FEV1. Percentages of CD3+, CD3+4+, CD3+8+, CD3+45RA+, CD4+8+, CD4+45RA+, and CD8+45RA+ cells were determined in peripheral blood by flow cytometry, before and 48 hours after the aspirin challenge. The results showed a significant decrease of CD3+45RA+ ($z=2.60$, $p=0.009$), CD4+45RA+ ($z=3.74$, $p=0.000$), and CD8+45RA+ ($z=2.47$, $p=0.014$) cells 48 hours after aspirin challenge. Similar results were obtained when ASA, ATA, NP, and HC groups were analyzed separately. No differences were found between the groups. The reduction of peripheral blood CD45RA+ cells in all patients as well as in healthy subjects indicates their sensitivity to aspirin, apart from their role in pathophysiology of allergic inflammation.

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F.9. Comparison of Efficacy of Salbutamol with Theophylline for Wheezy Adult Patients

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Objective: The aim of present study was to determine the comparison of efficacy and tolerability of salbutamol with theophylline in treating wheezy adult patients. Design: It was a parallel group study. Setting and Subjects: Wheezy adult patients admitted in internal medicine department from Jan 2007 - May 2007. Results: One hundred patients undergone in this study. Fifty were in salbutamol group and 50 were in theophylline group. Clinical problem at the beginning was recurrent wheeze (48%), wheeze with fever (35%) and spasmodic cough (20%). Salbutamol was more palatable ($P<0.001$) as compared to theophylline.The efficacy of both the drugs were nearly equal ($P>0.1$). Adverse effects were more frequent in theophylline treated patients (48%) than in salbutamol group (26%). Conclusion: Salbutamol is better tolerated drug than theophylline. Both the drugs were equally effective for bronchodilation in adult patients.

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F.10. HLA Inheritance and Asthma in HIV-infected Children

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Although HIV-infected children on highly active antiretroviral therapy (HAART) appear to be at increased risk for developing asthma compared to children not on HAART, genetic factors may also influence the development of asthma and other immunologic diseases. We propose that asthma in HIV-infected children may be associated with HLA alleles. We reviewed HLA haplotype data collected as part of the Women and Infants Transmission Study (WITS) for 124 HIV-infected children and their mothers. Associations of two-digit HLA Class I A, B, C and Class II DRB1 alleles with asthma medication use were examined using Fisher's Exact test. Candidate alleles and HAART medication use were then subjected to multivariate analysis using the Cox model to examine the independent associations of HLA haplotype and HAART treatment with asthma medication use. Preliminary analyses indicated an association of HLA A68 ($p=0.01$), B13 ($p=0.09$), B39 ($p=0.09$), and Cw6 ($p=0.06$) with asthma medication use. HAART therapy was also associated with asthma medication use ($p=0.04$). Multivariate analysis revealed that A68, B13, and HAART therapy were all independent, significant predictors for asthma ($p=0.007$, 0.03, and 0.04, respectively). Specific HLA alleles appear to modulate the risk of developing asthma, functioning independently of the specific actions of HAART therapy. Intriguingly, three of the alleles, B13, Cw6 and B39, are each associated with susceptibility to psoriasis and/or psoriatic arthritis, raising the question of whether the interactions of certain class I molecules that occur in psoriasis might predispose to asthma.

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F.11. Oxidative Constituents in Cigarette Smoke Extract Induce Macrophage Heme-oxygenase-1 Protein

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Heme-oxygenases [HO] catalyze the rate-limiting step in the oxidative degradation of heme to biliverdin, which is then converted to bilirubin, carbon monoxide, and iron. HO-1 is a 32 kD inducible isoform of the HO system that plays an important cytoprotective role during cellular stress. Macrophages play a key role in the pathogenesis of smoking-induced diseases. Although it has been demonstrated that macrophage HO-1 levels are induced in cigarette smokers and in patients with chronic obstructive pulmonary disease, the relative role of oxidative stress and nicotinic stimulation in the induction of HO-1 are not well-defined. To determine this, murine RAW 264.7 macrophages were exposed to freshly generated cigarette smoke extract [CSE] prepared from Kentucky 1RF4 research cigarettes or reportedly "reduced harm" Eclipse cigarettes. Following 6 hours of incubation with CSE, whole cell lysates were prepared and cellular HO-1 levels were determined by immunoblotting. Both CSE preparations induced substantial augmentation in macrophage HO-1 protein levels. Interestingly, augmentation of cellular HO-1 by CSE generated from "reduced harm" Eclipse cigarettes was even higher than that observed with research cigarettes. Nicotine [10-1000 ng/ml] failed to induce macrophage HO-1 protein. However, pre-incubation of macrophages with the anti-oxidant n-acetyl-cysteine completely abrogated the induction of HO-1 by either CSE preparations, suggesting oxidative stress as a primary mechanism for the observed augmentation of macrophage HO-1 levels by CSE. These data imply that soluble oxidative constituents in cigarette smoke induces HO-1 accumulation in murine macrophages, and that presumed "reduced harm" Eclipse