], PROF. KALU U.E. OGBUREKE[C]. [A] UNIVERSITY OF PORT PORT HARCOURT, [B] UNIVERSITY OF PORT HARCOURT, [C] UNIVERSITY OF TEXAS HEALTH SCIENCE CENTER AT HOUSTON

Objective: Ameloblastomas are benign but aggressive neoplasms of the jaw. The odontogenic keratocysts (OKC) are aggressive jaw cysts of odontogenic origin. A recent WHO classification designated OKC as a neoplasm but a current edition redesignated it as a cyst. The small integrin-binding ligand n-linked glycoproteins (SIBLINGs) are a family of molecules that some epithelial neoplasms use in furthering their progression. Members of the SIBLING family are bone sialoprotein (BSP), dentin matrix protein1 (DMP1), dentin sialophosphoprotein (DSPP), matrix extracellular phosphoglycoprotein (MEPE), and osteopontin (OPN). The objective of this study was to compare the expression of the SIBLINGs in ameloblastomas and odontogenic keratocysts.

Findings: Using immunohistochemistry, with appropriate controls, 49 cases of ameloblastomas and 35 cases of OKCs were screened for the expression of all five SIBLINGs in a retrospective study on archived paraffin sections. Immunoreactivity was scored as positive if > 10% of tumor/cyst cells stained for a SIBLING and negative if <10% of cells failed to stain for a SIBLING. For ameloblastomas 46 (94%) were immunopositive for BSP, 49 (100%) for OPN, 38 (76%) for DSPP, 16 (33%) for DMP1, 32 (65%) for MEPE, 47 (96%). For OKCs 32 (91%) were positive for BSP, 27 (77%) for DMP1, 35 (100%) for DSPP, and 14 (40%) for MEPE. The expression of DSPP and MEPE in ameloblastomas and OKCs respectively, were significant ($p<0.05$) compared with controls. MEPE ($\chi^2=6.15$, $p<0.05$), and BSPP ($\chi^2=26.06$, $p<0.05$) had positive predictive values greater than chance for ameloblastoma and odontogenic keratocyst, respectively.

Conclusion: While all members of the SIBLINGs are expressed in ameloblastomas and OKCs, DSPP expression in OKCs is significantly higher than in ameloblsatomas, whereas, MEPE expression in OKCs is considerably lower than in ameloblastomas. The differences in the expression of DSPP and MEPE between ameloblastomas and OKCs may indicate differences in the degree of aggressive behaviors.

GALECTIN-1 INHIBITION OF ORAL CANCER IN VITRO.

MS. PHILIPPA GREER[A], DR. DAWN COATES[A], PROF. ALISON RICH[B]. [A] SIR JOHN WALSH RESEARCH INSTITUTE, FACULTY OF DENTISTRY, UNIVERSITY OF OTAGO, [B] UNIVERSITY OF OTAGO

Galectin-1 is a carbohydrate-binding molecule that has been shown to be over-expressed in many types of cancer, including oral squamous cell carcinoma (OSCC). The higher the level of expression of galectin-1 by OSCC cells the greater the likelihood of invasion, distant metastasis and a poor survival rate.

Objectives: Investigation of the effect of galectin-1 in OSCC invasion, migration and epidermal-mesenchymal transition in vitro,and the effect of inhibition of galectin-1 using a small-molecule inhibitor (OTX008).

Results: One normal oral keratinocyte (NOK) cell line and three OSCC cell lines were cultured and the expression of galectin-1 protein in each quantified using an ELISA. All cell lines were found to express galectin-1, and one of the OSCC lines produced significantly more galectin-1 than the NOK cell line at 6, 24 and 48 hours.

All four cell lines were cultured with three concentrations of galectin-1 (50, 100 and 150 ng/mL) and four concentrations of OTX008 (12.5, 25, 50 and 100 μg/mL), and cell viability was assayed at 24, 48, 72 and 96 hours. Galectin-1 decreased cell viability at 24 hours in two of the OSCC lines, had no effect on the third, and increased cell viability in the NOK cells at 72 hours. OTX008 reduced cell viability in a dose-dependent manner in all cell lines, and this effect increased at each time point during a 96 hour culture period. OTX008 had the least effect on cell viability of the OSCC line with the highest galectin-1 levels compared to the other cell lines.

Conclusions: Galectin-1 is expressed by NOK and OSCC cell lines in vitro. OTX008 decreases the cell viability of OSCC and NOK cells in a dose-dependent manner, however this effect is reduced by higher endogenous levels of galectin-1.

DOWN-REGULATED YAP INHIBITS PROLIFERATION AND MIGRATION OF ORAL SQUAMOUS CELL CARCINOMA CELLS.

PROF. JAE IL LEE, MS. JI SOO HONG. DEPT. OF ORAL PATHOLOGY, SCHOOL OF DENTISTRY, SEOUL NATIONAL UNIVERSITY

Objectives: The network including Hippo signaling controls growth, proliferation, differentiation and apoptosis of the cell and tissue which also plays crucial role in organ size control in mammals and drosophila. In this network, tumor suppressor kinases include MST and LATS while YAP and TAZ exist as oncoproteins. Above all YAP is associated with the development of early embryo and the regeneration of the skin wound as well as abnormal growth of cancers in case of over-expression. However, there have been no reports on the effect of down-regulation of YAP in oral cancer cells. And further, research is needed to evaluate the role of YAP in oral squamous cell carcinoma (OSCC) cells. In the current study, we investigated the effects of YAP down-regulation on in vitro proliferation and migration of OSCC cells.

Methods: We screened 13 OSCC cell lines expression of YAP mRNA and protein were confirmed by PCR and western blot analysis. Among them 2 OSCC cell lines (HSC2, KOSCC11), YAP was expressed high levels comparing with other OSCC cell lines. HSC2 and KOSCC11 cell lines were transfected with sh.RNA compared to sh.RNA Control-transfected cells. Also we performed single cell cloning for cell line's clonal isolation. We checked that down-regulation of YAP. And in vitro cell proliferation and migration assays were used to investigate the effect of YAP down-regulation on cell proliferation and migration.

Findings: The YAP down-regulated OSCC cells grew significantly slower than the sh.RNA Control transfected cells ($p<0.05$). Additionally, migration of sh.HSC2 and sh.KOSCC11 cells decreased significantly compared with sh. Con cells. ($p<0.05$)

Conclusions: These results suggest that down-regulation of YAP induces anti-proliferative and anti-migratory effects in OSCC, and YAP may be a useful target molecule for the treatment of OSCC.

OVEREXPRESSION OF TWIST AND REDUCED E-CADHERIN EXPRESSION ARE ASSOCIATED WITH POOR BIOLOGICAL BEHAVIOR IN LOWER LIP SQUAMOUS CELL CARCINOMA: AN IMMUNOHISTOCHEMICAL STUDY.

MS. HELLEN SANTOS[A], MR. EVERTON MORAIS[A], PROF. JEAN NUNES DOS SANTOS[B], PROF. HÉBEL CAVALCANTI GALVÃO[A], DR. LÉLIA BATISTA DE SOUZA[A], PROF. ROSEANA DE ALMEIDA FREITAS[A]. [A] FEDERAL UNIVERSITY OF RIO GRANDE DO NORTE, [B] FEDERAL UNIVERSITY OF BAHIA

Objectives: This study aimed to evaluate the immunoexpression of Twist and E-Cadherin in 59 lower lip squamous cell carcinomas (LLSCC) and to verify their relationship with clinical and histopathological parameters (tumor size, regional lymph node metastasis, clinical stage, outcome, recurrence and tumor histological grade. Possible correlations between these two proteins were also evaluated.

Findings: Higher expression of E-Cadherin was observed in LLSCCs classified in early clinical stages (stage I) ($p < 0.05$) and in cases with disease-free survival after 5 years of follow-up ($p < 0.05$). Overexpression of Twist was found in lesions classified in advanced stages (II, III and IV), with recurrence and high grade of malignancy ($p<0.05$). Significant positive correlation between nuclear immunoexpression of Twist and cytoplasmic E-Cadherin expression ($p = 0.046$) was also found. In turn, there was a significant negative correlation between cytoplasmic expression of Twist and membrane expression of E-Cadherin ($p = 0.028$).

Conclusion: The results of this study suggest the potential involvement of Twist and E-Cadherin proteins in the modulation of events related to tumor progression and the poor prognosis of LLSCC.

MAMMARY ANALOG SECRETORY CARCINOMA OF SALIVARY GLANDS:A CASE REPORT..

DR. JIA ZHANG, DR. JIAFENG DUAN, DR. HONG QI, DR. SHUWEI LI. STOMATOLOGICAL HOSPITAL OF XI'AN JIAOTONG UNIVERSITY

Objective: Mammary analog secretory carcinoma (MASC), is a distinctive low-grade malignant salivary cancer. Microscopically, most cases of MASC consist of a circumscribed mass divided by thin fibrous septa into lobules composed of microcystic, tubular, and solid structures. Due to the scarcity of reported cases, however, little information exists regarding this lesion in the salivary gland. Here, we report a case of MASC occurring in the parotid gland.

Clinical Presentation: Our patient is a 28-yer-old male who presented with a $3.0 \times 2.5 \times 2.0$cm in the right parotid gland, referred slowly growing, painless approximately one year duration. The tumor is rubbery, with a white-tan to gray cut surface. On the cut surface of the mass, many small cystic spaces may be seen, containing yellow to whitish fluid. The borders of the tumor is circumscribed but not encapsulated.

Conclusion: Most cases of MASCs were diagnosed as AciCC or adenocarcinoma not otherwise specified. Many MASC were found to harbor an ETV6-NTRK3 fusion gene because of a t(12;15) (p13,q25) translocation, a finding identical to secretory carcinoma of the breast. The most recent version of the World Health Organization (WHO) Classification of Head and Neck Tumours utilizes the terminology of "secretory carcinoma" for consistency. In addition to the case report, we review the past and current cases enrolled of MASC. Awareness of such a clinical presentation is important for the clinician.

RESEARCH ON THE RELATIONSHIP BETWEEN O-GLCNAC AND ORAL SQUAMOUS CELL CARCINOMA.

PROF. JI-AN HU, DR. YI-NING LI. DEPARTMENT OF ORAL PATHOLOGY, SCHOOL OF STOMATOLOGY, ZHEJIANG UNIVERSITY

Objectives: Research on the relationship between O-GlcNAc and oral squamous cell carcinoma by the tissue and cells.

Findings: There were significant difference of O-GlcNAc and OGT between normal mucosa and oral squamous cell carci-noma ($p<0.05$). The expression of O-GlcNAc and OGT increased with the higher grade of the carcinoma. The expression of OGA was inconsistent with O-GlcNAc and OGT. TG could activated the expression of O-GlcNAc and OGT, DON could inhibited the expression of O-GlcNAc and OGT, in addition, DON could inhibited the proliferation of TCA8113 cells and the expression of PCNA.

Conclusions: O-GlcNAc could activate the oral squamous cell carcinoma. Inhibitor DON could depress the proliferation of TCA8113.