Sources of actions and efficacy of antiallergic drugs

Bernard Panaszek, Jerzy Liebhart

Department of Internal Medicine and Allergology, Wroclaw Medical University, Traugutta 57/59, PL 50-417 Wroclaw, Poland

Correspondence: Bernard Panaszek, e-mail: panaszek@akergolam.wroc.pl

Abstract:
Actions of antiallergic drugs depend on their ability to inhibit a particular stage of allergic reaction and to control symptoms of allergic inflammation. Drugs effective in early stage of allergic reaction prevent allergic cascade by acting on dendritic cells, by means of DNA-based therapies resulting both in down-regulation of Th2 cytokine profile release and immunoglobulin E (IgE) production, or by blocking IgE mediated activation of mastocytes and basophiles using monoclonal anti-IgE antibodies. Adhesion molecules and cytokines are also a potential target for therapeutic intervention in allergy. The relationship between drug concentration and pharmacologic response or effect is the essence of clinical pharmacology and the major mechanism of this relationships is that drugs or chemicals bind to or interact with macromolecules on cell surfaces (receptors) or in the cytoplasm to produce the effect. After receptor binding, the drug can activate and/or intensify a normal physiologic function and is termed an agonist, such as β2-receptor agonists and corticosteroids or can inhibit any intrinsic activity by competing for endogenous regulatory substances at the receptor and are called antagonists, such as antihistamines and anticholinergics. Through molecular biology techniques, most of the receptors for β2-agonists, corticosteroids, H1 antihistamines, anticholinergics, antileukotrienes (montelukast, pranlukast, zafirlukast) as well as enzyme inhibitors (methyIxanthines, zileutron), which are of interest for asthma and allergy treatment, have been identified and described, and much is still being learned about their molecular activity and interactions.

Key words:
allergic inflammation, DNA-based vaccines, monoclonal anti-IgE, adhesion molecules, cytokines, receptor agonists, receptor antagonists, enzyme inhibitors