**WCAT Decision Number:** WCAT-2013-01741
**WCAT Decision Date:** June 19, 2013
**Panel:** Andrew Pendray, Vice Chair

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## Introduction

[1] In early June 2011, the appellant worker was diagnosed with cancer in his larynx[1]. The worker attributed his cancer to his employment in an asbestos mine for approximately seven years in the 1980s, and as a result completed an application for compensation with the Workers' Compensation Board (Board).

[2] The Board determined, in an August 26, 2011 decision letter, that the worker's laryngeal cancer was not due to the nature of his employment (with particular reference to his exposure to asbestos). As a result, the worker's claim for compensation was denied. The Board's decision in that regard was confirmed in *Review Reference #R0133849*.

[3] Pursuant to section 239 of the *Workers Compensation Act* (Act), the worker now appeals *Review Reference #R0133849* to the Workers' Compensation Appeal Tribunal (WCAT). His position on appeal is, quite simply, that the medical evidence supports a conclusion that his laryngeal cancer is due to the nature of his employment and that his claim for compensation ought therefore to have been accepted.

[4] The appeal proceeded by way of an oral hearing, where the worker gave evidence and was represented by an adviser from the Workers' Advisers Office. The employer is no longer active, and, although the Employers' Advisers Office was named the deemed employer, it elected not to participate in this appeal.

## Issue(s)

[5] Is the worker's laryngeal cancer an occupational disease which is due to the nature of his employment?

## Jurisdiction

[6] WCAT has exclusive jurisdiction to inquire into, hear and determine all those matters and questions of fact, law, and discretion arising or required to be determined in an appeal before it (section 254 of the Act). It is not bound by legal precedent (subsection 250(1) of the Act). WCAT must make its decision on the merits and justice of the case, but, in so doing, it must apply a policy of the board of directors of the Board that is applicable in the case (subsection 250(2) of the Act), save for specific circumstances set out in section 251 of the Act. Subsection 250(4) provides that WCAT

[1] Squamous cell carcinoma.

must resolve the issue in a manner that favours the worker where evidence supporting different findings is evenly weighted.

[7] This is an appeal by way of rehearing. WCAT has jurisdiction to consider new evidence, and to substitute its own decision for the decision under appeal.

**Background and Evidence**

[8] In early 2010, the worker began to experience hoarseness in his voice. This was the initiating concern that led to the worker receiving treatment for laryngeal cancer.

[9] Dr. Higgins, the worker's family physician, saw the worker on April 6, 2010 in relation to the worker's hoarseness. It is apparent that he determined that the worker required a referral to specialist at that time. In an April 13, 2010 referral letter to Dr. Jewett, an otolaryngologist, Dr. Higgins set out the following regarding the worker's medical history:

> He has a history of a 30 pack year smoker, stopping smoking 12 years ago. He also drank heavily for a number of years and has not drunk alcohol for about 3 years now. He drank beer regularly but no hard spirits.

[10] Dr. Jewett performed two biopsies on the worker's throat in 2010: one in April and one in October. Pathology examinations of both of those biopsies failed to identify any cancer. In May 2011, however, the worker attended Dr. Jewett with a lump in the left side of his neck, a worsening of his voice, and pain radiating to his right ear. Dr. Jewett concluded at that time that the worker needed to be re-evaluated, and referred him to Dr. Gillis, another otolaryngologist, on an urgent basis. Dr. Jewett indicated that he was uncertain as to what was "going on" with the worker's larynx, but that the node was in his view, "somewhat ominous."

[11] The worker was seen by Dr. Gillis on May 19, 2011. In a consultation report to Dr. Jewett, Dr. Gillis indicated that he had concluded at that time that the worker likely had metastatic disease, and that the worker required further assessments including an endoscopic resection. Dr. Gillis further noted that the worker may have stage IV cancer, and that he had mentioned to the worker that he may require a laryngectomy if that were the case. Dr. Gillis indicated that the worker would be re-evaluated after a histological confirmation of cancer was obtained.

[12] On May 26, 2011, the worker contacted the Board to make an application for compensation. The Teleclaim operator who took the worker's application noted that the worker described having been an employee at an asbestos mine in northern British Columbia for a number of years in the early 1980s.

[13] A Board case manager contacted the worker via telephone on the date of his application. In a telephone memorandum detailing that conversation, the case manager indicated that the worker had reported that he had begun to experience hoarseness in his throat in early 2010, and as a result had been sent to a specialist. Two biopsies taken in April and October 2010 had been negative; however, a third biopsy taken sometime in either April or May 2011 had been found to be cancerous. Dr. Jewett, an otolaryngologist he had been seeing in his home community then referred the worker to see Dr. Gillis.

[14] The worker reported that Dr. Gillis had informed him on May 18, 2011 that he had stage III throat cancer and that he required surgery to remove his larynx as soon as possible. Chemotherapy would follow. The worker reiterated that he had worked with asbestos at the mine for approximately seven years from 1980 to 1987, and that Dr. Gillis had informed him that he believed that the worker's cancer may be related to his asbestos exposure. Of note, the worker indicated that he had not been diagnosed with asbestosis or mesothelioma, and that his chest x-rays were clear.

[15] Regarding the worker's employment at the asbestos mine, the case manager indicated that the worker had reported the following:

- He had worked in the mine garage, washing mud, grime, and asbestos off heavy equipment. When doing that work, he always wore a respirator, except while eating lunch. The worker would also use an extra filter with his respirator, even though he had been informed that working in the shop was low risk. He did this due to the fact that there was dust floating around everywhere throughout the mining town.
- After approximately five months working in the garage, he obtained an apprentice millwright position. That position involved working down in the mine. He stated that he was exposed to white, crystalline asbestos while working that position, which he continued in (eventually as a ticketed millwright) for six and a half years.

[16] The worker provided further detail regarding his employment in his October 4, 2011 submission to the Review Division. He described being exposed to asbestos fibres and dust "on a continual basis" from 1980 to 1987. He stated that while he did wear a respirator, it was not possible to wear it 100% of the time and that there were "several occasions" that he worked in the mills while not wearing the respirator. In a teleconference with the review officer, the worker explained that he had not worn a respirator until later in his employment, though he always tried to wear it when opening up machinery.

[17] At the oral hearing of this appeal, the worker further described his employment at the asbestos mine. He indicated that during that initial five-month period where he had worked in the garage at the mine, he had not worn a respirator at all. Once he had commenced his millwright apprenticeship, he did wear a respirator when getting inside machinery, but that he had not worn one when walking around the mine or working in the shops. The worker stated that he would spend a good portion of his day with a

respirator on, but that there would four to five hours on the worksite each day where he would not have been wearing one.

[18] With respect to his employment subsequent to 1987, the worker indicated that:

- From 1988 to 1989, the worker was employed in a copper, gold, and silver mine as a millwright, with no known exposure to asbestos;
- He was employed as a millwright at a soft wood lumber mill (Mill A) from 1989 to 1996, with no known exposure to asbestos (or hardwoods);
- He worked at another sawmill as a millwright from 1996-2000 (Mill B);
- In 2000, he obtained work as a sawmill supervisor, for three and a half years;
- The worker subsequently was employed at another sawmill, a food processing factory, and a mining shovel construction company until 2008. He worked as a millwright at all of those positions;
- He had been working as a long-haul truck driver since 2008.

[19] The case manager also documented the following general information regarding the worker's health:

- The worker had been a smoker for approximately 25 years, smoking a pack a day;
- He had quit smoking entirely 15 years prior to 2011;
- He had no history of gastroesophageal reflux disease, though he had occasionally taken over-the-counter medication for heartburn. The worker indicated that he would experience heartburn three times per year at most;
- He had quit drinking alcohol in 1991 or 1992. Prior to that, he had a drinking problem with respect to beer. He did not drink hard liquor;
- He had not been diagnosed with any other forms of cancer or other serious medical conditions.

[20] In his submissions to the Review Division, the worker further provided that his drinking habit had slowly escalated while working at the asbestos mine, thought he indicated that he had never been a "fall down drunk." The worker testified at the oral hearing of this appeal that at his worst, he was drinking between six and ten beers per day, and that that had lasted for approximately five or six years. The worker indicated that in 1991, he had sought professional help for his drinking and that he had remained alcohol-free until 1999, when he again began to drink socially. He noted that he ceased drinking altogether in 2006.

[21] In his conversation with the review officer, the worker indicated that he had started smoking when he was age 16 or 17 and that he had in fact smoked less than a pack of day. At the oral hearing of this appeal, the worker stated that he believed he had started smoking when he was 15 or 16 years old and that by the time he was 17, he was smoking between 5 and 10 cigarettes per day. He noted that he had quit smoking completely in 1997, when he was approximately 41 years old. The worker estimated

that he had smoked on average a half-pack to a pack a day from the time he was age 17 until he was 41. He noted that once in a while he would smoke a pack and a quarter of cigarettes per day.

[22] On May 31, 2011, Dr. Gillis undertook a resection of the worker's left hemilarynx. He indicated that his post-operative diagnosis was one of laryngeal cancer. Dr. Gillis confirmed that diagnosis in a June 16, 2011 letter to Dr. Jewett. Dr. Gillis also at that time recommended that the worker undergo a laryngectomy and neck dissection.

[23] In August 2011, Dr. Gillett, a specialist in internal medicine[2], reviewed the worker's claim file and associated medical records at the Board's request and provided an opinion as to whether or not the worker's laryngeal cancer was related to his employment. In providing his opinion, Dr. Gillett noted the worker's employment history at the asbestos mine, as well as the 30-pack year smoking history and drinking history noted by Drs. Higgins and Jewett. Dr. Gillett provided the following opinion:

> There have been a number of epidemiologic studies over the past 30 years looking for a relationship between asbestos exposure and laryngeal cancer. In 2000, a meta analysis was published in the Annals of Occupational Hygiene that looked at 24 previously published studies evaluating the risk for laryngeal cancer in asbestos exposed workers. The analysis showed that asbestos exposure did not increase the relative risk for laryngeal cancer. The analysis confirmed the most important factors increasing the risk for laryngeal cancer included smoking and alcohol consumption. The Annals study was referenced by the American College of Chest Physicians' in their consensus statement regarding the health effects of Asbestos exposure.
>
> **In summary, there is no evidence to suggest that asbestos exposure increases the risk of laryngeal cancer**. Further, there is wealth of data that suggests that cigarette smoking and heavy alcohol intake do convey marked increased risk for laryngeal cancer. In this case, it would appear that the worker's malignancy was a direct result of his smoking and alcohol exposures.
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> [emphasis added][3]

[24] Relying on Dr. Gillett's opinion, the Board issued the August 26, 2011 decision letter denying the worker's claim. In short, the Board concluded that the worker's laryngeal cancer was not occupationally caused.

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[2] Dr. Gillett had a further subspecialty in pulmonology.

[3] Relative risk (RR) is the ratio of the disease incidence or death among people exposed to an agent to the disease incidence or death among people who are not exposed. An RR of 2.0 if often equated with a 50% likelihood that an exposed person's disease was caused by the agent. An RR of greater than 2.0 is often seen as permitting an inference that an individual's disease was more likely than not caused by the implicated agent.

[25] The worker contacted the Board on September 8, 2011 in order to advise that he did not agree with the Board's decision. The worker at that time indicated that he had been drinking from 1981 to 1991, and subsequently had drunk only socially from 1999 to 2006.

[26] In response to the worker's call of September 8, 2011, the Board issued a further letter date September 12, 2011. That letter indicated that it was intended to "clarify" the August 26, 2011 decision denying the worker's claim. The Board noted that while it accepted that the worker was clearly exposed to asbestos while working at the asbestos mine, there was no presumption of work causation applicable to cancers of the larynx and exposure to asbestos. The Board explained that:

> Schedule B, item 4 (c) states:
>
> (c) Carcinoma of the larynx of pharynx associated with asbestosis … (*is presumed to be work caused*) where there is exposure to airborne asbestos dust.
>
> In my decision letter of August 26, 2011, I stated as follows.
>
> > (1) Section 6 (3), Schedule B, item 4 (c), provides that carcinoma of the larynx associated with asbestosis may be accepted where there is exposure to airborne asbestos dust. This section applies if there is evidence that a worker has asbestosis. There is no evidence that you have Asbestosis. Dr. Jewett's report of May 9, 2011 reports that you had a normal CT scan of the chest in April, 2010. The lungs were clear. The legal presumption of Section 6(3) Schedule B item 4(c) does not apply in your case, as there is no diagnosis of Asbestosis.
>
> Section 6 (3), Schedule B, item 4 (c), provides that carcinoma of the larynx associated with asbestosis may be accepted where there is exposure to airborne asbestos dust. This section applies if there was evidence that a worker had asbestosis. Although you were exposed to asbestos dust, you do not have evidence of asbestosis. This section does not apply in your case.

*Review Division*

[27] At the Review Division, the worker argued that Dr. Gillett had relied on a single, outdated study in reaching his opinion. He submitted that there were other studies (both older and more recent) which showed a direct correlation between exposure to asbestos and laryngeal cancer.

[28] Included with his request for review of the August 26, 2011 decision letter, the worker provided a number of letters from the physicians who were providing him with treatment.

[29] In a September 23, 2011 letter, Dr. Skarsgard, the worker's treating oncologist, indicated that:

> Although [the worker] was exposed to tobacco smoke, which is a well established risk factor for this type of cancer, he also had a long history of occupational exposure to asbestos. There is evidence to support a contributing role of asbestos in the development of a variety of cancers including of the larynx.

[30] In a September 22, 2011 letter, Dr. Gillis indicated that he understood that the worker had been exposed to asbestos during his working career and that:

> There is an increased incidence of laryngeal cancer in people who are exposed to asbestos. This is well supported in numerous studies and analysis throughout the world.
>
> I enclose eight brief abstracts as supporting evidence.

[31] In a November 16, 2011 letter Dr. Gillis described the worker's laryngeal cancer as being highly related to his exposure to asbestos. In a November 24, 2011 letter, Dr. Gillis indicated the following:

> [The worker] advised me that he worked for a number of years in an area of high asbestos particle exposure….
>
> **I believe that [the worker's] exposure to asbestos is a contributing factor to him developing laryngeal cancer.** I am fully aware that all people exposed to asbestos do not develop laryngeal cancer.
>
> [bold emphasis added]

[32] The worker provided a further letter from Dr. Gillis dated December 2, 2011. In that letter, Dr. Gillis clarified that it was his opinion that the majority of medical opinion (both in Canada and the United States) was that there was a high association between asbestos exposure and the development of laryngeal cancer. While Dr. Gillis noted that some studies had indicated that laryngeal cancer was not "highly associated" with exposure to asbestos, he went on to explain that the larger meta studies indicated that there was in fact such an association. Specifically, Dr. Gillis referred to a study carried out by Goodman, Ray, Malloy, and Zhao[4] which noted a suggestion of an association between asbestos and laryngeal carcinoma.

[4] *Cancer Causes Control*, 1999 Oct; 10(5): 453-65. "Cancer in asbestos-exposed occupational cohorts: a meta-analysis"

[33] The worker specifically asked Dr. Gillis to provide his opinion on whether the worker's asbestos exposure was a significant contributing factor to the development of his laryngeal cancer, having regard to his history of drinking and smoking. Dr. Gillis provided the following:

> All of these are contributing factors to the development of cancers in the head and neck area and in particular to laryngeal cancer. All patients exposed to asbestos, as all patients exposed to drinking, as all patients exposed to smoking do not necessarily develop laryngeal cancer. However, of the patients who do develop laryngeal cancer there is a higher association amongst these patients of having exposure to these contributing factors. It is my professional opinion that there is a stronger association with development of laryngeal cancer amongst patients who have had exposure to asbestos than those who have just had exposure to smoking and drinking. However the risk of developing laryngeal cancer is higher amongst people who have been exposed to asbestos than those who have had a history of smoking and drinking.

[34] The worker also provided a further undated letter from Dr. Skarsgard. In that letter, Dr. Skarsgard indicated that a significant obstacle for determining causation for cancer of the larynx was that there were so many well-established risk factors. Specifically, Dr. Skarsgard noted that the activities of smoking, and to a lesser degree alcohol consumption, were well known to increase the risk of developing laryngeal cancer. He explained that:

> The fact that many people who were exposed to asbestos were also smokers and/or drinkers makes it difficult to clearly see an association with asbestos exposure.

[35] Dr. Skarsgard further indicated that he did not dispute the findings of the study relied on by Dr. Gillett, noting that the findings of that study were based on the data available at that time. However, he indicated that a more recent study[5] was, in his view, of greater assistance in considering the causative association between asbestos exposure and laryngeal cancer:

> I refer you to a more recent and comprehensive analysis of this topic in a book from the Institute of Medicine of the National Academies….Of particular interest is chapter 8, p. 173 entitled "Laryngeal cancer and asbestos". Considering data from a large number of cohort and case control studies from the 1970s to 2000s, **the authors found a relative risk of laryngeal cancer of 1.40 (95% confidence interval 1.19 – 1.64)**

[5] Chapter 8, "Laryngeal Cancer and Asbestos": *Asbestos: Selected Cancers*; Committee on Asbestos: Selected Health Effects, Board on Population Health and Public Health Practices, Institute of Medicine of the National Academies, The National Academies Press; Washington. 2006, page 173-193. This will be referred to for the remainder of this decision as "the NAP study."

> **with any asbestos exposure compared to none, meaning a statistically significant 40% overall increase in risk compared to those who were not exposed. Persons with what was classified as a high level of exposure had a relative risk of up to 2.57 (i.e. more than 2.5 x that of the non-exposed population). Based on his history, I think this is probably the category in which the [the worker] belongs**. The conclusion of the chapter reads as follows: "Considering all lines of evidence…the committee concluded that the evidence is *sufficient* to infer a causal relationship between asbestos exposure and laryngeal cancer."
>
> **I recognize that [the worker's] lifestyle put him at increased risk for the development of laryngeal cancer, and that this could have happened in the absence of any occupational exposure. I believe the evidence is more than sufficient, though, to conclude that his risk of developing laryngeal cancer was considerably higher as a result of his occupational asbestos exposure**.
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> [bold emphasis added]

[36] At this juncture, it is appropriate to provide some further detail regarding the NAP study. That study, conducted by the Institute of Medicine[6], was undertaken to determine if there is a causal association between asbestos and, amongst others types of cancer, laryngeal cancer. The study authors noted that the most important risk factors for laryngeal cancer were tobacco-smoking and heavy consumption of alcohol, with the independent effect of tobacco being greater than that of alcohol consumption. Risk was further noted to increase with the number of cigarettes smoked per day, and the duration of smoking. The NAP study specifically noted that the effects of occupation on the risk of laryngeal cancer have often been difficult to study due to the relationship between laryngeal cancer with tobacco use and alcohol consumption. Of note, the NAP study referenced an earlier study which had found relative risk of smoking alone to incidence of laryngeal cancer was 7.1.

[37] In conducting its meta-analysis, the NAP study utilized 29 published cohort study papers that had examined the RR of a diagnosis of, or death due to, laryngeal cancer among people with any occupational exposure to asbestos compared with people in the general population without such exposure. From those studies, the combined RR associated with "any occupational exposure to asbestos" was identified by the NAP study to be 1.40. The aggregate RR estimate in the most highly exposed subjects[7] was 2.57 for the strongest association reported and 2.02 for the weakest association reported.

[6] Part of the United States of America's National Academy of Sciences.
[7] The definition of what constituted "highly exposed" varied from study to study.

[38] The NAP study concluded that the larger cohort studies consistently showed an increased risk of laryngeal cancer in asbestos-exposed workers employed in a wide array of industries, and that there was some evidence of a dose-response[8] relationship in the meta-analyses.

[39] The NAP study also reviewed 18 published case-control studies. The meta-analysis of 15 of those studies found an RR of 1.43 associated with "any" exposure to asbestos. A further meta-analysis of 10 of those case-control studies that provided "higher-quality exposure information" provided an RR of 1.21. The NAP study noted that most of the case-control studies made some attempt to control for tobacco use and alcohol consumption. Even after adjustment for those two risk factors, an association with asbestos exposure was found to appear to persist. Of interest, one study which controlled for covariates such as alcohol and tobacco noted that the association between asbestos exposure and laryngeal cancer was weakened when such controls were taken into account[9], while another study[10] which controlled for tobacco use and alcohol consumption noted that such controls made little difference.

[40] On the issue of the biologic plausibility that asbestos exposure may cause cancer of the larynx, the NAP study explained the following:

> The larynx, like the lung, is anatomically in the direct path of inhaled asbestos fibers. Inflammation or damage of the vocal folds could disrupt laminar airflow and predispose to the deposition and accumulation of asbestos fibers in the larynx. Squamous-cell carcinomas of the lung and larynx have histologic and clinical similarities. Cancers at both sites arise from the respiratory epithelium in regions of squamous metaplasia and dysplasia. Tobacco-smoking is the most important risk factor for both sites. Asbestos exposure is an established cause of lung cancer. On the basis of theoretical considerations, tobacco-smoking, alone or in combination with alcohol consumption, may predispose to the accumulation of asbestos fibers in the epithelial lining of the larynx. Aerodynamic turbulence at bifurcations of the large conducting airways is known to contribute to the deposition of long asbestos fibers in the lung (Asgharian and Yu 1988). Bronchogenic carcinomas commonly arise in those areas (Schlesinger and Lippmann 1978). The accumulation of asbestos fibers, together with smoking and/or drinking, could produce chronic irritation or inflammation and thus accelerates the progression of neoplasia.

[8] Dose-response refers to the phenomenon of higher exposed groups having higher rates of disease.
[9] Dietz A, Ramroth H, Urban T, Ahrens W, Becher H. 2004. Exposure to cement dust, related occupational groups and laryngeal cancer risk: Results of a population based case-control study. ***International Journal of Cancer*** 108(6): 907-911.
[10] Brown LM, Mason TJ, Pickle LW, Stewart PA, Buffler PA, Burau K, Ziegler RG, Fraumeni JF Jr. 1988. Occupational risk factors for laryngeal cancer on the Texas Gulf Coast. ***Cancer Research*** 48(7): 1960-1964.

[41] The NAP study concluded that there was biologic plausibility and sufficient evidence to infer that that a causal relationship exists between asbestos exposure and laryngeal cancer. The NAP study further indicated that even with adjustment for alcohol and tobacco consumption, an association between asbestos exposure and laryngeal cancer appeared to persist.

[42] Given this new information provided by the worker, the review officer conducting *Review Reference #R0133849* requested that the Review Division medical advisor, Dr. Prat[11], provide an opinion in response to the following questions:

> 1. Is there evidence in the medical literature of an association between occupational asbestos exposure and laryngeal cancer?
> 2. If so, does this association support a relative risk (RR) of 2.0 or greater, such that the worker had a greater risk (50% or more) than the general population for the development of laryngeal cancer?
> 3. What effect, if any, did the worker's tobacco and alcohol use have on the development of laryngeal cancer?
> 4. Is there evidence in the medical literature of an additive effect and enhanced risk for laryngeal cancer, due to asbestos exposure and smoking in combination?
>
> Having considered the above questions, please indicate whether the worker's occupational asbestos exposure from 1980 to 1987 was causally significant for the development of his laryngeal cancer.

[43] In a February 10, 2012 opinion memorandum, Dr. Prat provided a lengthy discussion of the nature of laryngeal cancer, cancer generally, odds ratios (ORs), and relative risk. At the outset, Dr. Prat noted that the latency period for the worker's type of cancer was typically 20 to 35 years, and that, as a result, the worker's exposure to asbestos (and smoking for that matter) was consistent with possible causation of his laryngeal cancer.

[44] In reviewing the medical literature, Dr. Prat concluded that smoking was by far the most highly correlated risk factor with the development of laryngeal cancer, with an OR of 11.03 in never drinkers for those who smoke more than 40 cigarettes per day and 8.36 for those that smoke 10 to 20 cigarettes per day. With respect to drinking, he noted an OR of 2.98 for heavy drinkers (more than five per day).

[45] With respect to a causal relationship between laryngeal cancer and asbestos exposure, Dr. Prat reviewed a number of meta-analyses, including the NAP study referred to by

[11] Dr. Prat is a family physician who holds a Master of Arts degree in occupational health and epidemiology.

Dr. Skarsgard. He also reviewed a number of other studies noting the following findings amongst others:

- In a study on construction workers in Sweden which adjusted for smoking, but not drinking, the RR for cancer of the larynx for those who were "ever" exposed to asbestos was 1.9, 2.4 for "moderate" exposure, and .8 for "high exposure"[12]. The study authors concluded that their findings offered further evidence that asbestos exposure increases the risk of laryngeal cancer.
- In a case control study looking at 315 cases of laryngeal cancer from 2000 which used regression analysis to account for smoking and drinking, the odds ratio was 1.24[13].
- In a 2011 case control study which examined 73 cases of laryngeal cancer, the OR after adjustment for smoking and alcohol was approximately 1.2. The study authors concluded that their results were not significant enough to conclude a causal relationship[14]. Nonetheless, the study authors concluded that moderately elevated risks for the development of laryngeal cancer after exposure to asbestos were confirmed.

[46] Dr. Prat summarized his conclusion on the association between laryngeal cancer and asbestos exposure as follows:

> Nevertheless, after year 2000, there has been more evidence supporting a possible causal relationship between cancer of the larynx and asbestos exposure. The NAP authors, who did the most recent comprehensive review, concluded that the evidence in 2006 was "sufficient" to accept a probable relationship between cancer of the larynx and asbestos. They proposed a RR of 1.4.

[47] Dealing with the worker specifically, Dr. Prat provided the following:

> [The worker] was a smoker until 12 years ago. He had a 30 pack year habit. This would be equivalent to one pack a day for 30 years. As per the review above, 10-20 cigarettes per day correspond to a RR for cancer of the larynx of 8.36 (5.18 to 13.51). A RR of 8.36 is very large. According to the family doctor's notes, [the worker] had been a heavy drinker at some point. This is another significant risk for cancer of the larynx.

[12] Purdue, MP. et al; "Occupational exposures and head and neck cancers among Swedish construction workers"; *Scandinavian Journal of Work, Environment and Health*, 2006 Aug; 32(4): 270-5.
[13] Marchand, JL. et al; "Laryngeal and hypopharyngeal cancer and occupational exposure to asbestos and man-made vitreous fibers: results of a case-control study"; *American Journal of Industrial Medicine*; 2000 Jun; 37(6): 581-9.
[14] Ramroth, H. et al; "Occupational asbestos exposure as a risk factor for laryngeal carcinoma in a population-based case-control study from Germany"; *American Journal of Industrial Medicine*; 2011 July, 54(7): 510-14.

> Dr Gillis and by Dr Skarsgard have provided letters in support of this appeal. Dr Skarsgard pointed out the most recent review done by the National Academy Press Committee on Asbestos Health Effects (2006).
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> **It is possible that asbestos exposure may have caused his cancer of the larynx. This possibility is supported by more recent studies and reviews. However there are other causes for cancer of the larynx.** The two main ones are cigarette smoking and alcohol consumption. [The worker] smoked and drank alcohol. A number of these cancers also have unknown causes.
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> In the case of this worker, if we use a aggregated RR of 1.4 (1.19 to 1.64) as quoted by Dr Skarsgard from the most recent review (NAP 2006), this gives him only a 28% chance that his cancer was caused by his exposure at work, assuming his exposure was similar to most of the ones in the studies quoted in that review. Further, if one look at his past cigarette consumption at 30 pack years (say one pack of 20 cigarettes per day for 30 years), his RR was at least 8.36, which is much higher than the RR of 1.4 due to asbestos exposure. **Hence it is clear that cigarette smoking is by far the most likely cause of this man's cancer of the larynx, much higher than any asbestos exposure he had in the past**. Alcohol may have made the situation even worse.
>
> [reproduced as written, emphasis added]

[48] In reply to Dr. Prat's opinion, the worker submitted that causation of the worker's laryngeal cancer may well have been multifactorial, and that it was speculative to rule out his workplace exposure to asbestos as a causatively significant factor on the basis that Dr. Prat had. The worker reiterated that work need not be the sole, or even predominant, cause of a worker's condition; rather, it need only be a significant cause (beyond that of a trifling nature). In this regard, the worker pointed to the fact that Dr. Prat had provided an opinion as to whether the worker's exposure to asbestos had "caused the cancer" rather than an opinion as to whether there was a 50% or greater probability that the work exposure was a significant cause of the worker's cancer.

[49] Dr. Prat provided a further comment on April 18, 2012, in response to a question from the review officer regarding a publication by the International Agency for Cancer Research (IARC). The review officer noted that on March 31, 2012, IARC had published its "List of classifications by cancer sites with sufficient or limited evidence in humans, volumes 1 to 104" and had indicated in that document that for cancer of the larynx, agents including alcohol, tobacco smoking, and asbestos (all forms) had been identified as "Carcinogenic agents with *sufficient* evidence in humans."

[50] As a result, Dr. Prat reviewed the IARC publication. He noted that it was not a meta-analysis, and that the IARC had not carried out a critical appraisal of the studies it had quoted. Rather, it had simply listed the results of the studies and provided some odds ration. Dr. Prat obtained the studies cited by the IARC publication, and after review, indicated that:

> **…**whichever way we look at the issue, the risk analysis numbers tend to stay well below a probability of 50% (i.e. a RR of 2.0) that the cancer was caused by the worker's exposure to asbestos for about 3-4 years in the 1980s. It is of note that the above cohorts were generally exposed for much longer than 3-4 years.

[51] Of interest, Dr. Prat provided the following in his April 18, 2012 memorandum:

> NB: My prior opinion did not discuss the issue of "causative significance" of asbestos exposure in the appearance of the cancer of the larynx for [the worker]. I limited myself to doing a risk analysis and to trying to arrive at some statistical estimates of the probability that the cancer was actually caused (or not caused) by the exposure.

[52] The review officer, in considering the question that is also at issue in this appeal, indicated that she approached the issue of the medical opinions before her as follows:

> In this situation, I have relied upon the "Protocol for the Assessment of Medical/Scientific Information – Industrial Diseases Standing Committee, Workers' Compensation Board of British Columbia", March 2, 1993 (9 W.C.R. 429, at 445), ("the Protocol"), which adopted standardized criteria for the evaluation of epidemiological evidence with respect to occupational diseases. Specifically, the Protocol adopted the *Bradford Hill Criteria* or guidelines, which require that the evidence for human studies demonstrate strength of association, consistency, dose-response, coherence, temporal relationship, specificity, and statistical significance. These are the factors to which the RDMA [Review Division medical advisor] has alluded in his cursory assessments of the literature.
>
> The Protocol noted the *Bradford Hill Criteria* finding that, "an RR of 1.4 or 1.5 is by itself not very impressive, because there are so many confounding variables, such as cigarette smoking, nutrition, genetic background, etc. A relative risk below about 2 quite often turns out to result from a different distribution of confounding variables in the exposed group compared to the control group." In order to be persuasive, the Protocol concluded that the other factors above needed to be present and most of the criteria must be met.

[53] The review officer acknowledged the worker's seven-year history of exposure to asbestos, but concluded that:

> With respect to this worker, I am unable to determine that his risk was similar to the workers included in the studies considered by the NAP and relied upon by Dr. S[karsgard]. Although the WA [worker's adviser] concluded that a 28% chance or aggregate RR of 1.4 was of causative significance, I find that the RR in this case, is well short of an RR of 2.0, and the 50% threshold currently required by the Board in order to confer a doubling of risk for laryngeal cancer with exposure. The epidemiological evidence does not consistently demonstrate the necessary strength, consistency and dose-response relationship (the *Bradford Hill Criteria*) required by the Board. Specifically, the various studies do not demonstrate a consistent threshold value of 2.0 or greater, with the necessary dose-response relationship, in order to conclude that asbestos-exposed workers have double the risk of laryngeal cancer, as compared to the general population.
>
> Having weighed all of the evidence, I am unable to conclude with confidence that that this worker's asbestos exposure had causative significance for the development of his laryngeal cancer.

*New Evidence at WCAT*

[54] Prior to and at the oral hearing of this appeal, the worker provided a number of publications[15] regarding the link between asbestos and laryngeal cancer. While I have reviewed those articles, I do not consider it necessary to summarize them here. It suffices to say that they generally indicated some association between asbestos exposure and the development of laryngeal cancer.

[55] The worker also provided an article entitled "Reversal of risk upon quitting smoking"[16] which indicated that the International Agency for Research on Cancer had noted that studies showed that:

> …relative risk for laryngeal cancer decreases steeply with time since stopping smoking, with reductions of about 60% 10-15 years after cessation and even larger after 20 years. However, former smokers still have higher risks as compared with never-smokers for at least 20 years.

**Submissions**

[56] In the worker's submission, his exposure to asbestos while working at the asbestos mine was more than a trivial cause in the development of his laryngeal cancer. He

[15] The worker provided either entire articles or abstracts of articles.
[16] *The Lancet*; Vol 368, Juy 29, 2006; p.348-9.

submitted that his laryngeal cancer was therefore a compensable occupational disease. The worker argued that the review officer had been looking for a 100% likelihood that his exposure to asbestos played more than a trivial role, whereas, in his submission, that likelihood need only be prove on a balance of probabilities.

[57] The worker referred to jurisprudence from the Supreme Court of Canada which sets out that, in determining whether a defendant's negligence had caused a plaintiff's loss, scientific proof of causation is not required[17]. The worker submitted that if scientific certainty was not required in proving causation on a "but for" test in civil cases of negligence, such scientific certainty certainly could not be required in proving "causative significance" as required in workers' compensation cases by the Act.

**Reasons and Findings**

[58] Section 6 of the Act provides that compensation is payable when a worker suffers from an occupational disease that is due to the nature of his or her employment. An occupational disease is defined by section 1 of the Act as:

- Any disease mentioned in Schedule B;
- Any disease that the Board has designated as peculiar to or characteristic of a particular process, trade or occupation; and,
- Any other disease which the Board, by regulation of general application or by order dealing with a specific case, may designate or recognize as an occupational disease.

[59] Subsection 6(3) of the Act sets out that if a worker, at or immediately before the date of the disablement, is employed in a process or industry mentioned in the second column of Schedule B, and the disease contracted is the disease listed in the first column of Schedule B set opposite to the description of the process or industry, the disease is deemed to have been due to the nature of that employment unless the contrary is proved.

[60] The first column of Schedule B includes item #4(c), which concerns "Carcinoma of the larynx or oropharynx associated with asbestosis." However, the evidence in this case fails to establish that the worker has been diagnosed with asbestosis. Therefore, while the worker's squamous cell carcinoma of the larynx satisfies one of the requirements found in the first column of Schedule B, the worker's squamous cell carcinoma does not satisfy all of the requirements. As a result, I find that the presumption set out in subsection 6(3) does not apply to the worker's claim.

[61] I turn, then, to a consideration of the compensability of the worker's cancer condition under subsection 6(1) of the Act.

[17] *Clements v. Clements*, 2012 SCC 32, para.46.

[62] There is no presumption under subsection 6(1). The issue is whether the worker has an occupational disease that is due to the nature of his employment. In reaching a conclusion on this issue, I must determine whether there is sufficient positive evidence demonstrating a causal link between the worker's employment and the worker's disease.

[63] Sufficient positive evidence of a causal link does not mean that the evidence must show that the worker's employment was the "only" or even the "most significant" causative factor. Rather, the question is whether the positive evidence is such that it can be concluded that the employment exposure was of causative significance in producing the worker's occupational disease. A speculative possibility that a worker's employment may have caused the disease is not sufficient to establish a causal relationship.

[64] The fact that there may, however, be more than one causal factor of a worker's occupational disease does not preclude acceptance of a claim. If the employment exposure has played more than a trivial role in producing a worker's disease, then, pursuant to subsection 6(1), that disease is a compensable one. In a case such as this, where there is clear evidence of potential non-occupational causal factors (such as the worker's drinking and smoking history), such a consideration is of particular importance.

*Association between Asbestos Exposure and Laryngeal Cancer*

[65] There are a number of medical opinions before me as to the connection or association between asbestos exposure and the development of laryngeal cancer, generally, as well as specifically as related to the worker in this case. Each of the physicians who have provided those opinions has also referred to various studies which have considered this issue. I have reviewed the conclusions of a number of those above.

[66] I will say at the outset of my consideration of whether there is an association of significance between asbestos exposure and the development of laryngeal cancer generally, that I place little weight on Dr. Gillett's August 2011 opinion that there exists "no evidence" to suggest that asbestos exposure increases the risk of laryngeal cancer. As was noted by a number of the physician's who have provided opinions in this case, and as was submitted by the worker, Dr. Gillett's opinion in this regard appears to have been based on a single (and perhaps outdated) study.

[67] My review of the literature on this issue leaves little question that there exists at least some evidence that exposure to asbestos increases the risk of developing laryngeal cancer. The findings of the NAP study are, in my view, clear in this regard. Dr. Gillett's opinion is simply inconsistent with the findings of the larger studies that have considered this issue, as well as being inconsistent with the commentary of any of the other physicians (including not only Dr. Skarsgard and Dr. Gillis, but also Dr. Prat) who have reviewed this issue in the course of the worker's claim.

[68] After reviewing the studies on the issue of a potential association between asbestos exposure and development of laryngeal cancer, it seems to me that the overall conclusion to be taken from the evidence is that such an association exists. The findings of the NAP study, which were based on a comprehensive review of more than 50 studies, provide particularly compelling evidence of this association.

[69] I find support for my conclusion in this regard in the opinions of Dr. Skarsgard and Dr. Gillis, which refer not only to the various medical studies on this issue, but also, in the case of Dr. Skarsgard, explain the biological plausibility of the association between asbestos exposure and laryngeal cancer. While not as definite in his opinion as were Drs. Skarsgard and Gillis, Dr. Prat also acknowledged that the more recent studies (since 2000) tend to indicate that a causal relationship between cancer of the larynx and asbestos exposure.

[70] In reaching this conclusion, I acknowledge that the review officer conducting *Review Reference #R0133849* concluded that she was unable to rely on either the NAP study findings or other submitted literature, as the RR that had generally been identified in that literature was not sufficient to find that an association between asbestos exposure and laryngeal cancer exists. The review officer explained that even if she accepted that exposure to asbestos led to an RR of 1.40 (as set out in the NAP study), such an RR was:

> …well short of an RR of 2.0, and the 50% threshold currently required by the Board in order to confer a doubling of risk for laryngeal cancer with exposure. The epidemiological evidence does not consistently demonstrate the necessary strength, consistency and dose-response relationship (the *Bradford Hill Criteria*) required by the Board. Specifically, the various studies do not demonstrate a consistent threshold value of 2.0 or greater, with the necessary dose-response relationship, in order to conclude that asbestos-exposed workers have double the risk of laryngeal cancer, as compared to the general population.

[71] With respect, there is no requirement in either the Act or in policy that an RR of 2.0 be found for the Board to accept that there is more than a trivial association between exposure to a substance and the development of a disease. It is worth noting that the Board's Industrial Diseases Standing Committee that issued the "Protocol" referred to by the review officer indicated that the criteria of causality in that Protocol were not a set of rigid rules, but simply guidelines, and that the relative importance of each criterion may vary according to the circumstances of each case.

[72] Further, I consider that in reaching her conclusion as to whether there exists more than a trivial association between asbestos exposure and laryngeal cancer, the review officer erred in her application of the comment contained within the Protocol (which is a reference to a comment in what are known as the *Bradford Hill Criteria*[18]) that:

> … a relative risk of 1.4 or 1.5 is by itself not very impressive, because **there are so many confounding variables, such as cigarette smoking, nutrition, genetic background, etc**. A relative risk below about 2 quite often turns out to result from a different distribution of confounding variables in the exposed group compared to the control group.
>
> [emphasis added]

[73] The next paragraph in the Protocol provides clarity to the above excerpted comment by setting out that:

> **The main exception to this rule is tied in with another criterion, dose-response, which will be examined later**. If, in a carefully designed study, there is a clear gradient from lower dose to a higher dose rising through figures such as 1.2, 1.4, and 1.6, these otherwise low and unimpressive relative risks become much more convincing.
>
> [emphasis added]

[74] While it is true that a dose relationship was not identified in each of the studies that have been placed before me (and were before the review officer), such a response was identified in a number of those studies. I consider the finding of the NAP study, which was that there existed some evidence of a dose-response relationship in the meta-analyses that it had completed, to be compelling. Further, I note that in a number of the studies identified in the NAP study, attempts were made to address the confounding variables (particularly those of smoking and alcohol), and that even in those studies, an association with asbestos exposure and the development of laryngeal cancer was found to appear to persist.

[75] In summary, I am satisfied that there is sufficient evidence to conclude that there exists an association between asbestos exposure and the development of laryngeal cancer.

[76] I turn, then, the worker's exposure to asbestos.

[18] A Bradford Hill, The Environment and Disease: Association or Causation? 58 *Proceedings of the Royal Society of Medicine*. 295.

*Exposure*

[77] The worker has been clear in stating that, in his view, he was exposed to asbestos dust and fibres for a period of approximately seven years. He has also clearly explained that there was a period of time of approximately three to four years while he worked at the asbestos mine during which he did not wear a respirator at all. Further, he has indicated that even when he did wear a respirator, it appeared as though asbestos dust was getting through the filters provided, and that he eventually used two filters at once.

[78] I found the worker to be a credible witness. I consider that it is more likely than not that, as he stated, he was exposed to airborne asbestos fibres on what was essentially a daily basis for a number of years while working at the asbestos mine. I accept, as did the Board and the Review Division, that the worker would have been exposed to asbestos fibres and dust for approximately seven years of employment while working at the asbestos mine.

[79] I also find that the worker was exposed to non-occupational risk factors for the development of laryngeal cancer, specifically, smoking and alcoholism.

[80] Specifically, I find that the worker engaged in a heavy alcoholism pattern for a period of approximately six years (more than five drinks per day) which ended in approximately 1991. I also accept, based on the worker's testimony, that he likely engaged in moderate alcoholism in the four years prior to that period.

[81] I also find that the worker was a long-term smoker, in that he was a daily smoker, for approximately 24 to 26 years. My finding in this regard is consistent with what the worker stated at the oral hearing. I note that I also accept the worker's evidence that his smoking habit varied over the course of that period of time, wherein he would at times smoke only a half-pack per day and at other times would smoke more than a pack of cigarettes per day.

[82] I note that the worker provided testimony regarding his subsequent places of employment, which included working at a welder in a variety of sawmills and production facilities. No evidence was placed before me, however, regarding whether the worker would have been exposed to asbestos or other chemicals which may play a causative role in the development of laryngeal cancer at any of those places of employment. I note further that none of the medical practitioners who have reviewed the worker's case were provided with this information nor did they consider his other employment. Finally, I note that it is clear that the Board was adjudicating the issue of whether the worker's employment at the asbestos mine (and the exposure to asbestos he experienced there) was of causative significance in producing his laryngeal cancer. I consider that that is the issue that I am also adjudicating on this appeal.

*Causative Significance*

[83] I find the opinion of Dr. Skarsgard on this issue to be compelling. I find that Dr. Skarsgard had a good understanding of the worker's exposure to asbestos (as it was based on the worker's reported history, which I have accepted), and accept his conclusion that the worker's exposure was likely on the higher end. In providing his opinion that the worker's risk of developing laryngeal cancer was considerably raised by his occupational exposure to asbestos, Dr. Skarsgard indicated that he was acutely aware of the worker's use of alcohol and smoking, as well as the fact that the worker may have developed laryngeal cancer in the absence of exposure to any of asbestos, alcohol, or smoking.

[84] Dr. Gillis indicated that although he was of the view that the worker was at increased risk for the development of laryngeal cancer as a result of his smoking and drinking, his opinion was that the worker's risk was increased by an even greater amount as a result of his occupational asbestos exposure.

[85] I consider the evidence before me to lead to the conclusion that, while it is true that the worker was at increased risk for developing laryngeal cancer as a result of his smoking and drinking, he was at further increased risk as a result of his occupational exposure to asbestos from 1980 to 1987.

[86] After considering the amount of the worker's exposure to asbestos during that period of time, the medical opinions of Dr. Skarsgard and Dr. Gillis, as well as my conclusions above that there exists an association between asbestos exposure and the development of laryngeal cancer, I find that it is at least as likely as not that the worker's asbestos exposure was more than a trivial cause in the development of his laryngeal cancer. As a result, I find that the worker's laryngeal cancer is due to the nature of his employment at the asbestos mine. While his occupational exposure may not have been the most significant cause of his laryngeal cancer, it was more than a trivial one. The worker's appeal is allowed.

[87] In reaching this conclusion, I note that I have placed little weight on Dr. Prat's opinion, as I consider that the worker was in fact exposed to asbestos for a period of seven years, rather than the three to four years that Dr. Prat identified.

[88] Further, I note that the fact that the worker's smoking habit may have been, as Dr. Prat put it, "by far the most likely cause" of the worker's laryngeal cancer does not mean that the worker's occupational asbestos exposure was not of causative significance in producing that condition. While Dr. Prat ultimately concluded that, from a statistical perspective, the worker's laryngeal cancer was not "caused" by his asbestos exposure, that finding was based on his view that an RR of 2.0 was required to reach a conclusion that that cancer was "caused" by that exposure. As I have indicated above, I do not consider that such an RR is required.

**Conclusion**

[89] I vary *Review Reference #R0133849* and allow the appeal. I find the worker's squamous cell carcinoma of the larynx is an occupational disease due to the nature of his employment.

*Expenses*

[90] The worker requested reimbursement for the cost of having to attend the oral hearing in Richmond, British Columbia. I noted that, based on the worker's evidence, he lived in a town in the interior of British Columbia, and that that town was one in which WCAT regularly held hearings. I queried why it was that the worker had not requested that his oral hearing be held in that location. Both the worker and his representative indicated that they had simply not thought to make such a request.

[91] Section 7(1)(a) of the *Workers Compensation Act Appeal Regulation* provides that WCAT may order the Board to reimburse a party to an appeal for the expenses associated with attending an oral hearing. The practice directive at item #16.1.2.1 of the WCAT *Manual of Rules of Practice and Procedure* notes that WCAT will normally order reimbursement for an appellant's travel expenses where WCAT schedules an oral hearing in a location that is not the closest to the community in which the appellant resides.

[92] In my view, the fact that it simply did not occur to the worker and his representative to request that the oral hearing take place in his home community is not a sufficient reason to order reimbursement for the cost of the worker's travel to attend the hearing in Richmond, British Columbia. It was at the worker's request that the oral hearing was held in Richmond. The WCAT notice of appeal form clearly provides that a worker may request the location of his hearing. In my view, the worker bears the responsibility for the location of his hearing, and, as such, this is not an appropriate case in which to order reimbursement for the expenses he incurred to travel to the Lower Mainland from Kelowna. I decline to exercise my discretion to order that the worker be reimbursed for those expenses.

Andrew Pendray
Vice Chair

AP/gw