

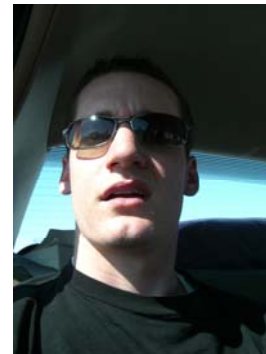
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QUANTIFICATION OF INTERLEUKIN 23 IN HUMAN PROSTATE CANCER

Many types of human epithelial cancers arise in a background of chronic inflammation. Infiltrating inflammatory cells may damage epithelium leading to chronic reparative cell turnover, release of oxygen free radicals causing DNA damage, degrade basement membranes, and trigger microvessel formation that may enable cancer cells to enter the bloodstream during metastasis. Using RNA expression microarray analysis, we have found that interleukin 23a (IL-23) expression correlates with poor outcome following prostatectomy in prostate cancer patients. IL23 promotes the development of T-helper 17 (Th17) cells, a recently identified subset of Th cells that appears to mediate chronic inflammation. We hypothesize that IL23 drives chronic inflammation in prostate cancer leading to metastatic disease. Using a cohort of prostate tumor samples from patients with variable clinical outcome, we are addressing the hypotheses that (a) the degree of chronic inflammation is correlated with poor prognosis in prostate cancer, and that (b) the number of IL23-expressing cells correlates with both the degree of chronic inflammation and poor prognosis. We are determining the amount of chronic inflammatory infiltrate present in the prostate tumor samples. And we are using PCR methods to clone a portion of the *IL23* gene into a vector suitable for *in vitro* synthesis of radiolabeled RNA probes for *in situ* hybridization studies to quantify IL23-expressing cells in prostate cancer specimens. Results of our study may provide insight into the role of chronic inflammation in prostate cancer biology, and may indicate a potential use for anti-inflammatory drugs being developed to block IL-23 function.



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