patients (p = 0.04). Kaplan-Meier survival analysis showed that higher density of CD163+ and CD79a+ cells were independently associated with shorter recurrence free survival (RFS). Notably, these differences in RFS remained in BCG immunotherapy-naive patients (n=170).

Conclusions These findings are the first evidence of sexual dimorphism in the TIME of NMIBC and may help to partially explain the worse clinical outcomes experienced by female patients. This study also provides the first evidence of the negative prognostic impact of B cells in NMIBC. Overall, this study provides insight into more rational implementation of immune-based therapies in female NMIBC patients.

Ethics Approval This study was approved by the Ethics Review Board at Queen’s University, Kingston, ON, Canada.

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IN VITRO IMMUNOREGULATORY EFFECT FROM CERVICAL CANCER DERIVED MESENCHYMAL STROMAL CELLS OVER MOLECULES EXPRESSION IN MONOCYTE DERIVED MACROPHAGE POLARIZATION

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Background Macrophages are immunological cells that sense microenvironmental signals that may result in the polarized expression of either proinflammatory (M1) or anti-inflammatory (M2) phenotype.1 Macrophages M2 are present in tumoral microenvironment and their presence in patients with cervical cancer (CeCa) is related with less survival.2Mesenchymal Stromal Cells (MSCs) are also present in tumor microenvironment of cervical cancer (CeCa-MSC), which have shown immunoregulatory effects over CD8 T cells, decreasing their cytotoxic effect against tumoral cells.3 Interestingly, MSCs from bone marrow (BM-MSC) decrease M1 and increase M2 macrophage polarization in an in vitro coculture system.4 Macrophages and MSCs are present in microenvironment of cervical cancer, however it is unknown if MSCs play a role in macrophage polarization. In the present study, we have evaluated the immunoregulatory capacity of CeCa-MSCs to induce macrophage polarization.

Methods CD14 monocytes were isolated from peripheral blood and cultivated in the absence or presence of MSCs from BM, normal cervix (NCx) and CeCa. Two culture conditions were included, in the presence of induction medium to favors M1 (GM-CSF, LPS and IFNγ) or M2 (M-CSF, IL-4 and IL-13) macrophage polarization. M1 (HLA-DR, CD80, CD86 and IFNγ) or M2 (CD14, CD163, CD206, IDO and IL-10) macrophage molecular markers were evaluated by flow cytometry. Finally, we evaluated concentration of IL-10 and TNFa in conditioned medium form all coculture conditions.

Results We observed that CeCa-MSCs and BM-MSCs in presence of M1 induction medium, decreased M1 macrophage markers (HLA-II, CD80, CD86 and IFNγ), and increase the expression of CD14 (M2 macrophage marker). Interestingly, in presence of M2 induction medium, BM-MSCs and CeCa-MSCs but not NCx-MSC increased CD163, CD206, IDO and IL-10 (M2 macrophage markers). We observed a decreased concentration of TNFa in the supernatant medium from all cocultures with MSCs, but only in presence of CeCa-MSCs, increased IL-10 concentration was detected in such cocultures.

Conclusions In contrast to NCx-MSCs, CeCa-MSCs similarly to BM-MSCs have in vitro capacity to decrease M1 and increase M2 macrophage phenotype.

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NEOADJUVANT CHEMORADIOThERAPY ENHANCES T CELL INFILTRATION IN PAnCREATIC DuctAL ADENOCARCINOMA BUT HIGH PERCENTAGE OF REGULATORY T CELLS ASSOCIATES WITH POOR SURVIVAL

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Background Currently, diagnosis with pancreatic ductal adenocarcinoma (PDAC) renders an almost intrinsically poor patient prognosis. Despite complete surgical resection and intense neoadjuvant and/or adjuvant treatment the great majority of patients will ultimately relapse and die from the disease. Further, PDAC has been characterized as highly immune resistant. It is speculated that radiation, chemotherapy, or chemoradiation cause the release of tumor antigens and inflammatory cytokines eventually leading to increased immunogenicity of PDAC.

Methods We used computational quantitative multiplex immune fluorescence (qmIF) (n=31) and the NanoString assay (n=34) to quantitatively analyze the effect of neoadjuvant