



## WORKPLACE SAFETY AND INSURANCE APPEALS TRIBUNAL

### DECISION NO. 2082/07

**BEFORE:** S. Ryan : Vice-Chair  
B. Wheeler : Member Representative of Employers  
D. Broadbent : Member Representative of Workers

**HEARING:** September 14, 2007 at Toronto  
March 25, 2008 (teleconference reconvene)  
July 7, 2009 (post-hearing investigation complete)

**DATE OF DECISION:** December 31, 2009

**NEUTRAL CITATION:** 2009 ONWSIAT 3035

**DECISION(S) UNDER APPEAL:** N. Kissore, Appeals Resolution Officer, January 16, 2006; and  
N. Kissore, Appeals Resolution Officer, May 13, 2006

#### APPEARANCES:

**For the worker:** G. Hoag, Office of the Worker Adviser (OWA)

**For the employer:** W. Lemay, a lawyer

**Worker Observers:** Articling Student, OWA representatives in training

**Employer Observer:** Occupational Health Physician

## REASONS

### (i) Introduction

[1] In the fall of 1989, the worker was diagnosed with squamous cell carcinoma of the left tonsil. On October 12, 1989, he underwent surgery following which he received radiation treatment.

[2] On November 29, 2002, the worker completed a Worker's Report of Occupational disease claiming that his tonsillar cancer was related to his employment between March 1975 and May 1976, and specifically to his exposure to second-hand smoke while travelling to and from the worksite on an employer bus.

[3] In this appeal, the Tribunal has been asked to determine whether or not the worker has initial entitlement for tonsillar cancer.

### (ii) Decision No. 2082/07I (October 24, 2008)

[4] In *Decision No. 2082/07I* (October 24, 2008), we provided a detailed history of this case which does not need to be repeated in this decision. We determined that our understanding of the medical issues in this case might benefit from an opinion of a Tribunal Medical Assessor specialising in occupational medicine. With the consent of the parties, the Tribunal's Medical Liaison Office (MLO) selected Dr. R. House, an occupational medicine specialist. Dr. House currently practises in the Department of Occupational and Environmental Health at St. Michael's Hospital. He graduated with a medical degree in 1974 from McGill University and obtained specialist qualifications in clinical epidemiology and occupational medicine in 1986 and 1988, respectively. He holds a number of current appointments including Assistant Professor in the Faculty of Medicine and Director of the Occupational Medicine Residency Program at the University of Toronto. He has written extensively on occupational medicine topics including second-hand smoke exposure in the workplace.

[5] In a report dated April 15, 2009, Dr. House reviewed the history of this case as documented in the Case Record and described in *Decision No. 2082/07I*. He answered specific questions posed by the Panel. Our questions and the Assessor's answers are set out in full below:

#### Questions:

1. *Please comment on any relationship between exposure to second-hand smoke (as described in the interim decision) and this worker's squamous cell carcinoma of the tonsil. Based on a review of the medical reports and epidemiological studies on file as well as the Panel's findings of fact, are you able to conclude that the second-hand smoke caused or significantly contributed to the development of the worker's cancer? Please explain.*

Oral and nasopharyngeal squamous cell carcinomas are often considered as a group because they share some similar risks factors. The tonsils are a ring of lymphoid tissue around the upper part of the pharynx. About 10% of oral squamous cell carcinomas being in the palate and tonsillar area.

The main risk factors for oral and nasopharyngeal squamous cell carcinoma are tobacco smoking and alcohol consumption. Other predisposing factors may include chronic viral

infection, in particular human papilloma virus infection. Immune deficient states, poor oral hygiene and genetic factors.

Due to the fact that smoking is a risk factor for oral and nasopharyngeal squamous cell carcinoma and that it is usually assumed that there is no threshold of exposure to most carcinogens below which there is no risk, it seems plausible that second-hand tobacco smoke exposure might be associated with increased risk of this general type of cancer and squamous cell carcinoma of the tonsil specifically. The International agency for Research on Cancer (IARC) has determined that second-hand tobacco smoke is a confirmed cause of lung cancer. However, a search of Medline using Pub Med did not indicate any published epidemiologic studies that reported an association between second-hand tobacco smoke and squamous cell carcinoma of the tonsil. If the research is expanded to include other oral and nasopharyngeal squamous cell carcinomas there is more epidemiologic evidence available but the results are conflicting. For example a case control study by Cheng et al in Taiwan (Cancer Causes Control. 1999; 10(3):201-207) found that exposure to second-hand tobacco smoke in child hood or adult life was not associated with increased risk of nasopharyngeal carcinoma whereas another case control study in China by Yuan et al (Int J Cancer, 2000;85(3):364-369) did find some evidence of an increased risk of nasopharyngeal carcinoma associated with second-hand tobacco smoke.

The time period of approximately 14 years between the initial exposure to occupational second-hand tobacco smoke in 1975 and the diagnosis of squamous cell carcinoma of the tonsil in 1989 is not problematic in terms of attributing the cancer to this exposure because a sufficient latency period is needed for the cancer to develop. As well the time in the early 1980's and the date of diagnosis of the cancer is not problematic for similar reasons.

When trying to determine whether a particular exposure caused a specific cancer, dose response is always a consideration. Second-hand tobacco smoke exposure is, in general, much lower than exposure from active smoking and hence the risks are expected to be corresponding lower. Within the general category of second-hand tobacco smoke exposure, the exposures described by the claimant do not seem to be particularly high or of long duration. These issues related to dose should be considered in the overall evaluation of risk.

Often it is difficult to find a likely cause of a specific cancer in a worker. In [the worker's] case he does not appear to have any of the obvious environmental or lifestyle risk factors for squamous cell carcinoma of the tonsil. Therefore it is reasonable to examine the possibility that second-hand tobacco smoke exposure may have caused the cancer. I think that this causal connection is biologically plausible but, it does not appear very likely that this cancer was caused by the exposure in question. The cancer is probably best described as being idiopathic.

2. *Do studies in the literature on second-hand smoke support a relationship between such exposure and tonsillar cancer? If so, is there any particular level or extent of exposure that is implicated?*

As mentioned above there do not appear to be any epidemiologic studies reported in the literature that support a relationship between second-hand smoke and the specific cancer of squamous cell carcinoma of the tonsil. However, there are some studies that have found an association between nasopharyngeal cancer and second-hand tobacco smoke and the causative factors for the various types of oral and nasopharyngeal cancers appear to be similar. The most relevant paper I found in terms of the risk associated with second-hand smoke was the case control study by Yan et al. mentioned above. This study included 935 cases of nasopharyngeal cancer and 1,032 community controls in Shanghai, China. Active smoking was found to be a statistically significant risk factor for nasopharyngeal carcinoma (Odds ratio 1.28; 98% of confidence interval 1.02 – 1.61).

As well the risk associated with second-hand smoke from various sources of exposure was examined in detail. I have included this paper with my report and the key information related to risk associated with second-hand tobacco smoke is found in tables II (page 366) and III (page 367). In particular table III summarizes the odds ratios associated with second-hand smoke in men due to smoking by their co-workers (which are probably most germane to [the worker's] case) as follows:

Hours exposed per working day	Odds Ratio (95% Confidence Interval)
≤ 3	1.21 (0.56-2.59)
≥ 3	1.48 (0.68-3.21)

These results for men were not statistically significant. It should also be pointed out that these estimates did not consider the years of exposure to second-hand smoke. However some of the other odds ratios for difference sources of second-hand tobacco smoke in men and women reported in the paper were statistically significant and the overall evidence presented gives an indication of what the risk associated with second-hand tobacco might be. I think it is reasonable to assume that, if there is risk associated with second-hand tobacco exposure, it should be less than the risk associated with active smoking and hence should lie in the range between approximately 1 – 1.25 times the baseline risk associated with no exposure to second-hand tobacco smoke.

3. *Can you provide any other medical information which you feel would be of assistance to the panel and parties in understanding the nature and aetiology of this worker's condition?*

There is considerable variation in the rates of oral and nasopharyngeal squamous cell carcinoma in different countries. For example Rosenquist (Swed Dent J Suppl. 2005;179:1-66) states that this type of cancer accounts for only about 1% of all cancers in Sweden but this is low in comparison to the incidence on the Indian subcontinent and in other parts of Asia, where it is one of the most common forms of cancer. Also for the last several decades the incidence of squamous cell carcinoma of the tonsil has been increasing in both the U.S. (Frisch M et al. Cancer Causes Control 2000;11(6):489-495) and Sweden (Hammarstedt et al. Acta Otolaryngol 2007;127(9):988-992). This increase has occurred when active smoking (and hence second-hand smoke exposure) was decreasing in both countries which suggests that tobacco smoke exposure has not been a key factor in this recent increase.

### **(iii) Submissions**

#### **(a) The worker**

- [6] On behalf of the worker, Mr. Hoag offered written submissions dated May 21, 2009. Attached to his submissions was a report dated May 19, 2009, addressed to Mr. Hoag, from Dr. N. Kerin, an occupational health physician at the Occupational Health Clinic for Ontario Workers (OHCOW) Inc. Under cover letter dated May 29, 2009, Mr. Hoag attached a second report from Dr. Kerin also dated May 19, 2009. The second report is identical to the first, but contains more information.

- [7] Dr. Kerin advised that he reviewed Dr. House's report and, in his second report of May 19, 2009, offered the following commentary:

Dr. House's discussion is confined to epidemiological considerations with respect to cigarette smoking and causation of oral pharyngeal cancers, specifically tonsillar carcinoma.

Dr. House goes on to describe this particular cancer. The cancer is probably best described as being 'idiopathic'. In medical literature, idiopathic means 'of unknown cause'. Upon review of my clinic notes of April 13, 2006, it is noted that [the worker] had periodic exposure to open pit mine face mining...as [the employer of record was an open pit and copper mine and processing plant. This type of mining operation exposes workers (including [the worker]) to the products of particulate matter crushing. The dust emanating from a crushing process will involve silica (including quartz silica) and varying levels of arsenic. It is known that northern Ontario's earth crust (Canadian Shield) may contain varying concentrations of arsenic, where the levels of arsenic seriously interfere with the amount of feed that may be fed directly into the refinery from these mines. It is unknown whether or not [the worker] would have been exposed to strong acids (H2SO2) which are also known to be carcinogenic.

I do not have information about what other processes were used at the open mine/processing plant, but would understand that the bus in which [the worker] travelled would likely have had some contamination from the clothing and work boots worn by miners during their two-hour travel each way daily.

As Dr. House and I have indicated in our submissions, the incidence of squamous cell carcinoma of the tonsil has been increasing in the United States and Sweden... As indicated, this increase has occurred when active and second-hand smoke rates have declined in both North America and Sweden, suggesting that tobacco smoke exposure alone is not the only contributing factor to this recent increase.

With respect to hours exposed per working day (page 3 of Dr. House's report), from his understanding of [the worker's] exposure he placed him at an odds-ratio of 1.2. My notes would suggest that [the worker] had approximately four hours' exposure to second-hand cigarette smoke on a daily basis (2 hours travel each way), this exposure time would indicate [the worker] had an odds-ratio of 1.48 and as such is significant with respect to second-hand smoke and nasopharyngeal cancer development.

- [8] Dr. Kerin opined that the worker would have been exposed to multiple chemical and dust exposures that were the norm in mining environments and that failure to mention them was a "serious oversight." He stated that the interaction between cigarette smoking and other carcinogens including ionising radiation, arsenic and asbestos, has an additive or multiplying-type effect on significant second-hand cigarette smoke. Dr. Kerin advised that there have been significant developments over the past 20 years in research techniques into the effects of mixture exposures. He concluded:

Generally, in occupational medicine, the use of the term idiopathic when describing malignancies, is used with more and more caution, as our understanding of weight of evidence from more sophisticated epidemiological studies are completed, for instance the recent *International Agency for Research on Cancer* (IARC) statement that asbestos is now considered to be a Class 1 carcinogen with respect to laryngeal and ovarian cancer reminds us of this caution. Just six months ago ovarian cancer would have been described as idiopathic.

In [the worker's] case i[t] would appear more appropriately to describe his exposure profile as mixed carcinogenic and the disease (tonsillar cancer) as more likely than not developed as a result of multiple chemical/dust exposure, given what we know about this particular case and the rise in incidence of this cancer while tobacco use is in decline.

### (b) Employer

- [9] On behalf of the employer, Mr. Lemay offered submissions dated June 12, 2009. The employer's representative reviewed in detail the report from Dr. House. He submitted that the report does not support the conclusion that the worker's exposures while employed by the

employer of record significantly contributed to the development of tonsillar cancer. Mr. Lemay emphasised Dr. House's observation that there has been an increase in nasopharyngeal squamous cell cancer cases in the Indian subcontinent despite declining smoking levels which suggests that second-hand smoke may not be a key factor in the development of that disease. Mr. Lemay emphasised that the conclusions reached by Dr. House were also reached by two other specialists, Drs. Senn and Brown.

[10] The employer's representative submitted that Dr. Kerin's report(s) should not be relied upon because he is not an expert in oncology or epidemiology. Mr. Lemay noted that no evidence or argument was raised on the issue of exposure to contaminants from open pit mines. He stated:

[T]here is no evidence whatsoever of any carcinogens in any dust that may have been coming from [the employer of record] mine.

[11] Mr. Lemay submitted that Dr. Kerin's estimation of the worker's exposure to second-hand smoke (four hours per day) is inconsistent with the Panel's findings of fact and the worker's testimony on that point. Accordingly, he submitted, Dr. Kerin's estimate of risk cannot be relied upon.

**(c) Worker (reply)**

[12] In correspondence dated June 25, 2009, Mr. Hoag submitted that biological plausibility between exposure to second-hand smoke and cancer was acknowledged by Dr. House and is one of the Bradford-Hill criteria making it an important factor in this case.

[13] Mr. Hoag cited *Decision No. 1645/99R* (October 31, 2000), in which a Vice-Chair commented on the significant contributing factor test applied widely in Tribunal jurisprudence in determining issues of causation which, she noted, was entirely consistent with the principles identified by the Supreme Court of Canada in *Athey v. Leonati*. Mr. Hoag submitted:

In this case, there is no positive scientific proof that [the worker's] cancer was caused by his work. However, it can also be said that there is no evidence to the contrary. Instead, there is biological plausibility, as noted by Dr. House. Thus, we ask that an inference of causation be drawn.

We would also urge the Panel to adopt the conclusions in [*Decision No. 1645/99R*], not only because of *Snell* – since positive scientific evidence is not necessary or determinative. Indeed, we would ask that a factual inference against [the employer] be made by the Panel, since evidence against the biological plausibility of a causal relationship has not been adduced by the employer.

Lastly, as noted above, we would also ask the Panel to adopt a robust and pragmatic approach to the evidence. We submit that there are no genetic or lifestyle causal factors, which would pre-dispose the worker to cancer, which have been adduced in evidence – which are likely to be a significant contributing factor to the worker's cancer. Absent those factors, and given the biological plausibility of the worker's exposure to second-hand smoke—which the Panel has accepted, we submit this should be quite compelling.

[14] Mr. Hoag acknowledged that Dr. Kerin is neither an oncologist nor epidemiologist. However, he stated that the OHCOW physician has significant experience in occupational settings, particularly in mines.

**(iv) Findings and conclusions**

[15] We have carefully considered all of the available evidence in the Case Record, testimony of the worker and submissions of the representatives.

[16] In *Decision No. 2082/071*, we discussed the legal standards of causation widely applied in Tribunal jurisprudence. We noted that the Tribunal has applied the significant contributing factor test in determining issues of entitlement including issues of entitlement in occupational disease cases. The Tribunal has also applied the civil standard of a balance of probabilities in determining whether or not there is a relationship between a workplace injury and a subsequent medical condition.

[17] We cited *Decision No. 661/93*, 33 W.C.A.T.R. 64, in which the Panel considered the question of how to approach the issue of causation in a context where there may multiple potential causes including the compensable accident to explain the emergence of a subsequent medical condition. The Panel concluded at page 77:

...the Panel is required to apply [the evidentiary standard of] a balance of probabilities... [where] there are...several possible scenarios, [the] Panel must decide what the most probable scenario is.

[18] We also cited the Supreme Court of Canada's ruling in *Snell v. Farrel* (1990), 72 D.L.R. (4<sup>TH</sup>) 289, that:

...[c]ausation need not be determined by scientific precision...it is not...essential that the medical experts provide a firm opinion supporting the plaintiff's theory of causation.

[19] In *Decision No. 2082/071*, we described the issue before as whether or not, on a balance of probabilities, the worker's exposure to second-hand smoke between March 1975 and May 1976, while employed with the employer of record, significantly contributed to the development of squamous cell carcinoma of the left tonsil diagnosed in September 1989.

[20] Our analysis of this issue necessarily includes consideration of the Bradford Hill criteria of biological plausibility and analogy. A worker's exposure to carcinogens in the workplace is significant when the applicable Bradford hill criteria have been met. In *Decision No. 429/02* (December 23, 2005), a Panel of the Tribunal succinctly described the nine Bradford Hill criteria:

The essential factor is temporality: that the exposure must precede the onset of the disease. The others are: the strength of the association (based on measures such as relative or attributable risk); consistency in the studies; specificity (which may support a finding of causation, but cannot be used to deny a causal relationship), dose-response; biological plausibility; coherence (i.e. that the cause and effect interpretation does not conflict with what is known); experimental evidence (to the extent available) and analogy.

[21] In our view, the applicable Bradford Hill criteria have been addressed, albeit implicitly, in this case in the medical reports of experts who have offered an opinion on causation.

[22] Our review of all of the available evidence and arguments before us leads us to conclude that on a balance of probabilities, the worker's exposure to second-hand smoke between

March 1975 and May 1976, while employed with the employer of record, *did not* significantly contribute to the development of squamous cell carcinoma of the left tonsil diagnosed in September 1989.

[23] In reaching this conclusion, we are persuaded primarily by the opinions of medical experts who have reviewed and understood the history of this case and have concluded that a causal relationship between the worker's exposure and his cancer is unlikely. We are also persuaded by the lack of any epidemiological evidence that supports a specific association between exposure to second-hand smoke and squamous cell carcinoma of the tonsil.

[24] We agree with Mr. Hoag that as per the *Snell* case, an inference of causation may be drawn where there is an absence of evidence to the contrary and it is not possible to establish causation with scientific certainty. We agree that a robust and pragmatic approach to the evidence is necessary in this case.

[25] Mr. Hoag argued that there is no identifiable minimum safe exposure level to second-hand smoke and, in the absence of any genetic or lifestyle contributing factors, an inference can be drawn that the worker's employment exposure caused his cancer.

[26] In assessing the likelihood of work-relatedness in a specific case it is necessary to consider whether the epidemiological evidence identifies any increased risk and, if so, what it suggests about the extent of that risk in a cohort group.

[27] In this case, three medical experts reviewed the available evidence and concluded that there was insufficient evidence of a causal link between the worker's exposure to second-hand smoke and the development of his tonsillar cancer. There is a paucity of epidemiological evidence relating cigarette smoking to squamous cell carcinoma of the tonsil. Although, as noted by Dr. House, there are some published (but conflicting) epidemiological studies that report an association between second-hand smoke and oral and nasopharyngeal squamous cell carcinomas, the preponderance of evidence indicates that the worker's exposures were not particularly high or of long duration. At a maximum, the worker was exposed to second-hand smoke generated by approximately 30 to 40 workers in a bus, two hours per day, five days per week between March 1975 and May 1976. Drs. Senn, Brown and House all concluded that, notwithstanding the lack of evidence of any non-occupational contributing factor, the worker's exposure was insufficient to relate to his tonsillar cancer.

[28] Dr. House noted the case control study by Yuan et al that active smoking was found to be a statistically significant risk factor for nasopharyngeal carcinoma with an odds ratio of 1.28 with a 95% confidence interval of 1.02 – 1.61. He noted that the same study indicated that second-hand smoke in men due to smoking by their co-workers had an odds ratio of 1.21 with a 95% confidence interval of 0.56 to 2.59 for less than three hours of exposure per day and an odds ratio of 1.48 with a 95% confidence interval of 0.68 to 3.21 for three or more hours of exposure per day. He opined that these results were not statistically significant.

[29] Dr. House's opinion is accurately based upon our findings of fact, namely, that the worker had no genetic or lifestyle pre-dispositions to the development of cancer and was exposed to second-hand smoke for less than three hours per day between March 1975 and May 1976.

Based upon his own review of epidemiological studies and the Yuan study in particular, he estimated that the probability of the worker developing tonsillar cancer as a result of his exposure to second-hand smoke for a duration of less than three hours per day, is equivalent to an odds ratio of 1.21. An “odds ratio” (also known as relative risk) of 1.21 is equivalent to a Standardized Incidence Ratio (SIR) of 121. The worker, therefore, had an SIR of 121 which translates into a probability of 17% ( $21/121 \times 100 = 17\%$ ) that the worker would not have developed tonsillar cancer, but for the workplace exposure.

[30] Tribunal decisions have held that an SIR of 100 or less is generally not recognized as supportive evidence of a relationship between exposure and cancer.<sup>1</sup> This is so because the entire incidence of cancer would have been expected to occur in the absence of the exposure.<sup>2</sup> Thus, according to Dr. House, while a relationship between the worker’s exposure to second-hand smoke and his tonsillar cancer may be plausible, it is not probable. Dr. House’s opinion is consistent with two other eminent experts who have reviewed this case.

[31] In a memorandum dated November, 18, 2003, Dr. J. Senn, Clinical Haematology/Oncology Specialist and Senior Internal Medicine Consultant,<sup>3</sup> reviewed the history of this case and noted that information regarding the carcinogenicity of second hand smoke in producing squamous cell cancer of the tonsil is “not convincing on epidemiologic grounds, according to the studies consulted.” He concluded that cancer of the tonsil in this case is unlikely to be occupationally related.<sup>4</sup>

[32] In a report dated April 27, 2005, Dr. D. H. Brown reviewed the claim file at the request of the Board. Dr. Brown is a professor of head and neck surgical oncology at the University of Toronto based at the Princess Margaret Hospital in Toronto. Dr. Brown summarised the history of this case in detail. He agreed that the evidence regarding carcinogenicity of second-hand smoke to cause a tonsillar carcinoma was very poor and “non-convincing on any grounds especially epidemiological grounds.” He opined that the worker’s tonsillar cancer was “extremely unlikely to have been specifically caused by second-hand smoke in his workplace.”<sup>5</sup>

[33] We also note that Dr. I. G. Taraschuk, a Board occupational medicine specialist, opined on July 22, 2003, that the worker’s exposure to second-hand smoke between March 1975 and May 1976 was unlikely to have contributed to the development of the worker’s squamous cell carcinoma of the tonsil. He repeated this opinion on September 3, 2003.

[34] The only medical opinion that links the worker’s exposure to second-hand smoke to the development of his tonsillar cancer is Dr. Kerin. He reported on April 13, 2006, that he interviewed the worker and reviewed the “limited epidemiological data on this rare form of oral

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<sup>1</sup> For example, *Decision No. 2232/05* (June 22, 2007)

<sup>2</sup> For detailed discussions on the calculation of risk see, for example, *Decision No. 600/97* (September 30, 2003) and *Decision No. 1574/05* (July 25, 2006).

<sup>3</sup> Dr. Senn is also a Tribunal Medical Assessor.

<sup>4</sup> For a more detailed description of Dr. Senn’s reasons and studies surveyed, see paragraph 59 of *Decision No. 2082/07I*.

<sup>5</sup> For a more detailed description of Dr. Brown’s reasons, see paragraph 60 of *Decision No. 2082/07I*.

cancer.” Dr. Kerin concluded that it was more likely than not the worker’s exposure to second-hand smoke “materially contributed” to or caused the worker’s left tonsilar squamous cell cancer.

[35] We acknowledge the opinion of Dr. Kerin expressed in the report of April 13, 2006 and in two reports dated May 19, 2009. As noted by Mr. Hoag, Dr. Kerin is not an expert in oncology or epidemiology. However, we accept that he has experience in the area of occupational medicine.

[36] Dr. Kerin’s opinion is not shared by other specialists who have offered an opinion in this case (discussed above). He estimated that the worker had an odds-ratio of 1.48 of developing nasopharyngeal cancer as a result of his exposure to second-hand smoke while working for the employer of record. Dr. Kerin’s estimate is based upon an express understanding that the worker “had probably in the region of four hours’ exposure to second-hand cigarette smoke on a daily basis.” Dr. Kerin’s understanding of the worker’s exposure to second-hand smoke is in direct conflict with the worker’s testimony under oath at the hearing and our findings of fact. At paragraph 47 of *Decision No. 2082/07I*, we noted the worker’s testimony on this point:

He stated that the travel time from his home to the mine was approximately one hour. Typically, he travelled to and from work five days per week. The worker testified that he did not work overtime (i.e. on weekends). (our emphasis)

[37] At paragraph 97 of *Decision No. 2082/07I*, we accepted that between March 1975 and May 1976, the worker travelled on a bus for two hours per day, five days per week, with between 30 and 40 mine workers, 90% of whom smoked on the bus. The worker did not travel on the bus four hours per day.

[38] However, even if we accepted Dr. Karin’s understanding of the worker’s exposure to second-hand smoke, according to Dr. House an odds ratio of 1.48 remains “statistically insignificant.” An odds ratio of 1.48 translates into an SIR of 148 and a probability of 32% that the worker would not have developed tonsilar cancer, but for the workplace exposure.

[39] Dr. Kerin raised for the first time in this claim in his reports of May 19, 2009, the issue of the worker’s exposure to other contaminants while working for the employer. He stated that the worker would have been exposed to multiple chemical and dust exposures that were the norm in mining environments and that the failure to mention them was a “serious oversight.” Dr. Kerin did not indicate who made the oversight. He stated that the interaction between cigarette smoking and other carcinogens including ionising radiation, arsenic and asbestos, has an additive or multiplying-type effect on significant exposure to second-hand cigarette smoke.

[40] At the hearing, the worker expressly denied any significant exposure to other contaminants during his employment between March 1975 and May 1976. At paragraph 46 of *Decision No. 2082/07I*, we noted:

The worker testified that he was hired by the employer or record, a copper mine, in March 1975. He recalled that for the first few weeks of employment, he lived in a trailer on the premises of the mine. He shared the trailer with a non-smoking co-worker. The worker testified that he worked in an office on the premises of the mine, but spent only

about 5% of his time in the mine itself. He denied any significant exposures to contaminants while in the mine.

[41] While we understand that the worker may not have been aware that he was exposed to some contaminants during his employment with the employer, there is simply no evidence of any significant exposure to carcinogens other than second-hand smoke. The worker spent only about five percent of his time in the mine between March 1975 and May 1976.

[42] We acknowledge Mr. Hoag's observation that Dr. Kerin interviewed the worker whereas Drs. Karaschuk, Senn and Brown did not. We do not find this fact to be particularly significant because our determination is based upon specific findings of fact about the worker's exposure and upon opinions of medical experts who reviewed relevant epidemiological studies. Their assessment of risk did not require an interview with the worker. Although Dr. Kerin's estimate of the worker's exposure is at odds with our findings of fact, we have already noted that his estimate of risk would still fall well short of statistical significance.

[43] We also acknowledge the numerous studies cited by Mr. Hoag and the worker (most of which are summarised in *Decision No. 2082/07I*). Some of these studies articulate the "no safe dose" principle: there is no level of exposure to cigarette smoke below which an increased risk of cancer is not anticipated. We accept that any exposure to cigarette smoke increases the risk of cancer. However, in considering the overall evaluation of risk for this worker, we have considered the particular facts of the worker's exposure, medical opinions from experts and relevant epidemiological data. As noted by Dr. House, second-hand tobacco smoke exposure is, in general, "much lower than exposure to active smoking" and, accordingly, "the risks are expected to be correspondingly lower."

[44] In this case, there is no evidence that identifies a specific cause of the worker's cancer. The most relevant epidemiological data to this case, that pertaining to oral and nasopharyngeal squamous cell carcinomas, indicates an association with second-hand tobacco smoke. However, as also noted by Dr. Howse, the association was not "statistically significant".

[45] For the above-stated reasons, we find that the worker's squamous cell carcinoma of the left tonsil was not causally related to his employment between March 1975 and May 1976.

**DISPOSITION**

[46]           The worker's appeal is denied.

DATED: December 31, 2009

SIGNED: S. Ryan, B. Wheeler, D. Broadbent