

Bespoke Latex Allergen Testing Improves Assessment of Respiratory Symptoms in Textile-Braiding Workers

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Background Latex allergy is poorly understood in latex-exposed textile workers.

Methods A cross-sectional study was carried out to better characterize respiratory symptoms, using personal breathing zone latex allergen measurement and specific IgE to latex allergens.

Results Forty-four of the 86 (51% participation rate) participated. Ten of 39 workers who gave a blood sample (25.6%) were found to have IgE to at least one workplace allergen (5/39 positive to either latex braiding coated with silica or talc, 4/39 were positive to the dyed cotton extract, and 1/39 to latex braiding coated with silica or talc and dyed cotton extract), whilst only 2 of these 10 had specific IgE to the commercial latex extract.

Conclusions The presence of symptoms with evidence of sensitization was strongly dictated by current latex exposure. Bespoke workplace allergen IgE testing identified cases of WR respiratory symptoms with sensitization that otherwise would not have been identified. Am. J. Ind. Med. © 2012 Wiley Periodicals, Inc.

KEY WORDS: latex exposure; braiding; respiratory symptoms; sensitization; bespoke

INTRODUCTION

Natural rubber latex is the milky sap produced by the laticiferous cells of the tropical rubber tree *Hevea brasiliensis* and forms the basis of a variety of household, industrial, and medical products. Latex exposure can cause a variety of allergic endpoints in sensitized individuals, including respiratory, cutaneous, ocular, or systemic effects [Beuers et al., 1990; Jaeger et al., 1992; American College

of Allergy, Asthma, and Immunology position statement, 1995], with healthcare workers being a major at risk occupational group. Health effects attributed to cross-reactivity with other allergens are also described [Wagner and Breiteneder, 2002; Blanco, 2003]. Much of the evidence base relating to latex allergy has been generated from the healthcare sector [Liss et al., 1997; Liss and Sussman, 1999; Garabrant and Schweitzer, 2002; Filon and Radman, 2006; Das, 2008], although non-healthcare sector occupational groups are also at risk, including those working as hairdressers, food handlers, latex glove manufacturers, and cleaners [Zuskin et al., 1998; Valks et al., 2004]. Although data from non-healthcare sectors are limited, textile-braiding workers have previously been the subjects of reports of latex allergy [Pisati et al., 1998; Lopata et al., 2007].

We describe the results of an investigation assessing respiratory symptoms and sensitization to latex in a group of workers employed at a latex-braiding workplace, using both standard clinical techniques, but including measurement of specific IgE to potential workplace allergens,

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using both standard and bespoke methods, in addition to measurement of airborne latex exposure.

METHODS

Each worker at the study worksite, a typical textile-braiding facility, was invited to participate. Following recruitment, an interviewer-led respiratory symptoms questionnaire was administered to all consenting participants. This was an adaptation of the Medical Research Council [Minette, 1962] and European Community Respiratory Health Survey designs [Burney et al., 1994], focusing on the presence or absence of both work- (WR) and non-work-related reported respiratory symptoms. WR symptoms were defined as those described as worse at work or improving on rest days. WR cough (any cough), shortness of breath (Medical Research Council (MRC) grade 2 or greater), chest tightness (any chest tightness ever) or wheeze (any wheeze ever) were categorized as WR lower respiratory symptoms and WR ocular and nasal irritation were categorized as WR upper respiratory symptoms. Demographic details, a full work history and smoking details were also recorded. It was not possible to further characterize these workers within this study, and consequently possible respiratory diagnoses such as asthma were based on questionnaire responses and lung function values.

The production process at the study site involved the manual tying of rubber thread, lycra, polyester, nylon, polypropylene, cotton, and antistatic yarns onto braiding machines. Threads were automatically fed through a braiding machine and knitted with elastic on the machine bed. The final product was expelled directly into packing boxes and wheeled to a packing and examining area, prior to final packing.

In order to measure worker sensitization to a variety of workplace-based allergens, protein extracts were prepared from dyed cotton and two types of latex braids collected from the workplace (BESPOKE method), and a commercial latex extract (Allergopharma Joachim Ganzer KG, Reinbek, Germany). The latter comprised the major latex allergens; including Hev b1, Hev b3, Hev b5, and Hev b6.02. Workplace samples were extracted in 10% (w/v) ammonium bicarbonate, filtered and centrifuged at 10,000 rpm for 30 min, the resulting supernatant then being freeze-dried and re-suspended in phosphate-buffered saline (PBS).

Radioallergosorbent (RAST) analysis was performed according to Ceska and Eriksson [1972], using cyanogen bromide-activated disks conjugated either with workplace allergens or commercial latex extract. Each worker provided a 10 ml sample of clotted blood. This was used for the measurement (by RAST) of atopic status and specific IgE to relevant workplace allergens.

Latex products were collected from the factory. Extracts were made for each by (i) extracting in 10% (w/v) ammonium bicarbonate; (ii) filtering the mixture; (iii) centrifuging the filtrate at 10,000 rpm for 30 min; and (iv) then removing the supernatant and freeze-drying to concentrate the protein. This was then re-suspended in 15 ml of PBS. BCA protein estimation was performed to determine the amount of protein in each extract in order to calculate the correct weight of activated disks to be used. The RAST disks were prepared from cyanogen-activated disks. One milligram of each extract per 100 mg of activated disks were rotated overnight in 0.1 M sodium bicarbonate buffer. The next day the disks were washed, blocked with ethanolamine, washed in acetate buffer, washed in bicarbonate buffer, and stored in PBS/0.1% Tween 20 at -20°C .

Two hundred microliters of test or control serum (diluted $\frac{1}{4}$ in PBS) was incubated with a disk overnight at room temperature. All the supernatant was then removed and three washes performed using 1 ml of PBS/0.1% Tween 20. After washing, 100 μl of ^{125}I labeled anti-human IgE was added to each disk and incubated at room temperature for 24 hr. Washing to remove unbound anti-human IgE was carried out as for the previous washing stage.

The radioactivity of the bound anti-human IgE was measured using a gamma counter (Packard, Meriden, CT). All assays were performed in duplicate and results were expressed as a RAST score. Samples were rejected if the duplicate results differed by more than 10% for samples binding 1% or more of the added ^{125}I (15% for samples binding less than 1% of the added ^{125}I). The corrected RAST score was calculated as the percentage score for the test serum disk minus that of the negative control disk. A RAST score of equal to or greater than 2 was considered positive.

A mixed disk was prepared, to define atopy, using equal amounts of grass pollen, cat dander, and house dust mite extracts. A RAST score of 2 or more to this mixed disk was considered positive.

Study participants were asked to perform a reproducible measurement of lung function using a calibrated spirometer (Alpha 2, Vitalograph, Buckinghamshire, UK), following the American Thoracic Society Guidelines [American Thoracic Society, Standardisation of Spirometry, 1995], with the forced expiratory volume in 1 s (FEV_1) and forced vital capacity (FVC) being recorded.

Personal exposures to inhalable dust were determined according to MDHS 14/2 using glass fiber filters (GF/A, 1.6 mm, Millipore, Billerica, MA) and Institute of Occupational Medicine (IOM) sampling heads [HSE, 2000]. Long-term personal samples were collected in order to measure full shift exposures, and individual worker pumps were set at a flow rate of 2 L/min. On completion of the sampling period and subsequent gravimetric analysis, sampling filters were eluted into 2 ml PBS/0.1% Tween 20

overnight and latex proteins were measured using the FITkit™ (FIT Biotech Oyj Plc, Tampere, Finland) enzyme-linked immunosorbant assay (ELISA) system, designed for the determination of hevein in natural rubber latex products. Currently, four of the identified latex allergens (Hev b1, Hev b3, Hev b5, and Hev b6.02) have been unequivocally demonstrated in manufactured products, in addition to being reported as four of the most important latex allergens [Wagner and Breiteneder, 2005] and FITkit™ provide assay kits for each of these allergens.

In order to stratify likely exposures within the workplace, information from a walkthrough hygiene survey, work-task observations, and personal inhalable dust sampling were combined to categorize worker exposure, a priori, to either a low or medium latex-exposed groups.

All data were entered into SPSS (Statistical Package for Social Scientists v 12, SPSS Inc., Chicago, IL) and

cleaned prior to analysis. In addition to descriptive analyses, tests of statistical inference included *t* testing for equality of means, Pearson's chi-squared analysis, or one-way ANOVA. Statistical significance was assumed at the 5% level.

This study was approved by the ethics committee of the UK Health and Safety Executive, and all workers provided written, signed, informed consent to participate.

RESULTS

The workplace employed 86 workers, 40 of who worked on the factory floor. A total of 44 of the 86 (51% participation rate) completed the questionnaire and 39 (45.3%) gave a blood sample. The characteristics of the study population are summarized in Table I. Twenty-two (50%) of the 44 workers were male, with a mean age of

TABLE I. Worker Characteristics by Exposure Group

Characteristics	All (N = 44)	Low exposure (N = 16)	Medium exposure (N = 28)	P-value
Age (years)				
Mean (SD)	37.2 (10.1)	36.7 (11.6)	37.4 (9.3)	0.964
Median	37.0	39.0	35.5	
Range	19–59	19–58	22–59	
Duration of work in industry (months)				
Mean (SD)	126.1 (108.7)	125.1 (122.8)	126.7 (102.2)	0.818
Median	126.0	97.0	132.0	
Range	1–504	1–396	2–504	
Gender				
Male (%)	22 (50.0)	10 (62.5)	12 (42.9)	0.210
Female (%)	22 (50.0)	6 (37.5)	16 (57.1)	
Smoking				
Current (%)	17 (38.6)	4 (25)	13 (46.4)	0.287
Ex (%)	11 (25.0)	4 (25)	7 (25.0)	
Never (%)	16 (36.4)	8 (50)	8 (28.6)	
Ever suffered from asthma				
Yes (%)	5 (11.4)	1 (6.3)	4 (14.3)	0.419
No (%)	39 (88.6)	15 (93.8)	24 (85.7)	
Ever suffered from eczema, hay fever, or other allergies				
Yes (%)	20 (45.5)	9 (56.3)	11 (39.3)	0.277
No (%)	24 (54.5)	7 (43.8)	17 (60.7)	
Characteristics	All (N = 39)	Low exposure (N = 12)	Medium exposure (N = 27)	P-value
Atopy				
Yes (%)	11 (28.2)	4 (33.3)	7 (25.9)	0.635
No (%)	28 (71.8)	8 (66.7)	20 (74.1)	
Characteristics	All (N = 39)	Low exposure (N = 12)	Medium exposure (N = 27)	P-value
Work-related sensitization ^a				
Yes (%)	10 (25.6)	4 (33.3)	6 (22.2)	0.463
No (%)	29 (74.4)	8 (66.7)	21 (77.8)	

^aWork-related sensitization; any RAST score ≥ 2 for any work-related allergen test.

37.2 years (SD; 10.1, range; 19–51 years), 38.6% were current smokers, and 25% ex smokers, with a mean time working in the industry of 10.5 years.

Twenty-four of the 44 (54.5%) workers reported either upper or lower WR respiratory symptoms. Twelve workers reported upper respiratory symptoms in the absence of any lower respiratory symptoms, three reported lower symptoms only, and seven reported both upper and lower symptoms. The most commonly reported WR symptoms were ocular [14/44 (31.8%)], followed by nasal [13/44 (29.5%)] and chest tightness [7/44 (15.9%)].

Eleven personal dust samples were taken, reflecting a broad range of work tasks. The results of this sampling exercise are shown in Table II. The latex assay used for Hev b 6.02, Hev b1, Hev b3, and Hev b5 had lower limits of detection of 0.08, 2.4, 0.21, and 0.55 ng/ml, respectively (which were similar to those reported by the manufacturer (FITkitTM; FIT Biotech Oyj Plc, Tampere, Finland). It was possible to detect Hev b6.02 in 9 of the 11 personal samples. There was no detectable Hev b1, Hev b3, or Hev b5 in any of the personal samples, although it should be noted that the lower limits of detection for these assays were considerably higher than that of Hev b 6.02.

Consequently, two exposure groups were defined a priori; (i) low exposure: mean total inhalable dust 0.45 mg/m³, with no detectable airborne Hev b 6.02 and (ii) medium exposure: mean total inhalable dust 1.2 mg/m³, with a mean Hev b 6.02 of 0.6 ng/m³. The latter value is already cited as a threshold above which asthma symptoms may occur in response to latex allergen exposure [Baur et al., 1998].

As shown in Table I, there was no significant difference in age, gender, smoking status, atopy status, or presence of specific IgE to workplace allergens between the 28 workers in the lower-exposed group and the 16 workers in the medium-exposed group. As shown in Table III, those workers in the medium-exposed category, however, reported higher levels of WR respiratory symptoms in comparison to those in the lower-exposed group, respectively, 18/28 (64.3%) compared to 6/16 (37.5%), although this difference did not reach statistical significance [$P = 0.086$, odds ratio (OR); 3.00, 95% confidence

interval (CI): 0.84–10.72]. The only symptom that was significantly increased in the medium-exposed group in comparison to the lower-exposed group was WR chest tightness [7/28 (25%)] in comparison to absence of this symptom in the lower-exposed group ($P = 0.029$).

Thirty-nine of the 44 workers (88.6%) consented to provide blood for the determination of specific IgE. Of these, 10 (25.6%) were found to have specific IgE to at least one workplace allergen (5/39 were positive to either latex braiding coated with silica or talc, 4/39 were positive to the dyed cotton extract, and 1/39 to latex braiding coated with silica or talc and dyed cotton extract). Only 2 of the 39 workers had specific IgE to the commercial latex extract (containing the major reported latex allergens including Hev b1, Hev b3, Hev b5, and Hev b6.02), both of whom also had specific IgE to both of the workplace-sourced latex braiding samples.

There was no observed association between exposure group and either sensitization to WR allergens or atopy. Specifically, 4 of the 12 workers in the low-exposure group had evidence of sensitization to workplace allergens in comparison to 6 of the 27 workers in the medium-exposed group ($P = 0.463$). Similarly, 4 of the 12 workers in the low-exposure group were found to be atopic in comparison to 7 of the 27 workers in the medium-exposure group ($P = 0.635$). Individuals sensitized to WR allergens were, however, more likely to be also atopic ($P = 0.010$; OR: 7.2; CI: 1.5–35.3).

The data were further analyzed to assess any relationships between worker demographic, lung function and sensitization, and self-reported WR respiratory symptoms (Table IV). Workers were categorized by the presence or absence of any WR symptoms or any WR lower respiratory symptoms. There was no association between age, gender, duration in industry, smoking status, or sensitization to WR allergens, and the reporting of WR symptoms.

However, there was an association between WR lower symptoms and both atopy ($P = 0.023$) and the individual reporting that they ever suffered from asthma ($P = 0.043$).

Current latex exposure strongly dictated the presence or absence of WR respiratory symptoms in sensitized workers, as shown in Table III. None of the four workers from the low-exposure group who were sensitized to WR allergens reported WR symptoms, whilst all of the six sensitized workers from the medium-exposure group reported such symptoms.

Mean lung function values, as expressed as a percentage of predicted of FEV₁, were significantly lower for the medium-exposed workers than those in the low-exposure group ($P = 0.006$), although there was no significant difference in the FEV₁/FVC ratio between exposure groups. The mean percentage predicted FEV₁ measured from workers reporting either lower or any WR respiratory

TABLE II. Results of Airborne Hev b6.02 Sampling

Sample characteristic	Total inhalable dust mg/m ³ , mean (range)	Hev b6.02 (ng/m ³), mean (range)
All (n = 11)	1.2 (0.3–1.8)	0.6 (LLD: 3.1, n = 9)
Office and stores (n = 2)	0.45 (0.3–0.6)	ND

LLD, below the lower limit of detection; ND, not detected.

TABLE III. Work-Related Respiratory Symptoms and Lung Function Measurements by Exposure Group

Characteristics	All	Low exposure	Medium exposure	<i>P</i> -value
Work-related cough	N = 44	N = 16	N = 28	0.113
Yes (%)	4 (9.1)	0 (0)	4 (14.3)	
No (%)	40 (90.9)	16 (100)	24 (85.7)	
Work-related chest tightness	N = 44	N = 16	N = 28	0.029
Yes (%)	7 (15.9)	0 (0)	7 (25)	
No (%)	37 (84.1)	16 (100)	21 (75)	
Work-related wheeze ^a	N = 41	N = 15	N = 26	0.558
Yes (%)	4 (9.8)	2 (13.3)	2 (7.7)	
No (%)	37 (90.2)	13 (86.7)	24 (92.3)	
Work-related shortness of breath	N = 44	N = 16	N = 28	0.682
Yes (%)	2 (4.5)	1 (6.3)	1 (3.6)	
No (%)	42 (95.5)	15 (93.8)	27 (96.4)	
Work-related ocular symptoms	N = 44	N = 16	N = 28	0.159
Yes (%)	14 (31.8)	3 (18.8)	11 (39.3)	
No (%)	30 (68.2)	13 (81.3)	17 (60.7)	
Work-related nasal symptoms	N = 44	N = 16	N = 28	0.235
Yes (%)	13 (29.5)	3 (18.8)	10 (35.7)	
No (%)	31 (70.5)	13 (81.3)	18 (64.3)	
Work-related lower respiratory symptoms ^a	N = 42	N = 15	N = 27	0.235
Yes (%)	10 (23.8)	2 (13.3)	8 (29.6)	
No (%)	32 (76.2)	13 (86.7)	19 (70.4)	
Work-related upper respiratory symptoms	N = 44	N = 16	N = 28	0.098
Yes (%)	21 (47.7)	5 (31.3)	16 (57.1)	
No (%)	23 (47.7)	11 (68.8)	12 (42.9)	
Any work-related respiratory symptoms	N = 44	N = 16	N = 28	0.086
Yes (%)	24 (54.5)	6 (37.5)	18 (64.3)	
No (%)	20 (45.5)	10 (62.5)	10 (35.7)	
FEV ₁ % predicted ^a	N = 42	N = 15	N = 27	0.006
Mean (SD)	98.0 (15.5)	105.3 (7.7)	93.9 (17.3)	
Median	100.5	107.0	96.0	
Range	57–21	91–19	57–121	
FEV ₁ /FVC (%) ^a	N = 42	N = 15	N = 27	0.595
Mean (SD)	82.9 (6.4)	83.6 (5.3)	82.5 (7.0)	
Median	83.3	84.0	82.6	
Range	60–95	73–95	60–94	

When rounded to 1 decimal place, percentages do not always total to 100%.

Bold denotes *P* values that are 0.05 or lower.

^aMissing data.

symptom were lower than for those workers not reporting symptoms, although this was only statistically significant for workers reporting any WR respiratory symptoms ($P = 0.030$). There was no significant difference in the FEV₁/FVC ratio and the reporting of WR respiratory symptoms.

DISCUSSION

The main findings of this study relate to the relatively high level of self-reported respiratory symptoms in this

group of latex textile workers. Whilst the levels of sensitization to workplace allergens did not differ between current allergen-exposure groups, respiratory symptoms in those sensitized were only reported in those workers with current allergen exposure. Additionally, the use of bespoke allergen testing, by estimating IgE levels to allergen extracts taken specifically from the workplace, identified a significantly increased number of sensitized workers in comparison to allergen testing using non-bespoke, commercially based methods. There are very limited previous data relating to allergen sensitization with an implied IgE

TABLE IV. Demographic, Smoking, Exposure, and Workplace Sensitization by Symptom Groupings

Characteristics	Work-related lower symptoms	No work-related lower symptoms	P-value	Any work-related symptoms	No work-related symptoms	P-value
Age (years)	N = 10	N = 32	0.908	N = 24	N = 20	0.764
Mean (SD)	37.9 (9.9)	37.5 (10.4)		37.6 (9.5)	36.7 (10.9)	
Median	35.5	37.5		35.5	37.5	
Range	28–59	19–58		23–59	19–58	
Duration of work in industry (months)	N = 10	N = 32	0.515	N = 24	N = 20	0.983
Mean (SD)	150.9 (147.9)	124.8 (95.7)		125.8 (109.2)	126.5 (111.0)	
Median	132.0	138.0		132.0	126.0	
Range	18–504.0	1–396		1–504	2–396	
Gender	N = 10	N = 32	0.147	N = 24	N = 20	0.545
Male (%)	7 (70)	14 (43.8)		11 (45.8)	11 (55.0)	
Female (%)	3 (30)	18 (56.3)		13 (54.2)	9 (45.0)	
Smoking	N = 10	N = 32	0.902	N = 24	N = 20	0.879
Current (%)	3 (30)	12 (37.5)		10 (41.7)	7 (35.0)	
Ex (%)	3 (30)	8 (25.0)		6 (25.0)	5 (25.0)	
Never (%)	4 (40)	12 (37.5)		8 (33.3)	8 (40.0)	
Exposure category	N = 10	N = 32	0.235	N = 24	N = 20	0.086
Medium (%)	8 (80.0)	19 (59.4)		18 (75.0)	10 (50.0)	
Low (%)	2 (20.0)	13 (40.6)		6 (25.0)	10 (50.0)	
Ever suffered from asthma	N = 10	N = 32	0.043	N = 24	N = 20	0.225
Yes (%)	3 (30)	2 (6.3)		4 (16.7)	1 (5.0)	
No (%)	7 (70)	30 (93.8)		20 (83.3)	19 (95.0)	
Atopy	N = 9	N = 29	0.023	N = 21	N = 18	0.442
Yes (%)	5 (55.6)	5 (17.2)		7 (33.3)	4 (22.2)	
No (%)	4 (44.4)	24 (82.8)		14 (66.7)	14 (77.8)	
Work-related sensitization	N = 9	N = 29	0.436	N = 21	N = 18	0.651
Yes (%)	3 (33.3)	6 (20.7)		6 (28.6)	4 (22.2)	
No (%)	6 (66.7)	23 (79.3)		15 (71.4)	14 (77.8)	
Work-related sensitization present AND	N = 3	N = 6	0.119*	N = 6	N = 4	0.005*
Medium-exposed	3 (100%)	2 (33.3%)		6 (100%)	0 (0%)	
Low-exposed	0 (0%)	4 (66.7%)		0 (0%)	4 (100%)	

Bold denotes *P* values that are 0.05 or lower.

*Fisher's exact test.

mechanism in textile workers, although lone IgE response to cotton have previously not been identified to be a useful clinical tool [Zuskin et al., 1992].

In order to better define the origin of the high prevalence of upper and lower respiratory symptoms, workplace measures were used to define the level of current allergen exposure. It was only possible to measure Hev b 6.02 in 9 of the 11 personal samples taken in various workplace locations, with five of these measurements below the suggested threshold limit [Baur et al., 1998] of 0.6 ng/m³. These levels were also considerably lower than a number of exposure studies reported in the healthcare sector where exposures of up to 978 ng/m³ have been reported [Swanson et al., 1994], although it is difficult to reliably

compare exposure levels between studies, given the lack of a standardized method to measure workplace latex allergen exposure. It is of note, however, that all measured levels were below the relevant current UK Workplace Exposure Limit [Health and Safety Executive: Occupational Exposure Limits, 2005] of 10 mg/m³ (8-hr TWA reference period). Clearly, exposure measures reflect only the single day of measurement, and it is likely that work practices at this site would have been associated with chronic intermittent allergen exposures.

The presence of latex-specific IgE also contributed to the understanding of reported WR respiratory symptoms. Of the 39 participants who provided a serum sample, approximately a quarter demonstrated sensitization to at least

one WR allergen. It is of particular interest that the presence of IgE to a workplace allergen was more readily identified using the bespoke method. Indeed, if only non-bespoke testing had been used, a fivefold underestimate of the prevalence of workers with workplace-specific IgE would have resulted.

It must be appreciated that the bespoke allergen disks would have bound to them a mix of workplace derived allergen and that the full extent of this mix was not possible to characterize within the scope of this study. Consequently, sensitization was measured to putative allergens in these extracts, for example, to latex Hev b proteins and likely other uncharacterized allergens.

Nevertheless, given that full characterization of all allergens was not possible, the results do have significance for assisting diagnosis of workplace allergic problems as all materials were source from the workplace itself, and thus the serum responses reflected an immunological reaction by certain workers to this mix of allergens actually derived from the worksite.

Furthermore, the additional cases of WR sensitization identified by bespoke allergen testing appeared to have clinical relevance, as judged by the strong association between the presence of WR respiratory symptoms and being not only sensitized but also currently allergen exposed for sensitized workers. Furthermore, the true level of sensitization to workplace allergens, as tested by the bespoke method, may in fact have been underestimated, raising the possibility that certain workers with occupational allergy could have been overlooked, as it was not possible to develop methods to detect specific IgE to all materials and products used.

There are a number of significant limitations to this study. These include the relatively low response rate, and as a consequence the risk of including both a selection and reporting bias. Despite a significant investment in time to meet with worker representatives, pressure of work was cited by the employer as the main reason for low response. Whilst information about lack of participation was not gathered from individual workers, it is possible that those who agreed to participate were more likely to complain of WR respiratory problems, although this effect might be considered generic between exposure groups, and as such may still allow a reasonable inference of differences in reported symptom levels across exposure groups. Specifically, workers would not have had prior knowledge of their sensitization status, and thus workers were unlikely to preferentially participate and report differently across sensitization groups. Similarly, the health worker effect could have influenced the findings of this cross-sectional study, reducing the levels of reported symptoms in a relatively fit surviving workforce. This would not, however, affect the individual relationship between reported symptoms and sensitization status.

Skin prick testing was not carried out in this study as the specific intent was to compare IgE response to standard latex allergen with bespoke workplace allergen testing. Testing for the latter with skin prick testing would be difficult, although the absence of skin prick testing in this study does raise the issue that sensitization status to latex may have been misattributed, if one assumes that a skin prick test is the gold standard for assessment of sensitization.

Similarly, individual breathing zone samples were not available for all workers within the study population. Nevertheless, walk through hygiene data were available, and, used in conjunction with current personal breathing zone sample results, exposure stratification was possible a priori.

In summary, we have described high levels of reported WR respiratory symptoms, certain of which are suggestive of occupational asthma, in a group of latex textile braiders. This study describes findings that are not traditionally associated with textile and cotton dust exposure, namely byssinosis and other consequences of organic dust and endotoxin exposure [Fishwick et al., 1994, 1996; Christiani et al., 2001; Latza et al., 2004; Oldenburg and Latza, 2007]. The presence of symptoms in workers with evidence of sensitization was strongly dictated by current latex exposure. Allergy testing performed to extracts developed from the workplace identified clinically significant cases of WR respiratory symptoms that otherwise would not have been identified, or misattributed to an irritant, or other, response. This finding has important implications not only for the identification and diagnosis of early cases of occupational respiratory disease, but also for the design of effective health surveillance programmes.

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