sion varies depending on the histological type of NSCLC. Identification of tumor immunophenotype makes possible to obtain more data about metastasis potential, malignancy rate and prognosis. This molecular markers panel can be used for early detection of LC recurring and metastasis.

doi:10.1016/j.ejcsup.2006.04.114

P55. IN SITU MOLECULAR ASSESSMENT OF CD8+ T CELL REACTIVITY IN NSCLC

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Background: While in cancer from colon and ovary number and reactivity of tumor infiltrating lymphocytes (TILs) were associated with survival, in NSCLC, the clinical significance of TILs is unknown. While TILs demonstrated antigen specific Interferon (IFN)-γ secretion and cytotoxicity in vitro, their in situ activation status remains difficult to determine. Here, we examine molecular assessment of CD8+ T cell reactivity in NSCLC.
Methods: We determined CD8+ T cell counts in 19 patients with NSCLC by immunohisto-chemistry and assessed T-cell immune reactivity by measuring mRNA IFN-γ levels by quantitative RT-PCR (TaqMan) from corresponding tissue. CD8+ T cells were classified into two groups: Intra-tumoral (distributed in cancer cell nests and stroma) and peri-tumoral (presented along the invasive margin). Five hpf were evaluated per each localization (IT and PT, respectively). Distribution of CD8+ T cells was semi-quantitatively classified into: 0-none or mild; I-moderate; II-severe. The relationship between the median CD8+ T cell density/hpf and the CD8-normalized IFN-γ mRNA expression was tested (Spearman's correlation).
Results: Semi-quantitative analysis revealed significantly higher CD8+ T cell counts within the tumor compared to the invasive margin ($p < 0.001$). However, immune activation status of TILs represented as IFN-γ/CD8 ratio was higher in the peri-tumoral than in the intra-tumoral compartments ($p = .022$).
Conclusion: In human NSCLC, IFN-γ reactivity of CD8+ T cells is mostly attributed to the tumor–host interface and indicates an inadequate activation of TILs within the tumor. This methodology can be applied to a variety of experimental trials if tumor tissue is available.

doi:10.1016/j.ejcsup.2006.04.115

P56. UPREGULATION OF COX-2 EXPRESSION OCCURS ALREADY IN GASTROESOPHAGEAL REFLUX DISEASE BUT IS FURTHER INCREASED IN BARRETT'S ESOPHAGUS AND BARRETT'S CANCER

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Background: Cyclooxygenase-2 (COX-2) mRNA expression is known to be progressively increased in the metaplasia–dysplasia–adenocarcinoma (MDA) sequence of Barrett's cancer (BC) development. Much less however, is known about COX-2 mRNA expression in patients with gastroesophageal reflux disease (GERD).
Methods: Endoscopic biopsies from 3 patient groups were analyzed: 43 patients undergoing evaluation for GERD (20 GERD positive/23 GERD negative) by 24-h pH-monitoring, 20 patients with Barrett's esophagus (BE) without dysplasia and 47 with BC. COX-2 mRNA expression was determined by quantitative real-time RT-PCR (TaqMan™) assays. The Demeester composite score (DS > 14.72) was used to match COX-2 mRNA expression levels with the degree of acid exposure.
Results: Median COX-2 mRNA expression was significantly upregulated in Barrett's metaplastic epithelium compared to matched normal squamous epithelium (NE) in the BE ($p = 0.03$) and BC group ($p = 0.001$). COX-2 mRNA expression levels in NE did not differ significantly between the 3 study groups however, within the group of patients evaluated for GERD, specimens obtained from patients with a mean Deemester score >14.72 showed significantly upregulated COX-2 mRNA levels in the distal acid-exposed esophagus ($p = 0.01$).
Conclusion: Our findings suggest that the induction of increased COX-2 mRNA expression occurs already in GERD without the presence of Barrett's metaplasia. A field effect as shown for other genes could not be detected for COX-2 expression in squamous epithelium in GERD positive, BE or BC patient groups. Chemoprevention strategies using selective or non-selective COX-2 inhibitors might be useful in patients with GERD to potentially prevent the development of Barrett's metaplasia.

doi:10.1016/j.ejcsup.2006.04.116

P57. ASSOCIATION OF CYCLOOXYGENASE-2 EXPRESSION WITH DEVELOPMENT AND PROGRESSION OF BARRETT'S METAPLASIA–DYSPLASIA–CARCINOMA SEQUENCE AND THE ENVIRONMENTAL INFLAMMATORY REACTION

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Background: Epidemiological data assume a reduction of risk for developing an adenocarcinoma of the esophagus for individuals taking non-steroidal anti-inflammatory drugs. One of the inhibited enzymes, cyclooxygenase-2, is supposed to be involved in the pathogenesis of Barrett's cancer. We examined a possible association between COX-2 protein expression and the progression of Barrett's sequence and the type and degree of the environmental inflammatory reaction.
Methods: Squamous epithelium, metaplastic, low-grade and high-grade dysplastic lesions and tumor tissue of 49 resection specimens from patients with Barrett's adenocarcinoma were analyzed. Immunohistochemical staining was performed with a