Work-Related Asthma in Health Care in Ontario

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Background *The health of workers in health care has been neglected in the past. There are few reports regarding occupational asthma (OA) in this group, and work-exacerbated asthma (WEA) has rarely been considered.*

Methods *We examined the frequency of claims for OA and WEA allowed by the compensation board in Ontario, Canada for which industry was coded as "health care" between 1998 and 2002, to determine the frequency of OA and WEA, causative agents, and occupations.*

Results *During this period, five claims were allowed for sensitizer OA, two for natural rubber latex (NRL), and three for glutaraldehyde/photographic chemicals. The two NRL cases occurred in nurses who had worked for >10 years prior to "date of accident." There were 115 allowed claims for WEA; health care was the most frequent industry for WEA. Compared to the rest of the province, claims in health care made up a significantly greater proportion of WEA claims (17.8%) than OA (5.1%) (odds ratio, 4.1, 95% CI 1.6–11.6; $P = 0.002$). The rate of WEA claims was 2.1 times greater than that in the rest of the workforce ($P < 0.0001$). WEA claims occurred in many jobs (e.g., clerk), other than "classic" health care jobs such as nurses, and were attributed to a variety of agents such as construction dust, secondhand smoke, and paint fumes.*

Conclusions *WEA occurs frequently in this industrial sector. Those affected and attributed agents include many not typically expected in health care. The incidence of OA claims in this sector in general was low; the continued low number of OA claims due to NRL is consistent with the successful interventions for prevention.* Am. J. Ind. Med. 54:278–284, 2011. © *2011 Wiley-Liss, Inc.*

KEY WORDS: occupational asthma; work-exacerbated asthma; health care workers; natural rubber latex; glutaraldehyde; cleaning agents; construction dust; molds; paint fumes

INTRODUCTION

The health of health care workers (HCWs) has been neglected in the past with the focus on infectious diseases/needlestick injuries, violence, and musculoskeletal problems. Occupational asthma (OA) has been recognized in this sector more recently, due to sensitizers present in hospitals such as natural rubber latex (NRL) [LaMontagne et al., 2006; Vandenplas et al., 2009], glutaraldehyde [Gannon et al., 1995; Curran et al., 1996; Di Stefano et al., 1999; Dimich-Ward et al., 2004], and medications [Malo et al., 1990]. We previously described work-related respiratory problems associated with Ontario health care settings. These reports included associations between work-related asthma symptoms and various tasks, chemical spills, and others exposures

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during radiographic processing/developing among medical radiation technologists or radiographers [Liss et al., 2003]. We have shown the successful reduction of OA due to NRL at the population [Liss and Tarlo, 2001] and clinic or institution [Tarlo et al., 2001] levels.

Work-related asthma is the most common chronic occupational lung disorder in North America. Ontario, Canada is one of the few jurisdictions in which work-exacerbated asthma (WEA) is compensated, in addition to OA. There are few population-based reports of work-related asthma in health care settings, and WEA has not often been considered. Moreover, in some past reports there have been few details on exposures or agents and the focus has been primarily on HCWs only, although there are many other occupations present in health care workplaces. In a prospective study of the employed population of Finland, Karjalainen et al. [2001] examined the incidence of asthma for various occupational categories relative to administrative work, and observed a slight increase in medical and nursing work (RR 1.1–1.2). Investigators from NIOSH [Moon Bang et al., 2005] examined asthma prevalence data based on the National Health Interview Survey 2001, and found a modestly increased odds ratio (OR) of 1.3 for physician-diagnosed asthma in the health care industry among white females with no increase among males.

More recently, Delclos et al. [2007] examined occupational risk factors and asthma among health care professionals with a detailed questionnaire mailed to a random sample of Texas physicians, nurses, respiratory therapists, and occupational therapists. In their final multivariate models looking at associations with occupational exposures, they observed moderate to strong associations with cleaning agents: for instrument cleaning, the OR was 2.2 based on reported asthma and 1.3 based on bronchial hyperresponsiveness (BHR)-related symptoms, respectively; for surface cleaners, 2.0 and 1.6, respectively; for aerosolized medications, 1.7 and 1.4; and for work with adhesives/solvents/gases, when used in patient care, 1.7 for both measures, and with spills at work, 2.0 for BHR-related symptoms. For use of latex gloves, an OR of 2.2 for reported asthma was observed during 1992–2000 but there was no increase after 2000. In an accompanying editorial, Rask-Andersen and Tarlo [2007] noted that cleaning products had been previously associated with professional cleaners but less recognized among health care professionals, emphasizing the importance of bystander exposure; and that this study underscored the possibility of mixed exposures that may occur in many work settings (exacerbating factors as well as sensitizers).

An international prospective population-based study (ECRHS-II) examined exposure to substances in the workplace and new-onset asthma [Kogevinas et al., 2007]. Asthma was assessed by methacholine challenge (MC) and by questionnaire data on asthma symptoms. Exposure was defined by high-risk occupations, an asthma-specific job exposure matrix with additional expert judgment and through self-report of acute inhalation events. Nursing was associated with an excess risk of asthma (adjusted RR 2.2, 95% confidence interval [CI] 1.3–4.0; $P = 0.007$).

These studies recommended [Moon Bang et al., 2005] that further details of agents and occupations associated with asthma in the health care industry are needed, preferably from a population-based perspective. An important proportion of the workforce is employed in health care. For example, in the province of Ontario, Canada with a population of 12.7 million in 2006, the number of health personnel (e.g., dental hygienists, dietitians, respiratory therapists, social workers, dentists, nurses, nurse practitioners, physicians, and others) was 193,223 persons in 2002 and 215,896 persons in 2006 [CIHI, 2007]. However, the total number of employees in health care facilities and services registered with the Ontario compensation board (Workplace Safety and Insurance Board, WSIB) was considerably greater (approximately 464,000 in 2006). As part of a review of work-related asthma that we conducted in the province of Ontario, we examined the frequency of claims for OA and WEA allowed by the Ontario compensation board in the health care industry over a 5-year period to determine the relative frequency of OA and WEA, the causative agents, and the occupations giving rise to the claims.

MATERIALS AND METHODS

Study Population

The study protocol was reviewed and approved by the Research Ethics Board of the University of Toronto. We first identified from the occupational disease database of the WSIB, claims that contained primary diseases that included asthma and/or Reactive Airways Dysfunction syndrome (RADS) for which the ''date of accident'' was between January 1998 and December 2002 (five consecutive years). We selected this time period because an analysis of asthma claims by the WSIB indicated that a 3-year lag was necessary to ensure that most claims had been filed with the WSIB and that an entitlement decision had been made.

Description of WSIB

The Workplace Safety & Insurance Board (WSIB) administers a single, no-fault workplace insurance system for the employers and workers of Ontario. Approximately 72% of the Ontario workforce is covered under the *Workplace Safety and Insurance Act*. The WSIB is entirely financed by employer premiums. In accordance with the *Workplace Safety and Insurance Act*, the WSIB provides benefits for loss of earnings, non-economic loss benefits for permanent impairment, payments for healthcare

expenses, assistance to facilitate return to work, and survivor benefits in case of work-related fatalities. Well over 90% of health-care organizations and workers, including all hospitals, long-term care facilities, and most community-based programs are covered by the WSIB.

Claims are adjudicated based on the merits and circumstances of each case. The adjudicator considers the nature of the work and the nature of the injury or illness in consultation with occupational hygienists, scientists and medical professionals to make an entitlement decision. Decisions are based on a balance of probabilities (more probable than not).

The ''date of accident'' is the date which is identified in the WSIB file as the date of exposure (in the case of a high accidental exposure leading to symptoms) or the date of symptom onset. This initial evaluation sample was then screened to exclude claims that were not allowed, were still pending WSIB decision, or were withdrawn by the claimants, and subsequently screened for duplicate entries, or erroneously entered date of accident outside the period of interest. The final data extraction was performed by a single trained physician reviewer who abstracted information from each claim file using a standardized abstraction form for each subject adapted from our previously published studies [Tarlo et al., 1997, 2002].

For the current report, we reviewed all claims allowed for OA and WEA in the WSIB database over this 5-year period, for which the industry was coded as health care. This may include jobs that are considered not directly health-care but are performed in a health care facility. The province of Ontario contains approximately 38% of the total population of Canada. Data were obtained as in our previous studies [Tarlo et al., 1997, 2002] for the following information if available: demographics (date of birth, gender, smoking status if available, occupation); exposure history including agents and duration; occupation; history of previous asthma before starting job for which the WSIB claim had been initiated; history of nasal, eye, and lower respiratory symptoms and their frequency; clinical diagnosis made by the most specialized assessing physician; WSIB decision; and objective tests (spirometry, peak flow studies, MC tests, skin tests to common allergens, and workplace substances, if available). The files were reviewed and abstracted by the physician research assistant, with subsequent discussion with the investigators for consensus as needed. That is, for those cases where the physician performing the extraction thought that there may be a different diagnosis from the WSIB claim decision, the data were presented by the extracting physician and there was discussion and further data review to come to an informal consensus regarding the most likely clinical diagnosis from the data available. The categorization of outcome in this study (i.e., final decision presented here after review and discussion) was different in some cases from that originally coded by the WSIB.

Definitions for purposes of this study (may not reflect subtle differences between the entities given the source from an administrative data base).

Sensitizer-Induced OA

Subjects developed asthma during working life; presence of work-related symptoms; presence of a known or presumed work sensitizer at the time of asthma onset/recurrence; asthma symptoms improved when an affected individual was off work (weekends or holidays); objective tests of asthma demonstrated at least 12%; and 180 ml post-bronchodilator improvement in FEV_1 or methacholine (or equivalent) PC_{20} of 8 mg/ml or less). The group was further subdivided into three subgroups:

- *Definite OA*: at least one positive work-related test (i.e., work-related changes on serial PEFR recordings, at least threefold improvement in PC20 during a period away from work versus during a work period, positive skin test to a relevant sensitizer, or positive specific challenge) with no negative work-related tests.
- *Probable OA*: at least one positive work-related test but there could be a negative response to another test (e.g., positive serial PEF findings but no significant improvement in methacholine response).
- *Possible OA*: one objective work-related test performed but was negative, but others were not performed.

Work-Exacerbated Asthma

Claimants with presence of pre-existing asthma (present before starting job for which the WSIB claim had been initiated), and exacerbation of symptoms at workplace.

RADS and Irritant-Induced Occupational Asthma (IIA)

RADS and IIA were defined as in previous publications [Brooks et al., 1985; Tarlo and Broder, 1989; Chatkin et al., 1999]. However no cases of RADS or IIA were identified in health care.

Statistical Analysis

We obtained for the year 2000 (the mid-point of the study period), numbers of workers in health care and in the rest of the provincial workforce that were covered by the WSIB from the ''Premium Rate Manuals'' on the WSIB website. We compared continuous variables with *t*-test or Wilcoxon test and categorical variables with the Chi-square test as appropriate. Analyses were conducted with Epistat and SAS versions 9.1 and 8.2.

RESULTS

Claims for the Province as a Whole

Of the 1,825 claims originally submitted for asthma for the province as a whole, between 1998 and 2002 inclusive, 589 were abandoned when the claimant did not respond to requests for information, and 281 claims were denied as considered unlikely to be related to work. After excluding claims that were pending with no decision (29) and duplicates, 893 claims remained that underwent file abstraction. Of these, after review, abstraction and consensus discussion, 645 were considered as WEA and 99 were classified as sensitizer-induced OA (plus 12 others were categorized as RADS or IIA). However, none of the latter 12 claimants had occupations or industries in health care and were not analyzed further here. In addition, 110 claims were classified as asthma-like reactions (i.e., transient asthma-like symptoms that were acute, emerging within 24 hr, persisting for less than 6 weeks in subjects with no pre-existing asthma and typically no objective documentation of asthma) and 27 as insufficient information for definitive categorization (the latter two categories are not further considered).

Claims in the Health Care Industry

In the health care industry, five claims were allowed for sensitizer-induced OA and 115 for WEA. Compared to the rest of the province, claims in health care made up a significantly greater proportion of WEA claims (17.8%) than did OA (5.1%) (OR 4.1; 95% CI 1.6–11.6; $P = 0.002$ by Chi-square) (Table IA). The most frequent industry for WEA in the province was the health care industry. The rate of allowed claims for WEA was significantly greater in health care than in the rest of the workforce (RR 2.1, 95% CI 1.7–2.6; $P < 0.0001$; Table IB). The claims occurred predominantly among females for both WEA (106/9) and OA (5/0). The WEA claimants were significantly younger than those with OA (41.6 $\pm$ 10.1 [mean $\pm$ SD] vs. 53.1 $\pm$ 4.1; $P = 0.013$ by *t*-test) and the time off work was much longer for those with OA than WEA (mean 309.6, median 108.0 days vs. mean 8.3, median 1.0 days; $P = 0.01$ by Wilcoxon test).

Details on OA Claims

Details regarding the agents and occupations for the OA claims are listed in Table II; four of the five were considered to have definite supporting evidence. Regarding OA due to NRL, the current study identified two allowed OA claims for NRL: one with year of accident in 2000, and one in 1998. Both were registered nurses who had positive skin tests to NRL and had worked in their jobs for more than 10 years, prior to the year of accident. The date of onset of sensitization was not clear. Additional information was sought from the WSIB files to explore this. Case 1 had started work in this location 13 years prior to year of ''onset'' likely using powdered gloves at that time. She had a 6-year history of cough worse at work, so the date of onset of sensitization is not clear but may have been considerably earlier than 2000. She had work-related changes in methacholine sensitivity and peak expiratory flow rates (PEFRs); however, the evidence for sensitization to NRL was weak. Case 4 initially had asthma symptoms worse at work but negative MC test prior to 1998. In 1998, the symptoms continued, she had a positive MC test, PEFRs worse at work, and a clearly positive NRL skin prick test. The other 3 OA claims identified were related to glutaraldehyde or photographic chemicals in occupations identified as radiological technicians or radiation therapist.

TABLE IA. Asthma Claims in Health Care, Ontario, 1998–2002 (No. [%])

	Health care	Ontario minus health care	Ontario total
WEA	115 (17.8)	530 (82.2)	645
OA	5 (5.1)	94 (95.9)	99
Total	120	624	744

$P = 0.002$ by Chi-square. WEA, work-exacerbated asthma.

TABLE IB. Rates of WEA Asthma Claims in WSIB-Covered Workforce, Ontario, 1998–2002

	Health care	Ontario workforce minus health care
No. of claims	115	530
Workforce population covered	346,743	3,399,575
Rate per 1,000	0.33	0.16

RR = 2.1 (95% CI 1.7–2.6), $P < 0.0001$. WEA, work-exacerbated asthma.

TABLE II. Details of Sensitizer Occupational Asthma Claims in Health Care, Ontario, 1998–2002

Case no. (year)	Occupation/setting	Agent	Evidence category
1 (2000)[a]	RN/hospital	Natural rubber latex (possible)	Definite
2 (2000)	Radiological technician/medical lab	Glutaraldehyde	Possible
3 (2001)	Radiological technician/hospital	Glutaraldehyde (photographic chemicals)	Definite
4 (1998)[a]	RN/hospital	Natural rubber latex	Definite
5 (1998)	Radiation therapist/hospital	Glutaraldehyde	Definite

[a]See text.

Details on WEA Claims

The frequency of reported occupations or jobs for the WEA claimants is shown in Table III. In addition to ''classic'' health care jobs such as RNs, nursing assistants, and medical and radiation technologists, workers in a variety of other occupations such as cleaners, laborers and clerks/receptionists were also affected.

Attributed exposures: There was a broad variety of agents to which the WEA cases were attributed (Table IV). Some of these occupations and exposures are unusual or unexpected for this industry, such as second-hand smoke and perfume.

TABLE III. Frequency of Occupations Recorded Among WEA Claimants in Health Care Industry, Ontario, 1998–2002

Jobs	N (%)
Registered nurses	34 (29.6)
Reception/clerk/telephone operator	13 (11.3)
Social workers	10 (8.7)
Nursing attendants/aides/assistants	10 (8.7)
Dietary/kitchen	9 (7.8)
Cleaners/housekeeping	7 (6.1)
Medical (lab) technician/technologist	4 (3.5)
Youth workers	4 (3.5)
Team leader/nursing supervisor/case manager	4 (3.5)
Homemaker/health care aide	4 (3.5)
Radiological technologist	3 (2.6)
Respiratory technician	2 (1.7)
General laborer	2 (1.7)
Personal service workers	2 (1.7)
Miscellaneous (community garden counselor; needs determination officer; health care records; pharmacy technician; discharge planner; day care worker; radiation therapist—1 each)	7 (6.1)
Total	115

WEA, work-exacerbated asthma.

DISCUSSION

These findings give some insight into the occurrence of work-related asthma in the health care industry from a population-based perspective using compensation data. Over the 5-year study period, the health care industry represented an important proportion of the total provincial claims for WEA (as a proportion of work-related asthma), and in fact, was fourfold greater than that for OA, as compared with the rest of the province. Furthermore, the rate of allowed WEA claims

TABLE IV. Frequencies of Exposure Agents in WEA Claimants in Health Care Industry, Ontario, 1998–2002

Agent	N (%)
Cleaning agents/bleaches/fresheners	23 (20.0)
Construction/renovation dust	18 (15.7)
Other fumes/odors	10 (8.7)
Molds	9 (7.8)
Paint fumes	9 (7.8)
Perfumes	9 (7.8)
Chemicals not otherwise specified (NOS)	5 (4.3)
Second-hand smoke	5 (4.3)
Latex	3 (2.6)
Airborne dust	3 (2.6)
Environmental factors (heat, cold, tight environment)	3 (2.6)
Carpet glue fumes	3 (2.6)
Ammonia products	3 (2.6)
Animals	2 (1.7)
Fire smoke	2 (1.7)
Unknown	2 (1.7)
Other wood dust	1 (0.9)
Other smoke	1 (0.9)
Toxic chemicals fumes	1 (0.9)
Exhaust fumes (carbon monoxide)	1 (0.9)
Glutaraldehyde	1 (0.9)
Carbon dioxide	1 (0.9)
Total	115

WEA, work-exacerbated asthma.

in this sector was more than double that in the rest of the province. As noted, many occupations besides RNs and other ''true'' HCWs were affected in these settings, such as reception/clerk, which represented the second most common job title. In addition, these workers attributed their condition (for WEA) to exposure to cleaning agents, construction/renovation dust, paint fumes, second-hand smoke, perfume, and other (irritant) exposures not typically considered associated with work tasks among nursing and other HCWs. This is consistent with the presence of bystander and mixed exposure as proposed by Rask-Andersen and Tarlo [2007]. It is possible that the spectrum of industrial settings and occupations included by the WSIB in Ontario in its definition of the ''health care'' industry is somewhat broader than is commonly considered, resulting in the inclusion of some unexpected occupations. However, the vast opportunities for jobs and exposures within health care settings is apparent and needs to be emphasized.

The number of claims for OA among HCWs was much smaller than that for WEA, similar to the relative frequencies of OA and WEA that we observed in the entire population for the province [Buyantseva et al., 2007]. The OA claims were due to NRL and glutaraldehyde, agents that have been reported previously among HCWs [Gannon et al., 1995; Vandenplas et al., 2009]. The observed low number of OA claims due to NRL (less than one per year) in the more recent time period (down from a peak of 11 cases per year among HCWs in the mid-1990s) is consistent with the decline observed by us previously [Liss and Tarlo, 2001], with the time windows reported by Delclos et al. [2007], and with the recent report from the same group that focused on nursing professionals [Arif et al., 2009]. Given that the two nurses that we identified had worked in health care for considerable periods of time prior to the year of accident, it is possible that they actually became sensitized and/ or developed their work-related symptoms some years prior to submitting their compensation claims, at a time when powdered moderate-protein NRL gloves were in use. Regardless, these data further illustrate the successful example of the primary prevention intervention involving the introduction of non-powdered, low-protein NRL gloves, and non-latex gloves [Liss and Tarlo, 2001; Tarlo et al., 2001; Allmers et al., 2002; LaMontagne et al., 2006; Vandenplas et al., 2009].

The other attributed agent (in three workers) over the time period in this study was glutaraldehyde or photographic chemicals in radiological technicians (also known as medical radiation technologists). As previously noted in our study of these HCWs [Liss et al., 2003], the change in process to digital imaging is expected to prevent OA in these workers associated with processing and developing chemicals, although glutaraldehyde and ortho-phthaldehyde are still used in other health care processes such as cleaning of endoscopes.

It is not surprising that the time off work was much greater for OA than for WEA, as patients with the latter can usually stay in the same job, but it may be necessary to reduce exposure to non-specific workplace triggers, for example, by optimizing medical management of the asthma, moving to a different work area, or making changes in the ventilation system [Tarlo et al., 1998, 2008; Tarlo and Liss, 2003]. On the other hand, for the management of (sensitizer-induced) OA, cessation of further exposure to the causative agent is recommended if feasible [Tarlo and Liss, 2003; Tarlo et al., 2008].

We did not observe any claims for RADS or IIA in health care but in our previous studies the proportion of OA cases due to these conditions was quite low (5–10%). So given the small number of OA cases, the absence of RADS cases was not surprising.

This study has limitations that should be noted. The estimation of both OA and WEA using compensation data may be incomplete due to cases not being recognized and/or reported by health care providers, and reluctance by workers to submit claims. WEA is not compensated in all jurisdictions so this aspect may not be comparable to all settings. Furthermore, the documentation available in the WSIB files for WEA tends to be limited compared to that for OA. An additional limitation is that we do not know the generalizability of these data, from 1998 to 2002, to the present day.

In conclusion, as noted by Delclos et al. [2007] and Rask-Andersen and Tarlo [2007], the health care setting presents opportunities for exposure to respiratory irritants and sensitizers. Health care was the most frequent industry for WEA in the province. Our findings add to the evidence to justify implementing and evaluating measures in health care settings that have been neglected in the past including identification of agents, implementing exposure controls, and medical surveillance as appropriate for early diagnosis.

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REFERENCES

Allmers H, Schmengler J, Skudlik C. 2002. Primary prevention of natural rubber latex allergy in the German health care system through education and intervention. J Allergy Clin Immunol 110:318–323.

Arif AA, Delclos GL, Serra C. 2009. Occupational exposures and asthma among nursing professionals. Occup Environ Med 66:274–278.

Brooks SM, Weiss MA, Bernstein IL. 1985. Reactive airways dysfunction syndrome (RADS). Persistent asthma syndrome after high level irritant exposures. Chest 88:376–384.

Buyantseva LV, Liss GM, Luce CE, Tarlo SM. 2007. Work-aggravated asthma and occupational asthma in Ontario: Relative frequency and exposures. Am J Resp Crit Care Med 175(Abstracts Issue): A804.

Chatkin JM, Tarlo SM, Liss G, Banks D, Broder I. 1999. The outcome of asthma related to workplace irritant exposures: A comparison of irritant-induced asthma and irritant aggravation of asthma. Chest 116:780–785.

CIHI (Canadian Institute for Health Information). 2007. Health personnel database. Number of health personnel in selected professions, by registration status, 2006. http://secure.cihi.ca/cihiweb/dispPage.jsp?cw_page=AR_1446_E&cw_topic=1446.

Curran AD, Burge PS, Wiley K. 1996. Clinical and immunologic evaluation of workers exposed to glutaraldehyde. Allergy 51:826–832.

Delclos GL, Gimeno D, Arif AA, Burau KD, Carson A, Lusk C, Stock T, Symanski E, Whitehead LW, Zock J-P, Benavides FG, Anto JM. 2007. Occupational risk factors and asthma among health care professionals. Am J Respir Crit Care Med 75:667–675.

Di Stefano F, Siriruttanapruk S, McCroach J, Burge PS. 1999. Glutaraldehyde: An occupational hazard in the hospital setting. Allergy 54:1105–1109.

Dimich-Ward H, Wymer ML, Chan-Yeung M. 2004. Respiratory health survey of respiratory therapists. Chest 126:1048–1053.

Gannon PF, Bright P, Campbell M, O'Hickey SP, Burge PS. 1995. Occupational asthma due to glutaraldehyde and formaldehyde in endoscopy and X-ray departments. Thorax 50:156–159.

Karjalainen A, Kurppa K, Martikainen R, Klaukka T, Karjalainen J. 2001. Work is related to a substantial portion of adult-onset asthma incidence in the Finnish population. Am J Respir Crit Care Med 164:565–568.

Kogevinas M, Zock J-P, Jarvis D, Kromhout H, Lillienberg L, Plana E., Kogevinas M, Zock JP, Jarvis D, Kromhout H, Lillienberg L, Plana E, Radon K, Torén K, Alliksoo A, Benke G, Blanc PD, Dahlman-Hoglund A, D'Errico A, Héry M, Kennedy S, Kunzli N, Leynaert B, Mirabelli MC, Muniozguren N, Norbäck D, Olivieri M, Payo F, Villani S, van Sprundel M, Urrutia I, Wieslander G, Sunyer J, Antó JM. 2007. Exposure to substances in the workplace and new-onset asthma: An international prospective population-based study (ECRHS-II). Lancet 370(9584): 336–341.

LaMontagne AD, Radi S, Elder DS, Abramson MJ, Sim M. 2006. Primary prevention of latex related sensitisation and occupational asthma: A systematic review. Occup Environ Med 63:359–364.

Liss GM, Tarlo SM. 2001. Natural rubber latex-related occupational asthma: Association with interventions and glove changes over time. Am J Ind Med 40:347–353.

Liss GM, Tarlo SM, Greene J, Doherty J, Purdham J, McCaskell L, Kerr M. 2003. Physician-diagnosed asthma, respiratory symptoms and associations with workplace tasks among radiographers in Ontario, Canada. Occup Environ Med 60:254–261.

Malo J-L, Cartier A, L'Archevêque J, Ghezzo H, Lagier F, Trudeau C, Dolovich J. 1990. Prevalence of occupational asthma and immunologic sensitization to psyllium among health care personnel in chronic care hospitals. Am Rev Respir Dis 142:1359–1366.

Moon Bang K, Hnizdo E, Doney B. 2005. Prevalence of asthma by industry in the US population: A study of 2001 NHIS data. Am J Ind Med 47:500–508.

Rask-Andersen A, Tarlo SM. 2007. Asthma among health care professionals [editorial]. Am J Respir Crit Care Med 175:633–634.

Tarlo SM, Broder I. 1989. Irritant-induced occupational asthma. Chest 96:297–300.

Tarlo SM, Liss GM. 2003. Occupational asthma: An approach to diagnosis and management. Can Med Assoc J 168:867–871.

Tarlo SM, Banks D, Liss G, Broder I. 1997. Outcome determinants for isocyanate induced occupational asthma among compensation claimants. Occup Environ Med 54:756–761.

Tarlo SM, Boulet L-B, Cartier A, Cockroft D, Côté J, Hargreave FE, Holness L, Liss G, Malo J-L, Chan-Yeung M. 1998. Canadian Thoracic Society Guidelines for occupational asthma. Can Resp J 5(4): 289–300.

Tarlo SM, Easty A, Eubanks K, Min F, Juvet S, Liss G. 2001. Outcomes of a natural rubber latex (NRL) control program in an Ontario teaching hospital. J Allergy Clin Immunol 108:628–633.

Tarlo SM, Liss G, Yeung KS. 2002. Changing rates of accepted compensation claims for occupational asthma due to diisocyanates versus other causes in Ontario 1980-1993 : A possible effect of intervention measures. Occup Environ Med 59:58–62.

Tarlo SM, Balmes J, Balkissoon R, Beach J, Beckett W, Bernstein D, Blanc PD, Brooks SM, Cowl CT, Daroowalla F, Harber P, Lemiere C, Liss GM, Pacheco KA, Redlich CA, Rowe B, Heitzer J. 2008. Diagnosis and management of work-related asthma: American College of Chest Physicians consensus statement. Chest 134(Suppl): 1S–41S.

Vandenplas O, Larbanois A, Vanassche F, François S, Jamart J, Vandeweerdt M, Thimpont J. 2009. Latex-induced occupational asthma: Time trend in incidence and relationship with hospital glove policies. Allergy 64:415–420.