Introduction Salivary gland tumours (SGTs) are important parts of human neoplasms. The most common SGT is the pleomorphic adenoma (PA); and the most common malignant SGTs are mucoepidermoid carcinoma (MEC) and adenoid cystic carcinoma (ADCC). TH17 cells (Th17) are characterised with the ability to secrete cytokines including IL17A, IL17F, IL9, IL21, IL22, IL23, IL26 and TNF-α. Th17 cells play an important role in inflammatory disease, infections, and cancers. This study aimed to investigate the role of Th17 in sera level of patients with SGTs.

Material and Methods The level of IL9, TNF-α, IL17A, IL17F, and IL21 were measured in the sera of 43 patients with malignant SGTs, 28 patients with benign SGTs, and 4 patients with inflammation in comparison with 25 healthy controls by using flow cytometry assays. The data were analysed by using Kruskal-Wallis and Mann-Whitney tests.

Results and discussions The findings revealed the IL17A to have increased in SGTs compared with the healthy controls (p=0.009 and p=0.002, respectively). There was no significant difference in the HSP27 serum levels between the patients with benign salivary gland tumours and healthy controls (p=0.2). No correlation was detected between the mean serum levels of HSP27 and clinicopathologic factors such as age, sex, stage, nodal metastasis (p>0.05), except for the tumour size (p=0.04).

Conclusion The HSP27 serum level was correlated with tumour size (p=0.04).

PO-386 DISSECTING THE ROLE OF REGULATORY T CELLS IN METASTATIC BREAST CANCER

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Introduction Breast cancer is the most frequent malignancy among women worldwide. More than 90% of breast cancer-related deaths are due to metastatic disease. Despite these facts, breast cancer metastasis remains incurable. A key player in metastasis is the immune system. Cancer-induced immunosuppression contributes to a tumour’s ability to evade immune destruction. A major cell type in this process is the regulatory CD4+ T cell (Treg). It has been reported that Tregs are found in metastatic disease, focusing on differences between (pre-)metastatic tissues and different steps in the metastatic cascade.

Material and methods To study primary spontaneous mammary tumours and the pre-metastatic niche, we primarily use the FVB. The conditional mouse model for invasive lobular carcinoma. For metastatic disease, KEP tumour fragments are orthotopically transplanted into wild-type syngeneic FVB mice. Following tumour outgrowth and mastectomy, widespread metastatic disease is present in lungs, lymph nodes and other distant organs, providing an accurate representation of the different steps of the metastatic cascade.
Results and discussions We observed systemically elevated levels of Tregs prior to metastatic disease. These tumor-educated Tregs display a distinct phenotype and specifically accumulate in the lung and lymph node metastases that arise in our metastasis models, perhaps indicating their possible importance for metastasis formation and progression.

In addition to these systemic changes, within primary tumours and metastases a large population of PD1high Tregs is found. Preliminary data suggest that tumor-associated myeloid cells influence this population.

Conclusion We are currently setting out to dissect the mechanism behind this interplay of intra-tumoural immune cells and the role of distinct Tregs found prior to metastatic disease.

**PO-387** ABSTRACT WITHDRAWN

**PO-388** THE GASTROINTESTINAL TRACT TUMOUR MICROENVIRONMENT DIFFERENTIALLY INFLUENCES MATURATION OF AND CYTOKINE SECRETION FROM DENDRITIC CELLS

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**Introduction** Oesophageal adenocarcinoma (OAC) and rectal adenocarcinoma are treated with neoadjuvant chemoradiotherapy in order to reduce tumour size prior to surgery however only 10%–30% of patients have a complete pathological response. Inflammatory and angiogenic mediators in the tumour microenvironment (TME) have many functions, such as enabling evasion of anti-tumour immune responses by disabling infiltrating dendritic cells (DCs) and have been linked with radioresistance. Tumour Conditioned Media (TCM) from colonic cancer has been shown to strongly inhibit DC maturation. Our aim was to understand if this DC inhibition extends to other cancers of the gastrointestinal tract, to investigate if radiotherapy influences this and to profile constituents of TCM that may influence DC maturation.

**Material and methods** TCM from 0Gy or 2Gy-irradiated cell lines or tumour biopsy explants, was used to pre-treat monocyte-derived DCs prior to stimulation with LPS to measure DC maturation based on DC cell surface markers (HLA-DR, CD86, CD54, CD80, CD83 and PD-L1) and two cytokine levels (IL12 p70 and TNF alpha). Inflammatory and angiogenic mediator multiplex ELISAs were used to profile the TCM of oesophageal and rectal adenocarcinoma.

**Results and discussions** DCs remained responsive to LPS following pre-treatment with OAC cell line TCM, whereas extensive inhibition was induced by CRC cell line TCM. *ex vivo* TCM from different gastrointestinal adenocarcinoma types induced different effects on DC maturation with oesophageal inducing DC activation, rectal inducing minor activation and colonic inducing inhibition of DC maturation markers. Interestingly, all cancer types induced DC inhibition of secreted TNF alpha. It was also found that 2Gy-irradiated TME induced significant inhibition of DC maturation for irradiated rectal adenocarcinoma and no effect with irradiated oesophageal cancer. Differential levels of inflammatory (IL2) and angiogenic mediators (Ang2 and bFGF) in TCM of GI tumours correlated with DC maturation.

**Conclusion** Overall, this study offers new evidence that there are differences in the human TME from different gastrointestinal (GI) cancers which can directly induce varying levels of inhibition of LPS-induced DC maturation markers, whilst all inhibit secreted TNF alpha.