

SERIES "CONTROVERSIES IN OCCUPATIONAL ASTHMA"

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Health and socioeconomic impact of work-related asthma

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ABSTRACT: There is accumulating evidence that the workplace environment contributes significantly to the general burden of asthma. The purpose of this review is to explore the respiratory health and socioeconomic consequences of work-related asthma by addressing a series of controversial issues: 1) what is the natural history of occupational asthma and in what ways does ongoing exposure to the causal agent impact clinical outcomes?; 2) how does the natural history of irritant-induced asthma differ in its health outcomes from immunologically-mediated occupational asthma?; 3) do working conditions have a significant impact on asthma regardless of the aetiology of the disease?; 4) what is the scope of work disability from work-related-asthma in social and economic terms?; 5) what is the clinician's role in reducing the respiratory health consequences of work-related asthma? 6) to what extent do existing compensation and other social insurance schemes successfully address occupational asthma and work-aggravated asthma?

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Asthma represents a considerable source of morbidity and economic burden [1, 2]. The workplace environment can initiate or trigger asthma and result in the development of different types of work-related asthma, among these the following conditions can be distinguished: 1) immunological occupational asthma (OA) induced by a variety of high and low molecular weight sensitising agents; 2) irritant-induced asthma (or "nonimmunological OA") resulting from the persistent effects of single or multiple exposures to high concentrations of irritant substances; and 3) work-aggravated asthma symptoms in workers with pre-existing or coincident asthma [3, 4]. Epidemiological surveys indicate that occupational exposures could account for ~10–15% of adult asthma and could contribute significantly to the cost of asthma [5]. This review will address a series of issues relating to the health and economic consequences of the different forms of work-related asthma.

What is the natural history of occupational asthma and in what ways does ongoing exposure to the causal agent impact clinical outcomes?

Follow-up studies of workers with immunological OA have shown that avoidance of exposure to the causative

agent results in a significant improvement in asthma symptoms, airway obstruction, and nonspecific bronchial hyperresponsiveness (NSBH). Nonetheless, removal from exposure does not necessarily lead to complete recovery from asthma. Approximately 70% of affected workers still experience asthma symptoms and retain NSBH several years after cessation of the offending exposure [6–21]. NSBH can, however, further improve >5 yrs after exposure cessation [18]. Persistence of asthma has been consistently associated with a longer duration of work-related symptoms before removal and with a more severe asthma at the time of diagnosis [9–18, 21, 22]. Atopy and tobacco smoking have not been identified as significant determinants of outcomes in OA.

The long-term health effects of reducing rather than eliminating exposure to the agent causing OA remain uncertain. Reduction of exposure can be achieved through relocation to less exposed jobs, improvement in workplace hygiene, modifying materials to reduce their allergenic properties, and use of personal protective devices. Short-term studies indicate that latex gloves with a lower allergen content [23, 24] and helmet respirators [24–28] can reduce asthmatic reactions in subjects with OA. A number of studies have compared the long-term outcome of subjects who reduce exposure with those who completely avoid further exposure

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Table 1.—Comparison of asthma severity after cessation or reduction of exposure to the causative agent

Causative agent	Persistence of symptoms	Persistence of NSBH	Improvement of asthma [#]	Worsening of asthma [#]	1st author [ref no.]	Year of study
Cessation of exposure						
Colophony	16/20	3/7	5/7	0/7	BURGE [7]	1982
Isocyanates	10/20	9/12	10/20	6/20	ROSENBERG [9]	1987
Isocyanates	31/43	31/43	22/43	5/43	PISATI [11]	1993
Isocyanates	NA	6/9	3/9	0/9	PAGGIARO [12]	1993
Red cedar	37/75	25/33	20/33	4/33	CHAN-YEUNG [14]	1982
Latex	13/16	16/16	8/16	1/16	VANDENPLAS [15]	2002
Total	107/174 (61)	90/120 (75)	68/128 (53)	16/128 (12)		
Reduction of exposure						
Colophony	5/8	4/4	1/4	0/4	BURGE [7]	1982
Isocyanates	7/7	7/7	4/7	0/7	ROSENBERG [9]	1987
Isocyanates	17/17	17/17	0/17	13/17	PISATI [11]	1993
Isocyanates	NA	4/7	3/7	0/7	PAGGIARO [12]	1993
Red cedar	50/50	22/22	0/22	9/22	CHAN-YEUNG [14]	1982
Latex	16/20	19/20	15/20	0/20	VANDENPLAS [15]	2002
Total	95/102 (93)	73/77 (95)	23/77 (30)	22/77 (28)		

Data are presented as number of subjects with outcome variable/number of subjects studied, data in the totals rows are presented as number of subjects with outcome variable/number of subjects studied (% with outcome variable). NA: data not available; NSBH: nonspecific bronchial hyperresponsiveness; #: improvement and worsening of asthma assessed on changes in symptoms and/or medication [9], NSBH [7, 12, 14, 15], or a combination of symptoms, spirometry, and NSBH [11].

(table 1) [7, 9, 11, 12, 14, 15]. Pooled analysis of these studies indicates that reducing exposure is more frequently associated with persistence of asthma symptoms (93%) and NSBH (95%), and with worsening of asthma (28%) than complete removal (61%, 75%, and 12%, respectively).

Exposure to the causative agent can lead to severe asthmatic reactions in subjects with OA, although deaths due to status asthmaticus following workplace exposure appear to be rare [29]. Data from the Ontario Workers' Compensation Board indicate that the rates of hospitalisation for respiratory disease and mortality among subjects compensated for OA are similar to those observed in asthmatic subjects without work-related asthma [30, 31].

How does the natural history of irritant-induced asthma differ in its health outcomes from immunologically-mediated occupational asthma?

A valuable series of case reports have provided convincing evidence that a single massive inhalation of a variety of irritant substances can induce the rapid onset of asthma in previously asymptomatic individuals [32–35]. Since the original description of this clinical entity under the term of "reactive airways dysfunction syndrome" or RADS by BROOKS *et al.* [32], the more inclusive concept of "irritant-induced asthma" arising from single or multiple exposures to high levels of irritants has gained wide acceptance [36, 37]. The pattern of initiation of irritant-induced asthma most strongly supports a primary role for acute or repeated toxic injury of the bronchial epithelium, although the pathophysiological mechanisms leading to the development and persistence of asthma and NSBH remain largely speculative [33, 34]. Relative risks for initiation, progression, or resolution of irritant-induced asthma have been difficult to ascertain. In recent years limited population-based data have begun to emerge that may be able to shed light on the development of irritant-induced asthma after acute exposure to respiratory irritants. Data from a USA poison control centre suggest an incidence figure of 6% for respiratory symptoms lasting ≥ 2 weeks following irritant exposures [38]. Pre-existing lung disorders and cigarette smoking were the only significant risk factors for persistent symptoms. The

Surveillance of Work-related and Occupational Respiratory Disease (SWORD) surveillance program in the UK found that inhalation accidents resulted in the development of RADS in 8.5% of the subjects (*i.e.* 33% of the subjects with symptoms persisting for ≥ 1 month [39].

Case reports, as well as compensation-related and other site-specific clinical referral series are likely to skew available data to greater disease severity. Studies of chemical accidents in which groups of subjects are transiently exposed to high concentrations of airway irritants in the community or workplace indicate that airway obstruction persists in a substantial, although highly variable, proportion (ranging from 10–60%) of subjects with RADS-like symptoms who have been evaluated from 3 months to 13 yrs after the inciting event [40–45]. Several cohort studies have examined the presence of NSBH following high-level exposure to irritants [42–44, 46–51] (table 2). In most of these studies [42, 44, 46–48] however, it cannot be formally established that NSBH was initiated by irritant exposure, since the level of nonspecific airway responsiveness was not assessed before the offending incident. Few cohort studies have assessed the time course of changes in NSBH after inhalation incidents. BLANC *et al.* [47] studied 10 of 45 subjects within 2 weeks after an irritant exposure reported to a poison control centre. Eight subjects demonstrated NSBH, while none had airway obstruction. Three months later, NSBH had reversed in only one subject. A cohort of construction workers who had been repeatedly exposed to chlorine over a 3-month period at a pulp mill have been surveyed prospectively [48, 49]. At the initial investigation carried out 18–24 months after the end of exposure, NSBH was found in 29 (41%) of 71 symptomatic workers considered at risk of developing RADS [48]. On subsequent evaluation of these 29 workers 1 yr later (30 to 36 months after exposure), NSBH persisted in 14 of 19 subjects who underwent repeated methacholine challenge [49]. Six subjects showed a significant improvement in NSBH, including five subjects whose level of airway responsiveness was no longer in the asthmatic range. Improvement in NSBH was not influenced by smoking habits, atopic status, or treatment with inhaled steroids. During a 4 yr prospective survey of 278 workers in a metal production plant [52, 53], 13 workers presented to the first aid unit with transient respiratory symptoms after accidental inhalation of chlorine [50]. Only

Table 2. – Natural history of reactive airways dysfunction syndrome: outcome of symptoms, airway obstruction, and nonspecific bronchial hyperresponsiveness in cohort studies

Type of exposure	No. studied/target population	Interval of time since exposure	Persistence of outcome variables		1st author [ref no.] yr of study
			Symptoms#	FEV1 <80% pred#	
Single, acute exposure to chlorine	13/20 exposed workers	12 yr	NA	NA	SCHWARTZ [42] 1990
Acetic acid spill (hospital workers)	51/56 exposed workers	8 months	8/51	NA	KERN [46] 1991
Inhalation accidents reported to poison-control centre	10/45 Sx>12–24 h (no pre-existing lung disease)	8±4 days [†]	9/10	0/10	BLANC [47] 1993
Single exposure to methyl-isocyanate (pesticide spill)	19/77/00 exposed public (includes pre-existing lung disease)	3 months [§]	4/9	0/9	BLANC [47] 1993
Repeated exposures to chlorine over a 3-month period (pulp-mill workers)	64/71 symptomatic workers ("at risk of RADS")	3–14 months	48/197	2/187 (1%) [¶]	CONE [43] 1994
Repeated exposures to chlorine over a 3-month period (pulp-mill workers)	20/29 subjects with NSBH	18–24 months	58/64	16/51 (23%)	BHERER [48] 1994
Acute exposure to SO ₂ (pyrite dust explosion)	6/7 exposed workers	30–36 months	18/20	8/20 (29%)	MALO [49] 1994
Acute exposure to chlorine (metal production plant)	13 presented to first aid visit over a 4-yr period	13 yrs	6/6	5/6 (71%)	PIRILA [44] 1996
		5–25 days [†]	13	1/13	LEROYER [50] 1998
		3 months [§]	NA	2/13 (15%)	LEROYER [50] 1998
				0/13	

NSBH: nonspecific bronchial hyperresponsiveness; NA: data not available; Sx: respiratory symptoms; FEV1: forced expiratory volume in one second; pred: predicted; RADS: reactive airways dysfunction syndrome; SO₂: sulphur dioxide; #: number of subjects with outcome variable/number of subjects studied (% of the target population); [†]: excluding 10 subjects with aggravation of pre-existing asthma; [‡]: initial assessment; [§]: follow-up assessment.

two of these workers demonstrated a significant increase in NSBH as compared with pre-exposure values. These changes in NSBH completely reversed within 3 months. Taken together, available studies suggest that the pattern of functional changes in irritant-induced OA may be remarkably similar to what has been described in subjects with immunological OA after cessation of exposure to causative sensitising agent, as NSBH can continue to improve for up to 3 yrs following an inhalation accident. A single study suggested that most of the improvement in spirometric values occurs within the first year after acute exposure [44].

Do working conditions have a significant impact on asthma regardless of the aetiology of the disease?

Prevalence estimates of work-aggravated symptoms vary largely according to the populations studied, including 5–17% in general practice [54, 55], 16–31% in tertiary asthma clinics [56, 57], 20% in patients hospitalised for asthma [58], and 20–57% in population-based surveys [59–62]. These findings provide convincing evidence that workplace exposure can cause exacerbation of respiratory symptoms in a substantial proportion of subjects with pre-existing or coincident asthma. In most of these studies, however, work-related changes in physiological indices of asthma were not investigated or, when they were investigated, objective changes could be documented only in a minority of the subjects experiencing asthma symptoms at work [57]. Thus, although widely used, terms such as "work-aggravated asthma" or "work-exacerbated asthma" most often refer to subjective worsening of asthma symptoms at work without objective lung function correlates. In addition, most controlled experiments among asthmatic volunteers have failed to demonstrate a physiologically relevant effect of exposure to irritant substances at permissible levels on airway calibre, level of NSBH, or airway inflammation [63–69], although some individuals may develop minimal changes in airway calibre [64, 65, 68]. Available data suggest that sulphur dioxide [70] and chlorine [71] can induce transient decrements in lung function in asthmatic subjects at lower levels than in those without NSBH. Moreover, it should be noted that several studies have identified considerable discordance between perceived symptoms and physiological parameters [66, 67], suggesting that lung-function tests alone may not adequately gauge the impact of exposure to irritant substances.

What is the scope of work disability from work-related asthma in social and economic terms?

The negative impact on the indirect cost of asthma can take many forms, including reduced workforce participation and employment rates, changes in employment or job duties as an adjustment to the asthmatic condition, asthma-related lost work days, and impaired work effectiveness while on the job. One cohort study of young adults showed that the rate of unemployment at the age of 23 yrs was slightly higher in those who reported a history of current or past asthma, independently from the severity of asthma [72]. In a random sample of 401 adult asthmatics, aged 18–50 yrs, seen by pulmonologists and allergy-immunologists in California, asthma was the reported cause of work cessation in 7% of the subjects [73]. In the study, work disability was related to asthma severity and job conditions [73]. When this cohort was assessed longitudinally and compared with an age-matched sample from the general population, however, asthma did not appear to affect employment status substantially, although

individuals who were not working at the time of disease onset were less likely to be employed in subsequent years [74]. Demographic and employment characteristics were the strongest determinants of continuity of employment, while medical variables did not drive employment status.

Partial work disability, defined as any change in job duties or reduction in work hours due to asthma, has been described in 5% to 20% of adult asthmatics attending specialist clinics [60, 73, 75]. In a population-based study of Swedish adults [61], partial work disability due to respiratory problems was reported by 2% of subjects without asthma, 13% of subjects with self-reported asthma, and 22% of asthmatics with documented NSBH. A number of studies have indicated that asthmatic workers have a higher incidence of absenteeism [59, 76, 77]. In a recent USA survey, employer expenditures for asthmatic subjects was ~2.5 times higher than those for control nonasthmatic subjects [78]. Wage-replacement costs for lost work days accounted for almost as much as medical care expenses. Even when asthmatic subjects are on the job, a substantial proportion of them (ranging from 9–27%) report a reduction in work effectiveness because of their condition (60, 76, 77). Impairment in self-reported work effectiveness has been associated with more severe asthma, marked NSBH, and the presence of work-related respiratory symptoms [60].

Immunological occupational asthma

However potent the impact of asthma is in general, the work impact appears to be even more dramatic among the subset of adults with OA. Multiple studies conducted in various countries have shown that immunological OA is associated with considerable professional and financial consequences [22, 79–87]. As summarised in table 3, 25–38% of subjects with OA suffer prolonged work disruption and 42–78% report a substantial loss of income [80–84, 87].

Financial consequences of OA are consistently more pronounced in workers who avoid further exposure to the causal agent [80, 84, 87]. This is likely to account for the finding that almost one third of workers with immunological

OA remain exposed to the causative agent (table 3). The medical consequences of exposure interventions have rarely been weighed against their financial impact. Only one prospective study has compared asthma severity, disease-related costs, and work-derived income after cessation or persistence of exposure to various agents causing OA [85]. About 40% of subjects who remained exposed had reduced levels of exposure to the causal agent. Complete avoidance of exposure to the causal agent resulted in a significant decrease in asthma severity and in healthcare expenses, but also in work-derived income, as compared with persistence of exposure [85]. In subjects with latex-induced OA [15], respiratory health outcomes improved similarly after reduction or cessation of exposure to airborne latex, whereas reduction of exposure resulted in a substantially lower professional and economic impact.

A number of vocational and sociodemographic factors adversely affect employment and socioeconomic status in workers with OA; these include unskilled jobs [80, 87], lower levels of education [84, 87], older age [81, 87], younger age [84], a lower number of economically-dependent subjects [81], and being employed in small-sized firms [84]. Interestingly, the clinical severity of asthma does not appear to be an important determinant of employment status in subjects with OA [79–85, 87].

The rate of unemployment among subjects with immunological OA seems higher than in the general population, although there is a need for further characterisation of the specific impact of OA as compared with nonwork-related asthma. In the only available study that compared immunological OA with nonwork-related asthma, the rate of unemployment was similar in the two groups, whereas a reduction of income was more frequently reported by subjects with OA (62%) as compared with those with asthma unrelated to work (38%) [83]. These two groups were not, however, matched for the severity of asthma and other relevant sociodemographic variables. Partial work disability, defined by any job change or work disruption due to asthma, occurred in 72% of Belgian subjects with ascertained OA and in 74% of Swedish subjects with claimed OA [88].

Table 3. – Socioeconomic consequences of occupational asthma

	Country of study					
	UK	British Columbia, Canada	Quebec, Canada	UK	France	Belgium
1st author [ref. no]	GANNON [80]	MARABINI [81]	DEWITTE [82]	CANNON [83]	AMEILLE [84]	LARBANOIS [87]
Population	112/140	128/181	134/211	87/113	209/257	86/95
Duration of follow-up yr	median: 1.4	mean: 4.8/5.6	range: 2–5	5	mean: 3.1	median: 3.3
Not working	35	41	25	39	34	38
Unemployment	20	38	8	NA	25	26
Early retirement	15	4	16	NA	3	2
Chronic sick leave	5	NA	NA	NA	6	10
Currently working	65	58	75	61	66	62
Other job, same employer	21	NA	31	21	18	15
New employer	14	NA	36	NA	16	26
Retraining for another job	NA	NA	8	NA	NA	0
Persistence of exposure	30	38	0	NA	32	33
Loss of income						
Exposed:	44 [#]	NA	NA	55	46	42 [#]
Unexposed:	74	NA	NA			78
Compensation	24	70	100	28	49	87
Loss of income offset by compensation [†]	NA	NA	NA	NA	44	22
Job retraining program	NA	NA	22	NA	NA	5

Data are expressed as per cent of the total number of subjects included in the study unless otherwise stated. [#]: per cent of subjects included in each exposure category; [†]: expressed as per cent of subjects who were compensated; NA: data not available.

Work-aggravated asthma

The socioeconomic impact of work-aggravated asthma symptoms has received relatively little attention given the potential magnitude of the problem. The results of the few available studies show that, even in the absence of OA or irritant-induced asthma, work-aggravated asthma is associated with a considerable socioeconomic impact. CANNON *et al.* [83] found that the rate of unemployment was not different in work-exacerbated asthma (31%), immunological OA (39%), or "asthma unrelated to work" (32%). In contrast, a reduction of income was reported more frequently by subjects with work-exacerbated asthma (65%) and OA (62%) than by those with "asthma unrelated to work" (38%). In this study, however, all subjects were investigated for possible OA and the criteria used for diagnostic categorisation were not precisely defined. LARBANOIS *et al.* [87] compared the socioeconomic outcomes of subjects with ascertained immunological OA with those of subjects who experienced work-aggravated asthma symptoms but failed to exhibit a positive response to specific inhalation challenges. The rates of unemployment (46%) and income loss (59%) in subjects with work-aggravated asthma symptoms did not differ significantly from those observed in subjects with OA (38% and 62%, respectively). Partial work disability, defined as any job change or work loss due to asthma, was slightly less common in subjects with work-aggravated asthma symptoms (54%) than in those with OA (72%).

What is the clinician's role in reducing the respiratory health consequences of work-related asthma?

Immunological occupational asthma

Targeting those factors that determine the persistence of asthma after removal from exposure is most relevant for improving respiratory health consequences of immunological OA. Indeed, making a reliable diagnosis of OA early after its onset could be the most straightforward way to reduce the long-term functional impairment resulting from OA. There is considerable evidence that OA remains largely underdiagnosed by healthcare providers. In a recent survey of asthmatic members of a health maintenance organisation in the USA, the work-relatedness of asthmatic symptoms was investigated by only 15% of general practitioners and 50% of allergists and chest physicians [55]. In addition, information from clinical studies, compensation statistics, and specific surveillance programs indicates a general lack of objective assessment of the work-relatedness of asthma [57, 89–92]. Once OA is suspected, the diagnosis should be promptly ascertained or excluded using the most reliable procedures, including monitoring of spirometry or peak expiratory flow at work and, where appropriate, specific inhalation challenges. As these diagnostic procedures require expertise and/or special equipment, they should be made more accessible through the formation of specialised diagnostic centres or through training a larger cadre of physicians with specific expertise in this area [92].

The impact of pharmacological therapy on the OA outcome remains uncertain. A retrospective study of subjects with OA found that treatment with inhaled steroids was not a significant determinant of changes in NSBH after withdrawal from exposure [18]. Only two studies have prospectively examined the effects of inhaled steroids in OA [93, 94]. These studies suggested that adding inhaled steroids to removal from exposure resulted in a small improvement in asthma symptoms, quality of life, peak expiratory flow rates [94], and

NSBH [93]. Worth noting is the fact that in one study, the beneficial effects of inhaled steroids appeared to be more pronounced when the treatment was started early after diagnosis [94].

Irritant-induced asthma

There are no controlled clinical data suggesting that irritant-induced asthma should be managed differently than any other form of asthma in terms of pharmacological treatment. Anecdotal reports indicate that early treatment with oral or inhaled steroids could exert a beneficial effect on the course of RADS [95–97], although prospective studies are required to determine the optimum dose and duration of treatment with inhaled steroids. When irritant-induced asthma has been provoked by acute exposure to agents that are also known to be sensitizers [36, 96, 98, 99], the possibility of specific bronchial hyperresponsiveness to these agents should also be taken into account [100, 101]. In most cases, however, workers with irritant-induced asthma are not, by definition, more reactive to the offending agent than other asthmatics. Accordingly, they can continue to work in the same environment, provided that levels of airborne irritants are kept below permissible levels at the workplace. Subjects with NSBH may, however, develop bronchial responses at lower levels of irritant substances, such as sulphur dioxide and chlorine [71], than it takes to provoke similar reactions in healthy subjects. The management of irritant-induced asthma may be further complicated by associated disorders, such as chronic rhinitis [102–104], perceived intolerance to multiple chemicals [105], hyperventilation syndrome [106], vocal cord dysfunction [107], and increased cough sensitivity [108], which can be associated with accidental exposure to irritant substances at work.

Work-aggravated asthma

The environmental and host factors that determine worsening of asthma symptoms at work should be further identified in order to improve the medical management of work-aggravated asthma symptoms. It has been proposed that work-related aggravation of asthma symptoms may reflect either exposure to higher-than-permissible levels of irritants at work, inappropriate treatment, or more severe underlying asthma [109]. Arguing against severity as a significant determinant, studies have observed that subjects with work-aggravated asthma show features indicating a milder asthma condition than what is found in those who do not experience worsening of symptoms at work [57] or those who have immunological OA [87]. Preliminary findings from a USA study comparing subjects with work-aggravated and occupational asthma suggest that subjects with work-aggravated asthma are younger, include a higher proportion of females and non-Whites, and are more often exposed to inorganic dust [110].

Levels of these irritants should be kept to acceptable limits at work. It has to be recognised, however, that most of these limits have been derived from healthy populations or extrapolated from animal experiments, while the concentrations that trigger airway irritation can be different in susceptible populations such as those with asthma. The efficacy of anti-asthma medications in improving work-related asthma symptoms and work performance warrants further prospective investigation using work-related symptoms and work-disability indices as endpoints.

To what extent do existing compensation and other social insurance schemes successfully address occupational asthma and work-aggravated asthma?

Physiological impairment resulting from asthma can be quantified using the degree of airflow limitation, the level of NSBH, and medications required to control symptoms, as recommended by the American Thoracic Society (ATS) [111]. Assessment of disability is much more complex, as it should take into account the impact that impairment may have in terms of function, such as the ability to work full time and without restriction of job duties or demands. Impairment does not lead to disability in a predictable manner. For instance, subjects with immunological OA should be considered completely and permanently disabled in terms of occupations that entail exposure to the sensitising agent that caused OA, even when their asthma demonstrates little physiological impairment without such exposure.

Objective assessment of the effectiveness of compensation systems, whether national or international, has seldom been carried out for occupational diseases in general and even less so for work-related asthma in particular. Available information (table 3) indicates that financial compensation for OA is currently inadequate in European countries, since about one third of subjects with OA remain exposed to the agent causing their asthma in order to avoid or minimise the adverse financial effects that would result from complete avoidance of exposure [80, 84, 87]. Even when assessment of impairment/disability is based on the ATS recommendations, the loss of income is offset by awarded indemnities in only 22% of subjects with OA [87].

It appears that the rate of unemployment is lower among subjects with OA in Quebec (25%) than in other countries (34–41%), despite removal from exposure of all affected workers (table 3). This lower rate of unemployment is coincident with a higher proportion of workers who benefit from professional retraining programs (22%) and/or find another job with the same employer (31% versus 15–21%). Extrapolating from this limited information, compensation schemes might be more effective to the extent that they can be redirected toward facilitating relocation into nonexposed jobs in the same company or retraining for other jobs rather than providing inadequate financial compensation. Furthermore, to be more equitable, compensation should ensure more appropriate income replacement when relocation or professional rehabilitation brings about a loss of earnings. Improving compensation for OA almost certainly requires that the cost and effectiveness of existing compensation systems be systematically evaluated.

A detailed review of international workers compensation schemes for OA and work-related asthma is far beyond the scope of this clinically-oriented summary. Differences among various systems are likely attributable to multiple factors, including economic forces, historical precedent, pattern of illness, cultural values, and political power balances. What is clear about compensation and asthma is that, by and large, all systems appear to address OA in a fairly heterogeneous manner.

Conclusion and needs for future research

Since the consequences of immunological occupational asthma in terms of respiratory health and socioeconomic status are now becoming better characterised, the major challenges for the clinicians and healthcare policy makers must shift from passive documentation of the problem toward the identification, implementation, and evaluation of

strategies aimed at minimising these adverse impacts. Because the understanding of the natural history of irritant-induced asthma is more limited, the need for descriptive data is more pressing and prevention is an even greater challenge. Work-aggravated asthma is an even more complex issue, since the very definition of what it entails continues to evolve as new data emerge. Clearly, understanding of the environmental and individual cofactors involved in the development and persistence of the various work-related asthma conditions should be enhanced in order to improve their management and reduce their health and economic impacts.

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