# Prevention of work-related asthma seen from the workplace and the public health perspective

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## Abstract

Work-related asthma (WRA) includes occupational asthma and work-exacerbated asthma. WRA is by definition preventable. This chapter discusses available tools for prevention of WRA, divided into primary and secondary prevention. For each tool, the available evidence for the effectiveness of the tool is summarized, and examples are provided. Primary prevention addresses healthy workers or persons with asthma due to causes unrelated to work. The principal tool is control of occupational exposure, reached by elimination or reduction in exposure, but vocational guidance and pre-employment screening are also regarded as primary prevention tools. Secondary prevention addresses early detection of work-related sensitization or WRA to prevent further progression. The principal tool for secondary prevention is medical surveillance. Prediction models represent a promising new tool in medical surveillance; this tool is described here in general and by an example. To set priorities for the prevention of WRA, the monitoring of occurrence in populations as well as in specific industries is crucial, and this chapter therefore briefly describes different sources for surveillance data including sentinel reporting systems, population studies, and occupational disease registers. In the future, focus should be on well-conducted intervention studies, improved exposure assessment, improved medical surveillance (e.g., using prediction models) and good quality national surveillance programs.

## Introduction

Work-related asthma (WRA) includes occupational asthma (OA) and work-exacerbated asthma (WEA) [1]. OA, or asthma caused by work, is the most common occupational lung disease in developed countries [2]. In addition, WEA, or pre-existing or concurrent/coincident asthma worsened by work factors, is probably even more prevalent and deserves increasing awareness due to the increase in asthma per se during the last 20 years [3]. Concurrent or coincident asthma has onset during employment but is not caused by conditions at work.

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WRA is by definition preventable. The following chapter discusses available tools for prevention of WRA, divided into primary (prevention of development) and secondary (prevention of progression) prevention. Tertiary prevention, or management of WRA, is dealt with in a separate chapter.

Suggestions for evidenced-based guidelines for prevention and management have recently become available for OA [4, 5]. For each tool, the available evidence for the effectiveness of the tool is summarized, and examples are provided.

Prevention of OA and WEA is in general covered together. With regard to primary and secondary prevention, tools for preventing OA and WEA are in principle identical. In order to set priorities for the prevention of WRA, the monitoring of occurrence in populations as well as in specific industries is crucial. This chapter therefore briefly describes different sources for surveillance data including sentinel reporting systems, population studies, and occupational disease registers.

## Primary prevention

Here primary prevention addresses healthy workers or persons with asthma caused by reasons other than work. The aim of primary prevention is to prevent development of work-related sensitization and, most importantly, WRA. The principal tool for primary prevention is control of occupational exposure, reached by elimination or reduction in exposure.

As we consider prevention of both OA and WEA as primary prevention, vocational guidance and pre-employment screening are also described in this section.

### Control of occupational exposure

According to Nicholson et al. [5], evidence based on well-conducted case-control or cohort studies suggests that reducing airborne exposure reduces the number of workers who become sensitized and who develop OA. In Table 1, different ways of controlling exposure are given focusing on source, room, or person, in decreasing order of preference.

In some industries comprehensive knowledge about determinants of exposure is available. A classic example from healthcare is substitution of low-protein powder-free natural rubber latex (NRL) gloves or non-NRL gloves for powdered NRL gloves. A well-conducted prospective cross-over trial in an operation room found that the mean aeroallergen level was decreased from 13.7 to 1.1 ng/m$^3$ on days where low-allergen gloves were used [6]. Other examples are studies in bakeries [7–9], wood industries [10–13], and hair dressing saloons [14], where for instance work task, cleaning procedures, quality of ventilation systems, and work routines determine the level of the exposure of interest.

*Table 1. Different ways of controlling exposure*

| | |
|---|---|
| The source | - Substitution for the harmful agent<br>- Enclosure, automation, or modification of the process |
| The room | - Ventilation<br>- Avoiding resuspension of the harmful agent (e.g., cleaning procedures, work practices) |
| The person | - Personal respirators<br>- Administrative initiatives (reduce number of workers or duration of work time close to the harmful agent) |

Several epidemiological studies have documented dose-response relations between high-molecular-weight (HMW) and low-molecular-weight (LMW) allergens and the occurrence of WRA or sensitization, e.g., in bakeries and flour mills [15–18], lab animal workers [19–22], wood workers [23, 24], and isocyanate workers [25–27], strongly suggesting that control of occupational exposure is effective in prevention of WRA.

Only a few studies have directly explored the effect of preventive measures on the occurrence of WRA or sensitization. NRL is the single most common agent addressed in primary preventive intervention studies, as reviewed by Lamontagne et al. [28]. The NRL studies all explored the effect of changing from high-protein powdered gloves to low-protein powder-free NRL or non-NRL gloves, upon either the occurrence of NRL sensitization [29–31] or the occurrence of NRL asthma or NRL-related symptoms [31, 32]. None of the studies fulfilled strict criteria for good quality intervention studies, i.e., they were observational studies without a randomized design and without a control group, but taken together they support assertions that substitution of NRL greatly reduces NRL sensitization and the occurrence of NRL-related asthma.

Smith [33] describes an attempt to prevent bakers' asthma in a UK food company. The intervention was a 5-year health surveillance program, and by no means a strict intervention study. Among other methods, they aimed at decreasing the general total dust level to $< 10$ mg/m$^3$, and the bread improver exposures to $< 1$ mg/m$^3$, to diminish exposure to mainly fungal amylases. They focused on information and training, installation of local exhaust ventilation, and wearing of respirators during handling of powdered bread improvers. During the 10 years following 1993, they found a decrease in the annual incidence rates of symptomatic sensitization (mostly flour and fungal amylase) from 2085 per million to 405 per million employees per year from the first 5 to the second 5 years. They did not measure the possible decrease in exposure from 1993 to 2003.

An ambitious intervention study was started in the Netherlands in the flour processing sector [9]. More than 900 personal measurements from four flour processing plants together with a thorough collection of control measures were used to model

the baseline exposure level and to rule out significant determinants of exposure. The Dutch government and the flour processing sector association agreed to participate in reducing exposure to flour dust. Dust reducing control measures were implemented in the different sectors along with monitoring of trends in exposure as well as sensitization and symptoms in the sector-wide health surveillance system.

In Ontario, the Ministry of Labour introduced a preventive program for diisocyanates in 1983, consisting of a mandatory 0.005 ppm airborne exposure limit for diisocyanates together with a medical surveillance program. Tarlo et al. [34] assessed retrospectively workers compensation data from 1980 to 1993. They showed an initial increase in compensation claims, which was attributed to increased case finding due to the medical surveillance program. The 50% decrease in accepted claims from 1991 to 1992–1993 was attributed to a combination of primary and secondary prevention measures. When measured levels of diisocyanate were compared among companies who had compensated claims for OA with companies without accepted claims, the former were more likely to have had measured levels of diisocyanates >0.005 ppm [35].

In the detergent industry, the occurrence of sensitization among employees apparently decreased dramatically from the late 1960s to the mid-1980s [36]. During this period the detergent industry association had published work practices, and at the same time medical surveillance programs were introduced. Two publications sponsored by major manufacturing companies described significant reductions in the prevalence of OA after introducing granulated proteases [37, 38]. Unfortunately neither study reported incidence rates. A Danish retrospective follow-up study reports decreasing incidence rates from the 1970s to 1990s for sensitization (0.2 to 0.06 per person year), but not for allergic diseases (0.03 to 0.02 per person year) among 1207 enzyme plant workers followed the first 3 year of their employment [39]. Cullinan et al. [40] reported an outbreak of asthma in a modern detergent factory that exclusively used encapsulated enzymes. As many as 90 (26%) of the workers were sensitized to a least one detergent enzyme, and 7% had a confirmed diagnosis of OA. Sensitization rate was clearly related to exposure level. This study indicates that the use of encapsulated enzymes is insufficient to control exposure and prevent enzyme-induced OA.

Case reports on OA caused by newly introduced enzymes [41, 42] highlight the importance of careful surveillance after introduction of new agents in the workplace. In addition, exposure to enzymes has increasingly shifted from the detergent industry to intermediate industries, e.g., the baking industry, where people are exposed to enzymes in low-technology environments [15].

In the US, a major pharmaceutical company introduced a preventive program for laboratory animal allergy (LAA) [43]. The program included education, engineering controls, administrative controls, use of respirators and medical surveillance. During a 5-year period, the incidence rate of asthma decreased from 10% to 0%. In the same period, the percent of workers using respirators increased from 86% to 100%

(workers with LAA) and from 50% to 81% (workers without LAA). No data on other specific preventive measures were available.

In the UK, Botham et al. [44] studied a retrospectively assembled cohort of new employees working with laboratory animals. In 1981, an education program for persons working with laboratory animals was introduced. From 1980 to 1984, the annual cumulative incidence proportion of symptoms consistent with LAA decreased from 44% to 16%, and this effect was at least partly attributed to the educational program.

## Use of respiratory protective equipment

There is only limited evidence, based on mainly two non-analytical studies, to support a reduction in the incidence of OA through the use of respiratory protective equipment (RPE). Grammer et al. [45] investigated the effect of introducing RPE devices among 66 newly hired workers in an epoxy resin producing factory using acid anhydrides. Only 4 of the 66 workers did not use RPE. From 1993 to 1999 the incidence rate of acid anhydride sensitization combined with respiratory symptoms decreased from 10% to 2%.

In a new wood plant that uses diisocyanate, Petsonk et al. [46] prospectively estimated respiratory health and work practices over a 2-year period. Workers who indicated that they had briefly removed respiratory protection had a five times higher prevalence of new-onset asthma-like symptoms, compared to individuals who reported never doing this (25% *versus* 5%).

## Vocational guidance and pre-employment screening

The main purpose of vocational guidance before job choice, and pre-employment screening, is to avoid persons at risk being exposed to sensitizers or irritant work exposures to prevent OA and WEA. According to Nicholson et al. [4] some evidence supports the claim that screening criteria do an inadequate job of identifying potentially susceptible individuals.

Knowledge of the effect of vocational guidance on career choice among teenagers is sparse. In some countries, e.g., Germany, Denmark and Sweden, vocational guidance is a well-established practice in primary schools, but only a few evaluations of the effects have been performed. In 1992 in Sweden, Bremberg et al. [47] evaluated whether medical vocational guidance among chronically ill preliminary school students (including asthma) had any impact on their choice of career. Only 5 of 235 students stated that vocational information from physicians and school nurses had been important for their choice of career, and the distribution of job choices among the chronically ill students did not differ from the job choices among

all students. A German study [48] and a Swedish Study [49] longitudinally elucidated career choices among asthmatics. They found self selection into low risk jobs to play a minor role in teenagers with asthma or allergy. This could indicate that vocational guidance among teenagers has a limited impact or has not been given. Knowledge about the quality and nature of vocational guidance and the impact on career choices is not available.

The effectiveness of using personal risk factors in pre-employment screening is low [21, 50–53], which has been very well illustrated by Sorgdrager et al. [54]. They used data on personal risk factors (atopic history, eosinophil count, lung function) and incidence of pot room asthma from a nested case-control study to estimate indicators of effectiveness of pre-employment screening. They calculated the positive predictive value (PPV), number needed to test and number needed to reject on a simulated population of 10 000 persons with high incidence rates (40 cases/1000 person-year) and 10 000 with low incidence (5 cases/1000 person-year) of pot room asthma. The atopic history prevalence was 6–12 times higher among asthmatics compared to controls depending on the incidence rate of asthma. In general, atopic history was the most effective indicator. The PPV was 20% at high incidence rates, and an even lower 7% at low incidence rates. At high incidence rates it was necessary to test 138 persons to prevent one case of OA, and for each prevented case of OA 5 persons were rejected from the job. They concluded that the personal risk factors were far from effective as a selection instrument.

There is an increasing focus on genetic testing for screening out susceptible subjects. Theoretically, genetic testing could be used for pre-employment screening of OA. So far associations between particular mutations and asthma occurrence are modest with low odds ratios. Thus, prediction of future occurrence is unlikely to be effective. Furthermore, asthma is caused by multiple genetic and environmental factors, and information obtained by a single gene test is limited for both diagnostic and preventive causes. Taken together, genetic testing is currently not useful for identifying susceptible subjects in pre-employment screening [55, 56]

In conclusion, screening out susceptible individuals for asthma seems to be inefficient, and it might discourage efforts to reduce risks and prevent diseases in the general working population. Vocational guidance and pre-employment screening should not be used to discriminate who should or should not get a job, but may be valuable tools to give persons at risk, e.g., atopic persons, an informed view of their chance of developing OA or work-aggravated symptoms.

## Secondary prevention

Secondary prevention addresses early detection of work-related sensitization or WRA in order to prevent further progression. The principal tool for secondary prevention is medical surveillance.

## Medical surveillance of work-related asthma

Individuals with high exposure levels to aeroallergens are more likely to have serious respiratory complaints and disability than workers exposed at lower levels. Even at very low levels a residual sensitization risk remains. Studies also suggest that the risk for work-related respiratory diseases cannot be avoided completely by exposure reduction [57, 58]. Although elimination of airborne allergens from the workplace is the ideal approach, it may not be possible in many workplaces such as bakeries, and animal care facilities. Even if a reduction of exposure has been shown feasible, there is no known no-effect level, other than zero, that will prevent sensitization in all exposed workers. As an example, the experience with NRL, involving the substitution of powdered gloves with "powder-free" gloves, has shown a dramatic reduction of new cases of contact urticaria among health care workers. However, allergic symptoms and asthma remained, although at lower prevalences, after this intervention [32, 59, 60].

The duration of symptoms while working was studied in Canada [60, 61]; the results showed a significant period of time between symptom onset and diagnosis of OA. The reported median time to the first suspicion of WRA by a physician was 1 year for WEA and 2 years for OA patients. The median time to a final diagnosis of OA after the onset of work-related symptoms was 4 years. Patients with OA waited on average 8 months (median 3 months) before discussing their symptoms with a physician. Lower education level and household income were significantly associated with an increased time to diagnosis.

Although exposure reduction may markedly reduce the incidence of OA, the ongoing high rates of work-related allergic diseases and the long time to diagnosis of WRA reinforces the need for secondary prevention by medical surveillance. Therefore, parallel to exposure reduction and exposure control, medical surveillance of the entire workforce should be conducted to detect early evidence of all work-related allergic and respiratory diseases.

However, different problems limit the successful use of medical surveillance programs for the identification of WRA, because asthma is characteristically a disease with exacerbations and remissions, and WRA may, therefore, be unnoticed. Self-reported symptoms and use of medication failed to identify WRA exacerbations as determined by serial peak exploratory flow measurements [62]. Routine surveillance programs in bakery workers showed that the use of questionnaire-reported respiratory symptoms could not discriminate bakery workers with and without clinical diagnosed asthma or specific IgE for baker-related allergens [51, 63]. In addition, disease outcomes are not identical in different workplace circumstances with different allergen exposures. In the case of low-molecular-weight sensitizers such as diisocyanates, the immunological mechanism and the respiratory health outcome are less clear.

Most of the evidence mentioned in the "Guideline for the prevention, identification, and management of occupational asthma" is, however, derived from (clinical)

case-control studies. The estimates of sensitivity and specificity are, therefore, biased by preferential referral of patients and distort the determination of predictive accuracies [64]. Besides, the predictive value of a test varies not only across different populations but also within a particular study population, and consequently may have different sensitivities and specificities [40, 65]. So, a generalized conclusion that a medical questionnaire is not a sensitive instrument for diagnosing OA can hardly be drawn.

The diagnosis of WRA is a phased and complex process that requires both the diagnosis of asthma and establishment of the relationship with work. It can only be made at an individual level in a clinical setting [1]. Medical surveillance programs should, therefore, not focus on clinically established allergic respiratory diseases, but on highly associated preliminary characteristics to identify workers at risk of having a work-related (allergic) disease. Sensitization to occupational allergens is one of these strongly related outcomes linked to OA and often the most appropriate preliminary characteristic that can easily be investigated. For occupations characterized by HMW allergens, a logical approach is therefore to identify sensitized workers first, followed by sequential diagnostic investigations only in these workers. Usually, sensitized workers are detected when they present themselves to the occupational physician with symptoms. However, for patients with WRA, only 6% consulted the company doctor first [66]. So, to find all sensitized workers, the whole population must be evaluated by a specific skin prick test (SPT) or IgE serology, which is less efficient and will result in high expenses for occupational health services.

Traditionally, "standardized" respiratory questionnaires used in epidemiological studies and medical surveillance programs contain questions about general and work-related respiratory symptoms, allergy, and asthma. Every answer to a simple question, such as "do you wheeze", can be considered as a test result. However, different questions often provide the same information because they are all associated with the same underlying disorder, and thus mutually correlated. For the occupational physician it is relevant to know which questions are redundant and which have true, independent additional predictive value for the presence or absence of, for instance, sensitization. Assessing the results of a particular test in view of other test results may even diminish its diagnostic contribution, simply because the information provided by that test is already provided by the other tests.

So far, none of the medical surveillance programs have made use of prediction research-derived diagnostic models in which personal and work-related characteristics are applied to estimate the individual probability of the presence (diagnostic) or occurrence (prognostic) of an outcome that is closely related to the disease(s) of interest. After detecting workers with an elevated risk, sequential diagnostic investigations are only necessary in these workers. So, by using a questionnaire as a first phase instrument, the probability of the occurrence of sensitization can be calculated and subsequently be followed by more advanced tests in the clinical evaluation.

## Prediction research and risk stratification

A diagnosis of a disease is the consequence of interpretations by individual medical doctors of consecutive test results, estimating the probability of the presence of a disease or other outcome of interest. As many test results generate more or less identical information it is important to evaluate the independent and additional predictive value of a test given the presence of earlier information. Prediction research offers a solution for this by using a multivariate approach that accounts for mutual dependence between different test results. The information of every item is translated into a predicted probability of the chosen outcome. This technique provides estimations of the probability of an outcome at present (diagnosis) or in the future (prognosis). Prediction models applied in occupational health practice may therefore enable occupational physicians to deal with uncertainties in considering workers at risk of having occupational diseases. The main goal is to optimize risk estimation at low costs, and may be the first step in the clinical evaluation and management of WRA [67, 68]. The models may initiate counseling and interventions, and are thus useful for identification of specific groups at risk. In Figure 1, a flow chart of medical surveillance of WRA using scores is outlined.

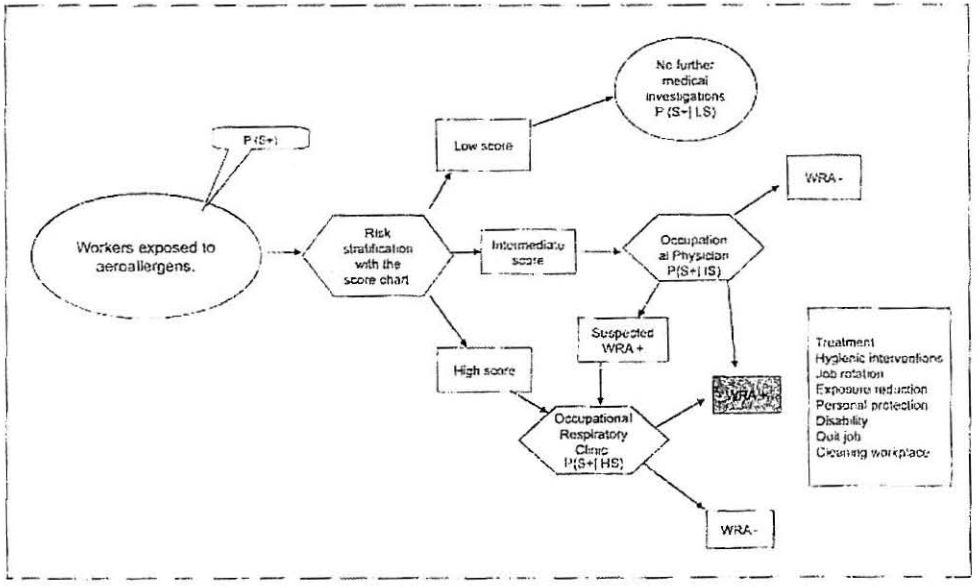


*Figure 1.*
*Medical surveillance of work related asthma (WRA). P (S+), prior probability of sensitization to aeroallergens; P(S+|LS), probability of sensitization conditionally a low score; OAD, occupational allergic disease.*

## An example of a prediction model in occupational health practice

Models to predict IgE sensitization have only recently been developed for workers such as bakers and laboratory animal workers who are exposed to HMW allergens [67–69]. The models were transformed into scoring rules with a restricted number of questionnaire items with different weighing factors. In a medical surveillance program among 5325 bakers in the Netherlands, a short questionnaire, containing 19 questions with four predictors for sensitization to wheat and/or fungal α-amylase allergens, was used as a decision tool in considering workers at risk of WRA. The results of the questionnaire were transformed into sum scores to predict the presence of sensitization in every individual worker as shown in Table 2.

*Table 2. Predictors and Predicted probability for sensitization (wheat and or α-amylase) among bakers*

| Score chart | | |
|---|---|---|
| **Predictors** | **Answer** | **Score** |
| "Have you ever had asthma in the past 12 months?" | If yes | 2 |
| "Have you ever had allergic rhinitis including hay-fever?" | If yes | 2 |
| "Have you ever had itchy and/or red eyes in the past 12 months?" | If yes | 1 |
| "Do you experience more of the following symptoms during work: shortness of breath, chest tightness, itchy eyes, itchy nose, and/or sneezing?" | If yes | 1.5 |
| Sum scores | Max | 6.5 |

| **Sum score** | **0** | **1** | **2** | **3.5** | **4.5** | **5.5** | **6.5** |
|---|---|---|---|---|---|---|---|
| Predicted probability (%) | 9 | 14 | 20 | 31 | 42 | 53 | 64 |

The scores were used to split bakery workers into three groups with different sensitization risk: a high-risk group in which detailed clinical investigations were required to set a diagnosis of WRA and other work-related allergies more accurately, an intermediate group in which medical follow-up by occupational physicians was essential for diagnosis and health protection, and a low-risk group comprising about 60% of the population in which medical investigations could be held back. The results are shown in Table 3.

*Table 3. General characteristics and questionnaire responses across low-, intermediate and high-score groups*

| | Low score (≤1.0) | Intermediate score (1.5–3.0) | High score (≥3.5) | Total |
|---|---|---|---|---|
| Participants, *n* (%) | 3059 (57.4) | 1282 (24.1) | 984 (18.5) | 5325 |
| Work duration, mean years (SE) | 13.8 (0.2) | 11.7 (0.3) | 12.9 (0.3) | 13.1 (0.14) |
| Upper or lower respiratory tract symptoms to common allergens (at least 1 positive answer), *n* (%) | 209 (6.8) | 448 (35.2) | 642 (65.8) | 1299 (24.5) |
| Symptoms suggestive for NSBHR (at least 2 positive answers), *n* (%) | 24 (.8) | 52 (4.1) | 144 (14.9) | 220 (4.2) |
| Use of medication to improve respiratory complaints in the last 12 months (e.g. inhalants), *n* (%) | 74 (2.4) | 186 (14.6) | 468 (48.1) | 728 (13.7) |
| Doctor visit for allergic complaints in the last 12 months, *n* (%) | 134 (4.4) | 197 (15.5) | 379 (38.7) | 710 (13.4) |
| Absenteeism due to allergic symptoms in the last 12 months, *n* (%) | 6 (0.2) | 34 (2.7) | 79 (8.2) | 119 (2.2) |
| Change of function or task due to respiratory symptoms, *n* (%) | 16 (0.5) | 19 (1.5) | 83 (8.5) | 118 (2.2) |

The predicted probability of sensitization can be calculated as:

$$= 1 / \{1 + EXP[-(-2.32 + 0.92 \times \text{asthma} + 0.90 \times \text{rhinitis} + 0.46 \times \text{conjunctivitis} + 0.62 \times \text{during work symptom})]\}.$$

Workers with high scores showed the highest IgE sensitization rate to wheat and/or α-amylase, and the highest rates in medication use, absenteeism, and doctor's visits.

This example illustrates that by using a score chart to predict the sensitization risk in workers exposed to HMW workplace allergens, a diagnosis of WRA can be considered more effectively and efficiently in the initial phase of a medical surveillance program performed by occupational physicians at the worksite. Prediction models based on IgE-mediated sensitization is not useful for LMW workplace allergens. To our knowledge no prediction models for LMW-related OA has been developed, but models based on bronchial hyperresponsiveness (BHR) could potentially be useful.

## Use of public health surveillance data to stimulate, guide, and document prevention of work-related asthma

Public health surveillance comprises more than the identification and counting of cases. It is "the ongoing systematic collection, analysis, and interpretation of health data essential to the planning, implementation, and evaluation of public health practices, closely integrated with the timely dissemination of these data to those who need to know." [70]. This section highlights examples of how public health surveillance has contributed to the prevention of WRA. While the examples are drawn from experience in the United States and Canada, this does not mean that similar surveillance activities are lacking in other countries.

Investigations of reported cases have identified the measures needed to prevent the onset and/exacerbation of asthma in specific workplaces, benefiting the index case, co-workers, and the employer. The Sentinel Event Notification System for Occupational Risks (SENSOR) is a state-based surveillance program for occupational diseases that is coordinated by the National Institute for Occupational Safety and Health in the United States. The SENSOR program in the state of Michigan registered 446 cases of WRA from 416 different workplaces during 1993–1995 [71]. Inspections were conducted at 185 (44.5%) of these workplaces, and air sampling for known agents were conducted at 123 (29.6%). Many recommendations or citations were issued as a result of the inspections: 60 for medical monitoring, 80 for engineering controls, 63 for air monitoring, 126 for hazard communication program, and 71 for respiratory protection program [71]. These figures suggest that workplace inspections stimulated by WRA case reports frequently identified deficiencies that inhibited prevention.

Ongoing surveillance programs facilitate the identification of and response to an increase in the number or severity of asthma cases. Deaths due to work-related diseases are powerful warnings for workers with similar exposures. The Fatality Assessment and Control Evaluation (FACE) program identifies and investigates work-related deaths in several states in the United States. The Michigan FACE (MIFACE) program reported a worker who died after he sprayed an isocyanate-containing coating inside a van [72]. This surface coating is normally sprayed onto the open cargo beds of trucks, but in this case was applied onto the floor and up the walls of an enclosed van. The MIFACE investigation revealed deficiencies in several factors that contributed to this unfortunate occurrence: product stewardship by the manufacturer of the materials used by the case, engineering controls, company health and safety program, and health care provider recognition that the asthma was work-related. The Michigan Occupational Safety and Health Administration (MIOSHA) issued 11 citations to the company. Also, MIOSHA initiated contact with over 100 other companies in Michigan that applied spray-on truck bedliners and provided educational and technical assistance. The surveillance program's expeditious investigation and dissemination of the findings likely had a positive impact on asthma morbidity and mortality.

Surveillance data can also be used to document the impact of preventive interventions. In the province of Ontario in Canada, legislation was passed in 1983 that required workplace monitoring of diisocyanate levels to maintain exposures within acceptable limits, as well as medical monitoring of exposed workers. The impact of this legislation was tracked using worker compensation data. The number of diisocyanate OA cases began to increase during the early 1980s, probably due to the increased medical screening of exposed workers [34]. The number of claims peaked in 1988–1990 and began to decline after that. Also, the cases were less severe as the number declined [34]. This decline in the number and severity of diisocyanate WRA cases was likely the result of many activities, including better control of exposures at work, and increased and earlier recognition of disease.

## Future developments

Most intervention studies are best characterized as "complex intervention studies" consisting of several intervention components, where the actual change in exposure is seldom monitored. It is therefore not realistic to assume that most future studies will fulfill strict criteria for good quality single intervention studies. However, it is desirable to put more effort into ongoing monitoring of changes in exposure. Many companies and organizations do collect data continuously on exposure, exposure determinants, and health outcomes. The use and extension of this valuable data source could be improved by establishing a closer cooperation between companies and experts in exposure assessment and occupational lung diseases.

There is still a need for development of more effective medical surveillance programs, and predictions models are a promising attempt to achieve that goal. A major future challenge will be to develop effective prediction models for LMW asthma.

Collection of surveillance data is essential in planning, implementation, and evaluation of the prevention of WRA. In the future it will be crucial to maintain resources for ongoing surveillance programs, and it will be of equal importance to initiate surveillance programs in countries where no surveillance data currently exist.

Finally, an increased focus on education of workers and health professionals is crucial to increase the possibilities for prevention of WRA.

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