were specifically expressed in tumor samples, but not in surrounding healthy tissue. Peptides derived from these genes could be used as a vaccine. Immune cell infiltrate populations were evaluated resulting in a major population of macrophages and T cell within the tumor. Cytokines and chemokines gene expression also correlated with several immune subpopulations. B memory cells correlates with better prognosis both in INCA and TCGA cohorts. BCR repertoire were predominantly IgG whereas IgA was in surrounding area. We also found germinative centers within the tumor by immunohistochemistry staining. TCR and BCR number of clonotypes correlated with neoantigen burden. TCR alpha and beta chains were the most abundant. Several clonotypes of TCR and BCR were shared between tumor and surrounding tissue, with very few shared between samples. By in silico molecular model we found a TCR clone that recognized a TAA derived peptide from MAGEA11. Concerning immune checkpoint blockade molecules, we tested several inhibitors and stimulatory receptors as lag3, pdcd1, pdl1 and ctla4 that were more expressed in tumor and higher lag3 expression correlated with a poor prognosis in INCA cohort.

Conclusion:
We described the complex role of several immune cell subsets, its inhibitory and stimulatory molecules in mucosa collaborating for the tumor microenvironment. Also, we found a TCR specific for a neoantigen. Altogether these results showed that each patient has its own collection of neoantigens derived from mutations, but shared peptides from TAA genes that can be proposed as a unique peptide vaccine for ESCC patients.

Keywords: Immune system; Esophageal Cancer; Neoantigen