WORKPLACE SAFETY AND INSURANCE APPEALS TRIBUNAL

DECISION NO. 484/06

BEFORE:	M. Kenny : Vice-Chair B. Wheeler : Member Representative of Employers R. W. Briggs : Member Representative of Workers
HEARING:	February 28, 2006, at Sudbury (oral hearing), and June 10, 2009 at Toronto (written hearing) Post-hearing activity completed on July 23, 2009
DATE OF DECISION:	November 30, 2009
NEUTRAL CITATION:	2009 ONWSIAT 2785
DECISION(S) UNDER APPEAL:	WSIB ARO decision dated August 25, 2003

APPEARANCES:

For the worker:	P. Hudyman, union representative (for the oral hearing) R. Hamilton, Office of the Worker Adviser (for the written hearing)
For the employer:	Did not participate
Tribunal Counsel:	C. Zimmermann

REASONS

(i) Introduction

[1] The worker is now 77 years old. He was 62 years old when he retired in 1994. He had worked as a labourer (beginning in the 1940s) and then in the plumbing field for over 40 years. The Board accepted that the worker had the potential for significant workplace exposure to asbestos, welding fumes, and dust.

[2] The worker was also a smoker. He smoked about 1.5 packs per day for 50 years (for a total of 75 pack-years).

[3] The Board accepted that the worker suffered from chronic obstructive pulmonary disease ("COPD") and it established August 23, 1999 as the "date of injury." The worker appealed the Board's decision about the "date of injury" to the Tribunal. In *Decision No. 484/06I,* the Tribunal accepted November 6, 1992, as the "date of injury/accident" (i.e., the date the worker was suffering from an impairment from symptoms related to the later-diagnosed COPD). The Board implemented the Tribunal decision by paying the worker full temporary disability benefits from July 1994 (when the worker retired) until June 1997 (when the worker was 65 years old).

[4] The Board also paid the worker non-economic loss ("NEL") compensation. It rated the degree of the worker's permanent respiratory impairment from COPD at 45%. It then applied a formula from the Board's 2001 *Adjudicative Support Material Binder I: Chronic Obstructive Pulmonary Disease (COPD)* to determine how much of the worker's permanent respiratory impairment resulted from smoking and how much of the impairment resulted from his work exposure. Applying that formula, the Board decided that 28% (of the 45% whole person impairment) resulted from the worker's work exposure, so he was entitled to a 13% NEL award.

[5] The worker appealed the Board's decision to "apportion" his permanent impairment from COPD, and to pay him NEL compensation for only the work-related portion of that impairment.

[6] The *Decision No. 484/06I* Panel decided to obtain additional information about the formula the Board applied, and additional medical information about COPD, as well as submissions on this additional material before deciding the NEL apportionment issue. Those post-hearing proceedings were completed in October 2008. In December 2008, Panel Member J. Robb died. Panel Member B. Wheeler was subsequently assigned to this appeal. The newly-constituted Panel considered the appeal on June 10, 2009.

(ii) The issue

[7] Is the worker entitled to NEL compensation for his total respiratory impairment from COPD (i.e., 45%)? Or was the Board correct when it apportioned that total impairment from COPD into a work-related percentage and a percentage related to smoking, and paid the worker NEL compensation for only the work-related percentage (i.e., 28% of 45% = 13%)?

(iii) The decision

[8] Because NEL compensation is only payable for permanent impairment that "results from" the work injury, the Board was correct when it paid the worker NEL compensation for only the portion of his permanent impairment from COPD that resulted from his work injury.

[9] We accept the evidence that the effects of dust and of cigarette smoking on chronic airflow obstruction ("COPD") are separate and additive. Thus, part of the worker's permanent impairment from COPD results from a work injury; part results from a non-work injury. Based on the best available medical and scientific evidence, we find that the extent to which the work injury and the non-work injury each independently contributed to the worker's global permanent impairment from COPD is measurable. The Board's COPD Binder formula provides a reasonable basis for measuring the relative contribution of dust and smoking to the worker's permanent impairment from COPD. On the facts of this worker's case, the rating of 28% (13% of the whole person) is a fair and reasonable estimate of the degree of impairment from COPD that resulted from his work injury.

(iv) How the Board calculated the worker's permanent respiratory impairment

(a) The worker's NEL assessment

[10] In 2001, Board Respirology Consultant Dr. Muir reviewed the worker's pulmonary function tests ("PFT"s) from August 23, 1999. Dr. Muir noted that the PFTs showed an FEV_1 (Forced Expiratory Flow Volume[1]) that was 44% of the value predicted (given the worker's height and age), and his FVC (Forced Vital Capacity[2]) was 103% of the value predicted. Dr. Muir concluded that the worker's PFTs provided "firm evidence of COPD."[3] The Board referred the worker to respirologist Dr. J. Roos for a NEL assessment.

[11] When he performed the NEL assessment, respirologist Dr. Roos recorded the worker's description of his symptoms, the physical findings on examination, and the spirometry test results from August 1999. Dr. Roos performed some in-office spirometry and he reviewed the worker's chest x-rays. He noted findings of "a flat hemidiaphragm consistent with emphysema but no acute lung lesion" as well as pleural plaques (the Board had determined the pleural plaques were not a source of impairment). He also noted the absence of x-ray changes consistent with asbestosis. Dr. Roos concluded:

> …This worker has severe obstructive ventilatory disease with some reversibility and with a low normal single breath diffusing capacity…The changes are consistent with COPD but not with changes caused by inhalation of asbestos dust…

[12] Thus, Dr. Roos concluded that the worker did not have asbestosis (i.e., because he did not have the radiological changes consistent with asbestosis, or the PFT findings such as reduced FVC that would be consistent with asbestosis). However, Dr. Roos did conclude that the x-ray findings (of changes consistent with emphysema) were consistent with COPD, as were the PFT findings (including FEV_1 measurements indicative of obstructive impairment).

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[1] FEV_1 (Forced Expiratory Flow Volume) is a measure of pulmonary function obtained using a spirometer. It is the most useful measurement for following *obstructive* pulmonary disease. It is obtained as follows: "The patient breathes in as deeply as possible and then blows out as quickly as possible into a chamber. The amount of air breathed out in 1 second (FEV_1) then gives a measurement of how relatively narrow the airways are; because the value obtained is compared with normal values from people of the same age, sex, and height." 2001 Board *Adjudicative Support Material Binder Series. Binder I: Chronic Obstructive Pulmonary Disease (COPD).* COPD General Disease Characteristics pages 9 and 10.

[2] FVC (Forced Vital Capacity) is the "maximal volume of air exhaled using maximal effort following maximal inspiration." It is the most useful measurement for diagnosing *restrictive* pulmonary diseases. Id. at page 10.

[3] When the FEV_1/FVC ratio is <70% in middle-aged adults, this indicates an obstructive disorder. Id. at page 10.

[13] In response to a question on the NEL Summary Report about whether there were medical conditions other than the work injury which impact on the extent of the worker's present work-related impairment, Dr. Roos wrote:

> Yes. Non-work-related 75% of COPD.

[14] Dr. Roos' response that 75% of the worker's COPD was non-work-related is consistent with the information the Board provided when it referred the worker for his NEL referral. The referral memo indicated that the NEL award would be adjusted for smoking and that "only 25% of the P.I. level is considered work-related."

(b) The NEL rating of the worker's permanent impairment from COPD

[15] Because *Decision No. 484/06I* decided that the "date of injury" was November 6, 1992, the pre-1997 *Workers' Compensation Act* (referred to in this decision as "the pre-1997 Act") applies in deciding this appeal[4].

[16] Section 42 of the pre-1997 Act describes when a worker will be entitled to compensation for non-economic loss (NEL), in addition to any other benefit receivable under the pre-1997 Act. It also describes how the amount of that NEL compensation will be calculated. Among other things, it states that the degree of a worker's permanent impairment will be determined "in accordance with the prescribed rating schedule." That rating schedule is the American Medical Association *Guides to the Evaluation of Permanent Impairment* (third edition revised) (the "AMA *Guides*")[5].

[17] After Dr. Roos' NEL assessment of the worker, a Board NEL Adjudicator applied the AMA *Guides* as follows:

Respiratory Disorder	***Impairment***	
Description of Impairment	**%**	**Reference**
Lower Respiratory Impairment: Class 3 – Moderate Impairment – PFT Date: 24 Aug 1999; FVC=103% FEV_1 = 44%; (FEV_1 /FVC) = 43%; D_{co} = 77%	45	P[age] 125, T[able] 8

> …
>
> Whole Person Impairment: Whole person 45%
>
> …
>
> Summary of NEL Benefit for this Claim:
>
> Rated at 45%. 25% (sic) to be attributed to workplace. 25% x 45% = 11.3% [changed by the ARO to 28% x 45% = 13%].

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[4] Section 102 of the *Workplace Safety and Insurance Act, 1997* (the "WSIA") states that the pre-1997 Act, as it is deemed to have been amended by the WSIA, continues to apply with respect to pre-1998 injuries.

[5] Section 15 of Ontario Regulation 1102. Now section 18 of Ontario Regulation 175/98.

[18] The NEL Adjudicator's reference to page 125, Table 8, is a reference to part of Chapter 5 of the AMA *Guides.* Chapter 5 is entitled "The Respiratory System" and it describes how a respiratory impairment will be evaluated under the AMA *Guides.*

[19] Table 8 provides the following criteria for establishing a rating for permanent respiratory impairment:

Table 8 - Classes of Respiratory Impairment

	Class 1 0% No impairment of the Whole Person	**Class 2** 10-25% Mild Impairment of the Whole Person	**Class 3** 30-45% Moderate Impairment of the Whole Person	**Class 4** 50-100% Severe Impairment of the Whole Person
FVC FEV_1 FEV_1/FVC (as percent) D_{co}	FVC ≥ 80% of predicted, *and* FEV_1≥ 80% of predicted, *and* FEV_1/FVC≥ 70% *and* D_{co} ≥ 80% of predicted	FVC between 60% and 79% of predicted, *or* FEV_1, between 60% and 79% of predicted, *or* FEV_1/FVC between 60% and 69%, *or* D_{co} between 60% and 79% of predicted.	FVC between 51% and 59% of predicted, *or* FEV_1, between 41% and 59% of predicted, *or* FEV_1/FVC between 41% and 59%, *or* D_{co} between 41% and 59% of predicted	FVC ≤ 50% of predicted, *or* FEV_1≤ 40% of predicted, *or* FEV_1/FVC≤ 40% *or* D_{co} ≤40% of predicted
	or	**or**	**or**	**or**
VO_2 Max	>25ml/(kg · min)	Between 20 and 25 ml/(kg · min)	Between 15 and 20 ml/(kg · min)	< 15 ml/(kg · min)

FVC is Forced Vital Capacity, FEV_1 is Forced Expiratory Volume in the first second, D_{co} is diffusing capacity of carbon monoxide. The D_{co} is primarily of value for persons with restrictive lung disease. In Classes 2 and 3, if the FVC, FEV_1, and FEV_1/FVC ratio are normal and the D_{co} is between 41% and 79%, then an exercise test is required.

VO_2 Max, or measured exercise capacity, is useful in assessing whether a person's complaint of dyspnea (see Table 1) is a result of respiratory or other conditions. A person's cardiac and conditioning status must be considered in performing the test and in interpreting the results.

[20] In this case, the NEL Adjudicator applied Table 8 by using the low FEV_1 and FEV_1/FVC findings (of 44% and 43% respectively of the predicted FEV_1 and FEV_1/FVC values) from the worker's August 1999 PFTs to rate the worker's respiratory impairment at the high end of a Class 3 impairment of the whole person. Those FEV_1 and FEV_1/FVC values were obtained before the worker was given a bronchodilator and re-tested (even though the worker's "significant response" to the bronchodilator may have suggested that some of the worker's impairment resulted from the asthma that the Board determined was non-compensable).

(c) Calculation of the percentage of the impairment related to smoking

[21] The Board originally calculated the work-related portion of the worker's respiratory impairment at 25%. However, the Board Appeals Resolution Officer recalculated this (using the same formula) and determined that 28% of the worker's impairment from COPD was work-related.

[22] Thus, the worker's NEL award was calculated as follows:

- Smoking history: 75 pack-years
- Work exposure: 42 years

Formula for calculating the percentage of workplace dust contribution[6]:

$$\frac{5.8 \text{ mL x no. of [workplace] dust-years x 100}}{8.5 \text{ mL x no. of pack-years [smoking]} + 5.8 \text{ mL x no. of [workplace] dust-years}}$$

$$= \frac{5.8 \times 42 \times 100}{8.5 \times 75 + 5.8 \times 42} = 27.6\% \text{ (rounded up to 28\%)}$$

Summary of NEL benefit: Rated at 45%

28% to be attributed to the workplace

28% x 45% = 13%

[23] Thus, the Board Appeals Resolution Officer decided that the worker was entitled to a NEL award of 13% (for total NEL compensation of about $3,700).

(d) The Board's formula for apportioning impairment from COPD

[24] The Board's formula for apportioning respiratory impairment from COPD is set out in the Board's 2001 *Adjudicative Support Material Binder Series. Binder 1: Chronic Obstructive Pulmonary Disease (COPD)*[7] (referred to in this decision as "the Board's COPD Binder"). The first part of this Binder consists of a medical description of "COPD: General Disease Characteristics" and a Scientific Review that reviews epidemiological studies that have examined whether a statistical association exists between exposure to various types of occupational dust and the incidence of diagnoses associated with chronic airflow obstruction (i.e., emphysema, chronic bronchitis, COPD) or PFT results consistent with reductions in lung function associated with chronic airflow obstruction (i.e., reductions in FEV_1); the second part of this Binder provides "Adjudicative Advice."

[25] According to the information in the Board's COPD Binder, COPD is a "group of diseases" characterized by certain symptoms. It can include any combination of the following:

- Chronic bronchitis

  Pulmonary emphysema

  Asthma, and

  Small airways disease (bronchiolitis) [8]

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[6] 2001 Board *Adjudicative Support Material Binder Series. Binder 1: Chronic Obstructive Pulmonary Disease (COPD).* Page 30 of the Adjudicative Advice portion of the Binder.

[7] April 2001, WSIB Medical and Occupational Disease Policy Branch, Policy and Research Division.

[8] The Board's COPD Binder, Adjudicative Advice chapter pg. 24.

[26] There is often an overlap between these conditions – "but as long as the dominant clinical feature is the limitation of expiratory airflow (obstruction), a diagnosis of COPD can apply."[9] Because COPD is diagnosed only when the chronic airway limitation is "obstruction," it is diagnosed only when the dominant feature is the limitation of airflow on breathing out (expiration).[10] Spirometry (included in PFTs) is used to measure airflow. The Board's COPD Binder defines some of the spirometry measures used in the AMA *Guides* Table 8, and it describes the spirometric measures used to determine whether an individual has an obstructive pulmonary disorder. It states:

> **Spirometry:**
>
> …
>
> FVC, FEV_1 and FEV_1/FVC ratio are the basic measurements required to interpret spirometry results. The measurement of FVC and FEV_1 are essential to diagnose COPD, as well as to assess its severity, progression and prognosis. The FEV_1/FVC ratio is reduced with obstruction. The FVC is reduced with restriction and the FEV_1/FVC ratio is preserved. COPD is confirmed if FEV_1 is reduced.[11]

[27] Thus, the most useful measure for confirming and following obstructive pulmonary disease is the FEV_1 measurement.[12]

[28] According to the "General Disease Characteristics" part of the Board's COPD Binder,[13] the "normal range" for FEV_1 and FVC values is 80% to 120% of the normal values for people of the same age, sex, and height. Accordingly, if a worker has a FEV_1 of 80% or more, he/she does not have a rateable impairment from COPD.[14]

[29] The introduction to the Adjudicative Advice chapter of the Board's COPD Binder explains that it is designed to give adjudicators a general understanding of COPD and to summarize the important features of the scientific review chapter, but it is not Board policy.

[30] The Adjudicative Advice chapter states the following with respect to permanent impairment rating for COPD:

> **Threshold for permanent impairment:** Currently the WSIB uses the AMA *Guides For the Evaluation of Permanent Impairment* 3rd Edition (revised) as the rating schedule for respiratory impairment. For COPD, the threshold for a rateable respiratory impairment is FEV_1 <80% of predicted values for gender, age and height.
>
> **Note:** For respiratory conditions in general, there is no functional impairment if the measured values for FVC and FEV_1 are >80%.

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[9] Id, pg. 26.

[10] The Board's COPD Binder, General Disease Characteristics page 8.

[11] The Board's COPD Binder, COPD General Disease Characteristics page 9-11.

[12] The Board's COPD Binder, COPD General Disease Characteristics page 10.

[13] Page 10.

[14] The *AMA Guides* Table 8 and the Board's COPD Binder, Adjudicative Advice pg. 29.

> **Proportional impairment rating**: Consider paying a portion of the worker's permanent impairment rating if the work-related component is measurable and distinct from the non-work-related component:
>
> - In COPD, measure the work-related impairment by applying the observed rate of loss of lung function found in the epidemiological studies for dust exposure compared with the rate found for cigarette smoking
> - Determine the relative portion attributed to each factor by:
>   - The number of years exposed to respirable dust, and
>   - The number of pack-years smoked.
>
> See the table on page 31 below.
>
> …
>
> **Impairment ratios:** Loss of expiratory flow lung function (FEV_1):
>
> - For dust, the estimated average loss is 5.8 mL per year of exposure
> - For cigarette smoking, the estimated average loss is 8.5 mL per pack-year.
>
> The table's percentage contribution values represent the relative contributions of smoking and dust to overall impairment. Reference for relative rates of lung function loss derived from the literature is found in the Scientific Review chapter.
>
> …
>
> For cases that fall outside the table, the formula for calculating the percentage dust contribution is:
>
> $$\frac{5.8 \text{ mL x no. of dust-years x } 100}{8.5 \text{ mL x no. of pack-years} + 5.8 \text{ mL x no. of dust-years}}$$

[31] Thus, the Board's COPD Binder notes that the WSIB uses the AMA *Guides* as the rating schedule for respiratory impairment. The AMA *Guides* chapter about the evaluation of respiratory impairment includes a reference to apportionment. In describing the "personal and medical history" that the examining doctor should obtain when evaluating impairment due to respiratory disease, it indicates that the examiner should obtain a detailed employment history (with specific information about exposures to dusts, gases, vapours and fumes), and the smoking history of the individual should be obtained. The AMA *Guides* chapter recommends that "the cumulative dose of exposure should be estimated in terms of 'pack-years,' which is the product of the usual number of packs of cigarettes the patient smoked per day multiplied by the total number of years the individual was a smoker." It states that "Data on environmental exposures and use of tobacco are especially important when the examining physician is asked to give an opinion on apportionment between causes of a lung disorder."[15]

[32] As indicated in the above-quoted excerpt on "proportional impairment rating," the Board's COPD Binder recommends that, where a worker has a permanent impairment rating for COPD, adjudicators consider paying the work-related portion of that permanent impairment if the work-related portion is "measurable and distinct from" the non-work-related component. It describes how to measure the work-related component of COPD. The COPD Binder formula does this by using the observed rate of loss of lung function found in the epidemiological studies for dust exposure (of 5.8 mL per year) multiplied by the number of years the particular worker

[15] The *AMA Guides*, Chapter 5: Respiratory Impairment, pg. 116.

was exposed to respirable dust at work, compared with the observed rate of loss of lung function found in the epidemiological studies for cigarette smoking (of 8.5 mL per pack-year) multiplied by the number of pack-years the particular worker smoked. This formula is intended to determine the relative contribution of smoking and of occupational dust exposure to the overall impairment from COPD, and thereby allow an adjudicator to pay for only the work-related part of the impairment from COPD.

(e) The MODP Scientific Review – the Basis for the Formula

[33] After the first day of hearing, the Tribunal Panel asked the Board to provide it with more information about the Board's COPD Binder formula for measuring the work-related component of a COPD impairment. In response, in addition to referring to part of the Board's COPD binder that provided some background on the Table, the Board sent the Tribunal a copy of the December 2000 document entitled *Chronic Obstructive Lung Disease and Dust Exposures.* It described this as "the background work for the smoking adjustment, by Reimar Gaertner, Senior Scientist [with the Board's Medical and Occupational Disease Policy Branch]" (referred to in this decision as "the MODP Scientific Review").

[34] The MODP Scientific Review noted that the effect of smoking is a much more significant cause of COPD in the general population than dust exposures – but that "in workers exposed to high levels of dust, occupational exposures have been shown in some studies to be as important as concomitant smoking in the development of COLD." [16]

[35] The MODP Scientific Review described how the Board derived the figures used in the formula it uses to decide what portion of a COPD permanent impairment results from workplace dust exposure and what portion results from smoking (i.e., using a 5.8 mL average loss of FEV_1 per year related to dust exposure, and a 8.5 mL average loss of FEV_1 per pack-year of cigarette smoking). To estimate those rates of loss, the MODP Scientific Review examined epidemiological studies of more than 30 populations of dust-exposed workers. It looked at the level of respirable dust to which the studied populations (of "dust-exposed workers") were exposed, and the loss of FEV_1 after one year and twenty years of exposure, as well as the loss of FEV_1 after 20 pack-years smoking.

[36] After reviewing the studies about loss of FEV_1 among dust-exposed workers (including hard-rock mining/quartz, coal mining, and asbestos workers), the MODP Scientific Review averaged the findings in those studies to calculate the following "overall grand total weighted averages":

Loss of FEV_1 after 20 years [dust] exposure, in mL:	116 (5.8 mL/year)
Loss of FEV_1 after 20 pack-years smoking, in mL:	170 (8.5 mL/pack-years)
(1.5 x dust)	

[37] It acknowledged that these overall averages for the loss of FEV_1 from smoking and the loss of FEV_1 from respirable dust exposure are not precise values – that there were wide variations in the results reported in the epidemiological studies reviewed, and the results were

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[16] The terms "COPD" (for "chronic obstructive pulmonary disease") and "COLD" (for "chronic obstructive lung disease") are used interchangeably in this decision.

derived from different types of dust exposures (and that different types of dusts might have somewhat differing effects on the lung). It also noted that some workers may be more susceptible to developing COPD – that most smokers do not develop COLD, but a minority are severely affected - and that some studies suggest a similar pattern of susceptibility with dust effects. The MODP Scientific Review concluded:

> …The majority of studies indicate that the effects of dust and tobacco smoke on COLD are additive. This means that, for example, a 120 mL FEV_1 loss due to dust and a 170 mL loss due to smoking will result in a total loss of about 290 mL for these two factors combined.
>
> …the results in Table 1 [the table setting out the quantitative data on the "Magnitude of the effect of dust, smoke, and age on FEV_1" in the listed epidemiological studies of dust-exposed workers] suggest that occupational exposure to dusty conditions over 20 years will result in an average FEV_1 loss of about 120 mL. By itself, such average exposures are unlikely to result in a significant permanent impairment. Due to the large reserve capacity of the lungs, the airflow limitation experienced by most workers after prolonged dust exposure is small and lung function is still within a normal range. Therefore, dust exposure by itself usually does not result in a rateable permanent impairment. But the combined effect of age, smoking, air pollution, occupational exposures and other factors could produce clinically significant impairment. Even when dust produces COLD with other gas or tobacco smoke exposures, or when the individual is otherwise predisposed to be more sensitive, the COLD disability may be said to be work-related if dust exposures contributed in a substantial way to the final impairment.
>
> …the data summarized in Table 1 suggests a method of determining the relative effects of cumulative dust exposure and smoking on lung function… Table 1 indicates that 20 years of dusty work would on average result in about a 116 mL loss of FEV_1. This is about 41% of the total loss in a 20 pack-year smoker, considering only these two factors. The relative contribution of dust to the loss of FEV_1 would decrease as the cumulative smoking exposure increases. For example, the effect of 20 years of work (giving rise to a theoretical 120 mL loss) would be relatively small compared to a 30 year, two-pack-a day smoking habit (60 pack-years at 8.5 mL/pack-year is a theoretical loss 510 mL FEV_1). In this case work would have contributed to about 19% of the FEV_1 loss, based on these two factors alone. In general, the number of years in dust is multiplied by 5.8 to get the loss due to dust, and the pack-years are multiplied by 8.5 to get the loss due to smoking. The relative dust contribution is then given by calculating the following equation: dust loss/(dust loss plus smoking loss).
>
> The above calculations assume that only two factors are relevant for the development of COLD – occupational dust exposure and tobacco smoke, although other environmental and personal factors are also likely to be relevant. The presence of other relevant factors in the development of COLD would tend to further reduce the percent contribution due to occupational dust exposures. This calculation merely indicates the relative contribution of the two factors, smoking and dust exposure, assuming that the worker will respond to tobacco smoke and dust in a way that is similar to the average response found in several working populations…

[38] The MODP Scientific Review also reviewed the epidemiological studies to estimate the threshold level of dust exposure that can cause COPD. On the basis of that review, it estimated that an exposure of about 2 mg/m^3 respirable dust for 27 years can normally be expected to double the risk of COPD (measured as FEV_1 <80%), but very dusty exposure to respirable dust would reduce the required duration of exposure (i.e., to only about 11 years for exposure to 5 mg/m^3 respirable dust); and exposure to lower respiratory dust levels would require a longer period of exposure (i.e., exposure to 1 mg/m^3 would require an exposure duration of more than 50 years).

[39] Thus, the MODP Scientific Review derived the formula for apportioning COPD permanent impairment by taking data from epidemiological studies of more than 30 populations of dust-exposed workers and using that data to estimate the relative contribution of dust-exposure and smoking to the overall reduction in FEV_1 among dust-exposed workers. It concluded that there was sufficient information to estimate the proportionate effect of dust and smoking on the permanent impairment due to COLD – and it used that information to develop the following formula:

$$\frac{5.8 \text{ mL x no. of dust-years x } 100}{8.5 \text{ mL x no. of pack-years} + 5.8 \text{ mL x no. of dust-years}}$$

[40] This formula was used to construct the Table that appears in the Board's COPD Binder.[17] It was the formula used by the Board to determine what percentage of this worker's global impairment from COPD was work related.

(v) Background medical evidence

[41] The file materials in this case included papers written before the Board's 2001 COPD Binder – during a time when there was considerable controversy in the medical community about whether there was any relationship between COPD and dust exposure. That controversy is reflected to some extent in this case by the fact that the earlier reports from the worker's treating doctors, and respirologist Dr. Roos, attributed the worker's emphysema and resulting shortness of breath to smoking (rather than to a combination of smoking and dust exposure).

[42] However, before the Board's 2001 COPD Binder was published, several papers prepared for the Tribunal by Drs. Holness and Muir described how evidence from epidemiological studies led them to conclude that occupational exposure to respirable dust – if heavy enough and for a prolonged enough period of time – could have an effect that was similar to smoking in the development of emphysema/COPD.

[43] In 1990, respirologist Dr. D. Linn Holness wrote that the "question of whether occupational exposure to dusts in a general sense, or specific types of dust can cause chronic obstructive pulmonary disease is controversial and has produced much debate within the medical community."[18] She acknowledged that the medical literature did not provide studies that provided clear-cut answers to all of the issues arising in addressing this question, but, after reviewing a number of epidemiological studies that then existed, she concluded that there was enough evidence to suggest a relationship between certain occupational dust exposures and COPD.

[44] In 1993, respirologist Dr. David C.F. Muir replied to a number of questions that the Tribunal asked him about COPD.[19] Dr. Muir indicated that the general medical view about whether there was a relationship between COPD and dust exposure had been a "bitterly disputed subject" and he referred to a paper he had co-authored that reviewed epidemiological studies

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[17] At page 31 of the Adjudicative Advice section. The Table uses years of respirable dust exposure @ 2 mg/m^3.

[18] Holness, Dr. D. Linn, *Chronic Obstructive Lung Disease (COPD).* 1990 Medical Discussion Paper prepared for the Tribunal.

[19] Id.

addressing that issue. That paper concluded that there was "good evidence that exposure to occupational dust can cause chronic bronchitis and clinically important losses of lung function in both smokers and nonsmokers, and that it can be a cause of marked COPD in smokers."

[45] Dr. Muir also answered specific questions about COPD that were posed by the Tribunal. He indicated that the overwhelming cause of COPD is cigarette smoking but that respirable dust exposure, if heavy enough and for a prolonged period of time, can have the same effect. He indicated that there is a dose/response relationship between COPD and both smoking and dust exposure and that there is probably no interaction between the effect of cigarette smoking and the effect of dust exposure. He wrote that the type of COPD caused by dust exposure cannot be distinguished from that caused by cigarette smoking, but he also indicated that (from a medical/scientific point of view) it is possible to apportion the degree of severity of COPD and exposure to dust or cigarette smoking – that, "what is required is an estimate of duration and concentration of dust exposure on the one hand and, if possible, the duration and intensity of smoking on the other."

[46] The file material also included a Discussion Paper that Dr. Muir prepared for the Tribunal in 1999. It provided his Overview of Current Opinion on the issue of Industrial Dust Exposure and Chronic Obstructive Airways Disease.

[47] In his 1999 Discussion Paper, Dr. Muir wrote that "the major 'event' that has dominated the legal and scientific dispute on the relationship of dust exposure to chronic obstructive airways disease has been the massive litigation in the U.K. between British Coal and a selected number of underground miners." In that case, the court investigated whether the respiratory illnesses of emphysema, chronic bronchitis, asthma, or small airways disease were caused by exposure to coal mine dust and, if so, whether such exposure was caused by actionable fault on the part of British Coal Corporation. After reaching findings on liability and medical issues, the court applied those findings to the facts of eight individual cases (the "lead plaintiffs").

[48] In his Discussion Paper, Dr. Muir noted that both sides in this litigation employed well known physicians and epidemiologists – and much of the litigation focused on the interpretation of epidemiological data derived by the Coal industry. Dr. Muir noted that the court in the British Coal case[20] reached certain findings on the generic medical issue of whether the small average decrements in FEV_1 of coal miners with heavy dust exposure were likely to cause significant disability, and he set out the court's findings as follows:

1. Coal mine dust (coal and stone) is a cause of *centriacinar emphysema*;
2. Such *emphysema* may, and usually does, lead to loss of ventilatory capacity most easily demonstrated by loss of FEV_1;
3. Confirmation that the causes and effects of tobacco smoke are as in findings 1 and 2 above;
4. It is probable, but not certain, that there is a common causal pathway to both cigarette and mine dust induced *emphysema* which usually gives rise to breathlessness;

[20] Re: the British Coal Respiratory Disease Litigation: *Griffith and Others v. British Coal Corporation*, January 23, 1998 judgement of The Honourable Mr. Justice Turner, High Court of Justice, Queen's Bench Division. Tribunal Library location JUR 1211.

> 5. Whether 4 is established or not, the effects are generally the same in that there is a spectrum of effect which in the majority is not clinically detectable but in the minority does produce a range of effects from simple impairment, frank disability and occasionally death.
>
> 6. In the individual smoker it is not possible to attribute the cause of breathlessness either to the one insult or the other, this is so whether or not there is a common pathway.[21]

[49] The British Coal decision granted damages to individuals who were susceptible to the effects of tobacco smoke/dust who were exposed to tortious dust to a material extent and who suffered from breathlessness where the underlying pathology was emphysema. However, the court reduced the amount of damages that would otherwise have been payable to the coal dust exposed plaintiffs by an amount that reflected the effects of smoking. To estimate the relative effects in any individual miner of smoking and of dust exposure, the court had regard to matrices and data (derived from epidemiological studies) that estimated the effect on FEV_1 of various levels of dust exposure among workers with varying smoking histories.[22] Dr. Muir commented on the result in this case as follows:

> …The court recognized that dust exposure and cigarette smoking had similar effects and that, in the individual it was possible to assign the proportion of causality according to the level of dust exposure and cigarette smoking. Compensation was then awarded on a proportional basis. This approach emphasizes the point that it is not valid to suggest that, in a dust exposed cigarette smoker, the disability can be treated as if it was due entirely to one cause or the other. It makes no scientific sense to ask a physician which is "the most likely cause of the disability" or whether one or the other made a "significant contribution." The appropriate scientific method is to estimate, as far as possible, the contribution by each cause.

[50] In his 1999 Discussion Paper, Dr. Muir proposed a table which (like the Table in the COPD Binder and the matrix presented in the British Coal litigation), estimates percentages of lung function abnormality attributable to dust exposure (with varying concentrations and varying years of exposure) in a cigarette smoker.

(vi) The Panel's questions to a Tribunal Medical Assessor

[51] After hearing submissions from the worker's then-representative (Mr. Hudyman) and Tribunal Counsel (C. Zimmermann), the Panel decided that, in determining the issue of whether the worker's NEL compensation should be reduced to reflect the portion of his respiratory impairment that resulted from cigarette smoking, it would be necessary to address the question of whether impairment from COPD is an impairment that results from one indivisible injury (with both compensable and non-compensable causes) or whether it is an impairment from two injuries (one of which is smoking; the other of which is workplace dust exposure) and, if it is from two injuries, whether each injury made a separate and measurable contribution to the impairment.

[52] In order to address these questions, the Panel asked for a Medical Assessor's opinion. The Tribunal's Medical Liaison Office ("MLO") referred the Panel's questions to Dr. Robert Neville Rivington. Dr. Rivington is a respirologist who has held both academic and hospital positions, including positions as an Associate Professor of Medicine at the University of Ottawa,

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[21] Id. Summary at page 30.

[22] Id. Summary at pg. 32.

and past Chief in the Division of Respirology at the Ottawa Civic Hospital. He has been a member of a number of professional associations, particularly in the field of thoracic medicine.

[53] The Panel asked Dr. Rivington to review the above-noted papers written in 1999 and 1993 by Dr. Muir and to provide an opinion about whether they are consistent with current studies/medical opinion about COPD. Dr. Rivington responded as follows:

> These papers are generally consistent with current opinion. An editorial and accompanying article in a recent American Journal of Respiratory and Critical Care Medicine[23] confirm a distinct but minor role of industrial exposures on the progression of COPD, especially fume exposures. Despite these findings, the editorial that accompanies the article suggests that such workers should usually be permitted to remain working.
>
> I believe the information in the articles by Dr. Muir can be considered accurate.

[54] The Panel asked Dr. Rivington the following question about the nature of the diagnosis of COPD:

> **Q: The diagnosis of Chronic Obstructive Pulmonary Disease ("COPD")**
>
> Is COPD a diagnosis of "impairment" or is it a diagnosis that requires identification of the particular pathological disease process that is causing the reduction in breathing capacity? Can COPD result from more than one disease/pathological mechanism?...
>
> If COPD is diagnosed on the basis of impairment (i.e., symptoms and spirometry), is it the case that a worker can have chronic obstructive airways impairment, but that impairment is not sufficient to be diagnosed as COPD? If so, what spirometry (and/or other) findings are necessary to diagnose COPD?

[55] Dr. Rivington responded as follows:

> COPD is an old term used to encompass diseases including chronic bronchitis and emphysema predominantly. Chronic asthmatic bronchitis likely can be included in this term. More recently the term CAO (chronic airflow obstruction) has been used as the common abnormality documented in these diseases is impaired expiratory airflow.
>
> COPD can be asymptomatic for years. Emphysema, when predominant, leads predominantly to dyspnea as seen in this worker. Chronic bronchitis, by definition, features chronic cough and mucous production most days for at least three consecutive months for two consecutive years... Chronic asthma can be included as COPD...
>
> Confirmation of COPD does require spirometry but knowledge of the natural history would make an earlier diagnosis strongly suspected...

[56] The Panel also asked Dr. Rivington about the possibility of differentiating between COPD from smoking and that from work exposures. It asked:

> **Q: Differentiation between COPD from smoking and COPD from work exposures**
>
> Is it possible to differentiate between lung damage or COPD caused by cigarette smoking and that caused by dust or fumes exposure? Do these produce different patterns or types of changes in the lungs? Please explain.
>
> Would the onset or course of COPD differ according to the cause?

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[23] Toren K, Barnes J. Chronic obstructive pulmonary disease: does occupation matter? Am J Respir Crit Care Med 2007; 176: 951-953; and Harber P, Tashkin DP, Simmons M, et al. Effect of occupational exposures on decline of lung function in early chronic obstructive pulmonary disease. Am J Respir Crit Care Med 2007; 176: 994-1000.

> Would the type of ventilatory impairment differ between COPD caused by smoking and that from other causes? Please explain.

[57] Dr. Rivington responded that differentiation between COPD from smoking and COPD from work exposure can be "challenging" and he then examined this question in light of the facts arising in this worker's case. He noted that the Board had found this worker's asthma to be non-compensable, and that it was unlikely (by the fact that the worker denied cough and mucous) that the worker had bronchitis. Thus, on the basis of this worker's history, Dr. Rivington concluded:

> As bronchitis is unlikely due to the recorded history, the (probable) emphysema caused by dust/fumes and cigarette smoke likely share the same pathophysiology. The course of emphysema would be accelerated in susceptible smokers who are exposed to dust and fumes.

[58] With respect to other possible causes of chronic airflow obstruction and other possible types of impairment associated with such airflow obstruction, Dr. Rivington wrote:

> Some patients who smoke develop chronic bronchitis. These patients have recurrent infection, abnormal blood gases and can develop pulmonary cardiac changes such as cor pulmonale and even ultimately right heart failure. The symptoms and impairments noted seem to be related to the noxious stimuli exposures. Emphysema seems to be more common in chronic bronchitis. Differentiation of the two is very difficult and they do produce the same patterns of abnormality.

[59] The Panel asked Dr. Rivington about how smoking and dust exposure produce obstructive respiratory impairment – whether they contribute to the impairment in an "additive" way or an "interactive" or "multiplicative" manner. The Panel asked:

> Both the Discussion Papers by Dr. Muir, and the Board's COPD Binder identify cigarette smoking as the most important cause of COPD. But they also find that certain workplace dust exposures can also cause COPD. These sources suggest that, in circumstances where a smoker also has workplace dust exposure, both the cigarette smoking and the dust exposure contribute to the respiratory impairment – and that they likely do so in an "additive" rather than an "interactive"manner.[24]
>
> Do you agree? Is the impairment from cigarette smoking and the impairment from workplace dust exposure likely to be additive or multiplicative? In other words, is this worker's COPD the product of impairment resulting from injury from smoking added to impairment from injury from workplace exposures? Or is it the result of impairment from the interaction between both smoking and workplace exposure?

[60] Dr. Rivington responded as follows:

> Except for gold miners in South Africa who seem to have a marked pulmonary impact from the work exposures, the usual industrial exposure to dusts and fumes is thought to be an additive to the adverse pulmonary effects of cigarette smoking.

[61] With respect to the facts of this case (in which the worker had a 75 pack-year smoking history and 42 years of work exposure), the Panel asked Dr. Rivington whether it is possible to estimate the likely level of the worker's chronic obstructive airways impairment from smoking alone (with no workplace exposures), or from workplace exposures alone (with no smoking history). Dr. Rivington responded as follows:

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[24] Dr. Muir's 1993 Addendum to the Tribunal Discussion's Paper on Chronic Obstructive Lung Disease (COPD); The Board COPD Binder (COPD: Adjudicative Advice section) page 6.

> It is difficult to speculate about the relative contribution to the observed severe obstruction. Overall, many susceptible patients with a 75 pack-year history of smoking could have similar spirometric findings. Others with 75 pack-year history may be more minimally impacted. I must say that in almost 30 years of pulmonary experience, I have never encountered this extent of abnormality solely due to workplace exposure. If the exposure in the workplace had led to such findings, it would be more likely due to organic dust or other contaminants leading to an asthmatic component to cause such severity and reversibility...

[62] The Panel also drew Dr. Rivington's attention to the fact that application of the Board's formula meant that 28% of the worker's total impairment from COPD had been attributed to his workplace and 72% had been attributed to his smoking. It asked Dr. Rivington whether, in his opinion, 28% was a reasonable estimate of the impairment arising from the worker's workplace exposure, and whether 72% was a reasonable estimate of the impairment arising from the worker's smoking history. The Panel also asked Dr. Rivington:

> Is it the case that the worker's chronic obstructive airway impairment likely resulted from the cumulative effect of both smoking and workplace exposure – and that impairment was properly diagnosed as COPD only when it reached a certain degree of impairment? Or did the combination of smoking and workplace exposures trigger an "injuring process" that, once started, had its own momentum and followed a normal course for the development of "disease" that was later diagnosed as COPD? Please explain.

[63] Dr. Rivington responded:

> I would feel the current attributions are quite fair to the worker by allowing 28% of the observed changes to be allocated to the workplace exposure. I also believe the cumulative impact of smoking and workplace exposure led to the gradual evolution of COPD that ultimately led to the severity causing symptoms and the diagnosis of this condition. I do not believe that the combination of smoking and workplace exposure triggered an injuring process that developed its own momentum…

(vii) Medical evidence about this worker

[64] Dr. Rivington's opinion that it is difficult to estimate the relative contribution smoking and dust exposure have made to a particular individual's chronic airflow obstruction is consistent with the view expressed in 1999 by Dr. Andersen from the Ontario Health Clinics for Ontario Workers Inc. ("OHCOW"). After conducting an extensive review of the worker's employment and exposure history, his medical history, and his medical records, Dr. Andersen wrote:

> [The worker] has a history of 42 years of exposure to multiple environmental dusts and gases and fumes, including asbestos...
>
> This coupled with his smoking history has probably from both his industrial exposures and his heavy smoking history resulted in his pulmonary emphysema and shortness of breath.
>
> The degree to which each has contributed to his current condition is difficult to estimate. It is safe to say, however, that his industrial exposures have played a significant, inestimatable role in the development of his pulmonary emphysema.

[65] Although Dr. Rivington also expressed the view that it was difficult to speculate about the relative contribution of smoking and of workplace exposure in the individual case, after reviewing the background material that the Board derived from the epidemiological studies, he concluded that the attribution of 28% to the workplace and 72% to smoking was quite fair to this worker. In doing so, he considered not only the nature of the worker's medical condition and the

facts about his employment – but also the nature of the workplace exposure in the worker populations of the epidemiological studies that generated the data that the Board used in developing its formula. The Panel's question about the application of the formula to the worker's case, as well as Dr. Rivington's response is as follows:

> **Q: Do you agree that the formula/Table used by the Board in apportioning the degree of impairment provides a reasonable estimate of the likely relative contribution of smoking and workplace dust exposure in this case? Why/why not?**
>
> **[Dr. Rivington's response]:**
>
> The formula/table setting out the relative contribution of COPD related smoking and impairment from workplace exposure was reviewed. I believe this table is quite fair to this worker who worked above ground in variable conditions. Aside from the asbestos plaques, I could see no other demonstrable asbestos-related disease. He does not have pulmonary asbestosis. He does not have any pleural tumors. The exposure to asbestos has not usually been reported to be associated with emphysema. Other dust could be present in the ambient environment and could be related to welding. This would be less intense than the usual nature of dust exposures that were used to generate the data on the tables. Often the workers were exposed to more continuous and more contaminated environments...

[66] In concluding his first report to the Panel, Dr. Rivington indicated that he would be interested in obtaining the worker's 2004 CT scan in order to determine the extent of morphological emphysema seen on that test. The Tribunal obtained this CT scan. After reviewing it, Dr. Rivington concluded that the CT scan showed only minor evidence of emphysema and that (in light of both the CT scan and the degree of reversibility reported on earlier PFT tests) the observed abnormalities in this worker were more likely related to chronic asthma than emphysema. Although the Board had concluded the worker's asthma was non-compensable (and Dr. Rivington agreed that asbestos exposure is not usually related to worsening of asthma), Dr. Rivington expressed the opinion that the worker's chronic asthma was likely worsened by his smoking history and that a dusty and dirty workplace could worsen a pre-existing asthmatic problem.

(viii) The submissions

[67] After the Panel received a decision that was released shortly after the first day of hearing (*Decision No. 865/92R4*), as well as all the post-hearing evidence, the worker's current representative (from the OWA) filed submissions. The worker's representative asked the Panel to follow *Decision No. 865/92R4* and decisions that have applied it.[25] The OWA asserted that these decisions on COPD apportionment reflect the common law principles confirmed by the Supreme Court of Canada in *Athey v. Leonati* (referred to in this decision as "the *Athey* decision").[26] In particular, the OWA noted that *Decision No. 865/92R4* concluded that "where there are essentially two co-existent factors causing simultaneous and indistinguishable injury to the worker's lungs, attribution is neither possible nor, given the nature of the impairment, fair."

[68] With respect to the facts of this worker's case, the OWA's submissions noted Dr. Rivington's evidence about the difficulty differentiating between the emphysema caused by

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[25] Tribunal *Decision Nos. 361/07, 1886/07, 895/07.*

[26] *Athey v. Leonati,* [1996] 3 S.C.R. 458.

dust and that caused by cigarette smoking as well as his evidence about the cumulative impact of smoking and workplace exposure in the gradual evolution of COPD that ultimately led to the severity of symptoms and the diagnosis of this condition – as well as Dr. Rivington's statement that it is difficult to speculate about the relative contribution to the severe obstruction that was observed in the worker's case. The OWA argued that the worker did not have any pre-existing disability or symptomatic prior disability that would permit the Board to apportion his NEL benefits for COPD, and that the Tribunal Medical Assessor confirmed that COPD caused by smoking is not medically divisible from COPD caused by workplace exposures, so his evidence supports a finding that that the worker's COPD is not "medically divisible."

[69] The OWA also argued that Dr. Rivington's opinion about the "fairness" of the Board's "apportionment table" is not relevant because, where a worker suffers a "single, non-divisible injury, the legal principles dictated by the Supreme Court of Canada support the determination that there is no apportionment between compensable and non-compensable causes."

[70] Tribunal Counsel addressed the "thin skull" argument raised in argument and in *Decision No. 865/92R4.* She noted that the "thin skull" rule arose from tort law. It means that a tortfeasor "must take the victim as the tortfeasor finds the victim" – that the tortfeasor becomes liable for the full extent of the losses sustained by the victim as a result of an injury, even though those losses are, because of the susceptibility of the victim, greater than they would be for the average person. She indicated that the Tribunal applies the thin skull rule – that, a worker who has a pre-existing frailty is entitled to full compensation for disability resulting from a work injury, even though the severity of the work injury (and the resulting disability) is, because of the worker's pre-existing condition, greater than it would be for the average person. Tribunal Counsel noted that the *Athey* decision is an illustration of the application of the thin skull rule in a case where the court found that a worker sustained one indivisible injury (a disc herniation).

[71] However, with respect to the issue of whether a worker's permanent impairment from COPD can be apportioned for NEL purposes, Tribunal Counsel reviewed a number of Tribunal decisions that have done this, including decisions dealing with impairment from COPD. She noted that these decisions distinguish non-compensable *co-existing* conditions (for which there is no general Board policy) from non-compensable *pre-existing* conditions (to which the "thin skull rule" and the Board policy[27] on pre-existing conditions apply). She noted that a number of Tribunal decisions have upheld the practice of apportioning a permanent disability or impairment award where compensable and non-compensable co-existing factors both contribute to the worker's disability in a way that is *identifiable* and *measurable*.[28] She summarized the decisions as follows:

> …the cases seem to distinguish between pre-dispositions, pre-existing disabilities, and co-existing factors. Where a worker is found to have a pre-disposition or susceptibility that did not become manifest until after a workplace accident, then the thin-skull doctrine applies and it would not be appropriate to apportion. Where a worker is found to have a pre-existing symptomatic disability, the amount of that disability is assessed and

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[27] Board *Operational Policy Manual* Document No. 08-01-05 "Second Injury and Enhancement Fund" and 18-05-05 "Effect of Pre-Existing Impairment."

[28] Tribunal *Decision Nos. 66/95, 303/02, 7/96, 1970/99.*

> deducted from the pension or NEL awarded for the compensable disability.[29] Where there are co-existing compensable and non-compensable factors, both of which are found to be contributing to the worker's disability in a way that is *identifiable* and *measurable*, some Tribunal decisions have found it appropriate to apportion between the compensable and non-compensable factors in determining the worker's permanent disability award.[30] Where it is not, however, possible to isolate the portion of the disability related to the workplace factors and the portion related to non-compensable factors, then the cases have held that it is not appropriate to apportion.

[72] In our view, the above is an accurate summary of the Tribunal's decisions.

[73] Tribunal Counsel suggested that Tribunal decisions that have apportioned permanent disability awards for COPD between compensable work exposures and non-compensable smoking[31] have done so because, either explicitly or implicitly, they have found that the compensable and non-compensable factors contributing to the permanent disability are distinct and measurable. These decisions (unlike *Decision No. 865/92R4*), accepted that it is possible to have a "divisible injury" to one body part. She noted that *Decision No. 865/92R4* did not disagree with the principle that the Board had the authority to "apportion" or pay a reduced permanent impairment award to reflect a distinct non-compensable injury, but the *Decision No. 865/92R4* Vice-Chair was not persuaded that the evidence (in the case before him) was sufficient to permit attributing a portion of the responsibility for the worker's COPD to cigarette smoking.

(ix) Tribunal decisions

[74] As indicated by Tribunal Counsel, there are Tribunal decisions in which a PD rating that has been given for the worker's global impairment is "apportioned" into a work-related portion (i.e., the percentage of the impairment that arises from the work injury) and into a non-work-related portion, and the PD award has been paid for only the work-related portion of the impairment. These decisions have been made in cases where the Tribunal concluded that a non-compensable factor/condition/injury was a separate and measurable source of a portion of the global impairment rating.

[75] These have included decisions in which the global impairment affected the same body part. For example, in *Decision No. 66/95*, a Tribunal Panel upheld a Board decision to reduce a PD award for hearing loss by an amount reflecting hearing loss from (non-compensable) Meniere's disease. Likewise, in *Decision No. 681/94*, a Tribunal Panel decided that a worker's permanent impairment from organic brain syndrome resulted from co-existing conditions – one of which was non-compensable (i.e., alcohol abuse) and one of which was compensable (i.e., long-term solvent exposure), and it decided that the worker was entitled to a PD pension for only the work-related portion of the total impairment (i.e., half of the 80% rating).

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[29] Board *Operational Policy Manual* Document No. 08-01-05 "Second Injury and Enhancement Fund" and 18-05-05 "Effect of Pre-Existing Impairment."

[30] There is no specific provision for this in Board policy. The rational for so doing is that the legislation is intended to provide benefits to workers for work-related injuries and disease, not to provide compensation for consequences that are not work-related.

[31] Tribunal *Decision Nos. 7/96, 1970/99, 1006/88.* Supported by obiter in *Decision No. 303/02.*

[76] And, in the case of noise induced hearing loss ("NIHL"), both Board policy[32] and Tribunal decisions have upheld the practice of reducing the PD rating (given for the global impairment from hearing loss) by an amount that is derived from studies on the effects of aging in the general population (rather than on some measurement of the effect of aging derived from the examination of the individual worker). For example, *Decision No. 66/95* referred to *Decision No. 132/90* – a Tribunal decision that explained its decision to apply the presbycusis factor as follows:

> The application of the presbycusis factor would seem to be consistent with the Board's general policy of apportioning entitlement to permanent benefits where a non-compensable condition can be identified and measured. The Board has simply adopted the presbycusis factor as a guideline for measuring the extent of the involvement of a particularly common non-compensable injuring process…
>
> The whole-person approach was never intended to require the Board to compensate identifiable, measurable, non-compensable conditions.

[77] There are also Tribunal decisions that have identified a portion of the global impairment from COPD that is non-compensable (from smoking) and reduced the PD award accordingly (by calculating the PD award on the basis of only the work-related portion of the rating for the global impairment from COPD).

[78] For example, *Decision No. 7/96* decided that the smoking habit of the worker in that appeal caused a condition which developed over many years, and that he was also exposed to fumes in the course of his employment that resulted in further damage. It concluded that smoking and work exposure to fumes were two independent co-existing causes of the worker's COPD. It therefore decided to follow the Board's practice with respect to apportionment, and to pay a PD pension calculated using the percentage of the total PD rating that reflected the work-related contribution.

[79] Likewise, *Decision No. 1970/99* "apportioned" a worker's respiratory impairment into portions attributable to compensable and non-compensable sources of impairment. The Panel in that case rated the worker's overall respiratory impairment at 60%. It then found that three factors (asbestosis, cigarette smoking, and dust exposure) contributed to this overall impairment. The *Decision No. 1970/99* Panel found that two of these factors (asbestos exposure with the resultant asbestosis, and dust exposure associated with obstructive lung disease) were compensable, but the smoking (associated with obstructive lung disease) was not. The Panel concluded that each of these factors was equal in significance. Because this meant that two-thirds of the worker's overall respiratory impairment resulted from compensable factors, the Panel found that the worker was entitled to a 40% NEL award (i.e., two-thirds of the 60% rating given for the overall respiratory impairment).

[80] *Decision No. 303/02* explained why the medical evidence about the nature of COPD raises the "apportionment" issue in cases where a worker smokes and suffers from chronic airways obstruction. *Decision No. 303/02* dealt with the appeal of a worker who had some occupational dust exposure, and who smoked 1 to 1½ packs of cigarettes a day for over 45 years

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[32] The Board's policy considers hearing loss due to aging to be non-compensable hearing loss, and it reduces the total measurable hearing loss by a presbycusis (aging) factor for every year a worker is over the age of 60 at the time of the audiogram. Board *Operational Policy Manual* Document No. 04-03-10.

before he was diagnosed with COPD. The decision reviewed the medical evidence about the nature of COPD – including the Tribunal Discussion Papers written by Drs. Holness and Muir (that expressed the opinion that the effects of smoking and exposure to dust are likely independent and additive factors in the development of impairment from COPD), as well as opinions about the medical nature of COPD from Medical Assessors quoted in other Tribunal decisions.[33] It concluded that smoking and workplace exposures were most appropriately considered to be co-existing factors that contributed concurrently to the worker's condition while he was working (with the smoking continuing to contribute to the condition after he stopped working).

[81] The *Decision No. 303/02* Panel contrasted this situation (of smoking and work exposure being *co-existing* factors that are separate and concurrent causes of impairment) with those situations in which a non-compensable condition *pre-existed* the work injury (and the "thin skull rule" and/or the Board policy[34] on *pre-existing* conditions applied). It also reviewed a number of Tribunal decisions that reduced the amount of compensation payable for permanent disability by an amount representing the contribution made to the disability by a non-compensable condition,[35] and it endorsed the approach used in *Decision No. 1006/88.*[36]

[82] With respect to apportionment, *Decision No. 303/02* stated:

> We consider the significant contributing factor test the appropriate test when there are several different causes that act together to result in a medical condition, and it is not possible to say whether or when the impairment would have developed or would have become symptomatic absent the compensable factor. Dr. Muir's opinions on file suggest to us that these facts may not apply in the impairment recognised in the condition of COLD [COPD], itself. In this case, the evidence suggests that a component of the disability is the result of a non-compensable co-existing factor. The evidence suggests that the contribution of that factor to the condition is irrespective of and separate from the effects of the compensable condition, and that the workplace factors do not contribute at all to that component of the impairment. In that case, in our view, Board policy and the above Tribunal decisions suggest that apportionment of permanent pension benefits between the co-contributing factors may be appropriate. We do not consider it a bar that the measurement of each component may not be exact, if the contribution can be reasonably estimated. Many measurements used in worker's compensation are less than exact, including measurements made under the pension rating schedule and the AMA guidelines…

[83] Because the *Decision 303/02* Panel decided that the level of the worker's COPD impairment was minimal when he stopped working (so he was not entitled to a PD pension regardless of whether the Panel considered the worker's entire impairment from COPD, or only the part of the impairment that resulted from work exposure), the Panel did not make a final decision on the issue of "apportionment."

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[33] Tribunal *Decision Nos. 178/98, 552/97, 1006/88*

[34] Board *Operational Policy Manual* Document No. 08-01-05

[35] *Decision Nos. 7/96, 1271/97, 1126/01, 681/94*

[36] The *Decision No. 1006/88* Panel accepted an opinion from Tribunal assessor Dr. Muir that the worker's COPD was caused in part by smoking and in part by dust exposure, and that the effect of each of these factors was independent and additive. It then adopted the test of "significant contributing factor" to determine whether that compensable portion of the worker's COPD was likely a significant cause of the worker's death from cancer.

[84] The decisions that have apportioned the PD rating into a work-related portion and a non-work-related portion and paid a PD award based on only the work-related portion noted the Board's position that it has no legal authority to provide benefits relating to consequences that are not causally connected to a work injury or disease.[37] And the decisions have emphasized the wording of section 45 of the pre-1989 Act that provides for "compensation…of a sum *proportionate to*" the permanent impairment of earnings capacity "*resulting from*" the compensable injury.[38]

(x) Decision No. 865/92R4

[85] In *Decision No. 865/92,* a Tribunal Panel considered an appeal by a worker who was diagnosed with chronic bronchitis and emphysema in 1965. After the worker was exposed to fumes at work in 1977, the Board granted the worker entitlement to benefits for an aggravation (from the fume exposure) of his pre-existing COPD. The Board granted the worker a 25% PD pension for the 1977 aggravation injury, but it found that the COPD that existed before the 1977 aggravation injury was not compensable. *Decision No. 865/92* concluded that there was very strong evidence that the worker's entire disease (COPD from bronchitis and emphysema, with some asthmatic component) could be attributed to his smoking history – and that the evidence linking the worker's COPD to his work exposure was weak – partly because it was "almost entirely based on relationships extrapolated from scientific studies which are still part of a medical controversy on causation." It therefore upheld the Board's decision that the worker's COPD condition was non-compensable before his 1977 work injury.

[86] In *Decision No. 865/92R2,* a Vice-Chair considered evidence about the concentration of dust to which the worker had been exposed at work before the date of the 1977 fume exposure in light of opinions from Drs. Holness and Muir that evaluated the facts of the worker's case in light of the scientific literature regarding the effect of dust and fume exposure on the development of obstructive lung disease. After noting Dr. Muir's opinion that it would be reasonable to suggest that up to half of the loss of the worker's FEV_1 could be the result of the worker's previously remarkably high dust exposure and that half (possibly more) of the FEV_1 loss could be due to the worker's cigarette smoking, *Decision No. 865/92R2* found that the worker's COPD condition was compensable before the 1977 "aggravation" injury. The Vice-Chair decided that the impairment the worker had in 1965 was likely due in equal measure to compensable exposure in the workplace and to non-compensable cigarette smoking. The Vice-Chair directed the Board to determine the amount of the worker's PD pension for COPD.

[87] The worker's representative subsequently asked the *Decision No. 865/92R2* Vice-Chair to make a determination about the issue of whether the Board could "apportion" the worker's pension before the Vice-Chair decided the issue of the quantum of the PD pension – and the Vice-Chair agreed to do so.

[88] Although the above-noted Tribunal decisions apportioned the PD ratings given for global impairment from COPD and paid PD pensions for only the work-related portion of the impairment, *Decision No. 865/92R4* did not do so – in part, because the Vice-Chair was not

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[37] *Decision No. 66/95* at page 12

[38] *Decision No. 681/94*

persuaded that the evidence established that smoking was a separate (co-existing) injury (with a measurable resulting impairment), and in part because the Vice-Chair considered the unfairness of doing so in terms of the impact that the combined effect of the two exposures had on the worker's earning capacity. With respect to the latter, the Vice-Chair wrote:

> …As I understand it…had the worker never been exposed to dust in his employment, it is unlikely that cigarette smoking alone would have brought the worker to the degree of impairment he reached when he sought a pension in 1977. His exposure to dust likely accelerated the impairment process, causing the worker to reach a point where his physical or functional abnormality or loss impaired his earning capacity at a much earlier point in time. This is not a situation, then, where a portion of the abnormality can be identified as being caused by the smoking, with the dust exposure creating a separate, identifiable disability. Rather, the worker's disability in 1977 resulted from both exposures, working together…

[89] Thus, although the Vice-Chair referred to evidence that smoking and dust exposure may have made separate and independent contributions to the worker's loss of lung function, the Vice-Chair concluded that the worker's disability (i.e., the worker's loss of earning capacity) was the result of both exposures working together, and that principles described in the *Athey* decision applied to ensure that the worker received compensation that would restore the worker to the position he would have been in "but for" the compensable injury.

[90] Although the Vice-Chair had, in his earlier decision granting entitlement to benefits for COPD, accepted a 1997 opinion from Dr. Muir that the worker's COPD impairment was likely due in equal measure to compensable exposure in the workplace and to non-compensable smoking, the Vice-Chair concluded that smoking and dust exposure constituted a single injury (instead of distinct and divisible injuries). He stated that he was not persuaded that the 50/50 attribution of responsibility "reflected more than a rough, formulaic estimate of the physiological impact of each of the contributing factors."

[91] Because the Vice-Chair concluded that the "two co-existent factors…jointly caused trauma to a single organ" and that the effect of smoking and dust could not be measured and distinguished from each other, he concluded that the worker had "a single injury causing an impairment of earning capacity in the form of chronic obstruction of his lungs" and the "thin-skull rule" applied. Because there was no evidence that the worker had any condition or disability that *pre-existed* his employment exposure (he had been smoking for only 2 or 3 years before he started working in the sinter plant at the age of 19), *Decision No. 865/92R4* concluded that the worker was entitled to a PD pension that was not reduced by a portion attributed to cigarette smoking.

[92] Three Tribunal decisions subsequently applied *Decision No. 865/92R4* and found that workers were entitled to full PD pension or NEL benefits for COPD.

[93] *Decision No. 361/07* noted that the *Workplace Safety and Insurance Act, 1997* ("WSIA") applied (because there was a 2002 "accident" date), but it did not refer to the difference in wording between the WSIA and the pre-1985 Act that the Vice-Chair applied in *Decision No. 865/92R4.* After quoting extensively from *Decision No. 865/92R4* and its analysis of the *Athey* decision, *Decision No. 1886/07* found that the worker was entitled to the full NEL award for COPD.

[94] Likewise, both *Decision Nos. 1886/07* and *895/07* quoted and applied *Decision No. 865/92R4.* These appeals dealt with dates of "accident" before the 1990 changes to the legislation.

[95] *Decision No. 1886/07* agreed that "apportionment" is permissible in the manner described in *Decision No. 865/92R4* - that is, "…where there are multiple divisible injuries, or a symptomatic prior disability, or a medically distinguishable co-existing injury." But it concluded that the *Decision No. 865/92R4* analysis applied to the circumstances of the appeal it was deciding, and found that the worker was entitled to a PD award based on the rating given for his total impairment from COPD.

[96] In *Decision No. 895/07,* the *Decision No. 865/92R4* Vice-Chair reiterated his earlier finding that apportionment is not appropriate where the injury is COPD because the non-compensable cigarette smoking and the compensable work exposure interact with each other to cause a non-divisible injury. He therefore found that the worker should not have his PD pension for COPD reduced to reflect the contribution of the cigarette smoking history.

[97] For the reasons that follow, we disagree with *Decision No. 865/92R4*. In our view:

1. Current medical and scientific evidence indicates that, where a worker is a smoker and he/she also has respirable dust exposure, the worker's global impairment from COPD does not result from one indivisible injury. Instead, it is the additive result of impairment resulting from a work injury, and impairment resulting from smoking (as well as other factors such as aging).

2. Although epidemiological evidence and medical opinion based on that evidence are imprecise, this is currently the best available evidence, not only with respect to whether occupational dust exposure has contributed to a worker's COPD, but also with respect to how to measure what percentage of the global COPD impairment results from smoking and what percentage results from occupational dust exposure.

   The formula set out in the Board's COPD Binder provides a fair and reasonable basis for measuring the relative contribution this worker's work injury (dust exposure) and his/her smoking injury made to the global impairment the worker suffers from chronic airways obstruction.

   There may be some circumstances where the facts of the individual case differ from those contemplated by the formula/Table (such as cases where the average yearly concentration of respirable dust to which the worker was exposed exceeds the $2mg/m^3$ used in the Board's Table). But there is nothing that requires the strict application of the formula/Table in such circumstances.

3. The *Athey* decision does not stand for the proposition that a worker must be paid compensation for any impairment affecting the same body part if that impairment develops after a work injury. To the contrary, the *Athey* decision is consistent with the principle that loss that results from one injury (or injuring process) should be separated from loss that results from a

> different injury in cases where the evidence establishes that those injuries each separately and independently resulted in loss/impairment. We note that, in circumstances similar to those arising in this appeal (i.e., emphysema resulting from smoking and from occupational dust exposure), the British Coal decision found that there could be apportionment where the evidence was sufficient to establish that certain effects could be attributed to smoking or to dust exposure (even when the attribution could not be precise).

(xi) The *Athey* decision

[98] In the *Athey* decision,[39] the Supreme Court of Canada ("the Court") dealt with an appeal in which the appellant had been working out at a gym when he suffered a disc herniation. The trial judge assessed a global amount for the worker's damages from the disc herniation but, because the trial judge had found that injuries from two previous motor vehicle accidents ("MVAs") were only a 25% cause of the disc herniation, she awarded to the appellant only 25% of the global amount assessed as damages. On appeal, the respondents argued that the trial judge was correct in apportioning the damages between tortious causes (i.e., the 25% attributed to the MVAs) and non-tortious causes (the worker's pre-existing back problems).

[99] The Court concluded that the appeal involved a "straightforward application of the thin skull rule." It found that this was a case involving "a single indivisible injury, the disc herniation." The Court indicated that the plaintiff had the burden of proving that the injuries sustained in the MVAs caused or contributed to the disc herniation – that it must prove causation by meeting the "but for" or material contribution test. Having found this causal link between the MVAs (for which the respondents were liable) and the later disc herniation, the court decided the respondents were fully liable for the damages resulting from the disc herniation. The Court therefore awarded the appellant the full global amount of the damages the trial judge assessed (instead of the 25% awarded by the trial judge).

[100] The Court described the reason for the "thin skull rule" that it applied as follows:

> It is not now necessary, nor has it ever been, for the plaintiff to establish that the defendant's negligence was the sole cause of the injury…As long as a defendant is part of the cause of an injury, the defendant is liable, even though his act alone was not enough to create the injury. There is no basis for a reduction of liability because of the existence of other preconditions: defendants remain liable for all injuries caused or contributed to by their negligence…

[101] The *Athey* decision involved "a single indivisible injury" and a "straightforward application of the thin skull rule." However, in its reasons, the Court explained how the facts of the *Athey* appeal differed from "apportionment" cases. The Court contrasted the facts of the *Athey* case in which there was "a single indivisible injury, the disc herniation" with cases in which a separation is made for "distinct and divisible injuries" – and the defendant is liable for only the injury he or she has caused. In this regard, the Court stated:

> …The respondents submitted that apportionment is permitted where the injuries caused by two defendants are divisible (for example, one injuring the plaintiff's foot and the

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[39] *Athey v. Leonati*, [1996] 3 S.C.R. 458

> other the plaintiff's arm)…Separation of distinct and divisible injuries is not truly apportionment; it is simply making each defendant liable only for the injury he or she has caused, according to the usual rule. The respondents are correct that separation is also permitted where some of the injuries have tortious causes and some of the injuries have non-tortious causes… Again, such cases merely recognize that the defendant is not liable for injuries which were not caused by his or her negligence.
>
> In the present case, there is a single indivisible injury, the disc herniation, so division is neither possible nor appropriate. The disc herniation and its consequence are one injury, and any defendant found to have negligently caused or contributed to the injury will be fully liable for it.

[102] The Court also contrasted the principles applicable to the facts of the *Athey* case to those in which some unrelated intervening event or unrelated medical condition was an independent cause of loss. The Court provided the following explanation of why damages for the injury caused by a defendant's negligence would be reduced in light of such independent intervening events:

> …The essential purpose and most basic principle of tort law is that the plaintiff must be placed in the position he or she would have been in absent the defendant's negligence (the "original position"). However, the plaintiff is not to be placed in a position better than his or her original one. It is therefore necessary not only to determine the plaintiff's position after the tort but also to assess what the "original position" would have been. It is the difference between these positions, the "original position" and the "injured position", which is the plaintiff's loss. In the cases referred to above, the intervening event was unrelated to the tort and therefore affected the plaintiff's "original position". The net loss was therefore not as great as it might have otherwise seemed, so damages were reduced to reflect this.
>
> In the present case, there was a finding of fact that the accident caused or contributed to the disc herniation. The disc herniation was not an independent intervening event. The disc herniation was a product of the accidents, so it did not affect the assessment of the plaintiff's "original position" and thereby reduce the net loss experienced by the plaintiff.

[103] The Court also contrasted the "thin skull rule" with the so-called "crumbling skull" rule on similar grounds – that, because the plaintiff is not entitled to be put in a better position than his or her original position, the defendant is liable for only the additional damage caused by the injury, and not for any debilitating effects of a pre-existing condition which the plaintiff would have experienced anyway.

[104] Thus, although the *Athey* decision applied the thin skull rule and held that the plaintiff was entitled to the full amount of damages flowing from the disc herniation, it did so because it found the disc herniation was a single indivisible injury that would not have occurred "but for" the injuries from the previous MVAs (for which the defendants were liable). The Court also noted that there was no finding of any measurable risk that the disc herniation would have occurred without the MVAs. The Court contrasted those facts with cases in which some of a plaintiff's loss results from a separate injury for which the defendant is not liable. Because a defendant will not be held liable for injuries it has not caused, in cases where the loss results from distinct and divisible injuries, the defendant is liable for only the loss flowing from the injury that would not have occurred "but for" the defendant's negligence.

(xii) Use of the term "apportionment"

[105] As indicated in the *Athey* decision, where there are distinct and divisible injuries, that is not really an "apportionment" of damages, but it is instead a matter of "making each defendant liable for only the injury he or she has caused."

[106] Likewise, the only consequences that are compensable under the pre-1997 Act are those flowing from the work injury. Thus, where there are separate and divisible injuries, one of which is the work injury and one of which is the non-work injury, it is (for the purposes of calculating NEL compensation) important to determine what impairment results from the work injury and what results from the non-work injury. Because both the occupational dust exposure and smoking injure the same body part (the lungs), the determination of what impairment results from the occupational dust exposure and what impairment results from smoking is more difficult in COPD cases than it would be in the *Athey* illustration of one injury affecting the plaintiff's foot and the other injury affecting the plaintiff's arm.

[107] In COPD cases, the medical examination that results in a PD rating or a NEL rating will provide a rating for a worker's global impairment from chronic airflow obstruction (COPD). Thus, in this appeal, the issue that has sometimes been referred to as a question of whether NEL compensation can be "apportioned" is really a question about whether that rating for the global permanent impairment from COPD can be divided into a work-related and a non-work-related component because dust and smoking are separate and divisible injuries, and NEL compensation is paid for only impairment that "results from" the work injury. The "apportionment issue" in this appeal is not a question of whether NEL compensation for permanent impairment that results from the work injury can be apportioned.

(xiii) The law and Board policy

[108] Substantial changes to the *Workers' Compensation Act* came into effect in 1990. Because the interim decision in this appeal decided that this worker was suffering an impairment from symptoms of COPD as of November 6, 1992, the legislation applicable to injuries occurring between January 2, 1990 and December 31, 1997 (the "pre-1997 Act") applies in this case.[40]

[109] Although many principles of tort law can guide us in interpreting the legislation, it is the pre-1997 Act that gives the Board the authority to pay this worker benefits for a work-related injury, and it is the pre-1997 Act that determines the amount of non-economic loss ("NEL") compensation he is entitled to receive. It is therefore important to consider the wording of the NEL provisions in the context of the 1997-Act as a whole in deciding this appeal.

[110] Before 1990, if a worker had a "permanent disability" ("PD") that "resulted from" the work injury, the Board paid the worker a PD pension – usually during the lifetime of the worker. The legislative provisions that described how the amount of the PD pension was to be calculated included wording relating to "impairment of earning capacity." That impairment was "estimated from the nature and degree of the injury." The Board could use a "rating schedule of percentages" as a guide in doing so.[41] The section therefore had some language directed at the

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[40] Section 144 of the *Workers' Compensation Act* R.S.O. 1990; section 102 of the *Workplace Safety and Insurance Act, 1997*

[41] Section 45 of the pre-1989 Act

impairment of earning capacity and other language consistent with rating a worker's physical impairment. Tribunal *Decision No. 915*[42] interpreted the wording of that legislation and concluded that the Board was correct in estimating a worker's "impairment of earning capacity" from the nature and degree of the injury only (rather than by considering the *actual* impact of the injury on a particular worker's earning capacity) – and that this could be done by comparing a particular worker's injuries to benchmark injuries on the Board's Rating Schedule (because those Rating Schedule ratings were estimates of the usual impairment of earning capacity caused by such injuries to the average unskilled worker). In some cases, other provisions of the legislation provided supplements that took into account a particular worker's actual loss of earning capacity[43] – but the calculation of the amount of the PD pension did not.

[111] The legislation that came into effect in 1990 (the pre-1997 Act) changed the nature of compensation payable to workers whose injuries resulted in permanent impairment. The pre-1997 Act clearly distinguished between compensation for economic loss (i.e., compensation for future loss of earnings arising from the injury) and compensation for non-economic loss ("NEL") for workers who suffered permanent impairment "as a result of" the compensable injury.

[112] In our view, the wording of the pre-1997 Act makes it important to distinguish between "injury" and "impairment," and between "impairment" and "disability" or "loss of earnings" in deciding what NEL compensation the worker is entitled to receive (i.e., whether the global rating for his permanent impairment from COPD can be apportioned and NEL compensation paid for only that portion resulting from his work-related dust exposure).

[113] Section 42 of the pre-1997 Act describes when NEL compensation will be paid. It states:

> **42. (1)** A worker who suffers permanent impairment as a result of an injury is entitled to receive compensation for non-economic loss in addition to any other benefit receivable under the Act.

[114] Thus, the pre-1997 Act provides for the payment of NEL compensation if a worker suffers permanent impairment "as a result of" a work injury, and this compensation is paid in addition to any other benefit the worker receives under the Act.

[115] Included among such "other" benefits are benefits paid for temporary disability[44] (and "disability" is defined as "the *loss of earning capacity* of the worker that results from an injury"[45]) and future economic loss ("FEL") compensation to compensate a worker for *future loss of earnings arising from the work injury*.[46] This decision does not consider the statutory provisions governing these other benefits, but it is noteworthy that, after the interim decision in this appeal was released, the Board paid the worker full benefits for loss of earnings from November 6, 1992 (the "date of injury") until he turned 65 years of age (the date at which an injured worker ceases to be eligible for FEL compensation).

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[42] (1987), 7 W.C.A.T.R. 1

[43] Section 45(5) and 45(7) of the pre-1989 Act, replaced by section 147 of the pre-1997 Act.

[44] Section 37 of the pre-1997 Act.

[45] Section 1(1) of the pre-1997 Act.

[46] Section 43 of the pre-1997 Act.

[116] In contrast with those loss of earnings provisions, the pre-1997 Act describes how the amount of NEL compensation payable to a worker is determined on the basis of the *percentage of permanent impairment arising from the work injury* (not on the extent of the worker's loss of earning capacity). The pre-1997 Act states:

> **1. (1)** In this Act, …
>
> "impairment", in relation to an injured worker, means *any physical or functional abnormality or loss including disfigurement which results from an injury* and any psychological damage arising from the abnormality or loss; …
>
> "permanent impairment", in relation to an injured worker, means impairment that continues to exist after maximum medical rehabilitation of the worker has been achieved;
>
> **42. (2)** The compensation for a worker's non-economic loss from an injury is determined by multiplying,
>
> > **(a)** *the percentage of the worker's permanent impairment arising from the injury* as determined by the Board; and
> >
> > **(b)** $45,000[47] [plus or minus certain amounts depending on age] …
>
> **42. (5)** The Board shall determine in accordance with the prescribed rating schedule and having regard to medical assessments conducted under this section the degree of a worker's permanent impairment expressed as a percentage of total permanent impairment.
>
> (italics added)

[117] Thus, the wording of the section of the pre-1997 Act directs the Board to determine the "percentage" or "degree" of the "physical or functional abnormality or loss" that results from the work injury. It does not define "impairment" in terms of the loss of earning capacity resulting from the work injury.

[118] Board *Operational Policy Manual* Document Nos. 18-05-03 and 18-05-04 describe how the Board will assess the permanent impairment and calculate the amount of NEL compensation. Board *Operational Policy Manual* Document No. 18-05-05 (the "Board's policy on pre-existing impairment) describes the effect that a Pre-existing Impairment will have on the calculation of NEL benefits. For the reasons that follow, we agree with *Decision Nos. 7/96* and *303/02* that smoking is not a pre-existing condition or a pre-existing disability or impairment for COPD, therefore we are satisfied that the Board's policy on pre-existing impairment does not apply to the facts of this case. Likewise, the Board's *Operational Policy Manual* Document No. 04-04-17 (the "Board's policy on COPD in smelter workers") – a policy that does reduce the amount of PD awards payable to smelter workers by a portion reflecting their smoking history – does not apply to the facts of this case because the worker was not a smelter worker.

[119] As previously indicated, the Panel considered the Board's COPD Binder, but the Adjudicative Advice chapter of that Binder states that the COPD Binder is not Board policy. The Tribunal may, however, still consider Board practice that is relevant and of assistance in analyzing an appeal. In this case, the Board's COPD Binder represents a significant collection of

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[47] $49,235 in 1992 (Board *Operational Policy Manual* Document No. 18-01-03).

medical and other information – including the formula the Board used to calculate the worker's permanent impairment rating – so it should be considered.

(xiv) The Panel's findings

[120] The Board has accepted that the worker is entitled to NEL compensation. It has therefore accepted that he has a "physical or functional abnormality or loss" that results from the work injury. It also, in the words of section 42(2) of the pre-1997 Act, determined that "the percentage of the worker's permanent impairment arising from the [work] injury" is 28% of the total impairment the worker suffers from COPD (which is a 13% impairment of the whole person).

[121] Is the Board correct in deciding that the worker is entitled to NEL compensation for 28% of the permanent impairment he suffers from COPD? Or is the worker entitled to NEL compensation for the entirety of the permanent impairment he suffers from COPD?

[122] To answer that question it is necessary to make certain findings about the nature of COPD. Of particular importance is the question about whether dust exposure causes an injury (airflow obstruction) that is separate from, and independent of, the injury (airflow obstruction) caused by cigarette smoking. If the injury and resulting impairment from dust exposure is separate and independent of the injury and impairment from cigarette smoking, it is then important to decide whether it is possible to isolate and measure the impairment that results from the work injury and, if so, whether the 28% work-related attribution derived from the formula in the Board's COPD Binder is an appropriate measure of the worker's work-related permanent impairment in this case.

(a) The Panel's findings about the medical nature of COPD

[123] On the basis of the medical and scientific evidence provided in this case, we find the following:

- Unlike diseases (such as cancer) where one medical disorder may result from the interaction of many causes, COPD is a term used to describe a type of impairment (chronic airflow obstruction). That type of impairment can result from a number of different, and often overlapping, diseases. These diseases include "emphysema" – the primary diagnosis in this worker's case (at least until post-hearing evidence suggested that asthma may have played a more significant role).[48]
- Medical examination and evaluation of an individual worker can provide evidence about the extent to which a worker suffers from chronic airflow obstruction and the resulting decrease in lung function can be measured (primarily by measuring the loss in FEV_1). In addition, a medical opinion can be formed about the nature of the underlying medical conditions associated with the observed chronic airflow obstruction (such as chronic bronchitis, emphysema, asthma, or bronchiolitis). But the clinical examination of a

[48] Although Dr. Rivington explained that COPD is an "old term" used to encompass a number of diseases, and that the term CAO ("chronic airflow obstruction") has been used more recently because the common abnormality documented in these diseases is impaired expiratory airflow, this decision continues to use the term COPD because that is the term used in the various documents presented in this appeal. However, the Panel has interpreted the term "COPD" in light of Dr. Rivington's explanation.

worker with chronic airflow obstruction from emphysema cannot detect whether any physical abnormality or decrease in lung function is related to occupational dust exposure.

- The evidence about whether occupational dust exposure has contributed to the development of impairment from chronic airflow obstruction (and, if so, under what circumstances and by how much) is derived primarily from epidemiological studies.
- Epidemiological studies study populations (i.e., populations of workers with occupational dust exposure, and populations of smokers) instead of the cause of an individual worker's condition. By making statistical comparisons between populations, epidemiological studies may demonstrate a statistical association between chronic airflow obstruction and an exposure – but that alone does not prove causation, even in the studied population. There must be a careful analysis of the statistical findings in light of other medical and scientific evidence[49] before an inference of cause and effect can be made – even with respect to the studied population.
- If an inference of cause and effect can be made with respect to a studied population, it is then necessary to compare the facts about an individual worker's circumstances with those in the studied population before making any inference about a causal connection (or about the relative contribution of factors such as smoking and dust exposure). Such evidence will, by its nature, be imprecise, particularly with respect to the individual worker.
- But it may, in some circumstances, provide the best available evidence about causation – especially in cases such as this where the actual examination of the individual worker does not provide reliable evidence about whether any (or how much) of the worker's chronic airflow obstruction was caused by occupational dust exposure.
- In this case, the medical and scientific evidence from the Board, Tribunal Discussion Papers, and Dr. Rivington identified smoking as a major cause of COPD in men and women in the general population. The COPD Binder indicates that it is estimated that 80% to 90% of COPD in the U.S. is caused by smoking[50] and 10% to 20% of smokers develop COPD.
- The issue of whether occupational dust exposure could cause COPD was very controversial (and, as indicated above, that issue could not be resolved on the basis of clinical findings from the examination of the individual worker). However, after extensively reviewing epidemiological studies of dust-exposed worker populations, the Board concluded that exposure to a high enough concentration of respirable dust for a prolonged enough period of time could have an effect on a worker that was similar to smoking in the development of emphysema/COPD (i.e., chronic airways obstruction with a resulting decrease in lung function as measured by a reduction in FEV_1).
- There was no evidence presented in this appeal that challenged the medical and scientific evidence presented in the Board's MODP Scientific Review or the Board's COPD Binder. The MODP Scientific Review and the COPD Binder were filed as evidence in

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[49] *Decision No. 600/97* (2003) 66 W.S.I.A.T.R. 1

[50] U.S. Surgeon General Report, reference included in the Board's COPD Binder: Adjudicative Advice section page 32.

this case, and the worker's representative had an opportunity to adduce evidence and make submissions. Similarly, the report from Dr. Rivington, the Tribunal's Medical Assessor, was provided and there was an opportunity to respond to it. The evidence in this case was consistent with the Tribunal Discussion Papers and Dr. Rivington's opinion. In our view, it established the following:

> **a)** The effect of respirable dust exposure on lung function (as measured by FEV_1) is cumulative (the higher the concentration of the exposure and the longer the period of exposure, the greater the reduction in FEV_1). Likewise, the effect of cigarette smoking on lung function (as measured by FEV_1) is cumulative (the more cigarettes smoked and the longer the smoking history, the greater the reduction in FEV_1).
>
> **b)** The effects of dust and tobacco smoking on chronic airflow obstruction ("COPD") are additive. For example, a 120 mL FEV_1 loss due to dust and a 170 mL FEV_1 loss due to smoking will result in a total loss of about 290 mL FEV_1 loss for the two factors combined. The effects are separate and independent.
>
> **c)** Some individuals are more susceptible to developing COPD than others – but the studies suggest that both smoking and dust exposure have a similar pattern of susceptibility.
>
> **d)** Taking measures of average lung function loss obtained from the epidemiological studies of dust-exposed workers that it reviewed, the Board estimated the loss of FEV_1 from dust exposure to be 5.8 mL/year and the loss of FEV_1 from cigarette smoking to be 8.5 mL/pack-year, and it developed the formula that appears in the COPD binder on that basis.
>
> **e)** Although the Board's formula was derived using the observed average rates of loss of lung function found in epidemiological studies, the formula does not calculate the individual worker's degree of impairment from COPD on that basis. It does not, for example, calculate the worker's loss of lung function by using the average rates of lung function loss and multiplying those average rates of loss by the number of years of the particular worker's exposure. Instead, the Board rates the individual worker's actual degree of impairment from COPD.
> Having determined the degree of the individual worker's actual global impairment from COPD, the formula is used to determine the relative contribution of smoking and of dust exposure to the worker's actual global impairment from COPD.
>
> Thus, a worker who is particularly susceptible to developing chronic airflow obstruction (and therefore loses more lung function than the average losses of 5.8 mL/year from dust exposure or the 8.5 mL/pack-year from smoking), will have that particular susceptibility taken into account because the Board will rate that worker's actual level of impairment when it rates the degree of the worker's global impairment from COPD. The formula is used only to determine the relative contribution of dust exposure and smoking to the individual worker's actual global impairment from COPD.

(b) The Panel's findings on the merits of this appeal

[124] Having accepted the evidence that the effect of dust exposure on lung function (as measured by FEV_1) is cumulative, and that the effect of cigarette smoking on lung function is

cumulative, and that the effects of each on the loss of lung function from chronic airflow obstruction is additive, we find that this worker's COPD does not arise from one indivisible injury. A portion of his impairment from COPD resulted from his work injury (occupational dust exposure resulting in chronic airflow obstruction) and a portion of his impairment from COPD resulted from the non-work injury (cigarette smoking resulting in chronic airflow obstruction). The effect of each injuring process is separate and additive. They are divisible injuries.

[125] In these circumstances, cigarette smoking is not, in our view, a "pre-existing" condition to which the thin skull rule applies. The worker started smoking when he was about 15 years old; he started working at a job where he was likely exposed to respirable dust when he was about the same age. After that, he suffered chronic airflow obstruction and a resulting loss of lung function from smoking, and he suffered chronic airflow obstruction and a resulting loss of function from respirable dust exposure. Both injuries co-existed and each resulted in increasing losses of lung function over the ensuing years – with the ongoing increase in loss of lung function from the dust exposure injury ending when the worker retired in 1994 and the ongoing increase in the loss of lung function from cigarette smoking ending about 4 or 5 years later when he quit smoking.

[126] If the worker had not been exposed to dust at work, we are satisfied on the evidence that his cigarette smoking would nonetheless have caused chronic airflow obstruction with a resulting decrease in lung function. The MODP Scientific Review and the COPD Binder support this conclusion. Dr. Rivington's opinion also states that, in 30 years of pulmonary experience, he has seen similar findings among susceptible patients with a smoking history that is comparable to that of the worker (i.e. 75 pack years) but he has never encountered the same extent of abnormality from workplace exposures alone (except where the mechanism of injury differed). Because the effect of cigarette smoking is cumulative, the decrease in lung function would have increased as the worker continued to smoke over the years. Such a decrease in lung function would normally not result in symptoms until the FEV_1 was <80% of the predicted value (for gender, age, and height). But, as he continued to smoke the resulting loss of lung function (and associated symptoms) from smoking alone would have increased – regardless of whether he was exposed to dust at work or not.

[127] Because the worker also had an injury from occupational dust exposure, he also had chronic airflow obstruction resulting from dust exposure, and the loss of lung function resulting from that work injury would have increased over the years he continued to be exposed to respirable dust at work. But according to the current medical and scientific evidence, the effects of the smoking injury and the dust exposure injury are independent of each other and additive. Thus, the worker would have suffered the same degree of lung function loss from occupational dust exposure regardless of whether or not he smoked. The physical or functional abnormality or loss (i.e., the "impairment") that arose from his work injury remained the same. It was separate and independent of that arising from the cigarette smoking.

[128] Is it possible to measure how much impairment results from the work injury (the dust exposure) and how much results from cigarette smoking?

[129] We accept the evidence from Drs. Rivington and Anderson (which is consistent with the other evidence presented) that, in a case such as this where the primary diagnosis for the chronic

airflow obstruction is emphysema, it is not possible to differentiate between the injuries on the basis of a medical examination of the individual worker. It is likely that the underlying physiological mechanism by which respiratory dust injures the lung is the same as that by which cigarette smoking injures the lung.

[130] However, both the Board and the Tribunal have relied on evidence other than the clinical findings in deciding whether workers have chronic airflow obstruction from occupational dust exposure. They have relied on opinions from specialists familiar with the epidemiological studies that have investigated the role of occupational dust exposure in the development of chronic airflow obstruction. Those specialists have considered facts about an individual worker's airflow obstruction (and the nature of the underlying diseases associated with that obstruction) in light of facts about a worker's work exposure and smoking history, and they have estimated the extent to which occupational dust exposure likely contributed to a worker's chronic airflow obstruction. In reaching their conclusions on causation relative to this particular worker, they have relied on the epidemiological evidence – evidence which is the best available evidence – and the extensive review and analysis conducted by the Board. Their opinions (by virtue of the fact that they rely on epidemiological studies of groups of exposed workers rather than clinical findings specific to the individual worker) are imprecise – but we agree with *Decision No. 303/02* that this is not a bar to the apportionment of the global permanent impairment rating. Many measurements used in workers' compensation are less than exact, including measurements under the pension rating schedule and *AMA Guides*.

[131] What is important is whether the estimate is reasonable, and well-founded given current medical and scientific knowledge.

[132] We also note that the Board used measures, albeit imprecise measures, of dust exposure from the epidemiological studies to estimate this worker's level of exposure to occupational respirable dust. Instead of requiring evidence of the worker's actual respirable dust exposure in the workplace during the worker's 40 year work history with various employers (evidence that would probably have been impossible to obtain) the Board extrapolated from the levels of exposure among workers in similar occupations that were included in the epidemiological studies on dust exposure and chronic airways obstruction.

[133] In our view, the epidemiological studies and the medical/scientific opinions founded on those studies provide the best evidence currently available about the role of dust exposure in the development of chronic airflow obstruction. Although imprecise, they provide evidence about causation that clinical findings from the examination of the individual worker cannot provide. In our view, they can provide a reasonable estimate of the relative contribution to the global COPD impairment of the impairment from smoking and that from occupational dust exposure.

[134] The formula set out in the COPD Binder estimates what portion of a worker's chronic airflow obstruction likely resulted from the occupational dust exposure and what portion likely resulted from cigarette smoking on the basis of the Board's thorough review of epidemiological studies that have studied that issue. It is clearly an estimate of the relative contributions (given what is known about a particular worker's exposure history). But the Board's MODP Scientific Review explains the basis for the formula – and there is no evidence that this does not, in this case, fairly represent the best possible estimate of the relative contribution of the work injury and the smoking injury to the worker's overall impairment from chronic airflow obstruction. Indeed,

there is specific evidence from Dr. Rivington that the current attributions are quite fair to this worker.

[135] As previously indicated, because the Board rates the individual worker's actual level of impairment from chronic airflow obstruction (rather than using the average lung function losses derived from the epidemiological studies), a worker who is more susceptible to developing chronic airways obstruction from smoking and from dust exposure will receive a higher rating because he will suffer a greater degree of impairment than will a worker who is less susceptible. The average losses of lung function from smoking and from dust exposure that are derived from the epidemiological studies are only used to estimate the relative contribution of each injury to the overall impairment – something that is quite consistent with studies that suggest that the pattern of susceptibility for both dust and cigarette smoking is similar.

[136] In our view, although the Board's formula is an estimate rather than a precise measurement, it nonetheless provides a reasonable basis for measuring the relative contribution of the work injury and the smoking injury to the worker's overall impairment from chronic airways obstruction.

[137] With respect to the relative impact of smoking and industrial exposure on this worker's airflow obstruction, Dr. Rivington's observations are consistent with the evidence that smoking usually makes more of a contribution to airflow obstruction than does industrial exposure. He expressed the opinion that more susceptible patients with the same smoking history as that of the worker could have spirometric findings similar to the worker's findings (even without dust exposure), but that he had not observed similar spirometric findings from industrial exposure alone (absent an asthmatic component to cause such severity and reversibility).

[138] In the worker's case, Dr. Rivington concluded that the "cumulative impact of smoking and workplace exposure led to the gradual evolution of COPD that ultimately led to the severity causing symptoms and the diagnosis of this condition."

[139] In expressing his opinion that the 28% that the Board allocated to this worker's work exposure was "fair," respirologist Dr. Rivington took into account the nature of the worker's medical condition, the worker's smoking history, and his own observations from 30 years of practice about spirometric findings of those with a similar 75 pack-year smoking history or with occupational exposure alone. Dr. Rivington also compared the nature of the worker's occupational dust exposure with that of workers in the studies used to generate the data used in the Board's Table/formula. Based on all the evidence, Dr. Rivington concluded that allocating 28% of the observed changes to work exposure was fair in the worker's case.

[140] Likewise, respirologist Dr. Roos accepted that 25% of the impairment the worker experienced from chronic airflow obstruction was work-related.

[141] We accept Dr. Rivington's opinion. We find that 28% of the worker's impairment from chronic airflow obstruction arises from his occupational dust exposure, and that his NEL compensation should be calculated on that basis.

[142] In view of the nature of the worker's employment history, and (as Dr. Rivington observed) the possibility that the worker's actual exposure may be somewhat less than many of

the workers in the epidemiological studies reviewed in the MODP Scientific Review that was the basis for the Board's Table/formula, we find that the dust exposure level used in that Table/formula (of years of exposure at 2 mg/m^3) likely does not underestimate the worker's actual occupational dust exposure. His 75 pack-year smoking history is not disputed.

[143] The rating of his global impairment from chronic airways obstruction was based on his own spirometric (and other) findings, so it takes into account any susceptibility he had with respect to developing chronic airflow obstruction. The rating was not reduced for other possible sources of chronic airflow obstruction. For example, it was not reduced to reflect a possible asthmatic component (i.e., the spirometric finding of 44% FEV_1 that was used to determine the level of the global impairment was the pre-bronchodilator finding). And the Board correctly applied the formula it derived from its review of the epidemiological studies.

[144] On the available evidence, we find that the Board's decision that 28% of the worker's global impairment from chronic airflow obstruction resulted from his work injury was correct. Because the amount of NEL compensation payable to the worker is based on a rating of the degree of "permanent impairment arising from the [work] injury," we find that the Board's decision to pay for only that portion of his chronic airflow obstruction that resulted from his work injury (occupational dust exposure) was correct.

[145] For the foregoing reasons, we disagree with the *Decision No. 865/92R4* finding that the chronic airflow obstruction of a smoker with occupational dust exposure results from one indivisible injury. We note that *Decision No. 865/92R4* did not have the benefit of Dr. Rivington's very thorough opinion. In our view, the best medical and scientific evidence that is currently available on this issue supports the findings of the earlier Tribunal decisions – that smoking causes an identifiable measurable co-existing condition that does not arise from the work injury, and is therefore not compensable.

[146] Furthermore, much of the *Decision No. 865/92R4* discussion of the unfairness that would result from reducing the amount paid for permanent impairment/PD by an amount representing the chronic airways obstruction from smoking was based on a discussion about how a worker's loss of earning capacity will reflect the combined effects of both smoking and dust exposure.

[147] We have no doubt that this is the case. A worker with chronic airflow obstruction from occupational dust exposure, added to chronic airflow obstruction from smoking, will have more chronic airflow obstruction and therefore more impairment, and therefore more symptoms that can impact on that worker's earning capacity. He/she will not be in the same position as a worker who never smoked. This does not, however, mean that the injury from smoking is part of the work injury. And, especially after the 1990 change in the legislation that specifically included compensation for "economic loss" (i.e., loss of earnings/earning capacity) that was separate from that provided for non-economic loss ("NEL"), the wording of the legislation makes it clear that the amount of NEL compensation is not based on any measurement of loss of earning capacity.

[148] There are other provisions of the pre-1997 Act that provide for loss of earning capacity or future loss of earnings resulting from the work injury. If a worker would not have had a loss of earnings "but for" the work injury, that worker can be compensated for the loss of earnings under those sections. For example, the interim decision in this appeal found that, as of

November 6, 1992, the worker was suffering an impairment from symptoms related to later-diagnosed COPD. The Board then investigated the evidence about the worker's ability to work. It found that the worker's pre-injury job was unsuitable given the moderate COPD he was suffering at that time, and other jobs he may have been able to perform were unavailable to him. It therefore decided that the worker had a loss of earnings that arose from his work injury (because 28% of his COPD resulted from the work injury) and it paid him full temporary disability benefits (in the amount of about $86,000 plus interest) from November 6, 1992 until June 1997 when the worker turned 65 years of age.

[149] To decide this appeal, it is important to consider the particular statutory language governing entitlement to NEL compensation. In our view, the wording of the pre-1997 Act makes it clear that NEL compensation is to be paid for the degree or percentage of physical abnormality or functional loss that "results from" or "arises from" the work injury. NEL compensation does not compensate for impairment that does not result from the work injury, or for a loss of earnings/earning capacity.

[150] We therefore find that the Board was correct when it limited the worker's NEL compensation to that portion of his chronic airflow obstruction that resulted from the work injury (dust exposure). In our view, 28% of the global impairment from chronic airflow obstruction is a fair and reasonable estimate of the permanent impairment that resulted from the work injury. The worker is therefore not entitled to an increase in his 13% rating for COPD (i.e. 28% of the 45% "whole person" rating).

DISPOSITION

[151] The worker's appeal is denied. The worker is only entitled to NEL compensation for the permanent impairment that resulted from his work injury. The rating of 28% (13% of the whole person) is a fair and reasonable estimate of the degree of that impairment.

DATED: November 30, 2009

SIGNED: M. Kenny, B. Wheeler, R. W. Briggs