the guinea pig with a respiratory virus caused a selective blockade of the β-adrenergic-mediated inhibition of antigen-induced contraction of airway smooth muscle. One plausible explanation is that the respiratory virus alters the airway mast cell's inhibitory response to a β agonist, thus ablating normal catecholamine suppression of mediator secretion. This conclusion awaits further confirmation. Moreover, we found that the intrinsic contractile and relaxing airway smooth muscle response is unaltered by virus. In studies to date, we have identified the major site of the virus effect to be on cells, eg, mast cells, which are critical in regulating bronchial smooth muscle tone.

**SUMMARY**

A number of important mechanisms have been identified by which viruses can provoke asthma. From the data available, there does not appear to be one single mechanism available to explain virus-induced asthma. The relationship between viral URIs and asthma is complex and involves many organ systems: airway epithelium, autonomic nervous system control, and the immediate hypersensitivity system. Identifying the effects of respiratory viruses on airway function remains an important undertaking as we try to better understand and control this precipitant of asthma.

**REFERENCES**


**Exercise-Induced Asthma**

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Many individuals with reversible airways dysfunction or asthma are troubled by symptoms of shortness of breath, cough, and wheezing when they attempt to exert themselves. These complaints are particularly frequent when exercising in environments that are both cold and dry. Physiologically, this abnormality is characterized by a development of obstructive airways dysfunction on pulmonary function testing. This article will focus on an overview of the problem, the physiology of exercise-induced bronchospasm (EIB), and the management of the condition.

**OVERVIEW OF THE PROBLEM**

It is probable that all persons with asthma are sensitive to development of EIB. It is characterized as a response to exercise in which there is a fall in the forced expiratory volume in the first second (FEV₁) or peak expiratory rate (PEFR) greater than 10 percent of preexercise values. Typically, the symptoms begin six to ten minutes after beginning exercise and are characterized by chest tightness, shortness of breath, wheezing, cough, and occasionally even stomach-ache. A second phase of EIB, which is usually the most severe, occurs after the exercise. When unaffected persons are recovering from the effects of exercise, those with EIB

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have a typical episode of bronchospasm. Without pharmacologic intervention, this lasts from 30 to 60 minutes. Fortunately, it rarely if ever leads to status asthmaticus. This type of response to asthma affects all ages and sexes. Furthermore, when EIB is provoked by exercise in the laboratory, about 70 to 80 percent of clinically recognized cases of asthma will have reduction in airflow typical of EIB. An interesting phenomenon associated with EIB is a refractory period, ie, light running, stretching, or calisthenics before exercise may reduce, or in some cases prevent, EIB. Furthermore, once a patient has experienced EIB, he has a refractory period of about one hour. Unfortunately, this response is not universal and only occurs in about 40 percent of patients. However, it can be used as an effective strategy to lessen the occurrence of EIB in some susceptible patients.

The occurrence of EIB depends on the type of exercise performed, its intensity and duration, and the atmospheric conditions under which it occurs. In general, the more demanding the exercise, the more likely it is that EIB will be provoked.

**Review of Physiology**

In general, EIB does not occur in normal individuals or in those with pulmonary diseases other than asthma. When exercise begins, there is initial bronchodilatation, with a fall in resistance to airflow in the lungs. This is thought to be due to release of endogenous catecholamines. This response is seen in normal persons, as well as patients with asthma. The contrast between the groups is that following this initial bronchodilatation, those with EIB have progressive airflow obstruction. This correlates with an elevation in airflow resistance. Lung volume mechanics also may be altered, and air trapping occurs. The result is elevated residual volume of gas in the chest and increased functional residual capacity. During the same time, there is a modest fall in arterial oxygen retention, but carbon dioxide retention is rarely present.

Many theories exist for the exact mechanism of EIB. An important factor appears to be cooling of the airways. In fact, the degree of EIB may correlate very closely with the amount of respiratory heat lost during exercise. EIB can be induced by having subjects merely hyperventilate extremely cold, dry air. Exactly how the cooling of the airway then results in frank bronchospasm still is a matter of much investigation. The importance of the observation that cooling of airways plays an important role in the pathogenesis of EIB is that it gives a clinical strategy for its management. Having patients perform exercises which require only relatively brief activity, such as football and baseball, will result in less sustained hyperventilation, loss of respiratory heat, and provocation of EIB. Furthermore, activities which are typically performed in a cold, dry environment, such as winter running and cross-country skiing, are more likely to produce EIB than indoor sports.

A number of mechanisms have been suggested for how airway cooling results in the airflow obstruction seen in EIB. The possible mechanisms have included chemical mediator release, lung mast cells, neural reflexes, and alterations in autonomic control of both airway smooth muscle and vascular function. It is even possible that the α-adrenergic system may have some role in the pathogenesis of the condition.

**Management of EIB**

Efforts to decrease airway cooling and rational use of pharmacologic premedication generally result in improved, if not total prevention of, EIB. A general approach for the management of EIB is as follows: First, the β-adrenergic medications given by inhalation appear to be particularly effective. Susceptible patients should take two to three puffs of a β agonist 15 minutes before exercise. Alternatively, the drug cromolyn, which is thought to stabilize mast cells, may be taken. In fact, some patients who do not respond to either drug alone may respond when both drugs are given in combination. Next, the patient should briefly warm up with moderate exercise, such as stretching for two to three minutes. The subject may then begin exercise. If symptoms do develop, some patients may "run through" their symptoms; if not, exercise should be stopped and remedication with inhaled β agonists performed. Finally, when performing exercise in cold, dry climatic conditions, such as cross-country skiing, breathing through a scarf or mask may decrease or lessen the likelihood of symptoms. In individuals who experience symptoms so severe that they cannot participate in outdoor sports during winter, alternative forms of exercise such as swimming or indoor jogging may be helpful.

**Summary**

Exercise-induced bronchospasm is a common condition of patients who have asthma. Its mechanism appears to be related to cooling of the airways. Clinically, it can be managed by pretreatment with β-adrenergic medications or cromolyn. Measures to protect the airway from excessive loss of heat, such as the use of scarves or participating in indoor activity, are also effective strategies in its management.

**References**