Occupational Asthma Induced by Exposure to Quinoa

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Quinoa seeds belonging to the plants of the Chenopodiaceae family are a pseudocereal that originates in the Andean region. They have recently begun to be used in several countries outside the region and are now cultivated in England, Italy, France, the United States of America, Russia, and Japan. Quinoa seeds are rich in proteins (>12 g/100 g), flavonoids, and dietary fiber, and their formulations contain lysine and methionine, 2 amino acids that are essential for proper functioning of the body [1]. They are consumed as a substitute to cereals by wheat-allergic or gluten-intolerant patients. Two cases of anaphylaxis after ingestion of quinoa have been reported [2,3].

We report the first case of occupational asthma induced by long-term exposure to quinoa flour. The patient was a 39-year-old man who is the owner of a company that mills and packages corn, rice, and, for the last few years, quinoa (total daily production of 40 tonnes). He started working in the family business at the age of 18, performing mainly commercial activities and, to a lesser extent, production activities. At the age of 28, he was diagnosed with seasonal rhinoconjunctivitis to grass pollens.

In July 2016, after ingestion of pasta made with quinoa flour, he presented urticaria, angioedema, and dyspnea. He reported episodes of wheezing during the processing of quinoa flour over the previous 2 years, although these were of mild-moderate severity and he recovered spontaneously after leaving the work environment.

In October 2016, the patient underwent skin prick testing (SPT) with corn and rice flour, which yielded a modest positive result. The SPT and specific IgE results for common pollens, including weed pollen, were positive only for grass pollens. Prick-by-prick testing with quinoa seed extract was positive (11 mm × 10 mm, according to the European Academy of Allergy and Clinical Immunology standardization position paper) [4]. The extract was prepared by homogenization in phosphate-buffered saline, dialyzation, and lyophilization, as previously described by Lopez et al [5], before being diluted in phenol glycerol saline solution at a concentration of 20 mg/mL. The SPT result was negative in 10 controls (5 nonatopic controls and 5 atopic controls).

Spirometry findings were normal, and the concentration of exhaled nitric oxide was increased (FeNO, 78 ppb). The methacholine challenge test confirmed the presence of bronchial asthma (PD20, 84 µg).

Given the presence of work-related asthma symptoms, the clear positivity to SPT, and confirmation of bronchial asthma and based on the diagnostic algorithm for occupational allergy by Vandeplas et al [6], we diagnosed the patient with occupational asthma. He started inhaler treatment (fluticasone dipropionate 500 µg/d, salbutamol spray 200 µg when necessary) and desloratadine 5 mg/d. He was asked to avoid occupational exposure to quinoa flour and ingestion of pasta made with this flour. The follow-up visits at the third, sixth, and twelfth months showed his asthma to be well controlled and his functional parameters to be stable (Figure, A and B).

Figure. A, FEV1 (L) values at follow-up visits. B, FeNO (ppb) measurements at follow-up visits.
FeNO continued to be increased, probably owing to the patient's partial adherence to therapy. In fact, the patient stopped inhaler treatment, continuing only the antihistamine for seasonal rhinitis. Meanwhile, he made improvements in the workplace, especially with the extraction system. He avoided direct exposure to quinoa flour and wore a mask while working in the production areas. However, he started to experience mild dyspnea from exposure to amaranth flour, which had been introduced into the production cycle a few months previously. Amaranth (*Amaranthus caudatus*) is also a pseudocereal of Andean origin. In March 2018, a prick-by-prick test with extract of amaranth flour (prepared as for quinoa [see above]) yielded a significantly positive result (7 mm × 10 mm) (Supplementary Figure 1). We confirmed a diagnosis of occupational allergy induced by quinoa and amaranth flour, assuming cross-reactivity between quinoa and amaranth in accordance with Aphalo et al [7]. The patient restarted therapy with inhaled fluticasone and subsequently avoided direct exposure to quinoa flour and wore a mask in the workplace.

To the best of our knowledge, this is the first case of occupational asthma due to exposure to quinoa flour. The working environment can lead to the development of different phenotypes of work-related asthma. Occupational asthma may result either from immunologically mediated sensitization to a specific substance at work, as in the case we report, or from exposure(s) to high concentrations of irritant compounds. Furthermore, pre-existing and coincident asthma can be exacerbated by nonspecific stimuli at work [6]. It was reported that up to 25% of cases of adult asthma were due to occupational exposure [8] and that up to 20% of cases were induced by flour [9]. Quinoa seeds have previously been reported to be associated with anaphylaxis in France [2] and the United States [3] in persons eating quinoa. Highly soluble proteins of quinoa are reported to be associated with a heat-stable allergen [2]. This is of particular interest in food allergy. The patient we report developed occupational asthma after long-term exposure to quinoa flour in the workplace. He also experienced food allergy after ingestion of pasta made with quinoa flour. Sensitization to food allergens through the airways has been described for some antigens, such as seeds and eggs, mainly as an occupational hazard resulting from release of airborne particles during food processing [10]. We confirmed an IgE-mediated mechanism for asthma and food allergy induced by quinoa; moreover, we hypothesized cross-reactivity between quinoa and amaranth flour based on the fact that the globulin-P fraction of amaranth shares some epitopes with quinoa globulins [7].

The development of organic and gluten-free food has increased the potential role of quinoa and other pseudocereals as risk factors in occupational respiratory diseases in the food industry. Therefore, we need preventive interventions both in the workplace and for consumers. These can take the form of reducing direct exposure to pseudocereal flours (improving extraction systems and automated cycles) for operators and introducing these flours as ingredients in mandatory labelling on all health foods.

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**Conflicts of Interest**

The authors declare that they have no conflicts of interest.

**Previous Presentations**

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**References**


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