Occupational asthma caused by dry metabisulphite

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Abstract

A case is described of occupational asthma in a worker with no previous history of asthma who sprinkled dry metabisulphite powder onto potatoes and developed work-related symptoms. Occupational asthma was confirmed by specific inhalation challenges. (Thorax 1995;50:585–586)

Keywords: asthma, occupational diseases, bronchial provocation test, bronchial hyperreactivity.

Sensitivity to oral metabisulphite is well known to occur in asthmatic subjects.1 Asthma and occupational asthma due to inhaled metabisulphite appears never to have been described.

Case report

A 27 year old man who had been working for 3–4 weeks for an agricultural producer developed respiratory symptoms. He was responsible for cleaning potatoes with water and sprinkling them with dry metabisulphite sodium powder. His symptoms included swelling, itching, redness and running of the eyes, nasal congestion and sneezing, with nausea and shortness of breath as soon as he was exposed to metabisulphite. He was asymptomatic in the evenings and at weekends. He paid three visits to an emergency department where a diagnosis of work-related toxic or allergic reaction was suspected. He was assessed six months after being away from work. He reported no nasal respiratory symptoms. Personal and familial atopic history was negative. He had never smoked. Chest auscultation and radiography were normal. Skin prick tests with 15 common inhalants as well as with diluted metabisulphite (0.1 mg/ml, 1 mg/ml, and 10 mg/ml) were negative. Although baseline forced expiratory volume in one second (FEV1) was reduced (3.41 with a predicted value3 of 4.71 or 72%), the FEV1/FVC ratio (3.4/4.4, 77%) was normal. The provocative concentration of metacholine causing a fall of 20% in FEV1 (PC20)4 was >128 mg/ml – in other words, showing no significant bronchial hyperresponsiveness.

Specific inhalation challenges were performed by exposing the subject to metabisulphite in powder form at 10% and 1% (respectively 10 g or 1 g of metabisulphite powder mixed with 90 g and 99 g of lactose powder) on two occasions separated by a two month interval, using previously described methods.5,6 Control exposure to lactose for 30 minutes did not cause significant changes in FEV1 in the minutes or hours after exposure. As shown in the figure, on each exposure to dry metabisulphite maximum falls in FEV1, of 35% and 52% were elicited 10 and 60 minutes after exposure periods of 35 seconds and four minutes. There were no late reactions and PC20 was >128 mg/ml seven hours after exposure ended. Exposing a control normal subject (PC20 >128 mg/ml) to metabisulphite 10% for a total period of 30 minutes did not cause any significant change (<10%) in FEV1, in the fol-

![Graph of respiratory response to metabisulphite and lactose challenges](http://example.com/graph.png)

Results of specific inhalation challenges with lactose and metabisulphite. BDT = inhaled β₂ adrenergic agent.
lowing hour. Challenges were also carried out with diluted metabisulphite using the methodology described by Wright and coworkers. As we expected that the subject would be extremely sensitive to metabisulphite, we gave him a concentration of only 1/100 of the first concentration of 0.2 mg/ml (which delivers 0.1 μmol metabisulphite) as proposed by Wright and coworkers. FEV₁ dropped from a baseline value of 3.31 to 3.01 (9%) and 1.91 (42%), 30 and 90 seconds respectively after inhalation. As the subject was very symptomatic with cough and dyspnoea salbutamol 400 μg was given by a standard metered dose inhaler. FEV₁ returned to baseline value five minutes later. There was no late reaction and PC₂₀ was still >128 mg/ml seven hours later.

Discussion

Sulphites used as preservatives in the food industry are well documented causes of anaphylactic reactions when ingested. To the best of our knowledge, metabisulphite has not been described as causing occupational asthma and should be added to the list of aetiological agents. Although the mechanism of the reaction to metabisulphite in our subject remains unknown (it did not seem to be IgE mediated as skin prick tests to metabisulphite were negative), we do not think that it could have been caused by a non-specific irritant mechanism because (1) he was exposed to low non-irritating levels of dried metabisulphite which did not elicit reactions in a control subject with normal responsiveness to methacholine, and (2) since he had normal bronchial responsiveness to methacholine we would not have expected any reaction when exposing him to dilute metabisulphite. Metabisulphite can, indeed, cause non-specific bronchospastic reactions in subjects with bronchial hyperresponsiveness to methacholine through the release of sulphur dioxide. Our subject clearly reacted to minute amounts of diluted metabisulphite (dose of metabisulphite estimated to be 0.001 μmol), far below the starting dose proposed for drawing dose-response curves to metabisulphite in subjects with increased bronchial hyperresponsiveness to methacholine (1/100).

Metabisulphite is widely used in the food and beverage industry as a preservative as both a dry and a wet aerosol. The frequency of occupational asthma due to this agent needs to be estimated.

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