



## The Anti-IgE Therapy: From the Known and the Unknown

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Asthma is described as a complex disease arising from the contribution of multiple genetic and environmental factors [1,2]. Asthma is caused by multiple interacting genes, some having a protective effect and others contributing to the disease pathogenesis, with each gene having its own tendency to be influenced by the environment. At the end of 2010, 100 genes including IL-1R1, IRN, 3, 4, 5, 8RA, 9, 10, 12, 13, CTLA-4, and ADAM33, among others had been associated with asthma in six or more separate populations [3]. We have no idea about the effect of omalizumab on most of these genes. In our previous study we described that omalizumab reduces systemic inflammation such as oxidative stress markers and circulating apoptotic ligands. In an analysis of 8793 genes, sensitization of mast cells with monoclonal IgE alone, was found to upregulate 58 genes more than 2-fold compared with their levels in unsensitized mast cells. These genes included those for cytokines, and colony-stimulating factor 1; chemokines; and cytokine and chemokine receptors [4]. The mechanism of action of Omalizumab in the treatment of asthma or urticaria is believed to be multifactorial, and includes effects mediated through altered production of redox metabolites, and regulation of production of known inflammatory proteins.

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### References

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