



---

# Asthma and the Workplace

Discussion paper prepared for

The Workplace Safety and Insurance Appeals Tribunal

November 1996

References updated January 2002

Prepared by:

Dr. Susan Tarlo

Respirologist

Dr. Susan M. Tarlo graduated from the Medical School of the London University, England, in 1969. She did post-graduate training in internal medicine at Westminster and Brompton hospitals in the UK and residencies in allergy and clinical immunology and respiratory medicine at Queen's University and McMaster University in Ontario. She joined the University of Toronto faculty in 1977 and currently holds the rank of Professor in the Department of Medicine. Her clinical and research interests are occupational respiratory disease and occupational allergy. She has published her research widely in these areas. Her main clinical appointment is Staff Physician in respiratory medicine at the Toronto Western Hospital, University Health Network, and she has a cross-appointment at St. Michael's Hospital. Dr. Tarlo serves as medical assessor at the Tribunal since 1986.

WSIAT literature search reviewed by Dr. A. Weinberg in 2010, who is of the opinion that this paper still provides a balanced overview of the medical knowledge in this area.

This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal's medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence : see *Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal)* [2009] O.J. No. 2080 (Ont Div Court).

*Asthma* is a condition which is characterized by increased responsiveness of the tracheobronchial tree (the airways leading to the lung). This is associated with inflammation (swelling and redness) of the lining of the airways and intermittent narrowing of the airways resulting from the swelling of the airway lining, increased production of secretions thick, sticky sputum) and contraction of muscle around the airways. The symptoms produced by this are episodic wheezing (noisy breathing on exhalation), coughing (usually with no sputum), a sensation of chest tightness and shortness of breath.

Asthma is common, occurring in about 5% of the general population. It can start at any age, from infants to elderly individuals. Usually the cause is unknown although there is often an association with allergy and many people with asthma also have allergic nose and eye symptoms, and/or a history of eczema. Allergic individuals who develop asthma often have their asthma triggered by common substances in the environment which can cause an allergic response (allergens) e.g. dust mites, animals, pollen and fungal spores. Such individuals also commonly have a family history of allergic disease with or without asthma. It is estimated that about 70% of children with asthma and about 50% of adults with asthma are allergic on the basis of their medical history and allergy skin tests to common airborne allergens.

About 50% of adults with asthma have no clear allergic component to their asthma. A proportion of these patients have a family history of asthma. About 11% of asthmatics are ASA-sensitive. This means that they develop a significant acute worsening of their asthma after taking medications containing ASA or other similar anti-inflammatory pain relievers. These patients often also have nasal polyps and sinusitis, and may or may not be allergic to common allergens.

Other common triggers of asthma exacerbations in allergic and non-allergic asthmatics include respiratory viral infections (colds or flu), cigarette smoke, dusts, fumes, sprays, cold air and exercise. The airway of the asthmatic is hyper-responsive (more reactive) than the normal airway to these factors.

In most patients with asthma the workplace has not caused their asthma. There are no reliable figures to determine the proportion of asthma in Ontario which results from the workplace. It has been estimated that about 2% of the working population develop asthma during their working life but unrelated to their work. Other reports state that 15% of a disability population attribute asthma to their work and about 8% of all asthma may be related to the workplace and about 2% of all asthma may be caused by the workplace

(although a Japanese estimate was up to 20% of all asthma, this was not based on actual patient data). In Ontario each year about 60 causes of asthma are compensated on the basis of occupational asthma which would represent about 1% of new asthmatics in the working population but would not include individuals who develop occupational asthma but are not eligible or do not apply for workers compensation.

Asthma can be caused by the workplace exposure to dusts or fumes, either through an immunological mechanism (an allergic response or unknown immune mechanisms) or by an irritant response from an accidental exposure to a high level of an agent causing irritation and inflammation in the airways. Over 300 agents (sensitizers) have been described which can cause asthma at work by a presumed immune mechanism e.g. wheat, animal proteins, latex, isocyanates, red cedar dust). They have the features of initially being tolerated by the worker for days/weeks or years before the person becomes sensitized and develops asthma symptoms. Once sensitized, even very low levels of exposure will worsen asthma. In contrast, the irritant induction of asthma occurs within 24 hours of the accidental exposure to the irritant agent (e.g. an acid spill or burnt paint fumes).

In Ontario, isocyanates are the commonest sensitizers and are used in spray paints, glues and polyurethane foam.

Since most adult-onset asthma is not caused by the workplace, the diagnosis of occupational asthma requires a history to suggest this diagnosis, and in addition, requires objective tests to confirm the diagnosis as indicated in the suggested reading. The addendum to this discussion paper gives a glossary to assist in understanding these objective tests.

In addition to occupational asthma i.e. asthma caused by the workplace, asthma can start coincidentally but be aggravated by work. In the same way that a dusty house, freshly painted room or smoky bar will aggravate asthma outside the workplace, dusts, fumes, sprays or cold air and exercise can cause asthma symptoms to be worse at work on a temporary basis (usually clearing within a day after leaving the environment but at times causing asthma to be less well controlled than usual for a few weeks).

Finally, asthma may vary co-incidentally to any work exposure. Viral infections, non-occupational exposure to allergens or irritant agents can worsen asthma symptoms and in a working individual, it might be difficult for the person to distinguish this from an occupational cause. In addition, asthma quite often spontaneously varies in severity without any identified environmental cause.

Objective tests to try to determine if there is an occupational cause for asthma or if asthma is co-incidental, but aggravated by the workplace can usually enable an accurate diagnosis to be made. In the absence of such tests, it may be helpful to consider the reported prevalence of occupational asthma with the individual workplace agent as compared with the prevalence of unrelated asthma; e.g. isocyanates, red cedar-and latex have been reported to cause asthma in up to 5% of exposed workers while formaldehyde and oil mists have only been reported to cause asthma in a total of few cases.

### Suggested Reading (available from WCAT Library)

American College of Chest Physicians (ACCP) Consensus statement on assessment of asthma in the workplace. Chest 1995; 108: 1084-1117.

Tarlo SM, Liss G., Corey P. A workers compensation claim population for occupational asthma. Comparison of subgroups. Chest 1995; 107:634-41.

Tarlo SM, Boulet L-P, Cartier A., et al. Canadian Thoracic Society Guidelines for Occupational Asthma. Canadian Respiratory Journal 1998 Volume 5, pgs 289-300

[http://www.pulsus.com/Respir/05\\_04/tarl\\_ed.htm](http://www.pulsus.com/Respir/05_04/tarl_ed.htm)

### Glossary of some terms used in medical reports or papers on occupational asthma.

**Occupational Asthma:** a disease characterised by variable airflow limitation and/or bronchial hyperresponsiveness due to causes and conditions attributable to a particular working environment and not to stimuli encountered outside the workplace.

**Work-Aggravated Asthma:** concurrent asthma worsened by non-toxic irritants or physical stimuli in the workplace.

**Dyspnea:** Shortness of breath/difficulty breathing.

**Wheeze:** noisy breathing, usually on exhalation, often a feature of people with asthma.

**Variable airflow limitation:** reduced flow of air through the tracheo-bronchial airways which varies in severity and can be measured by pulmonary function testing. It is a characteristic of asthma - both occupational and non-occupational.

**Bronchial hyperresponsiveness:** increased response of the airway to stimuli such as cold air, smoke, fumes, sprays, exercise, allergens, leading to transient airway narrowing and airflow limitation. This can be tested in a laboratory setting by inhaling increasing concentrations of histamines or methacholine and measuring airflow limitation.

**Spirometry (or flow/volume curves):** Pulmonary function measures to detect airflow limitation.

**FEV1:** a pulmonary function, it is the amount of gas breathed out of the lung in one second with maximum forced exhalation.

**VC:** vital capacity, a pulmonary function measure. The total volume of gas which can be exhaled after a full breath in. Methacholine (or histamine) PC 20: The concentration of methacholine (or histamine) which causes a 20% reduction FEV1 in a laboratory test, (a methacholine or histamine challenge test). This is used as a test for asthma or can be used to assess changes in asthma severity related to work exposure. A non-asthmatic person generally has a PC20 greater than 8mg/ml while a symptomatic asthmatic person generally has a PC20 less than 8mg/ml.

**Peak flow meter:** a small, hand-held breathing-test device which patients can use to directly monitor the peak flow of air which they can exhale. If the airways are narrowed, airflow is reduced and the peak flow is reduced as compared to the normal value for that person. Peak flow readings can also be reduced if the patient does not fully inhale or if a full effort is not given during exhalation. The peak flow readings on exhalation is displayed on the peak flow meter and generally then is transcribed by the patient onto a peak flow diary record. Electronic peak flow meters which store the results can be used but are very expensive at present.

**Specific laboratory chamber challenge:** Testing to measure changes in pulmonary function related to an exposure in a laboratory of the patient to the substance from work which is suspected to have caused occupational asthma. The results are usually compared with the results of exposure of the patient to an innocuous substance which will not affect the airways but looks the same as the suspected substance (i.e. a placebo).

**Single-blind Challenge:** the physician but not the patient is aware of the exposure substance on the day(s) of testing i.e. placebo versus work agent.

**Double-blinded Challenge:** neither the physician nor the patient are aware as to which is the placebo and which is the work agent during the challenge test.

**Prick Skin Tests:** a method of allergy skin testing.

**Wheal and Flare:** the skin response seen with a positive allergy skin test. To be significant, generally the wheal diameter needs to be at least 3mm greater than that produced by the negative control (diluent).

**Allergen:** a substance which can cause an allergic response in patients with specific antibodies to that substance.

**IgE Antibodies:** a type of antibody which can cause an allergic response.

**Atopic:** allergic.