

Occupational Asthma Induced by the Reactive Dye Synozol Red-K 3BS

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Various reactive dyes can elicit occupational asthma in exposed textile industry workers. To date, there has been no report of occupational asthma caused by the red dye Synozol Red-K 3BS (Red-K). Here, we report a 38-year-old male textile worker with occupational asthma and rhinitis induced by inhalation of Red-K. He showed positive responses to Red-K extract on skin-prick testing and serum specific IgE antibodies to Red-K-human serum albumin conjugate were detected using an enzyme-linked immunosorbent assay. A bronchoprovocation test with Red-K extract resulted in significant bronchoconstriction. These findings suggest that the inhalation of the reactive dye Red-K can induce IgE-mediated occupational asthma and rhinitis in exposed workers.

Key Words: Asthma; reactive dye; occupation

INTRODUCTION

Reactive dyes form covalent bonds with the fibers in textiles. A reactive dye (RD) can act as a hapten¹ and elicit occupational asthma (OA) in exposed workers.² Since the first report of RD-induced OA in 1978,³ the reported prevalence of RD-induced OA in Korea has been 2.5–5.9%.^{4,5} RDs were among the most frequent causes of OA in Korea until the 1990s.⁶ Of these dyes, Black GR is the most frequent sensitizer; others include Orange 3R, Blue GG, and Green 6B.^{4,7,8} There has been no previous report of OA caused by Synozol Red-K 3BS (Red-K). Thus, we report the first case of OA induced by Red-K, confirmed by skin-prick testing, bronchial provocation, and immunological testing in a dyer in the textile industry.

CASE REPORT

The patient was a 38-year-old male non-smoker. He had no history of allergic disease. He had worked in the textile industry for 15 years and had used reactive dyes, such as Synozol Black B 150 (Black B) and Red-K. Nine years after starting his job, he experienced rhinorrhea that progressed to a cough and dyspnea 1 year ago; these were aggravated at work.

At presentation, his blood differential count, serum biochemistry, total IgE (56 kU/L), and chest and paranasal sinus radiographs were normal. The sputum eosinophil count was elevated

to 84%, while the blood and nasal eosinophil counts were normal (220/ μ L, 0%). Skin prick testing was positive for mugwort and nettle pollens. Baseline results included a FEV1 of 3.84 L (120.0%) with a FEV1/FVC ratio of 90.61% and post-bronchodilator FEV1 of 3.92 L (122.3%) without reversibility. His initial methacholine PC20 was 1.25 mg/mL.

We considered that his asthma and rhinitis symptoms may be related to his occupation, especially using RDs. Two RDs used in his factory were Black B (color index name Reactive Black 5) and Red-K. The structures of Black B and Red-K are shown in Fig. 1. Skin-prick testing was performed with extracts of these two RDs (10 mg/mL) and histamine. He had a positive result for Red-K ($4 \times 4/31 \times 24$ mm), but a negative result (0 mm) for Black B, when compared with histamine ($3 \times 3/35 \times 29$ mm). Serum specific IgE antibodies to two RD-human serum albumin (HSA) conjugates were measured using an enzyme-linked immunosorbent assay (ELISA), as described previously.⁹ The positive cut-off value for a high serum specific IgE level was defined as the mean absorbance value plus three standard deviations.

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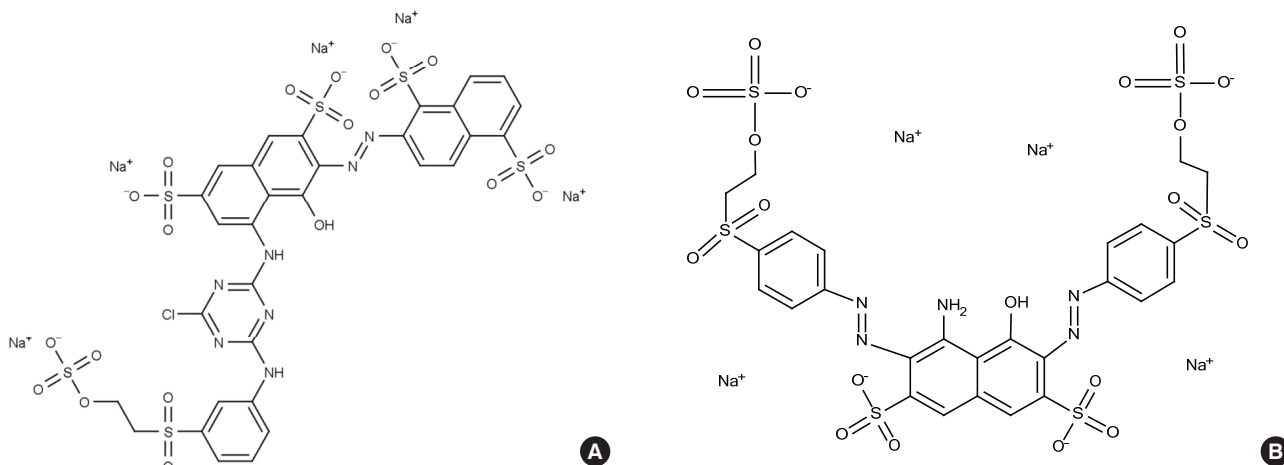


Fig. 1. Chemical structures of Synozol RED K 3BS (A) and Synozol Black B 150 (B).

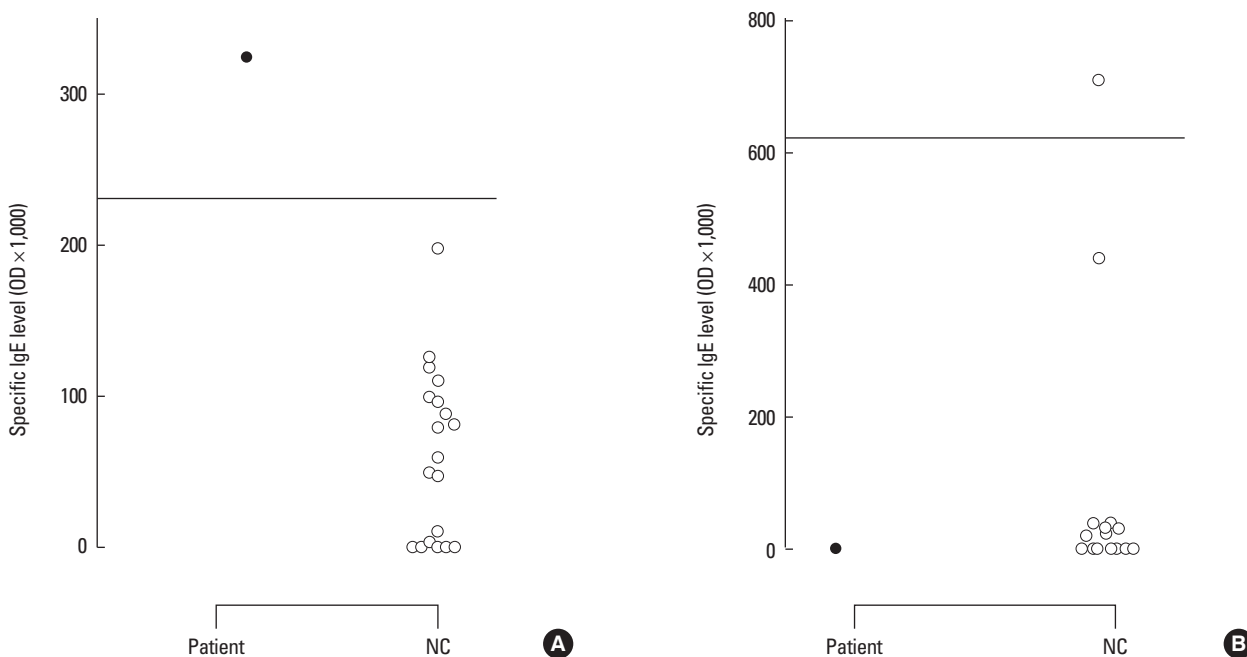


Fig. 2. Serum specific IgE levels to two reactive dye-human serum albumin conjugates by enzyme-linked immunosorbent assay: Synozol RED K 3BS (A) and Synozol Black B 150 (B). Horizontal bars indicate positive cut-off values. OD, optical density; NC, unexposed healthy controls.

tions for sera from 18 unexposed, non-atopic, healthy controls. He had a high level of IgE antibody specific to the Red-K-HSA conjugate, but no specific IgE binding was noted with the Black B-HSA conjugate (Fig. 2). To confirm OA, inhalation challenge tests with the two RD extracts were performed. No significant change in FEV₁ was noted after the placebo inhalation, while significant bronchoconstriction (a 45.6% fall in FEV₁ from the baseline value) with dyspnea and wheezing was noted 10 minutes after inhaling 10 mg/mL of Red-K extract; the response was negative after inhaling Black B extract up to 10 mg/mL (Fig. 3).

Based on these findings, he was diagnosed with OA and rhinitis caused by Red K. We recommended job relocation and the

use of an inhaled corticosteroid/long acting β -2 agonist with regular follow-up.

DISCUSSION

Positive skin-prick tests to extracts of dyes and a positive radioallergosorbent test to RD-bound paper discs suggest that RDs can act as haptens, provoking IgE-mediated hypersensitivity reactions.¹⁰ Both skin-prick testing and the detection of specific IgE to RD-HSA conjugate in serum are useful for screening, diagnosing, and monitoring OA resulting from exposure to RDs.¹¹ Our patient had positive responses to Red-K extracts on skin-

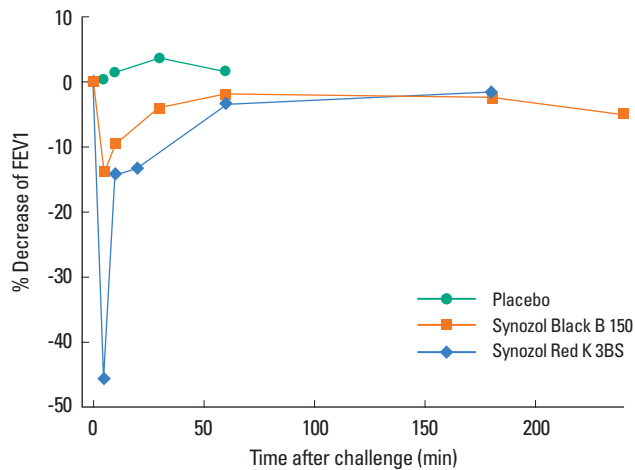


Fig. 3. Results of bronchoprovocation tests with two reactive dye extracts: Synozol Black B 150 and Synozol RED K 3BS.

prick testing and had high serum specific IgE antibody to Red-K-HSA conjugate. The bronchoprovocation test with Red-K extracts demonstrated immediate bronchoconstriction with the development of asthma symptoms. Thus, we confirmed the first case of OA caused by Red-K, not by Black B, in which an IgE-mediated response was suggested as a major pathogenic mechanism.

Reactive dyes contain a chromogen and reactive functional groups that form irreversible covalent bonds with the amino acid residues of cellulosic fibers.¹² There are many RDs with different reactive functional groups to which carrier proteins bind to induce immune responses.^{2,11,13} The vinyl sulfone RDs are major causes of OA and have one or two vinyl sulfone reactive groups.^{10,14,15} Black GR, the most frequent sensitizer, has two vinyl sulfone reactive groups,⁹ while Red-K has one. However, the allergenicity of an RD can differ according to the carrier protein and the conditions under which the hapten conjugation process occurs. The chromogen components, neoantigenic determinants, and reactive components all contribute to allergenicity.⁹ Our patient showed negative responses to skin-prick testing, ELISA, and bronchoprovocation testing for Black B, which is the most frequent sensitizer. Thus, we conclude that he was not sensitized to Black B.

He was atopic, although atopy is not a predisposing factor for RD-induced OA.⁷ He was a non-smoker, and smoking is a predisposing factor.⁸ Park et al.¹⁵ suggested that the duration from symptom onset to diagnosis was the only factor differentiating patients with normal and reduced lung function at diagnosis and that early diagnosis and treatment were essential for a good prognosis. Our patient's disease duration from asthma symptom onset to diagnosis was 1 year. Thus, his condition may improve if he moves to another workplace and takes anti-asthma medications.

In conclusion, we confirmed that Red-K powder inhalation

can induce OA, and an IgE mediated response was suggested in the pathogenic mechanism.

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