Background: Aspirin exacerbated respiratory disease (AERD) is a syndrome characterized by chronic hyperplastic rhinosinusitis, nasal polyposis, asthma and aspirin sensitivity. The mechanisms by which produce these manifestations of intolerance are not fully defined, the current research involve alterations in the metabolism of arachidonic acid, cyclooxygenase 1 (COX-1) inhibition and its deviation from substrate to the lipooxygenase (LO) pathway, inducing increased synthesis of leukotrienes (LT). Biological plausibility of this fact has led to the search for polymorphisms in genes responsible for LT synthesis however others factors such as genetics polymorphisms in pro-inflammatory cytokines like, IL1B and IL8 could be associated.

Methods: 78 patients with AERD, 135 aspirin-tolerant asthma (ATA) and 134 healthy control subjects participated. All participants who underwent a simple spirometry, methacholine challenge and nasal challenge with Lysine-aspirin (L-ASA), both tests performed according to international guidelines. Peripheral blood was drawn by venipuncture, genomic DNA was obtained using the commercial BIOTRACK DNA isolation kit. We selected 2 polymorphisms in 2 genes related to chronic inflammation rs16944 in IL1B, and rs4073 in IL8, Allelic discrimination of SNPs was performed by Real Time PCR (PCR-RT) on a 7300 Real Time PCR Systems. Statistical analysis was performed between groups of cases (AERD and ATA) versus control group with Epi-info v.6.04 by χ2 test to identify the difference between the allele and genotype frequencies of each polymorphism made, considering a significant P value <0.05, in addition to the calculation of odds ratios and confidence intervals of 95%.

Results: We find no association between IL1B (rs16944) to GG and GA genotypes in ATA patients versus control group neither AERD versus control group. Interestingly, the AA genotype showed increased frequency in the AERD patients versus the ATA patients (FG = 0.19 versus 0.07), this association remained significant (P = 0.018, OR 2.98, CI, 1.17-7.82)

Conclusions: This is the first observation that IL1B polymorphisms are involved in AERD, suggest that patients carrying out the IL1B-511 polymorphism (rs16944 AA genotype) may show enhanced susceptibility to develop AERD.

Fatty Acid Binding Protein 1 is Related with Development of Aspirin-Exacerbated Respiratory Disease
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Background: Aspirin-exacerbated respiratory disease (AERD) refers to the development of bronchoconstriction in asthmatics following the ingestion of aspirin. Although alterations in eicosanoid metabolites play a role in AERD, other immune or inflammatory mechanisms may be involved. We aimed to identify proteins that were differentially expressed in nasal polyps between patients with AERD and aspirin-tolerant asthma (ATA).

Methods: Two-dimensional electrophoresis was adopted for differential display proteomics. Proteins were identified by liquid chromatography-tandem mass spectrometry (LC-MS). Western blotting and immunohistochemical staining were performed to compare the amount of fatty acid-binding protein 1 (FABP1) in the nasal polyps of patients with AERD and ATA.

Results: Fifteen proteins were significantly up-(7 spots) or down-regulated in the nasal polyps of patients with AERD (n = 5) compared to those with ATA (n = 8). LC-MS revealed an increase in 7 proteins expression and a decrease in 8 proteins expression in patients with AERD compared to those with ATA (P = 0.003-0.045). FABP1-expression based on immunoblotting and immunohistochemical analysis was significantly higher in the nasal polyps of patients with AERD compared to that in patients with ATA. FABP1 was observed in epithelial, eosinophils, macrophages, and the smooth-muscle cells of blood vessels in the polyps.

Conclusions: Our results indicate that alterations in 15 proteins, including FABP1, may be related to the development of AERD.

The Correlation of Cholesterol Lowering Statin Drugs and Worsening Asthma Control in Mild Persistent Asthmatics
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Background: To show that pharmacological agents patient use, adversely alter the immunomodulatory activities that promise the worsening of the clinical course of allergic diseases such as asthma.

Methods: Two groups of 20 asthmatics patients each were compared from baseline values. Twenty patients with extrinsic asthma (group A) were prescribed statins for their lowering of their cholesterol necessity and 20 patients (group B) were controls who did not receive statins. Group A and group B were designed to compare FEV1, exacerbation asthma rates, beta agonists use, nocturnal awakenings, and daytime symptoms from baseline values.

Results: Statins treated asthmatic patients group A had significant worsening of FEV1 at 3 months, 6 months and 12 months, to almost no change in control asthmatic patients group B. Statins treatment patients group A were associated with more frequent use of rescue medication (albuterol inhaler), increased nocturnal awakenings, and increased daytime asthma symptoms, compared to group B.

Conclusions: Statin drugs may worsen asthma control in mild persistent asthmatics. Statins may cause possible immune alteration that promotes allergic diseases such as asthma.

Studies on the Relationship between Airway Inflammatory Responses in Patients With Asthma or Not-yet Onset Asthma and Air Pollution
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Background: Substantial data have demonstrated that air pollution is associated with cardiopulmonary mortality and morbidity in the world. Among a variety of pollutants, particulate components, particularly PM2.5, are especially suggested to be harmful to our lung health. Diesel exhaust particles (DEPs) are the major component of PM2.5, and therefore the relationship between PM2.5 or PM10 and airway inflammatory responses of asthmatic and people of not-yet asthma onset is important to be investigated. Recent findings suggested that susceptibility to DEPs is dependent upon certain genetic variations of anti-oxidative stress enzymes such as GSTP1, which is largely regulated by a transcription factor Nr2. By preliminary experiments, we found that exhaled breath condensates (EBC) are safely and repeatedly obtained from both disease and health persons, and that several biomarkers including growth factors, cytokines and oxidant stress markers could be measured.

Methods: In the present study, we attempted to study the airway inflammatory/fibrogenic responses from patients with asthma, and further, those from people who have suggestive, but not yet definite symptoms of asthma. Participants are asked to present exhaled breath condensates (EBC) by