Conclusions: These findings demonstrate that OTC and LA can inhibit activation of NF-kB, Nrf2, and HIF and thus attenuate allergen-induced airway remodeling, suggesting that antioxidants may provide therapeutic benefit in chronic asthma and other airway disorders.

We also showed the expression of procaspase-9 with the mAb was diminished compared with that of the control and of IL-5.

Conclusions: This study showed CD30 activation enhances the eosinophil apoptosis and the effect is mediated by Caspase-9 activation.

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CCL3L1 Protein Did Not Affect IL-6 Expression, but Significantly Up-regulated IL-10 Expression in the Allergic Response
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Background: Previously, we found that the mean copy number of CCL3L1 in patients with asthma was significantly lower than that of control subjects (3.13 vs 3.75, P = 0.001). We investigated its possible molecular mechanism using a human monocytic cell line stimulated with house dust mite extract.

Method: The THP-1 human monocytic cells were stimulated with various concentrations of HDM extract. After stimulation, assay-on-demand gene expression products (Applied Biosystems) were used to evaluate mRNA expression of CCL3L1 (Hs 00609691_ml), IL-6 (Hs00174131_ml), and IL-10 (Hs00961622_ml) levels as measurement of mRNA levels by real-time PCR.

Results: Treatment of THP-1 cells with various concentration of HDM extract induced marked up-regulation of the expression of cytokines IL-10 and IL-6, which indicated that allergic responses were efficiently induced. Recombinant CCL3L1 protein had no effect on cytokine expression of THP-1 Cells in absence of HDM extract stimulation. In the presence of HDM extract (10 ug/mL) stimulation, CCL3L1 protein significantly up-regulated IL-10 expression (Ratio to ng/mL CCL3L1) dose-dependently (0 ug/mL CCL3L1 + 0.3,12.4, 10 ug/mL CCL3L1: 15.8 ± 1.1, 50 ug/mL CCL3L1: 16.8 ± 0.3, 100 ug/mL CCL3L1: 18.0 ± 0.8, P > 0.05), but did not affect IL-6 expression (P > 0.05).

Conclusion: The significantly elevated asthma risk in subjects with a low copy number of the CCL3L1 gene which may be down-regulating IL-10 expression, not IL-6 expression.

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Caspase-9 is Involved in CD30 Activation Induced Eosinophil Apoptosis
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Background: We evaluated whether ligation of CD30 incite the apoptosis, and investigated the mechanisms of CD30 induced eosinophil apoptosis is dependent on caspase activation.

Methods: We purified eosinophils using MACS system. Expression of CD30 on eosinophils were measured and eosinophils were cultured in the wells pretreated with anti-CD30 mAb and isotype control IgG1, IL-5 and dexamethasone in RPMI 1640 media supplemented with 10% FBS, and the apoptotic rate were measured using flow cytometry. To evaluate whether caspase-9 is involved in CD30- induced eosinophil apoptosis, the apoptotic rate was evaluated with addition of caspase-9 inhibitor and the expression of procaspase-9 was also measured using Western blot.

Results: The apoptotic rates of eosinophils cultured in the presence of anti-CD30 mAb were significantly increased to 29.1 ± 6.1% and 47.3 ± 4.7% compared with 17.1 ± 6.7% and 29.4 ± 9.2% of the control at 4 and 24 hours, respectively (P ≤ 0.05). Caspase-9 inhibitor suppressed the mAb induced eosinophil apoptosis from 54.8 ± 6.9% and 71.5 ± 11.6% to 24.5 ± 6.0% and 47.8 ± 11.4% at 18 and 36 hours, respectively (both P ≤ 0.001).

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Role of NLR (Nucleotide Oligomerization Domain (NOD)—like Receptor) on Allergic Inflammation in a Mouse Model of Allergic Rhinitis
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Background: Recently, a new set of pattern-recognition receptors, the nucleotidebinding oligomerization domain (Nod)-like receptors (NLRs), have emerged. Their activation, either by allergens or microbes, triggers an inflammatory response. Objective: To investigate whether recognition of bacterial microbial-associated molecular patterns in the nose may result in susceptibility to developing allergic reactions, and to understand the molecular mechanisms by which such triggers block natural tolerance.

Methods: Ligands of intracellular microbial-associated molecular pattern recognition receptors—the nucleotidebinding oligomerization domain (Nod)-like receptors, Nod1 and Nod2—were given intranasally with antigen, and their ability to modulate airway tolerance was analyzed. Seventy 2 mice were randomized to one of 6 groups: control (n = 12), AR (n = 12), pre NOD1 group (n = 12), pre NOD2 group (n = 12), post NOD1 group (n = 12), and post NOD2 group (n = 12). All mice except for the control group were sensitized by an intraperitoneal injection of ovalbumine (OVA) and aluminum hydroxide. Two weeks after sensitization, all sensitized mice were challenged intranasally with OVA. The control group was received phosphate buffered saline intranasally. The allergic symptom after the final challenge was recorded. Interleukin (IL) -5, interferon-γ (IFN-γ), and IL-10 levels in nasal lavage fluid (NALF), as well as serum OVA-specific IgE levels were measured. The number of eosinophils in lamina propria was evaluated. The levels of T-bet, GATA-3, and Foxp3 mRNA expression in splenic mononuclear cells were determined by real-time polymerase chain reaction.

Results & Conclusion: We show that a Nod-like receptor is a novel, previously unrecognized, pathway that adversely links innate and adaptive immunity and leads to allergic rhinitis.