ORIGINAL ARTICLE

Changes in rates and severity of compensation claims for asthma due to diisocyanates: a possible effect of medical surveillance measures

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Objectives: A medical surveillance programme was introduced into Ontario for workers exposed to diisocyanates in 1983, but no mandated surveillance programme is in effect in this province for other occupational respiratory sensitisers. This study assesses changes in incidence and severity of compensated claims for occupational asthma (OA) due to diisocyanates compared with other causes, which have occurred since the introduction of this surveillance programme.

Methods: New claims for OA compensated by the Ontario Workers' Compensation Board (WCB) between 1980 and 1993 were retrospectively reviewed. Linkage was made between these data and an Ontario Ministry of Health database to assess hospital admissions for asthma from the date of onset of OA until the end of 1996.

Results: Numbers of claims for OA induced by diisocyanates ranged from 9–15/year in 1980–83, increased up to 55–58 claims/year in 1988–90, then fell to 19–20 claims/year by 1992–93. By contrast yearly numbers of claims for OA due to other causes increased up to 1985–87 then remained relatively stable. Duration of symptoms for OA induced by diisocyanates was shorter than for other claims and there were fewer hospital admissions among those with OA induced by diisocyanates than among those with OA induced by other causes. Occupational asthma from all causes was diagnosed earlier in claims for 1987–93 compared with 1980–86, and indicators of severity of asthma were also milder in accepted claims during 1987–93 than in earlier claims.

Conclusions: Although engineering and industrial hygiene measures may have contributed to these changes, our findings are also consistent with a beneficial contribution from the medical surveillance programme for workers exposed to diisocyanates.

Occupational asthma (OA) is the most commonly reported occupational lung disease in Ontario[1] as well as in several other regions and countries.[2 3] Diisocyanates have been recognised as most often causing OA, and are the most commonly compensated cause in industrialised countries such as the United Kingdom,[3] and in the Canadian Provinces of Ontario[4] and Quebec.[2]

In Ontario, legislation was introduced in 1983 by the Ontario Ministry of Labour, requiring monitoring of diisocyanate concentrations to maintain 8 hour average concentrations below 5 ppb and short term exposure concentrations below 20 ppb.[5] Also medical surveillance measures were introduced requiring a pre-employment respiratory questionnaire, and spirometry, with repeated respiratory questionnaires every 6 months and spirometry at least on an annual basis.[5] Workers with lower respiratory symptoms on questionnaire, or changes in spirometry were required to have a medical assessment as to their ability to continue work with diisocyanates. No legislation exists in Ontario to provide medical surveillance for other respiratory occupational sensitisers.

We have previously reported a greater risk of OA among diisocyanate workers from companies with higher measured concentrations of diisocyanates.[6] Also we reported that OA was diagnosed earlier among workers from companies known to comply with the medical surveillance programme than in companies who were not known to comply with it.[7] The present study was designed to examine whether this programme for workers exposed to diisocyanates was temporally related to changing annual numbers of compensated claims for asthma induced by diisocyanates compared with claims for OA induced by other causes. Also changes in severity of OA induced by diisocyanates were assessed. As there is no objective documentation as to the date of implementation of surveillance programmes in most facilities that use diisocyanates in Ontario, or even, in many cases confirmation of ongoing surveillance programmes, the present study aimed to assess changes in severity of OA induced by diisocyanates by comparing data from OA claimants whose claims were initiated in the first half of the study period, 1980–7—that is, the period before it would be expected that the regulations would have much impact—versus those whose claims were initiated in the second half of the study period, 1988–93. Results have been compared with data from claims accepted for other causes of occupational asthma during the same periods. This division of periods would, if anything, be likely to underestimate the effects of the diisocyanates surveillance programme, as some implementation probably occurred in the first period, but no information is available as to exact implementation dates or compliance with this programme.

METHODS

A retrospective review of Ontario Workers' Compensation Board (WCB (now renamed Workplace Safety and Insurance Board)) claims for all causes of OA which was previously performed for claims allowed between 1984 and 1988,[4] was extended to claims allowed for OA started in the 14 year period

Abbreviations: OA, occupational asthma; WCB, Ontario Workers' Compensation Board (formerly, Workplace Safety and Insurance Board); FEV_1, forced expiratory volume in 1 second; VC, vital capacity; FEF_{50}, forced expiratory flow at 50% vital capacity

Table 1 Most common attributed causes of occupational asthma in the 844 accepted OA claims 1980–93

Causative agent	n (%)
All agents	844 (100)
Diisocyanates	425 (50.2)
Flour	52 (6.2)
Metals	31 (3.7)
Red cedar	27 (3.2)
Natural rubber latex	22 (2.6)
Plastics	19 (2.3)
Grain	19 (2.3)
Non-cedar wood dusts	15 (1.8)
Welding fumes	12 (1.4)
Soldering fumes	9 (1.1)
Enzymes	9 (1.1)
Other foods or plant products	11 (1.3)
Other probable sensitisers (<1 each)	35 (4.1)
Acids or chlorine	16 (1.9)
Non-isocyanate paints or solvents	19 (2.3)
Not specified or not identified	122 (14.5)

between l980 and l993 inclusive. The questionnaire completed from each file was shortened from the one previously used[4] but included data on demographic features of claimants, work descriptors, symptoms of asthma, reported improvement on weekends and holidays, and duration of symptoms of asthma before the main medical assessment. Probable causative agent for each case of OA was determined from the clinical records. Spirometry results were recorded as well as results (when available) of allergy skin tests, peak flow readings, methacholine challenges during a working period, and off work and specific occupational challenges. As these tests were usually arranged by the claimant's attending physician in many different centres, the methods used for these tests were not uniform.

Linkage was made between the data from the WCB files and information from the health information discharge abstract data base (from the Ontario Ministry of Health), from the time of the WCB accident date—the date of onset of symptoms, up to 31 December l996 as previously described[8 9]—to compare the number of hospital admissions for asthma among those with OA from diisocyanates with that among those with OA from other occupational causes.

Statistical analyses

Data were entered into an SAS programme (release 6.12) for personal computer, using *t* tests and χ^2 tests as appropriate for analyses. To compare the annual rates of onset of accepted claims for OA induced by diisocyanates versus OA induced by other causes, the proportion of accepted claims for OA due to diisocyanates as a proportion of all accepted claims for OA was assessed for the period of 1980–86 and separately for 1987–93, using the Mantel-Haenszel χ^2 test for trend for each period. Subject variables were compared for subjects with new accepted claims, for OA attributed to diisocyanates and OA attributed to other causes. To assess changes over time, variables were initially compared for those whose onset of OA occurred in the first 7 years of the study period, with those whose onset of symptoms was in the last 7 years of the study. Analysis of variance (ANOVA) was performed to assess differences over period for those with OA caused by diisocyanates versus those with OA induced by other causes. Analysis of data on hospital admissions for OA from diisocyanates versus OA from other causes was based on the subset (810 out of the total of 844 OA claimants) with data available for smoking history and causative agent for OA. Multivariate analyses were performed adjusting for age, sex, smoking history, year of onset of symptoms, and work exposure (same work exposure, versus same workplace with reduced but possible exposure, versus no exposure) as well as exposure group (diisocyanates *v* other OA).

RESULTS

A total of 844 new claims for OA were allowed by the WCB with onset of symptoms between l980 and l993 inclusive. The number of accepted claims per year of onset steadily increased during this 14 year period for OA attributed to causes other than diisocyanates (419 claims) (fig 1). By contrast, claims allowed for OA attributed to diisocyanates (425 claims) initially rose parallel to OA induced by other causes, then rose more steeply than for OA induced by other causes between l988 and l990, and subsequently fell sharply by 1992–3. Overall, OA induced by diisocyanates accounted for 50.2% of all accepted claims during this period: initially around 50% in 1980–87, increasing to a maximum of 64% by 1988, then falling significantly to 29% in l992 and 35% in l993. There was no significant difference in the annual proportion of OA caused by diisocyanates from OA induced by other causes over the period 1980–86, but in the second half of the study period, 1987–93, there was a significant difference in this proportion over time (p=0.001) (fig 1). The most common attributed causative agents among the 844 claims are shown in table 1.

The characteristics of claimants with OA from diisocyanates and from other causes are compared in table 2. Those with OA attributed to diisocyanates included more women (40%), were significantly younger, were less likely to be atopic, and were more likely to have smoked. They were less likely to have reported associated nasal symptoms (42% *v* 53%), and the onset of symptoms of asthma after the onset of exposure at the workplace was significantly earlier than with OA from other causes (mean 5 years *v* 7 years, p=0 .0001). The mean duration of symptoms while continuing to work before the main medical assessment in the WCB file was significantly shorter in those with OA induced by diisocyanates (mean 2

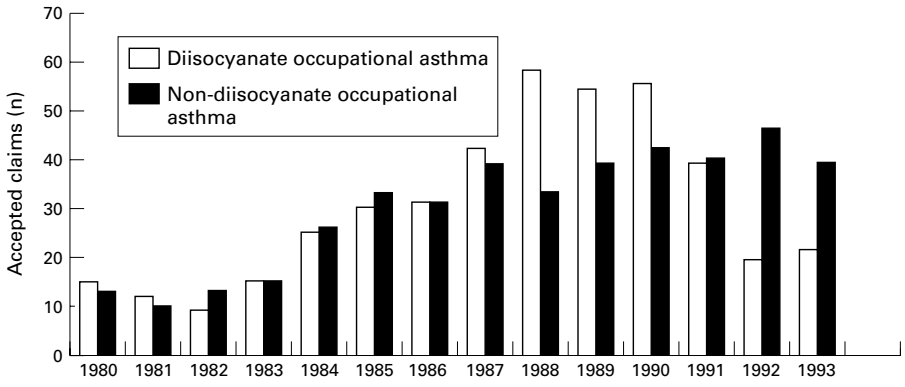

Figure 1 Number of allowed claims for OA induced by diisocyanates and OA induced by other causes by year of onset. A significant change occurred in the proportion of OA induced by diisocyanates and OA induced by other causes in the years 1987–93 (p=0.001).

Table 2 Features of workers with claims accepted for diisocyanate OA versus other causes of OA, 1980–93

	Diisocyanate OA	Other OA	p Value
n	425	419	
Age (y, mean (SD (median)))	37.4 (11.4 (35.25))	42.3 (12.6 (41.42))	0.0001
Male	60%	69%	0.005
Never smoked	37%	45%	0.001
Atopic on skin tests	33%	51%	0.001
Improve weekends	65%	60%	NS
Improve holidays	88%	78%	0.001
Nasal symptoms	42%*	53%	0.003
Exposure before symptoms (y, mean (SD (median)))	5.13 (6.1 (3.0))	7.45 (7.7 (4.87))	0.0001
Still exposed at assessment	23%	31%	NS
Exposure after onset of symptoms (y, mean (SD (median))	2.38 (3.5 (1.0))	3.38 (4.8 (1.50))	0.0008

*Number with information on nasal symptoms in the file: 382 diisocyanate OA, 391 other OA.

years versus 3 years (p=0.009)), and they were more likely to report improvement in symptoms at the time of the main medical assessment.

The proportion of each group who were still exposed to the implicated causative workplace agent by the main assessment in the WCB records were not significantly different (23% with OA induced by diisocyanates *v* 31% with OA from other causes). However, spirometric variables of forced expiratory volume in 1 second (FEV_1), vital capacity (VC), and forced expiratory flow at 50% vital capacity (FEF_{50}) at that visit, expressed as a percentage of the predicted normal, were significantly better for those with OA attributed to diisocyanates and they were less likely to be receiving inhaled corticosteroid treatment (p=0.001, table 3).

The differences between the OA due to diisocyanates and the OA due to other causes shown in table 3 remained significant after adjustment (for age, sex, smoking history, work exposure, and year of onset of symptoms) for FEV_1 (p=0.035), and for inhaled steroid usage (p=0.0003). Differences also remained significant between these groups for years of workplace exposure to the implicated work agent before the onset of symptoms of OA (p=0.0001), and duration of symptoms before leaving work (p=0.0008), when adjusted for year of onset of symptoms.

Linkage for hospital admissions with a primary or "most responsible diagnosis" of asthma up to the end of 1996 was analyzed for 414 accepted claimants for OA due to diisocyanates and 396 accepted claimants from OA due to other causes, for whom data were available on smoking history and likely exposure to an agent causing OA. A significantly smaller proportion of those claimants with asthma induced by diisocyanates had hospital admissions for their asthma (5.3%, table 3), than did those who had other attributed causes of OA (11.1%, p=0.003).

Changes in characteristics of occupational asthma over time

Comparison of the variables for those cases with onset of symptoms of OA induced by diisocyanates in 1980–6 was then made with those for whom the onset was in 1987–93 (table 4). Those with the onset of symptoms in the second half of the study period were younger (p=0.002) and there were relatively more women than in the first half of the study period. In the second half of the study period accepted claimants had a significantly shorter duration of symptoms of asthma before diagnosis or before leaving exposure to diisocyanates (mean 2.1 (3.2) years *v* 3.0 (3.9) years; p=0.014, table 4).

However, similar changes were found in those with OA induced by other causes. The proportion of women increased from 20% to 36% (p=0.001), and the period between the onset of symptoms and leaving exposure to the agent responsible in the second half of the study among those with OA induced by other causes, was 2.9 years versus 4.3 years (p=0.01)in the first half of the study. These differences over the two periods were not significantly different between OA induced by diisocyanates and OA induced by other causes.

Indices of severity of asthma at the time of diagnosis suggested milder asthma in those diagnosed in the second period of the study. Both for the group accepted for OA induced by diisocyanates (table 4) and the group accepted for OA induced by other causes, (data not shown), there was less limitation to airflow on spirometry in claims accepted in the last half of the study and less airway hyperresponsiveness on methacholine challenge while off work during the initial investigations (table 4). Again, these changes were not significantly different for OA induced by diisocyanates versus OA induced by other causes.

Table 3 Features reflecting asthma severity among those with diisocyanate OA versus other causes of OA, 1980–3

	Diisocyanate OA	Other OA	p Value
n	425	419	
Using inhaled steroids (n (%))	29% of 373*	43% of 386*	0.001
FEV_1 % predicted (mean (SD (median)))	97.6 (21.8 (99.35))	93.2 (25.3 (94.35))	0.01
VC % (mean (SD (median)))	102.5 (18 (103.5))	99.3 (19.4 (99.9))	0.02
FEF_{50} % (mean (SD (median)))	84.3 (36 (84.8))	75.8 (37.8 (74.7))	0.01
FEF_{25} % (mean (SD (median)))	73.3 (46 (63.1))	70.1 (44 (59.6))	NS
Actual FEV_1/VC (mean (SD (median)))	75.6 (10.8 (77.45))	73.6 (13.6 (75.29))	0.03
Methacholine PC_{20} during work period (mean (SD (median)))	4.6 (11.5 (0.9)) (n=101)	2.8 (6.7 (1.1)) (n=107)	NS
Methacholine PC_{20} off work (mean (SD (median)))	18.4 (23.5 (7.7)) (n=239)*	17.4 (24.1 (4.3)) (n=199)*	NS
At least one hospital admission for asthma up to 1996 (n (%))	5.3% of 414*	11.1% of 396*	0.003

*Number with information in the file.

Table 4 Differences in diisocyanate OA between those developing symptoms 1980–86 and 1987–93

	1980–86	1987–93	p Value
n	137	288	
Age (y, mean (median))	40 (38.3)	36 (33.9)	0.002
Male (%)	72	55	0.001
Exposure after onset of symptoms (y, mean (SD (median)))	3.03 (3.92 (1.3)	2.06 (3.17 (0.83)	0.014
Use of inhaled steroids (%)	41	25	0.003
FEV_1 predicted (%, mean (SD (median)))	96 (24 (100)	98 (21 (99)	NS
VC predicted (%, mean (SD (median)))	104 (21 (106)	102 (17 (103)	NS
FEF_{50} predicted (%, mean (SD (median)))	76 (40 (72.2)	88 (33 (90.3)	0.03
FEF_{25} predicted (%, mean (SD (median)))	71 (54 (58.8)	75 (42 (67.2)	NS
Actual FEV_1/VC (%, mean (median))	72 (73.9)	77 (78.5)	0.0003
Methacholine PC_{20} during work period (mg/ml, mean (SD (median)))	4.9 (11.1 (0.97)) (42)*	4.4 (11.8 (0.8)) (59)*	NS
Methacholine PC_{20} off work (mg/ml, mean (SD (median)))	13.2 (18.3 (6.0)) (77)*	20.8 (25.3 (8.0)) (162)*	0.02

*Number with information in the file.

The risk of hospital admissions for asthma was about 50% less among those with OA induced by diisocyanates compared with other claimants with OA. This reduced risk was found both for claims with an onset of OA during 1980–86 (6.8% *v* 13.6%; OR 0.46 (95% CI 0.20 to 1.07)) and during 1987–93 (4.6% *v* 9.9%; OR 0.44 (95% CI 0.22 to 0.88)). The reduced risk of a hospital admission among workers with asthma induced by diisocyanates remained significant when adjusted for differences in age, sex, smoking, or period of claim as previously reported.[9]

DISCUSSION

The increase in the absolute numbers and in the proportion of allowed claims for OA induced by diisocyanates relative to OA induced by other causes in the same period l986–90 (fig 1) in conjunction with a shorter duration of symptoms of asthma at the time of the claim, a younger mean age of subjects, and milder asthma in those diagnosed during the last half of the study period is consistent with an increase in case finding of OA induced by diisocyanates during this time, possibly attributable to the medical surveillance programme.

An earlier diagnosis was made during the second half of the study period as reflected by the shorter time between the onset of symptoms and the main medical assessment in the WCB file. There were similar findings in claims accepted for OA induced by other causes but the period before the main assessment and the submission of a claim remained shorter for OA induced by diisocyanates throughout the study time. This earlier diagnosis for OA in the second half of the study period may have been related to better general awareness of OA in this period by workers and physicians in Ontario, perhaps related to national recommendations for diagnosis.[10] It is also possible that for OA induced by diisocyanates the earlier diagnosis may have been related to the diisocyanates medical surveillance programme as suggested by our earlier study.[7] Although a significant difference was not detected in the time related changes between the group with OA induced by diisocyanates and the group with OA induced by other causes, we do not have information on the extent of medical surveillance and education programmes which might have been in place in companies whose employees were at risk for OA induced by substances other than diisocyanates.

If related to surveillance programmes, a benefit may have been directly attributed to the measures of symptom questionnaires and spirometry, or may have been related to associated health education of the workers relative to OA induced by diisocyanates or other sensitisers, to increased awareness of this entity by their physicians in conjunction with the medical surveillance programme, or to industrial hygiene measures associated with the companies' control programmes.

It is less likely that the increase in accepted claims for OA induced by diisocyanates during the central period of the study, and subsequent fall, were related to a change in acceptance practices of claims by the adjudicators and the WCB, as claims for OA from other causes continued to increase (with some fluctuations) on an annual basis.

The lower risk of hospital admissions for those with OA induced by diisocyanates than with OA from other causes was reported by us[9] in a fuller analysis of hospital admissions in this cohort of compensated claimants with OA compared with compensated injured workers and patients with non-occupational asthma. The reduced risk of a hospital admission among people with asthma induced by diisocyanates remained significant when adjusted for differences in age, sex, smoking, or period of claim, and therefore does seem to reflect milder asthma in these subjects, consistent with our findings from the other markers of severity of asthma in this study—such as spirometric variables, prescribed inhaled steroid therapy, and the trend to milder responses on methacholine challenge.

The decline in accepted claims for OA induced by diisocyanates after 1991 (during 1992 and 1993) down to lower numbers than those for OA induced by other causes cannot be explained only on the basis of a transient left shift of the incidence curve due to earlier finding of cases. The fall in annual numbers relative to the number of cases of OA induced by other causes may indicate a true fall in incidence of cases induced by diisocyanates, perhaps due to improved occupational hygiene measures in the workplace such as the use of robots in high exposure areas and routine monitoring of diisocyanate concentrations to conform with exposure guidelines. Another possible explanation for the fall in annual numbers of accepted claims might be random fluctuation, and a longer follow up period would be necessary to exclude this. Finally, it is possible that this apparent fall in incidence may reflect a decline in quality or availability of surveillance of diisocyanates in the last few years of the study. In the early years of the programme, the Ontario Ministry of Labour provided surveillance for many Ontario companies, but this was phased out in the early 1990s. Also, in 1991 participation by workers in the medical surveillance programme became voluntary rather than mandatory, as it had been before. Potentially this could result in delays in detection and a transient fall in accepted claims in the last few years of the study. In that event, the numbers would be expected to rise again in subsequent years, with an increase in duration of symptoms before diagnosis, and indices of severity of asthma.

Key messages

- Asthma induced by diisocyanates as reflected by compensation claims rose in frequency after introduction of medical surveillance measures and subsequently has fallen relative to accepted claims for occupational asthma from other causes.
- Results suggest increased cases after introduction of the surveillance programme and a subsequent fall in new cases, possibly related to better control of exposure.
- Between 1987 and 1993 compensated cases of occupational asthma were diagnosed earlier and were milder than cases over the preceding 7 years.
- This finding suggests an improved awareness of occupational asthma either related to the surveillance programmes or better awareness by attending physicians.
- Throughout the study period asthma induced by diisocyanates was diagnosed earlier and was milder at the time of diagnosis than occupational asthma from other causes, possibly related to the surveillance programme.

There is no evidence of a reduction of use of diisocyanates in Ontario to account for a declining incidence of OA, and imports of diisocyanates into Canada have continued to rise.[11] However, there is no direct information available as to the number of workplaces or workers using diisocyanates, and whether this may have changed over time. A previous study by us[6] reviewed data from 223 worksites that used diisocyanates, for which the Ministry of Labour of Ontario had exposure data on diisocyanates, but such monitoring after 1990 was not maintained by this Ministry in a systematic manner and there is no ongoing comprehensive record as to the compliance of companies that use diisocyanates with the medical surveillance and air monitoring requirements.

In conclusion, our findings indicate that OA due to diisocyanates in Ontario has been diagnosed earlier and has been associated with better pulmonary function and a better outcome as assessed by hospital admissions than OA induced by other causes. There has been an earlier diagnosis of OA, both induced by diisocyanates and by other causes especially in the second half of this study period. There is an apparent decline in incidence of OA induced by diisocyanates in the last few years of this study, which might reflect improved occupational hygiene measures at work or might reflect early identification of very mildly symptomatic workers and transfer of these workers to unexposed areas without submission of WCB claims, or without objective confirmation of the diagnosis. This would be consistent with our review of one company.[12] However, further follow up is needed to confirm this trend to a reduced incidence of OA induced by diisocyanates.

Policy implications

- Results of this study suggest a beneficial outcome from the Ontario surveillance programme for diisocyanates. However, further studies are needed to identify specifically which components of the programme may have contributed to the outcomes found, and also to assess the significance of the recent reduction in annual compensated claims for asthma induced by diisocyanates.

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