mortality increases between 1979 to 1985 and especially in people over 60 years old. These data did not support any direct relationship between increased β-agonist use and asthma mortality, because these patients have many health problems.4

The likely explanation for these changes in asthma mortality is the underdiagnosis and undertreatment possibly associated with factors such as race, urban crowding, and industrialization. Consequently, the data from Greece do not support the view that inhaled drugs and especially adrenergic drug consumption is related to asthma mortality.

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Occupational Asthma Caused by Pea Flour

To the Editor:

Flours derived from cereal grains such as wheat, rye, oat, and corn are an important source of allergens and a recognized cause of baker's asthma.1 Similar association has been reported in workers at flour mills. We report a case of occupational asthma from exposure to pea flour. Pea flour as a cause of occupational asthma has not been, to our knowledge, previously reported.

A 19-year-old male flour mill worker presented with a 3-month history of paroxysmal breathlessness with wheezing, chest tightness, and cough. Symptoms were work-related and progressive; they worsened by end of the work week. He recovered partially on weekends and recovered completely after 4 days off work. Salbutamol, beclomethasone, and theophylline provided symptomatic relief. The patient had been working for 1 year in a mill processing peas into bran, protein, and starch. He felt that pea protein dust was the most important symptom-provoking agent. He had no other medical history. His physical examination was unremarkable.

Hematology, chest roentgenogram, and pulmonary function tests were normal. Allergy skin prick testing with commercially available extracts was positive for pea dust (4×5 mm), egg, shell-fish, and cat. Skin prick test with a crude aqueous preparation of pea flour from the mill formed a 5×6 mm wheal with one pseudopod, and skin prick test with this extract in one atopic control was negative. Histamine challenge test revealed a PC20 of 0.5 mg/mL.2

Inhaled salbutamol 200 µg as needed was prescribed and a job change recommended. Three months later, after a job change, he was asymptomatic, not using salbutamol and his histamine PC20 had increased to 4 mg/mL.

Occupational asthma is one of the most common claims made to the Worker's Compensation Board.1,2 Often it is necessary to distinguish the group of people who develop asthma due to occupational exposure and sensitization from the asthmatics who have problems in the workplace because of the irritant effect. Nonspecific bronchial challenge tests with histamine or methacholine show airway hyperresponsiveness and support the diagnosis of asthma. And when done serially, these tests can provide objective evidence of exposure to the sensitizing substance.1,2 In our case, a three doubling concentration improvement in histamine PC20 on avoidance of pea flour exposure strongly suggests occupational asthma; histamine PC20 should not change in individuals whose nonoccupational asthma is exacerbated by irritants.

History, skin tests, serial histamine challenge tests, and relief of symptoms on avoiding exposure strongly supported pea flour as the allergen responsible, which obviated the need for bronchoprovocation test with pea flour.

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Adenosine Deaminase in Pleural Effusions

To the Editor:

I read the letters of Ramesh Chandra Sahoo1 and Luis Valdès et al2 in the June 1994 issue of Chest regarding the enzyme adenosine deaminase (ADA) in pleural effusions.

I disagree with their interpretation of ADA findings in pleural effusions because it is incorrect in three aspects. Surprisingly, these authors do not take into account that (1) ADA activity in human biologic fluids results from the action of two principal isoenzymes (ADA-1 and ADA-2), which have different pH, Km, and relative substrate specificities, (2) ADA-1 presence in biologic fluids is due to cell necrosis,3,4 and (3) ADA-2 is found only in monocyte-macrophages from which it is released when they are stimulated by the presence of living parasites in their interior. This explains why ADA-2 increases in biologic fluids in the course of infectious diseases due to intracellular parasites.5,6

Therefore, for a correct interpretation of ADA value in pleural effusions, it is necessary to establish whether ADA-1 or ADA-2 isoenzyme prevails. For example, in pleural effusion of tuberculous origin, it is ADA-2 that increases while in empyemas ADA-1 prevails. Given these data, many false-positive results seem related to an inappropriate evaluation of the two isoenzymes in the entire ADA activity.