

# Occupational Medicine Clinical Update

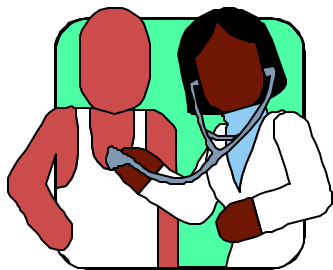
*Dedicated to the prevention of occupational illness and injuries, and promoting the well-being of all workers*

## This Issue:

- Case study – A significant contributing factor?
- The evolution of causality

## Next Month:

- Nuts and bolts of determining causality – the Bradford Hill method



## Occupational Health Clinics for Ontario Workers Inc, Samia-Lambton

### “Doc, do you think work caused my disease?”

Now that can be a pretty tough question to answer (unless you are seeing a roofer with a broken femur). The articles in this month’s *Update* will outline just how complicated answering such questions can be.

Last month we reviewed some cases of lymphoma where there is considerable evidence that occupation plays a role in the development of such malignancies: benzene and other solvents, silica, metals and/or metal cutting fluids, smoke, and pesticides.

But how does one go about *proving* that a particular exposure **caused** a disease or malignancy in a specific worker, or in workers exposed to that substance in general?

This issue outlines the evolution of the concept of **causation**, and the challenges faced by epidemiologists on this contentious issue. The case below illustrates some of the broad strokes in the decision-making process in trying to determine causality in compensation cases.

Does proving it matter? Certainly to the worker and any dependents there may be financial reasons where establishing a connection to work is important. What about society? If occupational diseases

are being recognized as such there are a number of benefits that accrue to society as a whole:

- ◆ Public health (OHIP) should be reimbursed by WSIB, lowering costs to OHIP and making more money available for health care.
- ◆ Increased WSIB premiums or decreased rebates to employers that inevitably lead to increased awareness and safer practices in the workplace. This benefits everyone ultimately.
- ◆ Workers and their families see something positive coming out of what is often a catastrophic event in their lives.

Many physicians fear getting involved with WSIB cases because of a combination of medical uncertainty, time, and workload concerns. However, it is worth noting that when completing a Physician’s First Report (Form 8) there is no legal obligation to produce any other reports or make appearances at any appeal/tribunal hearings in the future related to the case (although patient records may be requested).

Furthermore, if other medical support/consultation is needed the physicians at OHCOW are more than happy to help - not that we are looking for work, there is unfortunately far too much to go around.

## Benzene and MDS - Was the Workplace a “Significant Contributing Factor”?

The patient was a gasoline service station attendant from 1975 until 1995. In 1994, he began experiencing recurrent pneumonias and was found to have myelodysplastic syndrome (MDS). The worker filed a claim with the WSIB which was denied as is the case with non-Schedule 3 (non-established) cases. It was subsequently appealed. He died six weeks prior to the hearing from complications of MDS.

At the hearing, called the Workplace Safety and Insurance Appeals Tribunal (WSIAT), the key issues were: did the worker have the exposure, and does the exposure cause the disease?

### Significant Exposure to Benzene?

This was contested by the experts for the WSIB. Gasoline is acknowledged to be contaminated with benzene (1-2%) and service station attendants have been shown to have excess rates of hematological malignancies. However the WSIB attempted to show that this worker did not have excess exposure to benzene compared to study subjects. The

expert for the claimant, Dr. Abe Reinhartz from our clinic, showed evidence to dispute this and in the end, the tribunal judge sided with the claimant on this issue.

### Does Benzene Cause MDS?

This was the crux of the matter. Can an association be proven? If you read the accompanying article on page two, the short answer is ‘no’. In WSIAT hearings, the tribunal judge makes a ‘best estimate’ of proof of a causal relationship based on scientific and legal principles, precedent, and the scientific information presented at the time.

Again, Dr. Reinhartz presented a number of studies that supported an association between benzene and MDS. This included an extensive review on benzene and hematologic malignancies, in particular, leukemias and MDS, that had been produced by the Occupational Disease Panel of Ontario (ODP).

The ODP report concluded that a strong and probable connection existed between benzene and a wide variety of hematologic disturbances

*(Continued on page 2)*

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(Continued from page 1)

including leukemias and MDS. The methodology used to make this determination is often referred to as the 'Bradford Hill' method of evaluating causation and will be reviewed in the next edition of the *Update*.

The judge took into account that there were no other known factors in the worker's medical history that were associated with MDS (e.g. radiation, chemotherapy, smoking). Also important was the onset of disease, which developed 12-13 years after initial exposure to benzene, which was in keeping with the accepted latency period associated with MDS and benzene exposure.

In deciding causal relationships in these cases, the standard of proof is different from that applied in evaluating whether a new treatment should be used. The panels and courts involved in these decisions have recognized the theoretical inability to prove causal relationships conclusively (see other article on this page) and the epidemiological limitations that exist. The approach generally taken is to decide "whether it is more likely than not" that the disease was caused or aggravated by exposure to a chemical.

The judge summarized many previous court decisions and principles in arriving at his conclusion stating, "I acknowledge that, where epidemiological evidence exists, it is an important piece of evidence. However entitlement, in my opinion, cannot depend exclusively on such evidence being available. Its absence cannot be treated as determining that no causal relationship exists, particularly given how long it takes to develop epidemiological evidence."

He concluded, "In the present case, in my opinion, the evidence in its totality weighs in favour of a finding that exposure to benzene in the workplace was a likely **significant contributing factor** in the development of the illness resulting in the worker's death."



## A BRIEF HISTORY OF CAUSATION

Philosophical Arguments vs. Practical Needs

The issue of causation has been pondered and hotly debated since Aristotle (384-322 BC) first waded into the fray. He relied on *rationalism*, or simply put - reason. His approach to causation, and his views, impacted Western thought for centuries.

In the seventeenth century rationalism gave way to *empiricism*, combining observation with *induction*. According to the principle of induction, causation was an inferred relation between two events observed repeatedly in the same time sequence. This change from rationalism to empiricism marked the rise of modern science.

However, for many, the battle of causality had not been won with empirical principles. Philosophers like David Hume (1711-1776), led an ongoing debate (with other heavyweight thinkers such as Immanuel Kant [1724-1804]) arguing that the process of inductive 'logic' was faulty and prone to leading to incorrect inferences, and thus not logical at all. Hume argued that induction was in fact a *psychological process* whereby the only thing being modified by observations were the observer's convictions about the inferred causal relationships.



The philosophers continued to war two hundred years later, with Reichenbach (1951) and Carnap (1962) still defending induction. They acknowledged that certainty was not possible with empirical thought, but quantifying the degree of confirmation of a hypothesis was. They attempted to legitimize induction by applying the laws of probability.

While these philosophers found induction necessary, Popper (1968) found it quite dispensable. He argued that logically, scientific statements could only be conclusively decided in the negative. He rejected induction outright and showed, using deductive reasoning, that nothing could ever be irrefutably proven, only disproved.

This notion shook the supporters of inductivism but fit with the history of science: *accepted theories are replaced, after falsification, with new theories*. Theories are "accepted" or "established" but never conclusively proven. With this approach science becomes a Darwinian process, where only the fittest theory survives.

While philosophers debated these issues over the millennia, people still wanted answers. Scientists, governments, industries, the courts and the media demanded, out of necessity, that epidemiologists come up with some way of 'proving' whether causality exists for certain associations. As is illustrated in the case of benzene and MDS, different levels of proof are required depending on the question at hand.

The recognition that causation is "the elusive grail of epidemiology" has spawned various models of causation: *probabilistic, necessary and sufficient, sufficient-component, production and counterfactual*. All of these models acknowledge that we can never completely prove anything, only attempt to produce a model that provides the best supporting evidence for causation.

There is also a more pragmatic view of causation: *that a factor is a cause if its elimination improves health* (in some ways a corollary to The Precautionary Principle). This is the approach taken in 1849 by Dr. John Snow when he dismantled a water pump and stopped a cholera epidemic in England.

It should remind us to not let those with conflicts of interest argue about absolute 'proof', and forget to use the evidence we do have to make a change for the better.

### References

- Karhausen LR. 2000. Causation: the elusive grail of epidemiology. *Med Health Care Philos.* 3(1):59-67.
- Parascandola M, Weed DL. 2001. Causation in epidemiology. *J Epidemiol Community Health.* Dec;55(12):905-12.
- Scheutz F, Poulsen S. Determining causation in epidemiology. 1999. *Community Dent Oral Epidemiol.* Jun;27(3):161-70.
- Schottenfeld, D.; Fraumeni, J.F. Jr. 1996. *Cancer epidemiology and prevention, Second Edition.* New York, Oxford: Oxford University Press, 1521p