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# Regulation by the Transcription Factor STAT3 in the immunophenotype modulation monocyte-macrophage from M1 to M2 generate by culture supernatant of prostate cancer cells

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We investigated the regulation of STAT3 in the immunophenotype modulation macrophage from M1 to M2 generate by cell culture supernatant of prostate cancer line PC3.

Supernatant PC3 and macrophages were collected, and cytokines analyzed nitric oxide and cytotoxic tests. Macrophages were evaluated by STAT3 and membrane proteins by flow cytometry. Macrophage cultures were exposed to supernatant PC3 line, with and without Sttatic inhibitor.

Epidermal growth factor, fibroblast, granulocyte-monocyte colony, hepatocyte, platelet derivative A and B, cell stimulator, beta transformant, increased p <0.005 in the supernatant obtained from cultures of the macrophages exposed to the supernatant of the Line prostate cancer PC3 without inhibitor, like angiopoietin and erythropoietin. Cytokines IL-6, IL-4, IL-10 increased. IFN-γ and TNF-α decreased to undetectable values. Lactic acid increased 5 more times in both lines; nitric acid decreased p <0.005. Phosphorylated STAT3 increased 3-fold when macrophages were stimulated with PC3 supernatant, but not the total which maintained baseline values.

Cytokines present in the supernatant of the PC3 tumor line favor a change in the immunophenotype of macrophages to M2. Tumor-associated macrophages have been shown to play a key role in proliferation, progression, angiogenesis and metastasis. The results shown so far point to some points through which M2 may be constituted as therapeutic targets such as inhibition of STAT3 phosphorylation and suggest that the decrease in levels of these M2 or the reversion to an M1 phenotype can lead to in a decrease in tumor growth and spread.

## Article Information

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