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LETTER TO THE EDITOR

Histamine and indirect bronchoprovocation with adenosine monophosphate in asthma

Bucchioni and colleagues[1] examined the concentrations of cysteinyl-leukotrienes and histamine in exhaled breath condensate following bronchoprovocation with methacholine and adenosine monophosphate in healthy controls and asthmatic patients. Although the findings showing the lack of change in either histamine or cysteinyl-leukotrienes concentrations in breath condensate following methacholine bronchial challenge was not surprising, the absence of an increase in histamine concentration following adenosine monophosphate challenge was unexpected. Methacholine acts directly on airway smooth muscle cells leading to bronchoconstriction whereas adenosine monophosphate acts indirectly on surface A_2-purinoceptors on primed mast cells triggering the release of pro-inflammatory mediators such as prostaglandins, cysteinyl-leukotrienes and histamine.

Modern histamine H_1-receptor antagonists such as desloratadine, fexofenadine, and levocetirizine at clinically recommended doses as either monotherapy or add-on therapy to inhaled corticosteroids have been shown to attenuate bronchial hyperresponsiveness leading to significant improvement in the provocative concentration of adenosine monophosphate causing a 20% fall in forced expiratory volume in 1 s compared to placebo.[2–4]

Therefore, although the study serve to strengthen the fact that adenosine monophosphate acts indirectly by releasing cysteinyl-leukotrienes from primed mast cells, the findings of the authors should not be interpreted as casting doubt as to the involvement of histamine in mediating the response following indirect bronchial challenge testing. The study should thus act as a catalyst for future research into this area rather than providing definitive proof of mediators involved in the response following both direct and indirect bronchial challenges.

References

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