

Work-related asthma from cleaning agents versus other agents

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Background	Cleaning agents have been commonly implicated as causative or triggering factors in work-related asthma (WRA), mainly from epidemiologic studies. Relatively few clinical series have been reported.
Aims	We aimed to compare socio-demographic and clinical features among tertiary clinic patients with WRA exposed to cleaning and non-cleaning products.
Methods	Analyses were conducted on a patient database containing 208 patients with probable WRA referred to the asthma and airway centre at a tertiary centre hospital in Canada from 2000 to 2014. Chi-squared and independent samples <i>t</i> -tests were used to analyse categorical and continuous data, respectively.
Results	Twenty-two (11%) WRA cases were attributed to a variety of cleaning product exposures, 12 were diagnosed as occupational asthma (OA) and 10 as work-exacerbated asthma (WEA) (10% of all OA and 11% of all WEA). There were multiple exposures and the responsible agent(s) could seldom be clearly identified. Most frequent categories of exposure were surfactants, alcohols, disinfectants and acids. Compared to WRA with other exposures, those with cleaning agent exposures had a significantly larger proportion of females (82 versus 35%, $P < 0.001$), included a higher percentage of workers in healthcare (41 versus 4%, $P < 0.001$), and submitted more workers' compensation claims (86 versus 64%, $P = 0.05$). Other characteristics were comparable.
Conclusions	In a tertiary referral clinic, patients with WRA from cleaning agent exposure had clinical characteristics that were similar to those with WRA from other causes. Most frequent exposures were surfactants, alcohols, disinfectants and acids.
Key words	Asthma; cleaning products; healthcare workers; work-related asthma.

Introduction

Work-related asthma (WRA) remains one of the most common occupational lung diseases, reported to account for 16% of all adult-onset asthma [1]. It can be categorized into occupational asthma (OA) [2] and work-exacerbated asthma (WEA) [3]. OA is new-onset asthma that is caused by occupational exposure, while WEA is pre-existing asthma which is worsened by occupational exposure. WRA is a potentially preventable, yet debilitating condition that has serious socio-economic consequences attributed to healthcare costs, work disability, earning capacity, worker's compensation and psychosocial costs [4]. Since 2000, WRA cases caused by cleaning product exposures have increased in frequency in our tertiary clinic [5]. Cleaning products are the most common exposure identified in workers in healthcare

and education [5,6]. Given that primary prevention is the most effective way for preventing WRA [7], it is important to identify at-risk occupations/workplaces and investigate possible causative agents. Several suspected agents in cleaning have been identified, including glutaraldehyde, quaternary ammonium compounds (QATs), formaldehyde, bleach, chloramines, detergents, decalcifiers and fragrances [8–13]. Solvents and acids can also be used for cleaning in industrial processes. There have been relatively few reports detailing clinical features of cleaners with WRA. In a cross-sectional study of 142 Polish cleaners, 21% reported wheezing but all had negative skin and serum-specific IgE to disinfectants [14]. Another study of 50 Polish cleaners in a tertiary referral clinic identified WRA in 46%, of whom 15 had WEA, and eight had OA, based on a positive specific challenge (three to natural rubber latex, two to

chloramine and one each to glutaraldehyde, formaldehyde and benzalkonium chloride). Skin prick tests or serum-specific IgE were positive in two of the three with natural rubber latex and both with chloramine-induced OA [15]. However, comparison data between WRA from cleaning and non-cleaning product exposures are lacking and it may be useful to determine differences in order to understand risk factors. This study aimed to provide a comparison between socio-demographic and clinical features in patients exposed to cleaning and non-cleaning products, as well as provide a brief overview of their exposure agents and outcomes.

Methods

Analyses were conducted on a patient database containing 208 patients with probable WRA, i.e. those considered by a respiratory physician (S.M.T.) to be likely to have WRA after clinical investigations, as suggested in the American College of Chest Physicians Consensus Statement [7]. In most cases the diagnosis was reached from a combination of clinical history, objective confirmation of asthma, serial peak expiratory flow rates, symptoms, bronchodilator recordings, and methacholine challenges near the end of a working week and after 1 week or more off work. They had all been assessed by the senior responsible author in this study (a respiratory physician) after being referred to the asthma and airway centre of a tertiary centre hospital in Toronto, Canada from 2000 to 2015 [5]. Ethics approval was obtained from the hospital's research ethics review board.

The 'cleaning group' was initially identified from patients in our database whose reported exposure was coded as including cleaning products, including cleaning of machinery and equipment. The 'non-cleaning' group was defined as the remaining patients with WRA whose reported exposure did not include cleaning-related products. The database included gender, industry category, work status, smoking status, pulmonary function, methacholine challenge provocation concentration causing a 20% fall in FEV_1 (PC_{20FEV_1}), Ontario workplace safety insurance board (WSIB) compensation claim submitted, WRA caused by precipitating event, improvement when removed from work exposure, airflow obstruction at baseline and significant bronchodilator response. A chart review was then performed for those identified from the database with 'cleaners' as an exposure to identify other more probable causative exposures, type of cleaning agents and relation to symptoms (as reported by the patient) and chemical contents from material safety data sheets (MSDSs) included in the clinical files. Medication history and outcome were extracted through comparing the initial and most recent clinician review of the cleaning group. Medication categories included short-acting muscarinic antagonists and beta-agonists, long-acting

muscarinic antagonists and beta-agonists, inhaled corticosteroids, combination inhalers, leukotriene receptor antagonists and biologics. Diagnoses were based on the American College of Chest Physicians Consensus Statement [7].

Data were analysed using SPSS for Windows, version 22.0 (IBM Corporation, Armonk, NY, USA) and GraphPad Prism 6 (GraphPad Software, San Diego, CA, USA). Chi-squared or Fisher exact tests and independent samples *t*-tests were used to analyse categorical and continuous data, respectively. Cleaning product exposures were tallied and allotted into bins of cleaning-related products according to previously reported studies [8–11].

Results

Twenty-nine WRA cases included exposures to cleaning products in our database, but on chart review seven of these had other exposures more likely related to their WRA and were therefore excluded from further analyses (working in a pharmaceutical company, a wood mill, metal plating, yarn-making, leather work, papermaking and metal stamping). Of the 22 that were included, 12 were diagnosed as OA and 10 as WEA (10% of 114 with OA and 10% of 99 with WEA), Table 1. Among the 12 with OA from cleaning agents, five (42%) had a diagnosis of irritant-induced asthma. Eight WRA cases were seen between 2000 and 2007 (6% of WRA cases in that time), and 14 between 2008 and 2014 (18% of WRA cases seen in that time). Multiple cleaning products were identified including potential respiratory irritants and sensitizers. The most frequent exposures were surfactants, alcohols, disinfectants and acids. Immunologic evidence of specific sensitization to cleaning products could not be obtained and specific inhalation challenges were generally not performed, so a specific causative cleaner/chemical agent was usually not confirmed. In addition, the exposures were commonly mixed, and often included additional potential sensitizers or irritants. Compared with WRA with other exposures ($n = 191$), those with cleaning exposures had a significantly larger proportion of females (82 versus 35%, $P < 0.001$), had a higher percentage of workers in the healthcare industry (41 versus 4%, $P < 0.001$) and showed a trend to submitting more WSIB compensation claims (86 versus 64%, $P = 0.05$). Other characteristics as listed in Table 1 were comparable. Within the cleaning group, mean spirometry results showed mild airflow limitation (FEV_1 % predicted $72 \pm 17\%$, FEV_1/FVC $68 \pm 12\%$) and did not significantly differ from initial to follow-up visits. Sixteen were followed up at a mean time from diagnosis of 3.5 ± 3 years. Most of these patients required similar medication but four (25%) were able to discontinue asthma medications (three OA and one WEA) (Table 2). Of the 16 with follow-up, only one continued with the

Table 1. Patient group characteristics at time of diagnosis (total 213 patients with WRA)

	Cleaning group, <i>n</i> = 22 (10%)	Non-cleaning group, <i>n</i> = 191 (90%)	<i>P</i> -value, 95% CI ^a
OA	12 (55)	102 (53)	NS, 0.43 to 2.5
WEA	10 (45)	89 (47)	
Age (years)	49.5	45.7	NS, 0.88 to 8.35
Male/female	4/18 (18/82)	124/67 (65/35)	***, 0.04 to 0.37
Healthcare industry	9 (41)	8 (4)	***, 5.2 to 47.9
Actively working	17 (77)	146 (76)	NS, 0.37 to 3.0
Smoking status		(<i>n</i> = 189)	
1. Smoker	1 (5)	27 (14)	NS (smoker versus non-smoker, 0.04 to 2.5, smoker versus ex-smoker, 0.03 to 2.2)
2. Non-smoker	13 (59)	109 (57)	
3. Ex-smoker	8 (36)	53 (29)	
Pulmonary function (<i>n</i>)	<i>n</i> = 22	<i>n</i> = 191	
1. FVC % predicted	86.7 ± 14.6*	92.1 ± 16.3	NS, -1.8 to 12.6
2. FEV ₁ % predicted	72.2 ± 16.8	79.7 ± 18.9	NS, -0.8 to 15.8
3. FEV ₁ /FVC	68.5 ± 12.0	70.2 ± 11.0	NS, -3.2 to 6.6
Methacholine challenge (<i>n</i>)	<i>n</i> = 13	<i>n</i> = 121	
1. PC ₂₀ at provoking environment	3.7 ± 5.0	3.3 ± 4.6	NS, -2.3 to 3.9
2. PC ₂₀ while away from work >1 week	8.4 ± 6.3	7.1 ± 6.3	NS, -2.4 to 4.9
Submitted a WSIB claim	19 (86)	123 (64)	NS, 1.0 to 12.26
WRA caused by precipitating event	5 (23)	21 (11)	NS, 0.8 to 7.1
At least 3-fold improvement in methacholine	7 (32)	45 (25)	NS, 0.6 to 3.9
PC ₂₀ when removed from work exposure	<i>n</i> = 9 (78)	<i>n</i> = 105 (43)	NS, 0.9 to 23.6
Airflow limitation at baseline	12 (55)	92 (48)	NS, 0.5 to 3.1

^aCI, confidence interval; NS, not significant. *Error is expressed as mean ± SD. ****P* < 0.001.

same exposure and three reduced exposure while the others were able to avoid exposure. Among the four who no longer required asthma medications at follow-up, two had an initial diagnosis of OA, one had WEA (but a borderline specific challenge result) and one had irritant-induced OA. The latter had reduced exposure and the others completely avoided exposure to the suspected causes.

Discussion

Our data suggest that there are socio-demographic differences, but not clinical differences, between patients with WRA caused by cleaning and non-cleaning product exposures. Cleaning products or tasks affected a greater proportion of women and healthcare workers in this clinic review, consistent with the fact that most of the patients with WRA in the healthcare sector have exposure to cleaning products, and most of these are women [15]. These findings are also consistent with our previous findings of increased WEA in healthcare workers from review of Ontario workers' compensation claims [16]. A recent report of data from the USA has indicated that current asthma prevalence is highest by major industry in healthcare and social assistance (9%) [17]. The trend to a higher proportion of submitted compensation claims among those with WRA from cleaning agents might be explained by the relatively increased proportion of healthcare workers, and potential ability to be accommodated in alternative work within a

healthcare setting. It is possible that workers in a small company or those self-employed may have more difficulty with work-accommodation and may not wish to initiate a claim due to concerns about their career, although we did not assess that in this study. Also, there was a relatively high proportion of OA in the cleaners from accidental high-level exposures that perhaps led to initiation of a claim by an emergency department physician, or employer-initiated compensation claims for OA.

The relatively high proportion of OA patients with irritant-induced asthma in this series (42%) compared with previous series of WRA likely reflects the propensity for accidental high-level exposure to cleaning products. Previously reported triggers for asthma were found among the implicated cleaning-related chemicals, including glutaraldehyde, formaldehyde, QATs and bleach. While the most common exposures were surfactants, alcohols, disinfectants and acids (data not shown), the objective cause of the patients' asthma was seldom clearly identified since exposures were mixed, there are no validated immunologic tests for cleaning products, and specific inhalation challenges were not performed. Unlike Polish reports [14,15] and an older Canadian study [18] we did not identify natural rubber latex as a likely cause in this study, likely due to changes in latex glove use in Ontario since the late 1990s [19]. The range of job and exposure-agent descriptions of patients in this clinical report highlights the breadth and complexity of exposures involved in WRA

Table 2. Selected features and exposures of individual patients

No.	OA/WEA*	Rhino-conj. Y/N, atopy Y/N, smoker Y/N/Ex male/female	Exposure	Methacholine PC ₂₀ mg/ml work/off	Medications at diagnosis	Medications at follow-up	Methacholine PC ₂₀ mg/ml at follow-up	Follow-up** (mo)	Ongoing exposure to suspect agent(s)	Other comments
1	OA	N, Y, N M	Making personal cleaning products	2.8/>16	ICS, SABA	ICS, SABA		3	N	Ethylene oxide, formaldehyde, acrylic compounds
2	OA-IIA	N, Y, Ex (5 pk y) F	Sodium triphosphate, NaOH	0.1	ICS/LABA, SABA	ICS/LABA, SABA		11	N	Acute exposure in food processing plant
3	OA-IIA	N, Y, N F	Hospital cleaner	1.0	ICS, SABA	No follow-up			N	Bleach, toilet bowl cleaner, mixed
4	OA	Y, N, N F	Dry cleaner, perchlorethylene, trichloroethylene	3.0/16	ICS/LABA, SABA	ICS/LABA, SABA	1.5	36	Y	
5	WEA	N, N, Ex (7 pk y) M	Floor stripping/sealing, acids, ethanalamine, acrylics, formaldehyde	<0.13/nd	ICS/LABA, SABA, LTA, OCS	ICS/LABA, SAMA, LTA	FEV ₁ 32%	108	N	Left after 10 years of symptoms. Prednisone ~100 days/year when working, not needed off work
6	WEA	N, N, N F	Laundry worker, QATs, bleach, ammonia, NaOH	nd/0.3	ICS/LABA, SABA, SAMA, LTA	ICS/LABA, SABA, LTA		5	N	
7	WEA	N, N, Y (30 pk y) F	Nursing home RPN, QATs, Glu, latex	nd/>16	ICS/LABA, SABA	None		3	N	SIC borderline positive with Glu, negative with QAT, skin test latex negative
8	WEA	Y, Y, Ex (13 pk y) F	Radiation therapist with glutaraldehyde exposure	0.4/0.7	ICS, SABA	No further follow-up			N	Glutaraldehyde workplace challenge negative
9	OA-IIA	N, N, N F	Food packaging company, cleaning equipment	1.4/12	ICS/LABA, SABA	No follow-up			N	Acute exposure NaOH and acids to clean conveyor belts
10	OA	N, N, N F	Endoscope cleaning, Glu, OPA, enzymes	1.0/7.2	ICS/LABA	ICS/LABA	>16	72	N	Skin tests negative latex, enzymes. Developed ILS
11	OA-IIA	Y, N, N F	Hospital housekeeper	nd/PC _{100raw} 7.3	ICS/LABA, SABA	ICS/LABA, SABA	FEV ₁ 83%	16	N	Spill of formaldehyde, variable spirometric performance
12	OA	Y, N, N F	Hospital instrument cleaning, Glu, enzymes	6.9/>16	SABA	None except with colds		84	N	Skin tests negative latex, enzymes
13	OA	N, Y, N F	Nursing home kitchen worker, QATs, acids, bleach, NaOH	FEV ₁ 46%/PC ₂₀ > 16 (FEV ₁ 86%)	ICS/LABA, LAMA, SABA, LTA, OCS, omalizamab	ICS/LABA, LAMA, SABA, LTA, OCS, omalizamab	FEV ₁ 91%	30	N	Skin test negative QAT
14	OA	N, Y, Ex F	Hospital cleaning, QATs, acids, bleach	0.4/1.8	ICS, SABA	None		54	N	Skin test negative latex
15	OA-IIA	N, Y, Ex (4 pk y) F	Laundry in long-term care, bleach, acids, alkalis, enzymes	0.96/nd	ICS, SABA	None		12	Reduced exposure	Chemical leak at work
16	WEA	Y, N, Ex (7.5 pk y) F	Hairdressing salon, cleaners, shampoos, bleach, hair dye, spray	FEV ₁ 47%/45%	ICS/LABA, SABA, LTA, OCS	No follow-up				Also ASA sensitivity and nasal polyps
17	WEA	Y, Y, N M	Waste water plant, cleaning, acids	FEV ₁ 45%/41%	ICS/LABA, SABA	ICS/LABA, SABA	FEV ₁ 40%	98	Reduced exposure	Moved to outdoor work

Table 2. Continued

No.	OA/WEA*	Rhino-conj. atopy Y/N, smoker Y/N/Ex male/female	Y/N, Exposure	Methacholine PC ₂₀ mg/ml work/off	Medications at diagnosis	Medications at follow-up	Methacholine PC ₂₀ mg/ml at follow-up	Follow-up** (mo)	Ongoing exposure to suspect agent(s)	Other comments
18	WEA and COPD	Y, Y, Ex (30 pk y) M	School cleaner, QATs, monoethanolamine, latex, triclosene	FEV ₁ 44%	ICS/LABA, LAMA, SABA	No follow-up			Stopped work, long-term disability	PEFs worse at work, skin tests negative latex, QATs
19	WEA	N, Y, Ex (22.5 pk y) F	Hospital housekeeper, QATs, bleach	FEV ₁ 45%/67%	ICS/LABA, SABA, LTA, OCS, omalizamab	ICS/LABA, SABA, LTA, OCS, omalizamab	FEV ₁ 69%	5	N Able to avoid bleach	Skin test negative QATs
20	WEA	Y, N, N F	School laundry, tall oil, sulfonic acid, triethanolamine, sulphites	14/4.7	ICS/LABA, SABA	ICS, SABA		72	Reduced exposure and reported improvement but worsened with later ozone exposure in laundry (PC ₂₀ 0.4 mg/ml)	
21	WEA	N, Y, N F	Hotel cleaner, dust, smoke, air fresheners, QATs	nd/1.4	ICS, SABA	No follow-up				Skin tests negative QATs, latex, positive dust mites, fungi
22	OA	Y, Y, N F	Fast-food restaurant worker, QATs	1.9/6.2	ICS/LABA, SABA	ICS/LABA, LAMA, SABA	FEV ₁ /FVC 74%	60	N	Skin test negative QATs

*OA, occupational asthma; WEA work-exacerbated asthma; IIA, irritant-induced asthma; COPD, chronic obstructive pulmonary disease; rhino-conj, rhinoconjunctivitis; pk y, pack years; FEV₁, forced expiratory volume in 1 s; PC₂₀, provocation concentration causing a 20% drop in FEV₁; PC_{100raw}, provocation concentration causing 100% increase in airways resistance; mo, months; nd, not done; ICS, inhaled corticosteroid; LABA, long-acting bronchodilator; SABA, short-acting bronchodilator; LAMA, long-acting muscarinic agent; LTA, leukotriene antagonist; OCS, oral corticosteroids; SIC, specific inhalation challenge; Glu, glutaraldehyde; FVC, forced vital capacity; PEF, peak expiratory flow; RPN, registered practical nurse; OPA, orthophthalaldehyde; ASA, acetylsalicylic acid; ILS, irritable larynx syndrome; Y, yes; N, no; M, male; F, female.

**Follow-up in months after diagnosis, mean $\pm$ SD, 41.8 $\pm$ 36.5 months.

that may be attributed to ‘cleaning’ as a task or ‘cleaning agents’, such as may be reported in questionnaire studies. In many cases, whether the asthma was mediated through allergy or irritant mechanisms was unclear and difficult to determine from clinical findings and reported exposure.

At an average of 3.5 years after first clinic visit, most patients still required similar medication (Table 2). However, 25% of the WRA patients with follow-up and 25% of all the OA patients required no regular asthma medication at follow-up showing clinical resolution of asthma after modification in work exposure to avoid or reduce further exposure to the cleaning product(s). This is similar to the proportion of OA patients reported to clear on follow-up in systematic reviews [20,21].

As previously reported, we have seen an absolute and relative increase in cases related to cleaning products seen in this clinic from 2008 to 2014 compared with the previous 8 years [5], but overall the numbers seen and diagnosed are relatively low compared with the number of workers exposed to cleaning products, suggesting under-recognition. Some additional patients in this clinic had upper respiratory symptoms or other work-attributed symptoms without asthma, but our database and this report, only includes patients with WRA.

WRA is known to be a debilitating but preventable disease. Reducing exposure to cleaning products implicated in asthma, especially in relevant industries such as healthcare, may lead to a higher proportion of women and healthcare workers being able to continue their work, improving their quality of life and lowering socio-economic burden. A focus on prevention has been advised for these workers [19,22] as for other groups at increased risk and may result in less WRA.

Key points

- Eleven per cent of work-related asthma cases were among those exposed to cleaning agents: 55% of these were likely occupational asthma and 45% work-exacerbated asthma.
- Causative agents could not be determined with certainty, but the most common exposures were surfactants, alcohols, disinfectants and acids.
- Compared with work-related asthma from other exposures, those attributed to cleaning agents were more likely to be in women and healthcare workers, but other demographic features did not differ.

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Competing interests

None declared.

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