Occupational asthma induced by quinoa exposure

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Quinoa seeds belonging to the plants of *Chenopodiaceae* family are known as a pseudocereal that originated in the Andean regions. They have recently been introduced in several countries outside of Argentina, and they are also cultivated in England, Italy, France, United States, Russia, Japan. Quinoa seeds are rich in proteins (> 12 g/100g), flavonoids, and dietary fiber and their formulations contain two very important essential aminoacids for the proper functioning of the body: lysine and methionine [1]. They are consumed as a substitute to cereals by patients allergic to wheat or intolerant to gluten. Some authors have reported cases of food allergy; in particular two cases of anaphylaxis after ingestion of quinoa [2,3].

We present the first case report of occupational asthma induced by long-term occupational exposure to quinoa flour. A 39-year-old male who is the owner of a milling company and automated packaging of corn, rice and for some years quinoa (total daily production of 40 ton). By the age of 18 the patient worked in the family business, performing mainly commercial activities and a smaller quantity of production activities. The patient suffered from seasonal rhinoconjunctivitis to grass pollens by the age of 28 years. In July 2016, after ingestion of pasta made with quinoa flour, he presented urticaria, angioedema, and dyspnea. He mentioned episodes of wheezing during the processing of quinoa flour over the previous two years, but they were of mild-moderate severity and he spontaneously recovered after leaving the work environment.

In October 2016, the patient has been tested with prick tests (SPT) for corn and rice flour and modest positive resulted. The SPT and specific IgE for common pollens, including weed pollen, were positive only for grass pollens. Prick by prick with quinoa seeds extract was positive (11 mm x 10 mm, according to the EAACI standardization Position paper) [4]. The extract was prepared by homogenization in phosphate buffer saline, dialyzation and lyophilization, as previously described by Lopez D.E. et al. (5) and afterwards was diluted in phenol glycerol saline solution at a concentration of 20 mg/ml. The SPT resulted negative in 10 controls (5 nonatopic controls and 5 atopics).

Spirometry was normal: and the concentration of exhaled nitric oxide increased (FeNO 78 ppb). The methacoline challenge test confirmed the presence of bronchial asthma (PD_{20}FEV1 84 mcg).
Due to the presence of work-related asthma symptoms, the clear positivity to SPT and confirmation of bronchial asthma, in agreement with the diagnostic algorithm for OA by Vandeplas O et al. [6] we reached a conclusion of allergic Occupational Asthma (OA). The patient started inhalation therapy (fluticasone dipropionate 500 mcg/day, salbutamol spray 200 mcg prn) and desloratadine 5 mg daily. The patient 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 under control and functional parameters were stable (Figure 1a, 1b). FeNO continued to be increased, probably due to the patient's partial therapeutic adherence. In effect the patient stopped inhalation treatment, continuing only the antihistamine for seasonal rhinitis. Meanwhile, the patient made improvements in the workplace especially with the extraction system. He avoided direct exposure to quinoa flour and he wore a mask while working in the production areas. However, he started to experience mild dyspnea from amaranth flour exposure, introduced in the production cycle by few months. Amaranth belongs to the plants of Amaranthus caudatus, and is a pseudocereal of Andean origin. In March 2018, a prick by prick test with extract of amaranth flour was performed (prepared as previously done for quinoa) and it was significantly positive (7 mm x 10 mm) (Figure 1 supplementary). We then concluded for OA induced by quinoa and amaranth flour, assuming a cross-reactivity between quinoa and amaranth in accordance with previous reported by Aphalo P et al. [7]. The patient reintroduced fluticasone inhaled therapy and in the following period he presented occasionally non-work related exertional dyspnea; however, he reported face edema to indirect exposure to quinoa in the workplace.

To the best of our knowledge, this is the first case of Occupational Asthma (OA) due to exposure to quinoa flour. The workplace environment can lead to the development of different phenotypes of work-related asthma (WRA). OA may result either from immunologically mediated sensitization to a specific substance at work, as in our case report, or from exposure(s) to high concentrations of irritant compounds. Furthermore pre-existing or coincident asthma can be exacerbated by non-specific stimuli at work (WEA) [6]. It was reported that the workplace was responsible for the development of up to 25% of adult-asthma [8] and up of 20% of OA was induced by flour [9]. Quinoa seeds have been previously reported to related with anaphylaxis in France [2] and the United States [3] in subjects eating quinoa. It has been documented that highly soluble proteins of quinoa are associated with a thermostable allergen [2], and this fact is of particular interest for food allergy. Our patient developed OA after long-term exposure to quinoa flour in the workplace. He also experienced food allergy after ingestion of pasta made with quinoa flour. The 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 particulates during food processing [10]. We confirmed an IgE-mediated mechanism for asthma and food allergy induced by quinoa; moreover, we hypothesized a cross-reactivity between quinoa and amaranth flour according with the evidence that amaranth globulin-P fraction shares some epitopes with quinoa globulins [7].

The development of organic and gluten free food has increased the possible role of quinoa and other pseudocereals as risk factors for occupational respiratory diseases in the food industry. Therefore we need preventive interventions both in the workplace and for consumers. This can be done by 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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References


Figure 1 a) FEV1 (l) values at follow-up visits, b) FeNO (ppb) measurements at follow-up visits