antigen (LTRA) was 55.3%. Difference between initial steroid and/or LTRA contents and non-contents were age (60 vs 68.4 years old), positive BDT initially (31% vs 0%), history of asthma (52.4% vs 17.7%), history of allergic rhinitis (66.7% vs 35.3%), and elevated immunoglobulin E (IgE) and/or phadiotop (22% vs 0%) (all P<0.05). 26.5% patients in the group without initial steroid and/or LTRA did not have specific features of asthma or hypersensitivity airway diseases.

Conclusions: In the treatment of obstructive lung diseases, proper regimen can be initially given by careful evaluation. It is still difficult to make precise diagnosis initially in some patients. Closely following up and adjusting regimen are needed.

AP1246
ISOFлавONE AGLYCONES ATTENUATE CIGARETTE SMOKE-INDUCED EMPHYSEMA VIA SUPPRESSION OF NEUTROPHILIC INFLAMMATION IN A COPD MURINE MODEL
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Background and Aims: Chronic obstructive pulmonary disease (COPD), a lung disease caused by chronic exposure to cigarette smoke, increases the number of inflammatory cells such as macrophages and neutrophils and emphysema. Isoflavone is a polyphenolic compound that exists in soybeans. Daizein and genistein, two types of isoflavones, have been reported to have anti-inflammatory effects in various organs. We hypothesized that the daizein-rich soy isoflavone aglycones (DRIAs) attenuate cigarette smoke-induced emphysema in mice.

Methods: Mice were divided into four groups: the (i) control group that consisted of non-smoking mice on a normal diet (MF diet), (ii) isoflavone group that consisted of non-smoking mice on a MF diet containing 0.6% DRIAs including daizein, genistein, and glycitein, (iii) smoking group that consisted of smoking mice on a MF diet, and (iv) isoflavone+smoking group that consisted of smoking mice on a MF diet containing 0.6% DRIAs. The number of inflammatory cells in the bronchoalveolar lavage fluid (BALF) and emphysema levels using the mean linear intercept (MLI) were determined, 12 weeks after smoking exposure. Expressions of neutrophilic inflammatory cytokines and chemokines were also examined.

Results and Conclusions: In the isoflavone+smoking group, the number of neutrophils in BALF and MLI were significantly decreased than those in the smoking group. Furthermore, the gene-expressions of TNF-α and CXCL2 (MIP-2) in the isoflavone+smoking group were significantly decreased than those in the smoking group. Supplementation of the COPD murine model with DRIAs significantly attenuates pathological changes of COPD via suppression of neutrophilic inflammation.

AP1247
LNCrNA NEAT1 REGULATES IL-6 AND IL-8 MRNA EXPRESSION IN LUNG EPITHELIAL CELLS STIMULATED BY CIGARETTE SMOKE
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Introduction: Chronic obstructive pulmonary disease (COPD) is characterized by airflow limitation and abnormal inflammatory response of the lung. The most important risk factor of COPD is cigarette smoke. Lung epithelial cells exposed to cigarette smoke extract (CSE) causes oxidative stress and increases pro-inflammatory cytokines associated to disease progression.

Recently, long non-coding RNAs (IncRNAs) have emerged being important in controlling numerous biological and pathological processes, and many are abnormally expressed in human disease. Nuclear para-spectacle assembly transcript 1 (NEAT1) is an lncRNA that promotes the expression of IL-8 by stimulating with LPS, but nothing is known about NEAT1 in COPD. We focused oxidative stress and pro-inflammatory cytokines and hypothesized that macrodilids suppressed oxidative stress by CSE and NEAT1 expression was increased in epithelial cells stimulated by CSE, and therefore drove the production of inflammatory cytokines in COPD patients.

Methods: We employed BEAS-2B derived from human epithelial cells. The expression of IL-6, IL-8, and NEAT1 RNA were measured by qRT-PCR in BEAS-2B with and without 10% CSE. To confirm the validation of NEAT1, we transfected siRNA against NEAT1 and analyzed the expression of IL-6 and 8 by qRT-PCR and ELISA. We added azithromycin (AZM, 10ug/ml) and clarithromycin (CAM, 10ug/ml) then stimulated by 10% CSE in BEAS-2B.

Results: CSE increased IL-6 and IL-8 mRNA expression, and NEAT1 was also highly expressed. Silencing of NEAT1 significantly inhibited the expression of IL-6, -8 mRNA in BEAS-2B with CSE. CAM increased in Nr2 mRNA and protein in BEAS-2B stimulated by CSE.

Conclusion: Our study showed the IncRNA NEAT1 was highly expressed in BEAS-2B stimulated with CSE. Our results also indicated NEAT1 regulated IL-6, -8 mRNA expression. CAM also suppressed oxidative stress by CSE. Understanding how NEAT1 regulates IL-6 and IL-8 production and promotes antioxidant through Nr2 may elucidate mechanisms for the pathology of COPD.

AP1253
CORRELATION BETWEEN WOOD DUST LEVELS WITH INTERLEUKIN-8 IN BLOOD SERUM AMONG WOOD PROCESSING WORKERS AT X COMPANY, TANJUNG MORAWA-NORTH SUMATERA
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Background: Duration of exposure, number and size of wood dust in the respiratory tract of wood industry workers are associated with various pulmonary disorders such as, COPD, occupational asthma, allergic rhinitis, decreased pulmonary function and pulmonary fibrosis. Wood dust exposure can stimulate proinflammatory cytokines and increase IL-8 levels, which is a foreign body in the body will be phagocytosed by macrophages and excrete mediators such as chemokines, cytokines, and chemotactrats which can cause inflammation.

Aims: To determine correlation between wood dust levels with interleukin-8 in blood serum among wood processing workers at x company, Tanjung Morawa-North Sumatera.

Methods: A cross-sectional study which involved 40 workers from 3 different locations namely cutting, sanding and drafting. Cumulative wood dust exposure was calculated by measuring the dust content at the