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Phenotypic characterization of macrophages subpopulations CD3+ TCRαβ+/TCRαβ-

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Background: It was reported that 5-15% of macrophages isolated from peripheral blood mononuclear cells (PBMC) express the TCRαβ receptor. Using monocyte derived macrophages (MDM), from healthy donors, and infected *in vitro* with BCG, it was observed that the percentage of TCR+ macrophages increased in response to the infection. Preliminary results from our group, in mice, showed that after an intravenous BCG-infection there is an increase in the recruitment of two sub-populations of macrophages: CD11b+CD3+TCRαβ- and CD11b+CD3+TCRαβ+, at moment the characterization of these subpopulations has not been assessed.

Methodology: PBMC were obtained from healthy donors, CD14+ cells were obtained through immunomagnetic positive selection. After 7 days in culture MDM were obtained and characterized by flow cytometry. The MDM CD3+ TCRαβ+ and TCRαβ- subpopulations were evaluated for the coexpression of: CD80, CD86, CD11B, CD68, CD14, CD3, TCRαβ, TCRγδ, HLA-I, HLA-II, CD1a, CD1b, CD1c, CD1d, CCR4, CCR7, CXCR1, CD16, and tmTNF.

Findings: Our data shows that both of these sub-populations express efficiently the HLA-I, HLA-II. Moreover, the subpopulation CD3+TCRαβ+ showed an increase in the expression of CD1a, CD1b, CD1c and CD1d molecules, however in CD3+TCRαβ- this expression was absent. The expression of pro-inflammatory molecules CD16 and tmTNF had a stronger augment in the CD3+TCRαβ+ subpopulation. Chemokine receptors were measured and CCR4, CCR7 and CXCR1 were expressed 3 times more in CD3+TCRαβ+ compared to CD3+TCRαβ-.

Conclusion: The CD3+TCRαβ+ MDM are efficient cells to present peptide antigens; however the expression of CD1 family molecules suggests that CD3+TCRαβ+ could play an important role in the presentation of lipid antigens. Probably, overexpression of pro-inflammatory molecules and chemokines receptors on CD3+TCRαβ+ is used by the MDM to favor a pro-inflammatory function. The phenotypic characterization of CD3+TCRαβ+ provides evidence that this subpopulation could be crucial in pathologies where lipids and inflammatory environment trigger a signal, such as tuberculosis.

Biography

Adriana Rodríguez Cruz burned in Mexico, 1989. Awarded bachelor's degree in Biology with honors by Universidad Nacional Autónoma de México (UNAM), 2012. Education abroad in 2010 at University of California Berkeley, U.S.A. Research internships in 2013 at University of Arizona, U.S.A., and in 2014 at Charité Universitäts Medizin Berlin, Germany. Currently studying PhD in Biomedical Sciences at UNAM with the project "Evaluation of the signaling pathway of macrophages CD3+ TCRαβ+/TCRαβ- and their function as pro-inflammatory macrophages"; to identify the mechanisms of activation and cellular function of these macrophages, as well as their implication in the physiopathology of pulmonary tuberculosis.

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