Compensation for Occupational Asthma in Quebec*

Jean-Luc Malo, M.D.

FEV_1 = forced espiratory volume in 1 s; PEFR = peak expiratory flow rate

Bill 42, which deals with all aspects of workers' compensable diseases in Quebec, was adopted in 1985. Compensation is now handled by a central office at the Ministry of Labour, making it possible to obtain statistics on the extent and distribution. In 1988, occupational asthma represented the principal occupational respiratory ailment, both in terms of new claims for compensations and in the total number of claims (Table 1). As regards occupational asthma, 89 subjects were seen for reassessments of the percentage for disability or impairment. These figures demonstrate a major change compared with the situation in 1977 (Table 2).

Definition of Occupational Asthma

In Annexe I, Section V of Bill 42, occupational asthma is defined as an occupational disease wherein a worker is exposed to a specific (to the workplace) sensitizing (as opposed to irritant) product. Several factors led legislators to include this definition. There are indeed 2 types of definitions of occupational asthma in the medical literature: those that define occupational asthma as a type of asthma caused by a specific agent[1-3] and those that also specify that the agent is sensitizing.[4-6] Defining occupational asthma without differentiating between causal agents (sensitizing as opposed to irritating) did not seem justified, because there is no scientific basis for it and a huge increase in the number of claims could result. Scientific evidence indicates that exposure to a sensitizing agent such as pollen or occupational agents (isocyanates, red cedar, etc) in the everyday environment can increase the severity of asthma and lead to permanent bronchial obstruction and hyperresponsiveness. The evidence regarding the effect of other agents such as particles, gas, vapors, or nonspecific factors (exercise, cold air) is much less convincing. Exposure to ozone and other pollutants can increase airway resistance and bronchial responsiveness, but the effect is physiologically minimal and transient.

Compensating every patient with exacerbated asthma due to irritant factors (particularly atmospheric pollutants) would mean that 5% to 10% of the adult population (the proportion of asthmatic subjects in a random population sample) might be eligible. Indeed, most asthmatic subjects could argue that their symptoms are worse on exposure to irritants on their way to and from work or at the workplace.

*From the Department of Chest Medicine, Hôpital du Sacré-Coeur, University of Montreal, Montreal, Quebec, Canada

Table 1—*Extent and Distribution of Compensation*

Diagnosis	New Claims	Reassessments	Total
Occupational asthma	81	89	170
Asbestosis	30	111	141
Silicosis	36	103	139
Cancer	46	38	84
Occupational bronchitis	15	8	23
Other	20	37	57

Table 2—*Changes in Number of Claims From 1977 to 1987*

	1977		1987	
Diagnosis	Total No. of Claims	Accepted Claims	Total No. of Claims	Accepted Claims
Asbestosis	881	43	112	36
Silicosis	223	36	83	62
Occupational asthma	12	6	213	97

Compensation

Medicolegal Consultation: How It Works

Employee and physician claims for compensation are addressed to a regional office of the Workers Compensation Board. Claims concerning respiratory ailments are referred by the Medical Direction of the Board to a committee of 3 chest physicians. There are committees at 4 university hospitals in the province. All members are nominated for a 4-year term by the Minister of Labour after recommendation by the Corporation of Physicians and Surgeons. The chairmen of each committee meet to approve or reject decisions made by the original committee. Suspected cases of occupational asthma are referred to specialists for further investigation. These specialists work in 3 university hospitals that have facilities for investigating occupational asthma (challenge rooms, trained technicians).

Criteria Used for Compensation

To be accepted, every case of occupational asthma must have been investigated by objective means. Neither a questionnaire showing a work-related increase in asthma symptoms nor exposure to a known sensitizing agent nor the presence of antibodies is sufficient to confirm the diagnosis.

Table 3—*Use of Closed Questionnaire for Epidemiologic Assessment of Occupational Asthma (OA)*

Agent, Study	No. of Workers	No. of Workers with Questionnaire Suggestive of OA	No. of Workers with OA (% of those with history suggestive of OA)
Snow crab[7]	303	64	33 (52%)
Isocyanates[8]	48	14	6 (43%)
Psyllium[9]	130	39	5 (13%)
Spiramycin[10]	51	12	3 (25%)

Table 4—*Presence or Absence of Typical Symptoms of Occupational Asthma*

Final Diagnosis	Temporality of Symptoms: Present or Worse at Work	Present or Worse After Workshift	Improved on Weekends	Improved During Vacations
Occupational asthma ($n=47$)	43	27	35	40
Personal asthma ($n=30$)	29	15	17	20
Neither occupational nor personal asthma ($n=23$)	22	10	17	18
Total	94	52	69	78

There are several reasons for this.

First, the closed questionnaire used for epidemiologic assessments of occupational asthma is a sensitive but not specific tool for detecting occupational asthma (Table 3).[7-10] In our experience, based on the prospective assessment of 100 subjects referred for possible occupational asthma and in whom objective assessment (specific inhalation challenges, monitoring of PEFR) was performed, an open questionnaire administered by chest physicians trained in occupational asthma does not have a sufficient positive or negative predictive value to be useful for diagnostic purposes. The presence or absence of typical symptoms such as improvement on weekends or during vacations is not a satisfactory index for the presence of occupational asthma (Table 4). Furthermore, the chest physician's impression after administering an open questionnaire does not have a sufficient predictive value (Table 5).

Exposure of an individual to a known sensitizer does not mean that the diagnosis should be retained. Immunologic sensitization does not mean that the subject has occupational asthma. Although highly specific, the presence of specific antibodies is generally too sensitive in the case of an IgE-mediated phenomenon (Table 6).[7] For isocyanates, the presence or absence of antibodies is not sensitive nor specific enough for making a diagnosis of occupational asthma (Table 7).[11]

For these reasons, we feel that the diagnosis should be based on an objective assessment, preferably specific inhalation challenges in a laboratory if the occupational sensitizer has been identified or at work if the sensitizer is unknown or more than 1 agent may be present. These tests should be performed by specialists in centers designed for that purpose. Monitoring PEFR at work and away from work, alone or combined with assessment of nonspecific bronchial hyperresponsiveness, is an interesting tool[12,13] and may have sufficient sensitivity and specificity to confirm occupational asthma.[14]

Table 6—*Presence or Absence of Antibodies*

Test Result	Immunologic Sensitization to Psyllium	Occupational Asthma due to Psyllium
Immediate skin reactivity	23/120 (19%)	5/120 (4%)
Elevated specific IgE	31/118 (26%)	5/118 (4%)

Table 7—*Presence of Absence of Antibodies for Isocyanates*

Levels of Specific IgG Antibodies	Specific Inhalation Challenges: Positive*	Negative†
Increased	21	8
Normal	8	25

*Odds for increased specific IgG in a subject with a positive result on specific inhalation challenges = 72%.

†Odds for normal specific IgG in a subject with a negative result on inhalation challenges = 76%.

Types of Compensation

Two types of compensation are relevant. The first provides for an income replacement indemnity and readaptation. Subjects with occupational asthma are often young, and it appears mandatory to train them for a new job. Once the diagnosis is made, the indemnity generally lasts for 1 to 2 years. The second type is a permanent disability indemnity. Numerous retrospective studies have shown that occupational asthma may lead to permanent disability (Table 8).[15-22]

The criteria currently used to determine the permanent disability indemnity are baseline bronchial obstruction, baseline bronchial hyperresponsiveness, and need for medication. These criteria have been selected because they show a satisfactory correlation and they reflect the severity of the asthma.[23] Bronchial responsiveness to pharmacologic agents bears a satisfactory correlation to bronchial respon-

Table 5—*Predictive Value of Open Questionnaire*

Final Diagnosis	Occupational Asthma: Highly Likely*	Likely*	Uncertain	Unlikely†	Absent†
Occupational asthma ($n=47$)	32	5	6	3	1
Personal asthma ($n=30$)	7	8	8	4	3
Neither occupational nor personal asthma ($n=23$)	3	3	9	3	5

*Of 58 subjects with a highly likely or likely history, 37, or 64%, had occupational asthma (predictive value of a positive result).

†Of 19 subjects with an unlikely or absent history, 15, or 79%, did not have occupational asthma (predictive value of a negative test).

Table 8—*Permanent Disability*

Agent	No. of Subjects	Duration of Follow-up (yr)	Persistence of Symptoms (%)	Persistence of Hyperresponsiveness (%)	Reference
Red cedar	75	1-9	49	25/33 (76%)	15
Colophony	20	1.3-3.8	90	7/20 (35%)	16
Isocyanates	12	1-3	66	7/12 (58%)	17
Snow crab	31	0.5-2	61	28/31 (90%)	18
Snow crab	31	4.8-6	100	26/31 (84%)	19
Various	32	0.5-4	93	31/32 (97%)	18
Isocyanates	50	>4	82	12/19 (63%)	20
Isocyanates	20	0.5-4	50	9/12 (75%)	21
Isocyanates	22	1	77	17/22 (77%)	22

Table 9—*Criteria To Detrmine Permanent Disability*

Class	Level of Bronchial Obstruction[a]	Level of Bronchial Responsiveness[b]	Need for Medication	Percent Disability
1	0	0	None	0
2A	0	1	None	5
2B	0	1	BDT prn	8
2C	0	1	BDT reg	10
2D	0	2	None	10
2E	0	2	BDT reg or prn	13
2F	0	3	BDT reg or prn	15
3A	1	1	BDT reg or prn	18
3B	1	2	BDT reg or prn	20
3C	1	3	BDT reg or prn	25
4A	2	1-2	BDT reg or prn	28
4B	2	3	BDT reg or prn	33
5A	3	1-2	BDT reg or prn	50
5B	3	3	BDT reg or prn	60
6	4	1-2-3	BDT reg or prn Group with oral steroids and with or without inhaled steroids	100
			To be added:	
			Inhaled steroid	3
			Oral steroid	10

[a]Level of bronchial obstruction determined as follows:
0: FEV_1 (% pred) and/or FEV_1/FVC (% pred)<85% pred
1: FEV_1 (% pred) and/or FEV_1/FVC (% pred) = 71%-85% pred
2: FEV_1 (% pred) and/or FEV_1/FVC (% pred) = 56%-70% pred
3: FEV_1 (% pred) and/or FEV_1/FVC (% pred) = 40%-55% pred
4: FEV_1 (% pred) and/or FEV_1/FVC (% pred)<40% pred
for predicted (pred) results, see reference 24

[b]Level of bronchial hyperresponsiveness determined as follows:
0: PC_{20}>16 mg/ml
1: PC_{20} = 2-16 mg/ml
2: PC_{20} = 0.25-2 mg/ml
3: PC_{20}<0.25 mg/ml
PC_{20} assessed by the method outlined in reference 25.

siveness due to natural stimuli such as exercise and hyperventilation of unconditioned air. A flow chart has been developed and the percent of impairment is determined (Table 9).

The need for medication is set at the amounts normally required to control the subject's symptoms, as judged by the practitioner in charge. Subjects should be in a clinically steady state (absence of awakenings due to asthma symptoms, no extra need for inhaled β_2-adrenergic agent). The assessment is performed 2 years after the subject is removed from the workplace, as there seems to be a plateau of improvement after this interval.[19]

Costs

Costs related to compensation of cases of occupational asthma may vary according to several factors: duration of the income replacement indemnity, cost of medical consultation, possibility of finding another job, or severity of permanent disability or impairment. Three examples of findings and cost of compensation in actual cases of occupational asthma diagnosed in 1986 are presented in Table 10.

Conclusions

In order to be compensated for occupational asthma in Quebec, the diagnosis must be proved using objective means. The system provides direct help to affected workers in 2 ways: through readaptation with an income replacement indemnity, and by providing for permanent impairment/disability. The scheme of allowances for permanent impairment/disability has been used since 1985 and seems to be satisfactory.

References

1 Brooks, SM. Occupational asthma. In: Weiss EB, Segal MS, Stein M, eds. Bronchial asthma. Boston: Little, Brown & Co, 1985:461-69
2 Sheppard D. Occupational asthma and byssinosis. In: Murray

Table 10—*Cost of Compensation, Actual Cases*

Subject	Age at Diagnosis (yr)	Sensitizing Agent	Impairment (%)	Amount (CAN $)
No. 1	32	Snow crab	11	19,000,00
No. 2	52	Isocyanate	23	53,000,00
No. 3	62	Isocyanate	21	62,000,00

JF, Nadel JA, eds. Textbook of respiratory medicine. Philadelphia: WB Saunders, 1988:1593-1605
3 Parkes WR. Occupational lung disorders. London: Butterworths, 1982:415-53
4 Cotes JE, Steal J. Work-related lung disorders. Oxford: Blackwell Scientific Publishers, 1987:345-72
5 Burge PS. Occupational asthma. In: Barnes P, Rodger IW, Thomson NC, eds. Asthma: basic mechanisms and clinical management. London: Academic press, 1988:465-82
6 Chan-Yeung M, Malo JL. Occupational asthma. Chest 1987; 81:130S-136S
7 Cartier A, Malo JL, Forest F, Lafrance M, Pineau L, St-Aubin JJ, Dubois JY. Occupational asthma in snow crab-processing workers. J Allergy Clin Immunol 1984; 74:261-69
8 Séguin P, Allard A, Carier A, Malo JL. Prevalence of occupational asthma in spray painters exposed to several types of isocyanates, including polymethylene polyphenylisocyanates. J Occup Med 1987; 29:340-44
9 Bardy JD, Malo JL, Séguin P, Ghezzo H, Desjardins J, Dolovish J, et al. Occupational asthma and IgE sensitization in a pharmaceutical company processing psyllium. Am Rev Respir Dis 1987; 135:1033-38
10 Malo JL, Cartier A. Occupational asthma in workers of a pharmaceutical company processing spiramycin. Thorax 1988; 43:371-77
11 Cartier A, Grammer L, Malo JL, Lagier F, Ghezzo H, Harris K, Patterson R. Specific serum antibodies against isocyanates: association with occupational asthma. J Allergy Clin Immunol, in press
12 Chan-Yeung M, Lam S. Occupational asthma. Am Rev Respir Dis 1986; 133:686-703
13 Pauli G, Bessot JC, Dietemann-Molard A. L'asthme professionnel: investigations et principales étiologies. Clin Respir Physiol 1986; 22:399-425
14 Coté J, Kennedy S, Chan-Yeung M. Sensitivity and specificity of PC20 and PEFR in cedar asthma. J Allergy Clin Immunol 1990; 85:592-98
15 Chan-Yeung M, Lam S, Koener S. Clinical features and natural history of occupational asthma due to Western red cedar. Am J Med 1982; 72:411-15
16 Burge PS. Occupational asthma in electronics workers caused by colophony fumes: follow-up of affected workers. Thorax 1982; 37:384-53
17 Paggiaro PL, Loi AM, Rossi O, et al. Follow-up study of patients with respiratory disease due to toluene diisocyanate (TDI). Clin Allergy 1984; 14:436-69
18 Hudson P, Cartier A, Pineau L, et al. Follow-up of occupational asthma caused by crab and various agents. J Allergy Clin Immunol 1985; 76:682-88
19 Majo JL, Cartier a, Ghezzo H, Lafrance M, McCants M, Lehrer SB. Patterns of improvement in spirometry, bronchial hyperresponsiveness, and specific IgE antibody levels after cessation of exposure in occupational asthma caused by snow-crab processing. Am Rev Respir Dis 1988; 136:807-12
20 Lozewicz S, Assoufi BK, Hawkins R, Newman Taylor AJ. Outcome of asthma induced by isocyanates. Br J Dis Chest 1987; 81:14-22
21 Rosenberg N, Garnier R, Rousselin X, Mertz R, Gervais P. Clinical and socio-professional fate of isocyanate-induced asthma. Clin Allergy 1987; 17:55-61
22 Mapp CE, Corona PC, De Marzo N, Fabbri L. Persistent asthma due to isocyanates. Am Rev Respir Dis 1988; 137:1326-29
23 Chan-Yeung M. Evaluation of impairment/disability in patients with occupational asthma. Am Rev Respir Dis 1987; 135:950-51
24 Knudson RJ, Slatin RC, Lebowitz MD, Burrows B. Changes in the normal maximal expiratory flow-volume curve with growth and aging. Am Rev Respir Dis 1983; 127:725-34
25 Crockcroft DW, Killian DN, Mellon JJA, Hargreave FE. Bronchial reactivity to inhaled histamine: a method and clinical survey. Clin Allergy 1977; 7:235-43