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Review of the Workplace Safety and Insurance Board (WSIB) Adjudicative Support Material Binder (ASMB) for Chronic Obstructive Pulmonary Disease (COPD) used in the adjudication of claims from Ontario miners

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Last updated September 7, 2022

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Executive Summary

The Issue

At the present time, the Workplace Safety and Insurance Board (WSIB) of Ontario does not have a specific policy on chronic obstructive pulmonary disease (COPD); however, adjudicators use the outdated WSIB COPD Adjudicative Support Material Binder (ASMB) that consists of two documents: COPD: Scientific Review (SR) and Adjudicative Advice (AA) in the adjudication of COPD claims to guide their decisions on COPD claims. The SR and AA documents were prepared by the Medical and Occupational Disease Policy (MODP) Branch of WSIB in 2001 (WSIB 2001). Although not explicitly stated in the WSIB COPD ASMB material, a "guideline" requiring a cumulative respirable dust (RD) exposure of 40 to 50 mg/m^3- years has come to be adopted by WSIB and WSIAT as a criterion that has to be met in order for a COPD claim to be allowed. This appears to have been calculated by multiplying an average duration of 20 to 25 years of exposure by an average RD concentration of 2 mg/m^3. However, the specific studies that were used to derive the average durations and intensity of exposure are not identified in the ASMB. The epidemiological literature that was used to develop the ASMB is now more than 20 years old.

The Present Review

The present review focused on the exposure considerations in the AA document and the epidemiological studies in the SR document (Section 4) that may apply to Ontario hard rock miners.

Before the early 1980s, dust exposures in mines were assessed by collecting samples of dust with instruments such as konimeters and thermal precipitators, followed by counting of the dust particles under a microscope. Konimeters were widely used in Ontario mines and thermal precipitators and konimeters were used in South African gold mines. Conversion of particle counts (ppcc) to equivalent gravimetric units (mg/m^3) required a series of complex calculations and assumptions. The dust exposure information used in the epidemiological studies of South African gold miners was based on the early surveys using konimeters and thermal precipitators by Beadle and Bradley (1970) and summarized by Page-Shipp and Harris (1972).

Use of Ontario mining dust sampling data in the adjudication of COPD claims is also fraught with limitations and uncertainties. The Ontario Mining Exposure Database (OMED) is a recently available important source of exposure data for dust and other contaminants in Ontario hard rock mines.

The WSIB relied primarily on studies of South African gold miners to draw conclusions about the association between exposures to RD and RCS and risk of COPD. Concerns about the accuracy of the South African gold mining dust sampling results were raised when results for silicosis among South African miners were found to be substantially different from the silicosis studies of Ontario miners. It was suggested that the South African risk estimates for silicosis were overestimated because the RD and

RCS exposures were underestimated. A detailed analysis of the Beadle and Bradley (1970) and Page-Shipp and Harris (1972) calculations by Gibbs and DuToit (2002) identified three factors that may have contributed to the underestimation of the RD and RCS exposures. After adjusting the South African RD and RCS by a factor of 2, the cumulative exposures that were associated with a statistically significant increased risk of COPD were still well below the WSIB COPD exposure guideline of 40 to 50 mg/m^3-years.

The WSIB COPD SR is incomplete as it does not include other relevant studies that were published in the period considered by WSIB (1980 to 2000). A reexamination of these and additional studies published since the ASMB found statistically significant increased risks for COPD morbidity defined as emphysema, chronic bronchitis and impaired lung function and mortality at cumulative RD exposures much lower than the WSIB COPD exposure guideline of 40 to 50 mg/m^3–years. In particular, recent studies observed an exposure-response relationship between a cumulative RCS exposure of about 2.0 mg/m^3-years and a decrease in lung function (FEV_1/FVC) to the lower limit of normal (LLN) that meets the Global Initiative for Chronic Obstructive Lung Disease (GOLD) definition of COPD.

For further details, the reader is directed to the conclusions for each of the COPD morbidity and mortality sections in this document.

Occupational Exposures Not Considered Previously

The WSIB COPD ASMB does not consider the contribution of McIntyre aluminum powder (MP) to the cumulative RD exposures of Ontario miners. In October 2017, the WSIB acknowledged that the Time-Weighted Average (TWA) exposure of MP experienced by at least 27,500 Ontario hard rock miners was 0.74 mg/m^3 and that the daily TWA exposure likely ranged from 0.5 to 1.0 mg/m^3. If a miner was exposed for 30 years to MP, this exposure alone would have contributed from 1/3 to 2/3 of the cumulative RD exposure of 40 to 50 mg/m^3–years required by WSIB COPD guideline.

Similarly, exposures to diesel exhaust (DE) have also not routinely been taken into account by WSIB in the consideration of COPD claims from Ontario hard rock miners. Recent studies have suggested that exposure to DE may be directly associated with an increased risk of COPD. DE also fits into the category of vapours, gases, dusts and fumes (VGDF) that covers a wider range of occupational exposure and for which there is increasing evidence of an association with COPD. A recent independent scientific review on COPD commissioned by the WSIB concluded that increased intensity or duration of exposure to VGDF with or without smoking result in greater lung function impairment and a greater likelihood of developing COPD (WSIB 2021a).

Conclusions

The basis for the current WSIB COPD exposure guideline requiring a cumulative RD exposure of 40 to 50 mg/m^3-years is uncertain. The information supporting it is outdated, having been last considered in 2001.

Important exposure assessment considerations in the WSIB SR and AA documents for COPD include:

- Historical measurement of dust concentration in Ontario hard rock mines
- Estimating respirable mass concentrations from konimeter and thermal precipitator samples
- Extrapolating results from South African gold mining studies to Ontario hard rock miners
- Use of Ontario mining dust sampling data in the adjudication of COPD claims
- Limitations of Ontario mining exposure data in the WSIB COPD ASMB

Findings from seven South African gold mining studies published between 1986 to 1996 appear to form the main basis for the WSIB COPD exposure guideline as it applies to Ontario hard rock miners. After taking the possible underestimation of RD exposures in these and other South African gold mining studies into account, the cumulative dust exposures associated with statistically significant increased risk of COPD morbidity and mortality remained below the WSIB COPD exposure guideline of 40 to 50 mg/m^3 – years.

The guideline requires updating, taking into account more recent epidemiological studies of the association between RD, RCS, VGDF and COPD morbidity and mortality published in the last 20 years. Based on this more recent evidence, it is recommended that in addition to RD and RCS, MP, DE and the broader category of VGDF should also be included in the assessment of cumulative exposures that contribute to the risk of COPD.

OHCOW strongly urges the WSIB to reconsider its existing COPD exposure guidelines for Ontario miners and other occupations in light of new information and in the need for the more equitable adjudication of COPD claims filed by workers and their families.

Introduction

The purpose of this review is to examine the exposure criteria and the epidemiological evidence that are used by the Workplace Safety and Insurance Board (WSIB) in the adjudication of chronic obstructive pulmonary disease (COPD) claims for Ontario hard rock miners. Of particular interest is the basis of an unpublished, internal guideline requiring a cumulative RD exposure of 40 – 50 mg/m^3-years in order for a claim for work-related COPD to be considered.

The Background section provides an overview of the two documents used by the WSIB occupational disease adjudicators in the adjudication of COPD claims. These documents are contained in the COPD Adjudicative Support Material Binder (ASMB) and consist of: COPD: Adjudicative Advice (AA) and Scientific Review (SR). The AA and SR documents were prepared by the Medical and Occupational Disease Policy (MODP) Branch of WSIB in 2001 [WSIB (2001)][A]. The focus throughout this review is on the portions of the AA and SR documents that are relevant to COPD related to Ontario hard rock mining exposures.

The AA section of the WSIB ASMB is discussed first as there are several important exposure assessment considerations that have a bearing on the interpretation of the epidemiological studies that were considered in the SR section of the WSIB ASMB. This is followed by a summary of the epidemiological studies considered in the SR section of the WSIB ASMB and other relevant studies on the association between exposures to RD and respirable crystalline silica (RCS) in hard rock mining and COPD. The contribution to the risk for COPD from exposure to McIntyre aluminum powder (MP), diesel exhaust (DE) and Vapors, Dusts, Gases and Fumes (VGDF) are also considered.

Background

Occupational diseases are adjudicated under Section 2 (1) and Section 15 of the Workplace Safety and Insurance Act and by Regulation in Schedules 3 and 4 of the Act. If a disease is not listed in the Schedules and a relevant policy has not been developed, entitlement to WSIB benefits and services is determined based on the real merits and justice of the individual claim. It must be established that it is more probable than not that the circumstances of a worker's employment and exposure history significantly contributed to the development of the medical condition being claimed.

At the present time, the WSIB does not have a policy on COPD; however, many WSIB Decision letters and Workplace Safety and Insurance Appeals Tribunal (WSIAT) appeals for COPD claims often refer to a "WSIB COPD Guideline" that has to be met in order for the claim to be allowed. Although not explicitly stated in the WSIB COPD ASMB material, this "guideline" has come to be adopted by WSIB and WSIAT as a criterion for a cumulative RD exposure of 40 to 50 mg/m^3- years that has to be met in order for a

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[A] *SR in this document refers to the WSIB COPD Scientific Review section of the WSIB COPD Adjudicative Support Material Binder (ASMB). AA in this document refers to the Adjudicative Advice section of the WSIB COPD Adjudicative Support Material Binder.*

claim for COPD to be considered. This appears to be calculated by multiplying an average duration of 20 to 25 years of exposure by an average RD concentration of 2 mg/m^3.

Section 1 of the ASMB is entitled “Occupational Risk Factors: A Literature Review”. It states the following under Magnitude of Exposure:

> *The **average intensity** of respirable dust/fume exposure in the study groups reviewed ranged from 0.89 mg/m^3 to 12 mg/m^3 with a mean of 2 mg/m^3.*
>
> *The **average duration** associated with significant risk of COPD from all studies reviewed is estimated to be 25 years. The increased risk of COPD impairment is most closely associated with **cumulative exposure** to respirable dust. Cumulative exposure reports the worker’s total dose of **respirable** dust by proportionally combining exposure intensity with duration (mg/m^3 years). If the intensity is extremely high, then shorter duration can produce a significant risk for COPD. Low intensity dust exposure can also create similar risks, if there is a corresponding increase in the duration of exposure.*

A review of the SR and AA documents in the WSIB COPD ASMB did not reveal which studies were used as the basis for the stated range of average dust intensities of 0.89 mg/m^3 to 12 mg/m^3 or the mean of 2 mg/m^3. It is also not clear how the average exposure duration of 25 years was calculated since there is such a wide range of results among the epidemiological studies. The COPD ASMB also does not provide the rationale for applying such a broad estimate for a cumulative dust exposure of 40 to 50 mg/m^3- years as the exposure criterion for all types of occupations and exposures.

WSIB Adjudicative Advice (AA) Exposure Assessment Considerations

This section discusses some of the issues around the exposure assessment considerations outlined in the WSIB ASMB and begins with a brief discussion of historical dust measurements in Ontario and South African gold mines. This is followed by a more detailed discussion of the dust exposure assessments in the epidemiological studies of South African gold miners as these are the main studies used to support the assessment of COPD risk for Ontario hard rock miners in Section 4 of the ASMB.

Other exposure assessment considerations will include; how dust sampling data from Ontario hard rock mines are used in the WSIB exposure assessments and the importance of accurate conversion from particle counts in units of particles per cubic centimeter (ppcc) to respirable mass concentrations in units of milligrams per cubic meter (mg/m^3).

Historical measurement of dust concentrations in Ontario hard rock mines

Before the early 1980s, dust concentrations in Ontario mines were measured with particle counting with a konimeter that was an instantaneous dust sampler having a sampling duration of approximately one-third of a second. Airborne dust particles were drawn through a small opening by a spring-loaded piston and deposited on a microscope slide or a circular disc that could be rotated so that up to 29 individual dust samples could be collected. An example of a konimeter is shown in Appendix 1.

Dust sampling in South African mines used either the konimeter or a thermal precipitator to collect dust particles. A thermal precipitator collects dust particles differently than the konimeter. Dust is drawn into the thermal precipitator and as the dust particles pass through a chamber that is heated on one side (or a heated wire in the middle of the chamber), the particles move towards the lower temperature side(s) of the chamber and are deposited on glass slides that are then analysed under a microscope and the particles are counted in the same way as they are for konimeter sampling. The collection efficiency of thermal precipitators is quite high for small particles and is virtually 100% for particles in the range of 0.01 micrometers to 10 micrometers. An example of a thermal precipitator is the standard head type that was manufactured by Casella. The Casella model of the thermal precipitator utilizes a built-in water reservoir and calibrated jet aspirator to pull 7 ml/min of air through the collection slot. The wire may be heated with a small dry cell battery making the instrument completely self-contained, easily portable, and safe for use in explosive atmospheres. The Casella thermal precipitator contains two 19 mm diameter cover glass slides onto each of which a strip of dust about 1.5 mm by 10 mm is deposited. Examples of a thermal precipitator are shown in Appendix 1.

Konimeter sampling is useful for obtaining instantaneous or “grab” samples of airborne dust particles at a particular location to assess the effectiveness of ventilation and other exposure control methods but is not representative of personal exposure to RD. Thermal precipitators were also used for area sampling and had the advantage of collecting dust samples for longer periods than konimeter sampling but they were not portable enough to measure personal exposure to RD. One advantage of the thermal precipitator is that it is able to collect samples over a longer period of time (up to a full work shift) whereas konimeters only provide instantaneous or “spot” measurements. However, both methods have the limitation of only providing particle counts. Personal size-selective dust samplers consisting of a battery-operated pump and a cyclone that collected respirable sized particles onto a filter were introduced in the late 1970s. The dust collected on the filters could be weighed to provide mass concentration of RD in units of milligrams per cubic meter (mg/m^3). The collected dust sample could also be analyzed with X-ray diffraction to determine the RCS content and became the sampling method that is used for modern exposure standards and in more recent epidemiological studies. An example of a size-selective gravimetric dust sampler is shown in Appendix 1.

Ontario mining dust sampling data provided in the COPD ASMB

Assessment of dust exposures in hard rock mining is outlined on page 14 to 17 in Section 2 "Exposure Description and Assessment" of the WSIB COPD ASMB and in more detail in Appendix 1 "Procedure for Assessing Mining Exposure". Appendix 1 instructs the adjudicator to use the "Job Classification System"[B] in Appendix 9 to determine which of 8 job categories match the miner's job titles with category 1 representing the highest dust exposures and 8 the lowest dust exposures. The next direction to the OD claims adjudicator is to check Table 1 (gold mines data summary) Table 2 (uranium mines data summary) and Table 3 (nickel mines data summary) on page 36 of the COPD Adjudicative Support Material Binder. These tables (reproduced below) are intended to help the adjudicator assess the magnitude of the miner's exposure pre-1960 and post 1960.

Figure 1

Tables 1-3 reproduced from pg. 36 of the WSIB COPD ASMB

Table 1: Gold mines data summary

Era	Mine Camp and Job	Dust Level
Pre 1960	Raise drilling, drift crews, all crusher operations, stope, tram; load, motorman (MOT), scoop, Load Haul and Dump, (LHD), skip, other underground jobs	High
	Timber, hoist, cage, sampler, hydraulics, track	Low
Post 1960	Raise drilling	Medium-High
	All crusher, drift crew, stope, tram, load, MOT, scoop, LHD, skip, other underground jobs	Medium
	Timber, hoist, cage, sampler, hydraulics, track	Low

Table 2: Uranium mines data summary

Era	Mine Camp and Job	Dust Level
Pre 1960	Drilling (stopers, jacklegs, lyeners), blasting, headings, raises, stopes, chutes, loader, slushing, dumping, crushing, mills, grinding	High
	Shafts, drifts, mills	Low
Post 1960	Raise drilling	Medium-High
	Drilling (stopes, raise), loading and dumping, mucking, slushing, crushing, grinding, service, conveyor, leaching	Medium
	Milling, laboratory (assay)	Low

Table 3: Nickel mines data summary

Era	Mine Camp and Job	Dust Level
Pre 1975-77	Raises, crushing/conveying and hoisting, underground development, stoping, slushers, LHD	High
	Mills (ore dressing), mills (concentrator)	Medium-high
Post 1975-77	Contact the company for exposure data	

[B] *The "Job Classification System" appears to be based on the Ontario Mining Study by Muller et al (1983)*

Appendix 10 in the COPD ASMB provides five summary tables of dust counts (ppcc) & gravimetric measurements for gold & uranium mines from 3 sources:

- Ministry of Labour (MOL) Muller personal communication 1985 (summary of average dust counts for selected job categories in gold mines by mining camps for different periods; before 1955, before 1960 & after 1960)
- MAPAO semi-annual reports 1956-1958 (average dust counts for selected locations/operations in 5 gold mines)
- MAPAO semi-annual reports 1955-1957 (average dust counts for selected locations/operations in 5 uranium mines)
- MAPAO (no names of mines) for 3 years (1958/59/60) (annual & quarterly average dust counts for selected locations/operations in all uranium mines combined)
- Ontario Ministry of Health Uranium Mine Dust Survey 1973-74 (average of total dust counts, RD & RCS in mg/m^3 & % RCS for selected locations/operations at two uranium mines; Rio Algom & Denison)

Exposure data from these tables was further summarized by jobs & time period of operation (pre & post 1960 for gold & uranium mines) using the following steps:

The mean exposure level for the different jobs from the different mining camps or mines for a given period were first combined together

Jobs with similar exposure levels were then grouped together into high, medium or low exposure categories for the different periods & job categories 1-8 from Appendix 9 were added to the high/medium/low categories

This appears to be a qualitative assessment of exposure (high, medium, low) yet the calculation of the cumulative exposure outlined in the ASMB requires a quantitative estimate of the cumulative RD exposure in terms of mg/m^3 – years.

Simplification of the RD exposure levels for the convenience of the claims adjudicators obscures the true range of RD and RCS exposures for specific mines, occupations or tasks.

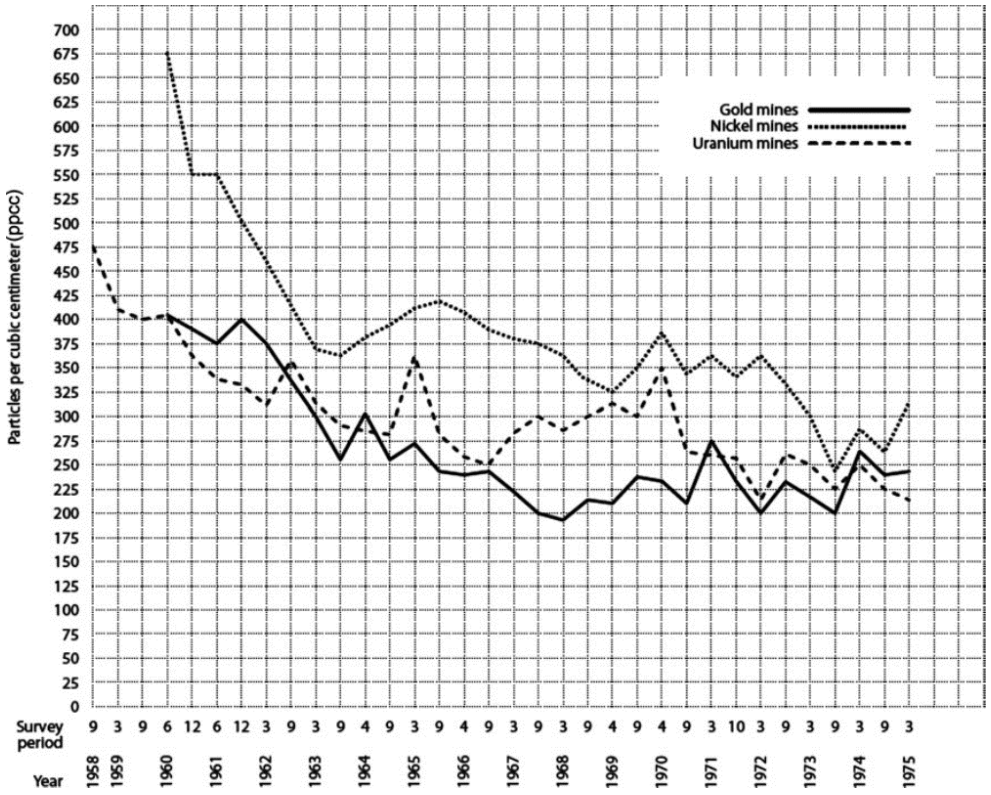
Some generalized conclusions about RD and RCS exposures in Ontario mines are incorporated in these tables. For example, it was generally assumed that before 1960, RD exposure in most Ontario gold mines was in excess of 2 mg/m^3, a concentration classified as high exposure and that after 1960, there it was assumed that there was a dramatic decline in RD exposures with a few specific job categories such as raise driller continuing to have high exposure and that most other mining jobs fell into the medium (~1

to 2 mg/m^3) and low exposure (< 1 mg/m^3) categories. Overall estimates of RCS content in the RD were assumed to be ~10% for gold mines, ~20% for uranium mines and ~5% for nickel mines.

These widely held assumptions are reflected in the oft-referenced figure in the 1976 Royal Commission on the Health and Safety or Workers in Mines reproduced below that shows the trend between 1960 and 1975 of reduced average dust counts based on annual MAPAO reports.

Figure 2

Average dust counts (ppcc) for gold, nickel and uranium mines 1958-75



Source: Figure 9 in 1976 Royal Commission on the Health and Safety or Workers in Mines

Figure 3 Comparison of average dust levels (ppcc) for nickel, gold and uranium mines in the first half of 1960 and first half of 1975

Type of mine	Average dust level in ppcc: 1st half 1960	Average dust level in ppcc: 1st half 1975	Quartz in total dust (average %)	MAPAO guideline in ppcc by % of quartz in total dust
Nickel	680	310	Up to 10	500 (up to 10% quartz)
Gold	400	250	15 to 35	300 (10 to 30% quartz)
Uranium	400	220	60 to 70	200 (over 30% quartz)

Source: Mines Accident Prevention Association of Ontario (MAPAO), Semi-Annual Survey of Dust Conditions, 1958–1975.

Source: Table11 in 1976 Royal Commission on the Health and Safety or Workers in Mines

MAPAO Semi-Annual Reports of Air Conditions

The primary source for konimeter particle count data for Ontario mines is the semi-annual reports of the Mines Accident Prevention Association of Ontario (MAPAO) that were collected between 1959 and 1982. Konimeter measurements were made by personnel at each mine (not MAPAO staff) & results were recorded on the MAPAO "Summary of Semi-Annual Survey of Air Conditions" forms and submitted to the MAPAO. The purpose of the MAPAO surveys was to:

- provide summarized data to the mines for comparison to the rest of the mines (e.g., Kerr Addison gold mine could compare their results to other Ontario gold mines combined)
- provide summarized data to WSIB for compensation purposes and the Ontario Ministry of Labour (MOL) for compliance/enforcement purposes

Paper originals of the MAPAO semi-annual dust survey reports were later copied onto microfiche and stored by the Mines and Aggregates Safety and Health Association (MASHA) – the organization that superseded MAPAO and in turn became part of Workplace Safety North. At the present time there is limited access to these microfiche copies of the MAPAO forms.

An example of a MAPAO semi-annual report is shown in Figure 4.

Figure 4

Example of Semi-annual Report by the Mines Accident Prevention Association of Ontario (MAPAO)

FORM 1

SUMMARY OF SEMI-ANNUAL SURVEY OF AIR CONDITIONS

UNDERGROUND

FIRM KERR ADDISON MINES LIMITED MINE OR PLANT KERR ADDISON PERIOD ENDING APRIL 15th, 19 71

	OPERATION	NO. ACTIVE WORKINGS SAMPLED	DUST – P.P.C.C. NUMBER OF SAMPLES	AVERAGE	NO. OF SAMPLES 0-300	300-500	500-800	800+	AIR ENTER	AIR LEAVE	FILTERED AIR BEFORE SHAKE	FILTERED AIR AFTER SHAKE	SILICA ANALYSIS % TOTAL LOW	% TOTAL HIGH	% TOTAL AVG.	% FREE LOW	% FREE HIGH	% FREE AVG.	VENT. F.P.M. AT OPERATION	USE OF WATER NO. WATER BLAST	NO. USING SPRAYS INTER	SPRAYS CONT.	RESP. USED VOL.	RESP. USED COMP
1.	HEADINGS - DRILLING	1	1	372	0	1	0	0	95	388									40	1	1	0	0	0
2.	- LOADING																							
3.	RAISES - DRILLING	1	1	421	0	1	0	0	55	612									30	1	1	0	0	0
4.	- SCRAPING																							
5.	STOPES - DRILLING	8	8	418	3	2	3	0	169	450									26	8	8	0	0	0
6.	- SCRAPING	13	13	525	4	3	3	3	166	497									27	12	13	1	0	0
7.	- TIMBERING	16	16	234	11	4	0	1	107	238									26	16	16	0	0	0
8.	- FILLING	1	1	95	1	0	0	0	50	50									30	1	1	0	0	0
9.	SHAFTS - DRILLING																							
10.	- LOADING																							
11.	DUMPING	8	8	498	1	5	1	1	180	591	0	0							39	0	0	0	6	0
12.	PULLING CHUTES	8	8	395	1	5	2	0	262	434	0	0							28	0	8	0	8	0
13.	MUCKING - DRAW POINTS																							
14.	- SCRAMS	3	3	499	1	0	2	0	225	554	0	0							37	0	3	1	3	0
15.	SAMPLING	11	11	394	6	2	2	1	164	351									23	11	11	0	0	0
16.	UNDERGROUND CRUSHERS	3	9	250	7	0	2	0	50	105	50	50											3	0
17.	UNDERGROUND CONVEYORS	1	6	137	6	0	0	0	50	183	0	0											1	0
18.	SHAFT LOADING POCKETS	3	9	354	3	5	1	0	174	450	0	0							30	0	0	0	1	2
19.	FRESH AIR SPLITS	8	8	145	7	1	0	0																
20.	RETURN AIR SPLITS	8	8	293	5	2	0	1																
21.	SHAFTS AND STATIONS	13	13	144	10	2	1	0																
22.	MAIN DRIFTS AND X-CUTS	9	9	240	7	1	0	1																
23.	TOTALS	115	132	328	73	34	17	8	146	369	50	50							28	50	62	2	24	2

24. NO. OF DRILLS OBSERVED DURING SURVEY: JACKLEGS 16 STOPERS 2 SINKERS 0 MOUNTED 2
25. NO. OF ABOVE HAVING HANDVALVES IN WATERHOSE AT DRILL: " 0 " 0 " 0 " 1
26. NO. OF ACTIVE DEAD ENDS ON DAY SHIFT: HEADINGS 1 RAISES 1 STOPES 3 SHAFTS 0
27. NO. OF ABOVE SERVED BY AUXILIARY VENTILATION: " 0 " 1 " 0 " 0
28. VENTILATION UNDERGROUND TOTAL CFM 86,000 AVERAGE TONS, ORE + WASTE PER DAY 1,319 CFM/TON 65
29. IS DUST DISPERSAL AT DUMPS AND PASSES CONTROLLED? Yes HOW? Isolated by Doors
30. FRESH AIR SUPPLIED TO OPERATOR - CFM U/G CRUSHER: 25000 CONVEYOR: 2,000 LOADING POCKET: 10,000
31. RE ITEM 22. CALCULATE WEIGHTED AVERAGES EXCLUDING FRESH AND RETURN AIR SPLITS.
32. RE ITEM 14. EXCLUDE FROM AVERAGES IF USE OF RESPIRATORS IS COMPULSORY.
33. STATE REMEDIES BEING PLANNED OR APPLIED TO REDUCE UNDESIRABLE EXPOSURES.

Ron Slater SURVEYOR

MANAGER

The sampling data that was recorded on the MAPAO semi-annual survey forms included;

- number of active workings sampled
- number of samples collected
- average dust concentration in ppcc for each operation
- overall mine average[C]
- number of samples in the following ranges; 0-300 ppcc, 300-500 ppcc, 500-800 ppcc and > 800 ppcc.[D]

Active workings that were regularly sampled included;

- Headings - drilling, loading
- Raises - drilling
- Stopes - drilling, scraping, timbering and filling
- Dumping
- Pulling Chutes
- Sampling
- Underground crushers
- Underground conveyors
- Shaft loading pockets

The konimeter measurements were intended to assess the amount of dust generated by different processes or in different areas of the mine & to judge the overall effectiveness of ventilation or other control measures such as using water sprays. They were not originally intended to assess personal dust exposures of individual miners; however, these were the only dust exposure data available up to the mid to late 1970s.

The main limitation of the MAPAO summarized results is that the "averages of averages" approach obscures the true range and extent of dust and RCS exposures (e.g., number of samples > MAPAO guidelines or MOL and other OELs, range of dust exposures for each operation, location of operations with high exposures)

The example of the MAPAO semi-annual report in Figure XX above, illustrates these limitations. Although the overall average is slightly above the MAPAO guideline of 300 ppcc, the upper range is 525 ppcc. In addition, 45% of the samples were above the MAPAO guideline, 20% were above > 500 ppcc and 6% were above 800 ppcc (see Figure XX below).

In addition, the ventilation air entering some of the production areas of the mine contained a significant about of dust. For example, the air entering the area where the task of pulling chutes was done had 262 ppcc of dust that is equivalent to 0.3 mg/m^3 of RD containing about 0.03 mg/m^3 of RCS.

[C] *Overall mine average is a weighted average calculated as the sum of the number of samples for each active working x average ppcc of those samples ÷ total number of samples collected from all the active workings.*
[D] *These ranges used likely coincided with the MAPAO guideline for gold mines of 300 ppcc, the WSIB Gold Mining policy definition of 500 ppcc as "dusty gold mining" exposure and 800 ppcc as reaching the upper limit of reliable konimeter measurements.*

Figure 5 Illustration of the Limitations of using averages from the MAPAO reports (using the MAPAO semiannual report in Figure 4)

Overall average 328 ppcc Range 95-525 ppcc

59/132 or ~ 45% samples > 300 ppcc (MAPAO guideline for gold mines)

25/132 or ~ 20% samples were > 500 ppcc (WSIB definition of "dusty gold mining")

8/132 or ~ 6% samples > 800 ppcc (upper limit of konimeter measurement)

Conversion of Total Particle Counts (ppcc) to RD and RCS concentrations (mg/m^3)

Epidemiological studies of miners exposed to dust prior to the early 1980s only had particle count data available to estimate dust exposures and is the only data available for this period. Conversion from particle counts to equivalent gravimetric units was challenging and was usually based on theoretical calculations and assumptions about the relationship between surface area of total dust particles, respirable mass and other factors (Page-Shipp Harris 1972, Gibbs and Dutoit 2002).

The exposure assessment portion of an epidemiological study of silicosis among Ontario hard-rock miners during 1982–1986 by Muir et al (1989a, 1989b) included the conversion of historical dust counts (ppcc) into gravimetric units (mg/m^3) of RCS (Verma et al 1989). This was done by side-by-side sampling of the dust in one gold mine and one uranium with konimeters and size-selective gravimetric sampling instruments (cyclone and filter). The sampling included ~ 2,360 gravimetric samples and ~90,000 konimeter measurements collected over two years.

The correlation between the dust counts and RCS measurements was found to be nonlinear. The following equation that included linear and exponential functions was the best fit to the data and allowed for the conversion of gravimetric RCS values to konimeter values for gold and uranium mines:

$$K = \beta_0 \times [1 - \exp(\beta_1 \times G)] + \beta_2 \times G$$

where: K = konimeter counts in ppcc
G = gravimetric RCS in mg/m^3
B_0 = 459 for gold mines; 437 for uranium mines
β_1 = -27.4 for gold mines; -9.25 for uranium mines
β_2 = 159 for gold mines; 86.3 for uranium mines

Based on this equation, Verma et al (1989) generated a table of gravimetric RCS values for konimeter counts ranging from 0 to 1,200 ppcc.

Figure 6

Table for converting RCS concentrations (mg/m^3) to konimeter counts (ppcc) for gold and uranium mines

TABLE A. Table of Conversion Based on the Gravimetric to Konimeter Equation ($K = \beta_0 \cdot (1 - \exp(\beta_1 \cdot G)) + \beta_2 \cdot G$) for Two Ore Types, Using the Parameter Estimates Given in Table II.

	Konimeter count (ppcc)	
Respirable silica (mg/m^3)	Gold	Uranium
0.00	0	0
0.01	112	39
0.02	197	76
0.03	262	108
0.04	312	139
0.05	350	166
0.06	380	191
0.07	403	214
0.08	420	235
0.09	434	255
0.10	445	272
0.11	454	289
0.12	461	303
0.13	467	317
0.14	471	329
0.15	475	341
0.16	479	351
0.17	482	361
0.18	484	370
0.19	487	378
0.20	489	386
0.30	507	436
0.40	523	461
0.50	538	476
0.60	554	487
0.70	570	497
0.80	586	506
0.90	602	515
1.00	618	523
2.00	777	610
3.00	936	696
4.00	1095	782
5.00	1254	869

The WSIB modified the original Verma et al RCS to ppcc conversion table to include RD values based on his estimates of average RCS concentration in the various mines. Corresponding values for RD in nickel, gold and uranium mines were calculated using average RCS values of 5%, 10% and 20% respectively[E]

- nickel mines 0.1 mg/m^3 RCS ~ 2.0 mg/m^3 RD [(0.1 mg/m^3 ÷ 5) x 100 = 2.0]
- gold mines 0.1 mg/m^3 RCS ~ 1.0 mg/m^3 RD [(0.1 mg/m^3 ÷ 10) x 100 = 1.0]
- uranium mines 0.1 mg/m^3 RCS ~ 0.5 mg/m^3 RD [(0.1 mg/m^3 ÷ 20) x 100 = 0.5]

Table 1 in Appendix 10 of the ASMB, reproduced in Figure 7 below allows for the conversion of RCS concentrations (mg/m^3) to particle counts (ppcc) and RD concentrations (mg/m^3).

[E] *Verma et al 1989 observed about a 2-fold higher RCS content in the uranium mine compared to the gold mine where they did the sampling and from unpublished data, he observed that nickel mines had about twice the respirable dust exposures but half the RCS content of respirable dust in gold mines*

Figure 7

Table developed by WSIB to convert RCS concentrations (mg/m^3) to konimeter counts (ppcc) and RD (mg/m^3) for gold and uranium mines

Table 1: Standardized Conversion of Gold, Nickel and Uranium Mine Dust Counts in ppcc to Respirable Dust Levels in mg/m^3

Respirable Silica (mg/m^3)	Gold Mine*		Nickel Mine**		Uranium Mine***	
	ppcc	Resp. Dust (mg/m^3)	ppcc	Resp. Dust (mg/m^3)	ppcc	Resp. Dust (mg/m^3)
0.01	112	0.1	112	0.2	39	0.05
0.02	197	0.2	197	0.4	76	0.10
0.03	262	0.3	262	0.6	108	0.15
0.04	312	0.4	312	0.8	139	0.20
0.05	350	0.5	350	1.0	166	0.25
0.06	380	0.6	380	1.2	191	0.30
0.07	403	0.7	403	1.4	214	0.35
0.08	420	0.8	420	1.6	235	0.40
0.09	434	0.9	434	1.8	255	0.45
0.10	445	1	445	2	272	0.50
0.11	454	1.1	454	2.2	289	0.55
0.12	461	1.2	461	2.4	303	0.60
0.13	467	1.3	467	2.6	317	0.65
0.14	471	1.4	471	2.8	329	0.70
0.15	475	1.5	475	3.0	341	0.75
0.16	479	1.6	479	3.2	351	0.80
0.17	482	1.7	482	3.4	361	0.85
0.18	484	1.8	484	3.6	370	0.90
0.19	487	1.9	487	3.8	378	0.95
0.20	489	2	489	4	386	1.00
0.30	507	3	507	6	436	1.50
0.40	523	4	523	8	461	2.00
0.50	538	5	538	10	476	1.00
0.60	554	6	554	12	487	3.00
0.70	570	7	570	14	497	3.50
0.80	586	8	586	16	506	4.00
0.90	602	9	602	18	515	4.50
1.00	618	10	618	20	523	5.00
2.00	777	20	777	40	610	10.00
3.00	936	30	936	60	696	15.00
4.00	1095	40	1095	80	782	20.00
5.00	1254	50	1254	100	869	25.00

An important observation is that because of the nonlinear relationship between ppcc and mg/m^3, there is a rapid increase of RD and RCS with small increases in ppcc. For example:

for gold mines:
489 ppcc = 2.0 mg/m^3 RD and 0.20 mg/m^3 RCS
507 ppcc = 3.0 mg/m^3 RD and 0.30 mg/m^3 RCS
18 ppcc or a 4 % increase = 50% increase in RD and RCS

for uranium mines:
386 ppcc = 2.0 mg/m^3 RD and 0.20 mg/m^3 RCS
436 ppcc = 3.0 mg/m^3 RD and 0.30 mg/m^3 RCS
50 ppcc or 14 % increase = 50% increase in RD and RCS

Limitations of using the Ontario mining exposure data in the WSIB COPD ASMB

Some of the significant limitations with the mining exposure data in the COPD ASMB include:

1. The tables (e.g., Tables 1 to 9B) are difficult to interpret and no guidance is given to the adjudicator on how to interpret or use these tables. For example, it is not clear how to interpret the range of dust particle counts for jobs in category 4a in Table 7A (range 194 to 2000 ppcc). It is also not clear which semi-annual MAPAO reports were the source for this data.

2. The data from the MAPAO semi-annual reports summarized in the ASMB tables is incomplete and is not an accurate reflection of all the sampling data that is available since the MAPAO collected konimeter measurement data from 1959 until the early 1980s (Verma et al 1989).

3. The particle counts provided in Tables 2C to 9B of the ASMB tables are either overall averages for some of the mining processes or locations where the samples were collected or are the overall average for the individual mines or by type of mine; i.e., all gold, uranium or nickel. The overall mine averages included measurements in locations such as the clean air supply provided by the ventilation system. This results in a much lower average than if it were calculated from only areas where the miners worked (i.e., productions areas). The averages calculated for the mine types (i.e., gold, uranium and nickel) are meaningless because these were calculated by combining the overall averages of all the individual mines within each type (e.g., the overall average for all gold mines was calculated by combining the overall mine averages for each individual gold mine).

4. It is generally assumed that dust exposures in Ontario mines were significantly reduced after 1960 and overall averages for gold mines were considered to be < 300 ppcc by this time. However, this is not supported by the data in the individual MAPAO semi-annual reports that also provided particle counts for specific operations or locations in the mines and the number of samples above 300 ppcc, 500 ppcc and 800 ppcc for each of the locations that were sampled and for the mine overall.

   For example, a review of the MAPAO semi-annual reports for the Kerr-Addison gold mine found that as late as the mid-1970s more than 50% of konimeter samples for drilling and up to 25% of all samples for the mine were above 500 ppcc. This assumption also does not take into account that some mines introduced more effective ventilation and enforcement of practices such as using water sprays to reduce dust exposures during certain processes (e.g., drilling) earlier than other mines.

5. An important consideration is that the conversion from particle counts (ppcc) to mg/m^3 of RD as determined by Verma et al (1989) and reproduced in Table 1 (pg. 69 of the ASMB) is a logarithmic **not** a linear relationship which means that small changes in the estimated dust

concentration in ppcc can have a significant effect on the resulting mass concentration in mg/m^3 that in turn can have a significant effect in the calculation of cumulative RD exposure.

For example, using Table 1, 489 ppcc is equivalent to 2.0 mg/m^3 of RD in gold mines and 523 ppcc is equivalent to 4.0 mg/m^3. Therefore, an increase of only 34 ppcc or 7% increase in the konimeter count results in a 2-fold increase in RD exposure. A gold miner who worked for 15 years at an average concentration of 489 ppcc or 2.0 mg/m^3 (just below the WSIB definition of "dusty gold mining") would have a cumulative RD exposure of 30 mg/m^3 – years and he would not meet the WSIB COPD exposure criteria of 40 mg/m^3 to 50 mg/m^3 – years. However, if his average exposure was determined to be 523 ppcc or 4 mg/m^3, his cumulative RD exposure would be 60 mg/m^3 – years and he would exceed the WSIB COPD exposure criteria.

Dust exposure data for Ontario hard rock miners since the WSIB COPD ASMB

Since the 2001 WSIB COPD ASTM, three additional sources of dust exposure data specific to Ontario mines are now available:

- 2014 Verma et al paper on the 1978-79 Ministry of Labour (MOL) survey of Ontario gold mines
- 2015 Verma et al paper on the 1974 Ministry of Health (MOH) survey of uranium mines
- Ontario Mining Exposure Database (OMED)

Two comprehensive surveys of RD and silica were conducted in 1974 and 1978-79 at Ontario uranium and gold mines by the MOH and MOL respectively; however, these results remained largely unknown and were not readily available until they were summarized and published in the open literature (Verma et al 2014, Verma et al 2015).

Summary of Verma et al 2014 paper

The main objective of the 1978-79 MOL survey of 8 gold mines was to determine the most suitable gravimetric sampling device that could be used for routine sampling in Ontario mines. The main findings of the Verma et al paper included:

- as shown in the summary Table IV reproduced below from the paper, the mean RD & RCS concentration for the 8 gold mines were ~1.0 mg/m^3 and 0.08 mg/m^3 that were below their contemporary MOL Occupational Exposure Limits (OELs) of 3.0 and 0.1 mg/m^3 respectively; however, the maximum values reached 5.73 mg/m^3 for RD & 0.85 mg/m^3 for RCS
- mean RCS concentrations for 7 out of 8 operations/occupations sampled were > 0.025 mg/m^3 (current ACGIH TLV and the proposed new MOL OEL for RCS)

TABLE IV. Summary data from eight gold mines and seven work categories[A] combined for respirable dust, respirable quartz, and percent silica in respirable dust

	Mean	Median	Min	Max
Respirable Dust (mg/m^3)	0.99	0.76	0.08	5.73
Respirable Quartz (mg/m^3)	0.08	0.04	0.01	0.85
% Silica	7.5	5.7	0.10	37.5

[A] Includes six samples from category "others".
Note: The source of these data is a 1978–1979 Study by the Ontario Ministry of Labour.[22]
N = Number of samples Total = 277.

It is unclear whether the 1978-79 MOL survey data appears to have been included in the WSIB COPD ASMB.

Summary of Verma et al 2015 paper

The 1974 MOH survey at 2 uranium mines (Rio Algom & Denison) included ~ 1,000 personal & area dust samples that were collected under normal working conditions. Several methods of dust sampling were used including; midget impingers, konimeters, U.K. Mine Research Establishment (MRE) gravimetric RD samplers, & personal RD samplers (Micronair & Casella) *(this was before size selective gravimetric sampling became the standard method for dust sampling)*.

The paper provided summary tables of ranges of RD and RCS concentrations measured by these different methods for various operations (e.g., drilling, loading, slushing, crushing, milling etc.) at each mine. As summarised in Table 1 below, the tables also provided the percentage of samples that were found to exceed the respective OELs and presents a different interpretation of WSIB's summary of the 1974 MOH survey.

The Verma et al 2015 paper concluded:

> *"The data described in this article would be useful in future epidemiological and health studies. It will also be useful in estimation of exposure for individual miners in the assessment of compensation claims for diseases such as silicosis, chronic obstructive pulmonary disease (COPD), and autoimmune diseases such as renal disease and rheumatoid arthritis."*

Table 1 Summary of Percent of Samples Exceeding OELs by Type of Sampling Method (based on data in Tables 1-5 in Verma et al 2015 paper)						
Sampling Method	**Units**	**Relevant OEL**	**Mine**	**Number of active operations sampled**	**Number of Samples (all operations combined)**	**Percent of samples exceeding relevant OEL**
midget impinger	mppcf	5 mppcf [A]	Rio Algom	7	120	96%-100%
			Denison	7	190	83%-100%
konimeter	ppcc	176 ppcc [B]	Rio Algom	7	185	36%-100%
			Denison	8	276	43%-82%
MRE Gravimetric Respirable Dust Sampler	mg/m^3	0.27 mg/m^3 [C]	Rio Algom	5	28	57%-100%
			Denison	5	9	0%-100%
Micronair Respirable Dust Sampler	mg/m^3	0.27 mg/m^3 [C]	Rio Algom	5	14	50%-100%
			Denison	6	21	50%-100%
Casella Respirable Dust Sampler	mg/m^3	0.27 mg/m^3 [C]	Rio Algom	6	49	54%-100%
			Denison	6	30	86%-100%

[A] respirable dust OEL in mppcf = 300 ÷ (% quartz + 10)

[mean silica content was 46.6% therefore 300 ÷ (46.6 +10) = 5 mppcf]

[B] respirable dust OEL in ppcc = mppcf x 35.2 [therefore 5 mppcf x 35.2 = 176 ppcc]

[C] respirable dust OEL in mg/m^3 = 10 ÷ (% silica + 2) [based on chemical & X-ray diffraction results and a mean silica concentration of 35% was considerable reasonable by the survey authors, therefore 10 ÷ (35 + 2) = 0.27 mg/m^3]

Ontario Mining Exposure Database (OMED)

OMED is a recent database that contains air sampling data for Ontario mines from three sources: (MAPAO) semi-annual reports of konimeter air sampling results from 1959 to 1975[F], gravimetric sampling data collected by the MOL from the late 1970s to early 1990s, and from individual mining companies. The Occupational Cancer Research Centre (OCRC) consolidated these data from paper records into the electronic version of OMED that now includes measurements for over 140 hazards in the Ontario mining industry, totaling more than 119,000 sampling records collected over the period 1950-1991 (Blagrove-Hall et al 2021). Approximately 65% of the records in OMED are for RD, RCS, and radon. The remainder of

[F] *MAPAO collected semi-annual reports from Ontario mines from 1959 to 1982*

the records are for a wide range of other contaminants in the mining environment such as various metal dusts and diesel exhaust.

OMED has been developed further by OHCOW to make it searchable using a number of criteria (i.e., mine, type of contaminant, type of sample, task or location, etc.). Although some sampling data are available from the mid-1950s, the majority of data are from 1970 to 1991. More than 4,200 RCS measurements from gold, uranium, and nickel mines are contained in the OMED database.

A 2021 OCRC paper that analyzed the RCS data in OMED from 1974 to 1991 concluded that although RCS exposures generally decreased during this period likely due to increased regulatory requirements, improved ventilation and other exposure control methods, the probability of overexposure to RCS remained high in Ontario's mining industry (Blagrove-Hall et al 2021). It is generally believed that surface miners do not experience the same high levels of RCS exposures as underground miners. However, the OCRC analysis found that there was no overall difference in the mean RCS concentrations of surface miners compared to underground miners and that some of the highest measurements were observed in the surface operations such as crushing.

A review of the MAPAO data contained in the Occupational Mining Exposure Database (OMED) found that there is significantly more data available for individual mines and for specific processes or tasks than is reflected in the WSIB COPD ASMB. For example, for one gold mine alone (Kerr Addison) there are at least 6500 individual measurements for 29 exposure categories mostly for the period 1969-1983 with some konimeter sampling data available from as early as 1952. In addition, OMED includes some of the sampling data collected by the Ontario Ministry of Labour and individual mining companies.

OMED may be a very useful tool to estimate an individual miner's exposure to RCS or other contaminants and can provide much more information than what is outlined in the WSIB ASMB for COPD.

A limitation of OMED is that sampling data may not be not be available for all mines, jobs or tasks or all time periods. However, in situations where specific information is not available, a broader search may be used. For example, if there are some years in which limited or no sampling data is available for an operation or job task at a specific gold mine, a wider search may be used that combines the sampling data from all gold mines. In addition, the OHCOW version of OMED allows for some statistical analysis such as calculating the mean, standard deviation or confidence intervals for a set of sampling data.

WSIB Scientific Review (SR)

The Introduction to the SR document in the WSIB COPD ASMB states:

> *This module reviews the literature published after 1980 on dust and COPD. Based on this information, this module assesses the causal connections between exposure to mineral dust (silica, cement, coal and asbestos) or mixed inorganic dusts related to industrial processes (smelting, foundry and welding operations) and the development of impairment due to COPD.*

The SR included retrospective cohort and case-control studies as well as some literature reviews.

The purpose of the SR is to support the information in the AA section in the COPD ASMB. The SR states, "We have reviewed the epidemiological studies of COPD undertaken since 1980." The full date range that was considered in the literature search is not explicitly stated; however, a review of the reference list at the end of the document suggests that the latest article that was included was from 2000. The section "Dust Containing Silica; Hard Rock Dust" included 16 studies published between 1986 and 1999. Therefore, the literature review on which the COPD ASMB is based is at least 20 years old. A considerable number of scientific papers on occupationally related COPD have been published during this period.

To estimate the volume of literature that has been published since the 2010 WSIB COPD SR document that supports the advice and information in the COPD ASMB used by occupational disease (OD) adjudicators, a PubMed search was done for the period January 1, 2000 to December 9, 2021. Using the terms "chronic obstructive lung disease" OR "COPD" AND "occupational exposure" and limiting the search to articles on humans written in English with at least an abstract available resulted in 1,235 citations. Narrowing the search using the terms "chronic obstructive lung disease" OR "COPD" AND "mining" with at least an abstract identified 245 papers published since 2010.

A recent review of the epidemiological literature on occupational exposure and COPD titled "Chronic Obstructive Pulmonary Disease: Occurrence and Associations with Occupational Exposures and Smoking" was prepared for OHCOW by Dr. Christine Oliver (2020) and should be considered as a companion document to this review. In addition, the WSIB commissioned an independent scientific review, that was released on February 1, 2021, on issues associated with the interaction between smoking and occupational exposure to RD and the broader category of vapours, gases, dust and fumes (VGDF) exposures (WSIB 2021a). [This review showed interactive effects between smoking and occupational exposures on risk for COPD to be at least additive and in some case, multiplicative.] As a result, WSIB will no longer reduce non-economic loss and permanent disability benefits for workers with COPD claims who also have a smoking history (WSIB 2021b).

A closer review of the studies on COPD related to RD and RCS in hard rock mining considered in Section 4 of the COPD ASMB and additional, more recent published studies found that there is a significantly increased risk of COPD morbidity due to emphysema, chronic bronchitis and/or pulmonary function impairment, as well as increased mortality due to COPD at cumulative RD concentrations below 40 to 50 mg/m^3- years.

The present review will also consider the contribution of exposure to aluminum McIntyre powder (MP) that was used a prophylactic measure to prevent silicosis among Ontario miners to the cumulative RD exposures as well as the association between exposure to diesel exhaust (DE) or other vapours, gases, dust and fumes (VGDF) and COPD.

As will be described below, Section 4 of the WSIB COPD SR document focused mainly on South African gold mining studies; however, the limitations associated with these studies may have resulted in an underestimation of the exposures to RD and RCS.

Limitations of Dust Exposure Assessments in the South African Gold Mining Studies

In contrast to the conversion equation derived by Verma et al (1989) used for estimating RD and RCS exposures in Ontario mines, the conversion of particle counts to respirable mass concentrations in the South African gold mining studies were derived by Beadle and Bradley (1970) and summarized by Page-Shipp and Harris (1972) using largely theoretical approaches. To date, the theoretical conversion of particle number concentrations using konimeters or thermal precipitators to respirable mass concentrations as measured using modern size-selective gravimetric sampling instruments such as cyclones, has not been validated with side-by-side measurements in South African gold mines as was done by Verma et al (1989) for Ontario gold mines. Some of the issues associated with converting particle counts (ppcc) into respirable mass units (mg/m^3) are discussed in further detail later in this document.

The dust exposure estimates in many epidemiological studies of South African gold miners were based on a survey undertaken in 1956–60 by Beadle and Bradley (1970). The objective of the survey was to establish average shift-long dust particle exposures for mining workers grouped into 11 occupational categories. Konimeters and thermal precipitators (standard and modified[G]) were used to collect dust samples in 20 gold mines that were randomly selected. It was estimated that the numbers of konimeter samples and modified thermal precipitator (MTP) samples were about 22,000 each. In addition, 650 standard thermal precipitator (STP) samples were also collected.

The konimeter, MTP and STP instruments were carried and operated by an observer who accompanied the miner whose exposure was being measured. In all, 650 men were each followed through a single

[G] *Modified thermal precipitators were smaller than the standard thermal precipitators which made them more portable to allow sampling in more areas of the mines.*

work shift to approximate personal exposures to dust and precautions were to taken to ensure the work patterns of the miners were typical of normal practice. Konimeter samples were taken at 10-minute intervals (6 samples per hour) throughout the shift and during the operations in progress in the vicinity of the sampling. The STP samples were taken intermittently throughout the shift and the MTP instruments operated throughout the full work shift and collected individual samples with a duration of 10 minutes each (6 samples per hour). Table 2 summarizes the konimeter sampling results reported in Table II and Table III in Page-Shipp and Harris (1972).

Table 2 Summary of Konimeter Sampling Data from Beadle and Bradley (1970) as summarized in Tables II and III of Page-Shipp and Harris (1972)													
Data	**Occupational Group**												
	1. Shaft Sinkers	**2. Developers**	**3. Stopers**	**4. Assistant Miners Trammers** (incl timbermen, locomotive drivers, pipes & tracks)	**5. Shift Bosses** (incl Learner Officials)	**6. Other Officials** (incl mangers, ventilation & other engineers, surveyors, samplers)	**7. Banksmen/ Skipsmen/ Onsetters**	**8. Workers near Shafts** (incl shaft timbermen, winding, engine drivers, pump attendants)	**9. Boiler-makers** (incl truck repairers)	**10. Other Artisans** (incl carpenters electricians, fitters, masons, riggers, handymen)	**11. Misc.** (incl diamond drillers, blasters, sanitation)	**Production Groups (1-5)**	**All Groups (1-11)**
Mean Dust Levels[H] [measured by konimeter (ppcc) after acid treatment]													
Mean	280	210	210	180	160	200	140	100	210	150	160	208	180
Standard Deviation	270	120	100	100	90	120	180	90	170	130	90	206	120
Range	50-800	50-600	0-600	0-650	50-350	0-750	0-850	0-400	0-700	0-850	0-350	0-800	0-850
Daily Dust Exposures[I] [measured by konimeter (ppcc x hours per shift - ppcc-hours) after acid treatment]													
Mean Shift length (hours)	7.7	8.0	7.8	7.7	5.2	4.0	7.5	6.5	6.3	5.7	7.2	7.3	6.7
Mean	2130	1780	1560	1360	860	810	1030	760	1230	800	1030	1538	1190
Standard Deviation	1740	1030	830	870	470	560	1310	790	920	590	570	988	900
Range	500-6500	500-5000	0-5000	0-5000	500-2000	0-3000	0-6500	0-3500	0-4500	0-3000	0-2500	0-6500	0-6500

[H]Mean Dust Levels = mean of konimeter samples collected at 10-minute intervals every hour (6 samples per hour)

[I] Daily Dust Exposures = mean shift-long dust levels x mean shift length in hours: e.g., for shaft sinkers; mean daily dust exposure = 280 ppcc x 7.7 hours = 2130 ppcc-hours

Page-Shipp and Harris (1972) summarized the konimeter data gathered in the 1956–60 survey by Beadle and Bradley (1970) as shown above in Table1. They observed the following from the mean-shift-long dust levels:

- Although the actual values ranged as high as 850 ppcc, the mean values did not exceed 250 ppcc.
- The range of exposures in any occupational group was very wide which the authors attributed to differences in mining methods (e.g., high or low speed development) and variability between tasks done by the miners during sampling. The wide dispersion of the values from the mean are also illustrated by the large values in the standard deviations.

They also summarized the mean dust levels measured in the 1956 to 1960 survey by Beadle & Bradley (1970) for operations in progress at the locations where the konimeter samples were collected (e.g., drilling, scraping, tramming etc.) for each of the 11 occupational groups. For example, 13 locations and operations where shaft sinkers worked were assessed, a total of 480 konimeter samples were collected and the overall mean dust level was 252 ppcc. For further details the reader is referred to Table IV of Page Shipp and Harris (1972).

Page-Shipp and Harris (1972) put greater emphasis on the konimeter sampling because at that time konimeter sampling results were most meaningful to the South African mining industry. Gravimetric sampling using size-selective instruments such as cyclones were not developed until the early 1980s. Since mostly konimeter sampling results were available, they were used extensively for epidemiological studies of South African gold miners, despite their limitations.

The paper also estimated respirable mass in milligrams per cubic meter (mg/m^3) from 649 standard thermal precipitator samples collected in the 1956-1960 survey by Beadle & Bradley (1970) through a series of calculations as described in Appendix 1 of Page-Shipp and Harris (1972) and by Gibbs and DuToit (1992). The following is a brief description of the main steps in obtaining particle counts and converting these into respirable mass. Full details of these steps, including a worked example for converting particle counts into mass concentration (mg/m^3) for a shaft sinker using the Beadle and Bradley (1970) data, are provided in the paper by Gibbs and DuToit (1992).

1. The dust sample on the thermal precipitator glass cover slip was ignited to remove any combustible particulate matter
2. Total particles and respirable sized particles with diameters in the range 0.5-5.0 micrometers (μm) were counted under a microscope.

3. The thermal precipitator coverslip was then acid washed to remove any acid soluble material. It was assumed that what remained was mostly crystalline silica (quartz), silicates (e.g., mica) and metal oxides.
4. Total particles and respirable sized particles with diameters in the range of 0.5-5.0 micrometers (µm) were counted again and recorded as "after acid treatment" respirable particle concentration as particles per cubic centimeter (ppcc), respectively.
5. The projected surface area[J] of the total and RD particles before and after acid treatment was calculated in units of square micrometers per cubic centimeter ($\mu m^2/cc$).
6. The respirable surface area (RSA) of particles ($\mu m^2/cc$) before and after acid treatment was calculated (see Appendix in Gibbs and Du Toit 2002).
7. The conversion of RSA ($\mu m^2/cc$) into respirable mass in milligrams per cubic meter (mg/m^3) involved a series of complex calculations based on theoretical assumptions[K].

A more simplified conversion equation was developed as described in Hnizdo et al (1992); respirable mass (mg/m^3) = 0.027 x $(RSA)^{0.474}$. Hnizdo et al (1992) concluded that this equation explained 76% of variation in RSA and that this formula was applicable to occupational group means. They also concluded that the error associated with the conversion from RSA over the range 100-1000 $\mu m^2/cc$ is generally less than 0.1 mg/m^3. For example, when this equation is applied to the mean respirable surface area of 609.1 $\mu m^2/cc$[L], after acid treatment, for shaft sinkers as summarized in Appendix 1 of Page-Shipp (1972), the result of 0.56 mg/m^3 is in close agreement with the result of 0.57 mg/m^3 using the more complex calculations ($0.027 \times 609.1^{0.474} = 0.56$).

Concerns were raised that the RD and RCS exposures in South African gold mines were underestimated when compared to North American hard rock mines. Hughes and Weill (1995) estimated that the risks of silicosis after 40 years of exposure to 0.1 mg/m^3 of RCS based on studies of Ontario miners by Muir et al (1989) and South African gold miners by Hnizdo and Sluis-Cremer (1993) were 1.2% and 60% respectively. Hughes and Weill (1995) suggested that one possibility for this large difference in silicosis risk for the same RCS exposure was due to the South African exposures being underestimated. Another possibility may have been in the difference in RCS concentration in the RD reported in Ontario gold mine (6%) and South African gold mines (30%).

[J] The method used to convert particle number concentrations to surface area and to respirable mass was largely based on the work of Sichel (1957), Joffe and Sichel (1965) and is summarized in Appendix II of Page-Shipp and Harris (1972).

[K] Full details of the conversion of RSA($\mu m^2/cc$) into respirable mass in milligrams per cubic meter (mg/m^3) are described in Page-Shipp and Harris (1972) and Gibbs and Du Toit (2002).

[L] Respirable surface area of 4,690 $\mu m^2/cc$ adjusted for average of 7.7 hours per shift (4,690 ÷ 7.7 = 609.1 $\mu m^2/cc$)

A study by Gibbs and DuToit (2002) reviewed the Page-Shipp and Harris (1972) summary of the South African gold mining sampling results gathered by Beadle (1970) and examined the factors that may have been responsible for the underestimation of RD and RCS exposures.

Table 3 that follows is a summary of the RD and RCS concentrations in South African gold mines from Page-Shipp and Harris (1972) and Gibbs and DuToit (2002) and the discussion that follows refers to this table.

Table 3 Summary of RD and RCS Concentrations in South African Gold Mines from Page-Shipp & Harris (1972) and Gibbs & DuToit (2002)						
Occupation	**A Respirable Dust Concentration (mg/m^3) as estimated by Page-Shipp & Harris (1972) from Beadle (1970) data mean (range)**	**B Respirable Dust Concentration (mg/m^3) adjusted by Gibbs & DuToit (2002) mean (range)**	**C Respirable Dust Concentration (mg/m^3) equivalent personal cyclone sampling results estimated by Gibbs & DuToit (2002) mean (range)**	**D Respirable crystalline silica (RCS) Concentration (mg/m^3) (assuming 30% silica) based on Page-Shipp & Harris (1972) data mean (range)**	**E Respirable crystalline silica (RCS) Concentration (mg/m^3) (adjusted to 54% silica) estimated by Gibbs & DuToit (2002) mean (range)**	**F Respirable crystalline silica (RCS) Concentration (mg/m^3) (adjusted to 15% silica) estimated by Gibbs & DuToit (2002) mean (range)**
1. Shaft Sinkers	0.58 (0.10-1.56)	1.04 (0.18-2.81)	2.13 (0.90-5.80)	0.17 (0.03-0.47)	0.56 (0.10-1.52)	0.32 (0.14-0.87)
2. Developers	0.25 (0.04-1.00)	0.44 (0.07-1.80)	0.90 (0.14-3.71)	0.08 (0.01-0.30)-	0.22 (0.04-0.97)	0.14 (0.02-0.56)
3. Stopers	0.20 (0.03-0.77)	0.35 (0.05-1.39)	0.76 (0.11-3.02)	0.06 (0.01-0.23)	0.19 (0.03-0.75)	0.11 (0.02-0.45)
4. Assistant Miners/Trammers (incl timbermen, locomotive drivers, pipes & tracks)	0.16 (0.00-1.03)	0.27 (0.00-1.85)	0.56 (0.00-3.77)	0.05 (0.00-0.31)	0.15 (0.00-1.00)	0.08 (0.00-0.28)
5. Shift Bosses (incl Learner Officials)	0.17 (0.04-0.77)	0.29 (0.07-1.39)	0.65 (0.16-3.16)	0.05 (0.01-0.23)	0.16 (0.04-0.75)	0.10 (0.01-0.21)
Occupations 1-5 classified by Page-Shipp & Harris (1972) as production	0.27 (0.00-1.56)	0.48 (0.00-2.81)	1.00 (0.00-5.80)	0.08 (0.00-0.47)	0.26 (0.15-0.56)	0.15 (0.00-0.87)
6. Other Officials (incl mangers, ventilation & other engineers, surveyors, samplers)	0.19 (0.03-1.00)	0.29 (0.05-1.80)	0.64 (0.11-4.05)	0.06 (0.01-0.30)	0.16 (0.03-0.97)	0.10 (0.00-0.27)
7. Banksmen/Skipsmen/Onsetters	0.17 (0.00-1.07)	0.31 (0.00-1.93)	0.53 (0.00-3.38)	0.05 (0.00-0.32)	0.17 (0.00-1.04)	0.08 (0.00-0.28)
8. Workers near shafts (incl shaft timbermen winding engine drivers, pump attendants)	0.09 (0.00-0.62)	0.14 (0.00-1.12)	0.33 (0.00-2.61)	0.03 (0.00-0.19)	0.08 (0.00-0.33)	0.05 (0.00-0.17)
9. Boilermakers (incl truck repairers)	0.16 (0.02-0.63)	0.27 (0.04-1.12)	0.68 (0.10-2.84)	0.05 (0.01-0.19)	0.15 (0.02-0.61)	0.10 (0.01-0.17)
10. Other Artisans (incl carpenters, electricians, fitters, masons, riggers, handymen)	0.11 (0.02-0.70)	0.18 (0.04-1.26)	0.37 (0.08-2.56)	0.03 (0.01-0.21)	0.10 (0.02-0.68)	0.06 (0.01-0.19)
11. Misc. (incl diamond drillers, blasters, sanitation)	0.14 (0.00-0.56)	0.22 (0.00-1.01)	0.39 (0.00-1.81)	0.04 (0.00-0.17)	0.12 (0.00-0.54)	0.06 (0.00-0.15)
Total (Occupations 1-11)	0.17 (0.00-1.56)	0.27 (0.00-2.81)	0.56 (0.00-5.80)	0.05 (0.00-0.47)	0.15 (0.00-1.74)	0.09 (0.00-0.87)

Column A = Time Weighted Average (TWA) concentration calculated as respirable mass (mg/m^3) after acid treatment from Appendix 1 in Page-Shipp & Harris adjusted for average hours worked per shift typically used in epidemiological studies of South African gold mines
Column B = TWA respirable dust concentrations recalculated by Gibbs and DuToit (2002) [values rounded to 2 decimal places from original 3 decimal places in Table 4 of Gibbs & DuToit (2002)]
Column C = Column B x ratio of respirable surface area before and after acid treatment as shown in Table 3 of Gibbs & DuToit (2002) who concluded that this was the closest approximation to respirable dust concentrations that would be measured using a size-selective personal cyclone sampler
Column D = RCS concentration based on assumed 30% silica content by Beadle (1970) and Page-Shipp & Harris (1972) (Column C x 0.30)
Column E = (Column B x 0.54) Estimated RCS concentration based on adjustment to 54% RCS content of acid treated respirable dust as estimated in Table 4 of Gibbs & DuToit (2002)
Column F = (Column B x 0.15) Estimated RCS concentration based on 15% silica content as estimated in Table 4 of Gibbs & DuToit (2002)
Columns A to F are TWA concentrations over the average duration of the shifts
Shaded columns C, E and F are the corrected concentrations of respirable dust and RCS as estimated by Gibbs and DuToit (2002) from the Beadle (1970) and Page-Shipp & Harris (1972) data, and are more likely representative of the actual concentrations of respirable dust and RCS used in the South African gold mining studies

Factors Contributing to Underestimation of South African RD and RCS Exposures

1. Inaccuracies in converting particle counts to respirable mass concentrations

When the complex calculations that were based on theoretical assumptions to convert respirable surface area RSA ($\mu m^2/cc$) into respirable mass in milligrams per cubic meter (mg/m^3) were reviewed by Gibbs and DuToit (2002), they found some inaccuracies in the surface area and volume calculations. The recalculated respirable mass concentrations from Appendix 1 in Page-Shipp and Harris (1972) are shown in column B of Table 3 above (column A are the original values).

For example, the recalculated mean RD concentration for shaft sinkers increased to 1.04 mg/m^3 compared to 0.57 mg/m^3 from the Beadle (1970) data summarised in Appendix 1 of Page-Shipp and Harris (1972). This illustrates that the RD exposure for shaft sinkers was underestimated by a factor of 1.8 (1.04 mg/m^3/0.57 mg/m^3). After applying this correction factor to all the 11 exposure categories it was found that the average RD exposures were underestimated by a factor of 1.6.

2. Respirable mass concentration estimates

The respirable mass concentrations were calculated by Page-Shipp and Harris (1972) from particle counts of the respirable sized particles (0.5–5 µm) after incineration and acid washing. As noted earlier, the acid washing process removes any acid soluble particles such as silicates and metal oxides. RD concentrations using size-selective gravimetric instruments such as cyclones measure the concentration of all the RD (equivalent to pre-acid washed respirable particle samples). Thus, comparisons between South African studies using particle measurements and other studies that used gravimetric measurement of RD may be difficult.

Gibbs and DuToit (2002) determined that the airborne RD concentrations reported in the South African gold mining studies may be adjusted by multiplying the acid treated RD concentrations by the ratio of the respirable particle concentrations before acid washing to that after acid washing. Various ratios were examined and pre-acid-treated particle concentrations were found to be approximately twice the post-acid-treated concentrations. However, using a common conversion factor of about 2 to convert respirable acid-washed concentrations to respirable non-acid-washed gravimetric concentrations assumes that particles removed by acid treatment (i.e., soluble particles) have the same particle size distributions and densities as those not removed by acid treatment (i.e., insoluble particles).

Gibbs and DuToit (2002) estimated that the corrected RD concentrations shown in Column B in Table 3 multiplied by the ratio of the respirable surface area before and after acid treatment are probably the closest approximation to those that would be measured using a personal cyclone sampler as shown in Column C of Table 3.

In summary, according to the work by Gibbs and DuToit (2002), the RD concentrations used in the South African gold mining studies are underestimated by a factor of 2 when adjusted from the original values

reported by Beadle (1970) and Page-Shipp and Harris (1972). Estimates of equivalent gravimetric concentrations that would be measured by a personal cyclone sampler would increase the original respirable mass concentrations by an additional factor of about 2.

3. Assuming the RD contained 30% RCS

After studies of silicosis in Ontario miners were published in 1989 [Muir et al (1989a, 1989b), Verma et al (1989)], concerns were raised by Hughes and Weill (1995) that the risk of silicosis in South African gold miners as reported by Hnizdo and Sluis-Cremer (1993) may have been over-estimated because of underestimates of RCS exposures.

The epidemiological studies of South African gold miners by Hnizdo and others, typically estimated the RCS content of the RD by multiplying the heat-treated, acid washed RD mass concentrations by 30% as estimated in the Beadle and Bradley (1970) data. However, Beadle and Bradley (1970) reported that the percentage of RCS in the dust before heat treating and acid-washing was 30%. Gibbs and DuToit (1992) observed that after heat-treating and acid washing, the percentage of RCS increased to 54%. Therefore, applying 30% as the percentage of RCS in the heat-treated, acid-washed RD concentrations would result in the RCS exposures being underestimated by a factor of 1.8 (54%/30%).

Gibbs and DuToit (2002) estimated that the RCS exposures reported in the study by Hnizdo and Sluis-Cremer (1993) were probably underestimated by a factor of between 1.3 and 2.3. They concluded if it is assumed that the RCS exposure levels in the Hnizdo and Sluis-Cremer (1993) silicosis study were underestimated by a factor of 2, the adjusted cumulative risk of 1.4% of silicosis would have corresponded to a cumulative RCS exposure of ~2.7 mg/m^3-yr. This brings the risk estimate closer to the risk estimated by Hughes and Weill (1995) for Ontario gold miners (i.e., 1.2% risk of silicosis at a cumulative RCS exposure of 4.0 mg/m^3-yr).

4. Other Factors

Gibbs and DuToit (2002) also identified two additional factors that may have contributed to the underestimation of RD and RCS concentrations in South African gold mines.

Mine Differences

Page-Shipp and Harris (1972) assumed that miners doing the same type of work in different mines were exposed to the same dust levels. However, dust controls at some mines were considerably better than at others. Mine differences were not taken into account when assessing exposures in the epidemiological studies of South African gold mines.

Mining Conditions

As reported by Gibbs and DuToit (2002), dust conditions in South African gold mines have generally been assumed to be lower than in other gold mines for two reasons. Firstly, mining regulations as early as 1911 required wet mining methods such as using water sprays during rock drilling and removal of broken

rock. Secondly, the large Witwatersrand gold mining operations (included in many of the South African gold mining studies) involved deep mines. By 1954, stoping operations at the large Witwatersrand gold mines were being done at a depth of 2,749 meters and development was being done at depths up to 2,960 meters. By 1965 stoping was being done at 3,002 meters and development at 3,304 meters. Because of the heat at these great depths, considerable volumes of ventilation air were necessary to cool the mines and that would have also reduced dust exposures.

Page-Shipp and Harris (1972) assumed that the dust conditions did not vary greatly in the period 1935-1960 based on mean dust levels reported by the South African Chamber of Mines for this period. However, as summarised in Gibbs and Du Toit (2002), the dust concentrations began to increase after the period 1957 to 1960 to levels like those measured in 1941, likely due to increased production and better instrumentation. Gibbs and DuToit (2002) estimated that these assumption about the dust levels over time in the South African gold mines may contribute a factor of about 1.3 to the underestimation of the true dust levels.

Implications for the WSIB COPD Exposure Guideline

If the corrections to the RD and RCS suggested by Gibbs and DuToit (2002) were applied to the COPD studies of South African gold miners considered in the WSIB COPD Adjudicative Support Material Binder [Wyndham et al (1986), Hnizdo et al (1990, 1991), Reid and Sluis-Cremer (1996)], adjusting the cumulative RD and RCS exposures by a factor of 2, still results in statistically significant COPD at cumulative RD or RCS exposures well below the WSIB COPD guideline of 40 to 50 mg/m^3 – years.

For example, Reid and Sluis-Cremer (1996) estimated a statistically significant doubling in the risk of COPD mortality among smoking gold miners after 27 years of underground exposure to 0.14 mg/m^3 resulting in a cumulative RD exposure of 3.8 mg/m^3 – years. Increasing the cumulative dust exposure by a factor of 2 to 7.6 mg/m^3 – years, is still well below the WSIB guideline of 40 to 50 mg/m^3 – years.

If an additional correction factor of ~ 2 were applied to the RD values used in the South African gold mining studies in order to estimate the equivalent personal cyclone measurements as calculated by Gibbs and DuToit (2002) and shown in Column C in Table 3, the cumulative RD exposures associated with statistically significant increased risk of COPD morbidity or mortality are still below the WSIB COPD exposure guideline (7.6 mg/m^3 – years x 2 = 15.1 mg/m^3 – years).

Using the Reid and Sluis-Cremer (1996) study referred to above, a 2-fold increased risk of dying from COPD would be associated with a cumulative RD exposure of 14.4 mg/m^3 – years.

The effect of adjusting the cumulative RD and RCS exposures on the risk of COPD is discussed further in the Review of Epidemiological Findings section of this document.

Review of Epidemiologic Findings for the Association between Hard Rock Mining Exposures and COPD

RD and RCS

Table I in Section 4 “Dust Containing Silica; Epidemiologic findings for hard rock dust” of the SR section of the WSIB COPD ASMB summarizes the findings from 9 mining studies. Six were studies of South African gold miners (Reid & Sluis-Cremer 1996, Wyndham et al 1986, Hnizdo et al 1990b, Hnizdo et al 1991, Becklake et al 1987 and Cowie & Mabena 1991); one, of Australian gold miners (Holman et al 1987); one, of Colorado uranium miners (Roscoe et al 1997) and one, of Colorado hard rock miners (Kreiss et al 1989). The table included a 1993 review by Oxman et al that compared the evidence for COPD from 8 studies of British coal miners and 3 of south African gold miners (Hnizdo et al 1990a, Hnizdo et al 1990b and Hnizdo et al 1991)

Table 1 also included studies of stone workers (Costello et al 1995), diatomaceous earth miners and millers (Checkoway et al 1993), English and Scottish heavy clay industry workers (Love et al 1999), and Quebec workers exposed to silica and asbestos (Begin et al 1995).

Only mining studies that assessed COPD associated with exposure to RD or respirable silica are considered in the following discussion as they are most relevant to Ontario hard rock miners. The South African gold mining studies were based on data gathered between 1969-1987 by the South African Medical Bureau for Occupational Diseases (MBOD) that required an annual medical examination for all miners who worked in dusty occupations.

The studies summarized in Table I in Section 4 of the ASMB are difficult to compare for several reasons:

- studies of different types of COPD (emphysema and chronic bronchitis) and aspects of COPD (pulmonary function impairment and mortality) are all grouped together.
- risk estimates are difficult to compare as studies include cohort studies, case-control studies and cross-sectional studies that estimated risks in different ways[M]. The risk estimates in Table I include: relative risk (RR), standardized mortality ratio (SMR) and odds ratios (OR).

M *In cohort studies, populations exposed to a specific risk factor are followed forward in time (examined prospectively), or examined retrospectively over time, and outcomes of interest are compared with an unexposed reference population. Data from cohort studies may include incidence of disease and/or mortality. Statistical tests of significance that may be performed include standardized incidence and mortality rates (SIR and SMR), relative risk (RR), and hazard ratio (HR). SIR and SMR are generally standardized by comparison with the general population using a demographic variable such as age.*

In case-control studies, persons suffering from the disease of interest are compared with controls (or referents) who do not have the disease. Exposure is recorded retrospectively. The odds ratio (OR) is calculated as a comparative probability of having a given exposure (e.g., RCS for lung cancer).

Case-control studies are often performed when the outcome of interest is rare (e.g., angiosarcoma of the liver). Exposures are assessed retrospectively in cases and controls and the “odds” of having a particular exposure compared and the significance of the difference analyzed.

- confidence intervals are not included with the risk estimates
- quantitative RD or RCS data or estimates of cumulative exposure are not consistent; units include; mg/m^3, mg/m^3 – years, particle-years, particle-years/1000, gram-hours/m^3
- not all of the findings from the studies are contained in the Measure and Relative Risk or Comments columns in Table I

The following discussion of the studies included in Table I in Section 4 of the ASMB and other relevant studies address some of these issues by:

- separating the studies on COPD into more specific categories such as; emphysema, chronic bronchitis, pulmonary function impairment and mortality
- providing more analysis of these studies than is provided in the WSIB SR and AA documents
- presenting the risk estimates in a more understandable way
- including the confidence intervals as reported in the original articles
- wherever possible converting units of RD or RCS exposure and cumulative exposure into more commonly used units of mg/m^3 and mg/m^3 - years

Similar to the approach taken by the National Institute for Occupational Safety and Health (NIOSH) (2002) and the Occupational Safety and Health Administration (OSHA) in its discussion of silicosis in 2016, the following discussion of the epidemiologic findings on RD and RCS and COPD is separated into the three main aspects of COPD morbidity: emphysema, chronic bronchitis, pulmonary function impairment, and COPD mortality.

Emphysema

Of the four studies that are included in this section, only two, Becklake et al (1987) and Hnizdo et al (1991) and the 1993 review by Oxman et al are included in the WSIB COPD SR document.

Becklake et al (1987)

An early case-control study by Becklake et al (1987) of emphysema in white South African gold miners (44 cases of emphysema, and 42 controls without emphysema), found that the strongest predictors of emphysema at autopsy were number of shifts worked in high dust jobs, smoking, and age.

Exposure information was derived from service records at the approximately 80 gold mines where the miner may have worked and were reported to the Medical Bureau for Occupational Disease (MBOD) in South Africa. All job titles were categorized as high, medium, low or surface dust exposure using data collected and reported by Beadle and Bradley (1970) and summarized by Page-Shipp and Harris (1972). Number of shifts worked in high dust, calculated on the basis of 280 shifts per year, was also calculated

In cross-sectional studies, the exposure and disease status are examined at a given point in time for a defined population. Disease prevalence but not disease incidence can be determined and risk factors identified.

by decade (before 1950, 1950-1960, 1961-1970, 1971-1980). However, the dust exposures in particle counts or equivalent gravimetric units were not provided.

Multiple logistic regression analysis showed that age, average number of cigarettes consumed before 1960 and total number of shifts worked in high dust were important and independent predictors of emphysema at autopsy. The presence or severity of silicosis had no effect on the prediction of emphysema at autopsy. The main finding of this study was that a miner who had worked in high dust for 20 years had a nearly 13-times statistically significant greater chance of having grade 2 or higher emphysema at autopsy than a miner who had never worked in high dust exposures; odds ratio (OR) = 12.7; 95% confidence interval (CI) 3.0 to 52.0.

de Beer et al (1992)

A reanalysis of the data from the Becklake et al (1987) study by de Beer et al (1992) added back cases and controls thought to have been removed because of possible selection bias in the original study. The cases that had been originally excluded worked fewer shifts in high-dust jobs. There was no difference in number of shifts in high-dust jobs between the controls in the initial data and those added back.

After including these new cases, miners who had worked in high-dust jobs for 20 years still had a statistically significantly greater chance of having emphysema at autopsy, but the OR decreased from 12.70, 95% CI 3-52 to 3.06, 95% CI 1.14-8.23. The authors concluded that their study “highlights the need to maximize the inclusion of eligible subjects and the danger of differential exclusion of subjects in an analytical study”.

The study by deBeer et al found that South African gold miners working for 20 years in jobs with high RCS dust exposure had a greater than 3-fold increase in the risk of emphysema (RR = 3.5, 95% CI 1.6 to 6.6)

Hnizdo et al (1991)

Hnizdo et al. (1991) conducted a retrospective analysis of South Africa’s MBOD computerized records of 1,553 white gold miners who had undergone autopsy examinations between 1974 and 1987, who were 40 years of age or more, who had worked in underground gold mines for ≥ 10 years and any other type of mine < 2 years. Emphysema grade was assigned as none, moderate (≥ 35%) or marked (≥ 65%) and sufficient data and smoking history were available from the MBOD annual medical examination records.

For each miner, information was available on the number of shifts worked in each occupation and mine. Occupations were grouped into five categories that were assigned dust factors proportional to previously evaluated RD measurements by Beadle and Bradley (1970) and Page-Shipp and Harris (1972). Each miner’s dust exposure was calculated in terms of number of dusty shifts, years spent in high-dust

occupations, and cumulative dust exposure. These three dust exposure measures were cumulated to 35, 45, and 55 years of age and to death.

After controlling for smoking, it was found that years of high dust exposure and cumulative dust exposure up to 45 years of age were the strongest predictor of emphysema. Using the duration of exposure variable, the authors estimated that a gold miner who smoked and worked for 20 years in a high-dust occupation up to 45 years of age had a statistically significant 3.5 (95% CI 1.7 to 6.6) times higher odds of having a moderate to marked degree of emphysema at autopsy than a miner not working in a dusty occupation.

The authors reported that the average dust exposure for miners working in high-dust occupations such as shaft-sinkers and developers was about 3.12 mgh/m^3 (milligram-hours/cubic meter) over an 8-hour shift or about 0.4 mg/m^3 as a time-weighted average (TWA) (i.e., 3.12 mgh/m^3 ÷ 8 = 0.4 mg/m^3). This is in good agreement with the values derived from the Page-Shipp and Harris data summarized in Table 3 above (shaft sinkers 0.58 mg/m^3 + developers 0.25 mg/m^3 ÷ 2 = 0.415 mg/m^3).

The cumulative RD exposure for a miner who worked for 20 years in a high-dust exposure occupation can be estimated to have been about 8.0 mg/m^3-years (20 years x 0.4 mg/m^3) which is well below the current WSIB COPD exposure guideline of 40 to 50 mg/m^3 - years.

No association between dust exposure and emphysema was observed in nonsmokers. However, there were only four non-smokers in the cohort; two spent about 15 years in high dust exposures and the other two had very few dusty shifts.

In terms of RCS, it may be estimated that gold miners who smoked and were also exposed to average RCS concentrations of 0.12 mg/m^3 (0.4 mg/m^3 RD x 30% RCS content) for 20 years resulting in a cumulative exposure of 2.4 mg/m^3 (0.12 mg/m^3 x 20) may be associated with a statistically significant greater than 3-fold increased risk of developing emphysema.

The exposures in the Hnizdo et al (1991) study that were based on the Beadle (1970) and Page-Shipp and Harris (1972) data can also be evaluated in terms of the adjusted RD and RCS concentrations estimated by Gibbs and DuToit (2002) described earlier and summarized previously in Table 3. Applying the correction factor of 1.8 to the RD concentration of 0.55 mg/m^3 calculated by Oxman et al (1993) for the high-dust occupations in the Hnizdo et al (1991) study, would result in a value of 0.99 mg/m^3. The corresponding value for RCS adjusted to 54% of silica in the RD, would be 0.53 mg/m^3. The cumulative exposures for 20 years of work in high dust occupations such as shaft sinkers or developers would be 19.8 mg/m^3-years for RD and 10.7 mg/m^3-years for RCS. Even after this adjustment to the higher cumulative RD exposure, the resulting 3.5-fold significantly increased risk of developing emphysema would occur at 50% or less of the current WSIB COPD guideline of 40 to 50 mg/m^3-years.

Hnizdo et al (1994)

Hnizdo et al (1994) conducted a retrospective cohort study of 242 non-smoking South African gold miners who had an average of 23.1 years of mining (range 1-48 years) with a mean cumulative RD exposure of 6.8 mg/m^3-years (range 0.5-20.2 mg/m^3-years). Based on a content of 30% RCS in the RD, the cumulative RCS exposures could be estimated to have ranged from 0.015 mg/m^3 – years to 6.06 mg/m^3-years with a mean cumulative RCS exposure of 2.04 mg/m^3 – years. Only a minimal, insignificant grade of emphysema was found at autopsy and that grade was not associated with a statistically significant degree of lung function impairment or with RD exposure (years of gold mining or cumulative dust index).

The authors concluded that the mild emphysema found in non-smokers exposed for many years to RD containing RCS would be unlikely to cause a significant impairment of lung function. Based on the earlier study by Hnizdo et al (1991) the authors also concluded that the statistically significant association between exposure to silica dust and the degree of emphysema in smokers suggested that tobacco smoking potentiates the effect of silica dust.

Conclusions for Emphysema

Based on the findings from the studies discussed above, it may be concluded that exposure to RD and RCS in hard rock mining can increase the risk of emphysema, regardless of whether silicosis is present. This appears to be clearly the case for smokers. However, it is less clear whether nonsmokers exposed to RD and RCS would be at higher risk for emphysema and if so, at what levels of exposure. Some of the studies also found that smoking potentiates the effect of RD, and RCS in particular, resulting in an increased risk of emphysema.

After exposure units of gram-hours per cubic meter (gh/m^3) were converted to the more typical units of milligrams per cubic meter (mg/m^3) of RD or respirable RCS, it was found that a miner who smoked would have had a 3.5 times statistically significantly increased risk of emphysema at considerably lower levels of cumulative RD exposure (11.0 mg/m^3) than the WSIB COPD guideline of 40-50 mg/m^3. These findings suggest that gold miners who smoked and were also exposed to average RCS at about 1.6 times the current MOL OEL of 0.1 mg/m^3, for 20 years, may have a greater than 3-fold significantly increased risk of developing emphysema. Even after correcting the RD concentration for the high dust occupations in the Hnizdo et al (1991) study by using the data from Gibbs and DuToit (1992), the cumulative RD exposure associated with a statistically significant 3-fold increased risk was less than 50% of the current WSIB COPD guideline of 40 to 50 mg/m^3-years.

Chronic Bronchitis

A number of cross-sectional studies have examined the relationship between RD or respirable RCS exposure and chronic bronchitis. The 2001 WSIB SR document for COPD, considered the following three studies that are discussed below; Holman et al (1987), Kreiss et al (1989), Cowie and Mabena (1991).

Additional studies by Sluis-Cremer et al (1967), Wiles and Faure (1977), a review by Hnizdo and Vallyathan (2003), and epidemiological assessments by the American Thoracic Society (ATS 1997), NIOSH (2002), and OSHA (2016) are also discussed below.

Sluis-Cremer et al (1967)

A community-based study by Sluis-Cremer et al (1967) of 827 male residents of a South African gold-mining town was done to determine whether chronic bronchitis occurred more frequently among a group of 562 dust-exposed miners and ex-miners than among 265 males in the non-mining (non-exposed) group. The dust-exposed group contained more smokers and fewer persons who had never smoked than the group without exposure to dust. The prevalence of chronic bronchitis (obtained by questionnaire) was compared among dust-exposed smokers or non-smokers and non-dust exposed smokers or non-smokers. There was a significant difference ($p < 0.01$) between the prevalence of chronic bronchitis in dust-exposed and non-dust exposed miners among smokers only. Chronic bronchitis was shown to be significantly more common in miners than non-miners for every age and smoking category (present or past smoker), with the exception of non-smoker. No increased prevalence of chronic bronchitis was observed among non-smokers. The authors suggested that the increased prevalence of bronchitis in miners who smoke was the result of synergism between smoking and dust inhalation rather than just the dust inhalation or smoking alone.

Wiles and Faure (1977)

In contrast to the Sluis-Cremer et al 1967 study, this study of South African gold miners 10 years later found that the prevalence of chronic bronchitis increased significantly with increasing dust concentration and cumulative dust exposure in smokers, nonsmokers, and ex-smokers (Wiles and Faure, 1977). This was a larger cross-sectional study of 2,209 South African gold miners who were 45 to 54 years of age and had 10 or more years of employment in gold mines, 653 who had been ex-miners for one year or more and 483 non-miners. Information on mean dust counts (expressed as respirable surface area) for each occupation and the number of shifts each miner had worked in an occupation was available from the data collected and reported by Beadle and Bradley (1970) and summarized by Page-Shipp and Harris (1972). However, specific dust count exposures were not provided in the study. The study found a statistically significant increased prevalence of chronic bronchitis with increasing average dust concentration ($p < 0.001$) and with cumulative dust exposure in nonsmokers ($p < 0.05$), ex-smokers ($p < 0.05$), and smokers ($p < 0.001$).

Holman et al (1987)

A later study observed an overall prevalence of chronic bronchitis of 14% in the cohort of 1,363 gold miners in Western Australia (Holman et al 1987). Years of underground mining experience was used as a surrogate for dust exposure and the prevalence of bronchitis was compared to lifetime non-miners. After controlling for age and smoking the odds ratio for chronic bronchitis was statistically significant for all exposure durations, and increased with increasing exposure durations consistent with a positive exposure-response relationship:

OR = 1.8 (95% CI, 1.0-3.3) for 1 to 9 years of underground mining,

OR = 2.5 (95% CI, 1.2-5.2) for 10 to 19 years of mining, and

OR = 5.1 (95% CI, 2.4-10.9) for more than 20 years of mining.

This observation was consistent with the findings in the earlier studies by Sluis-Cremer et al. (1967) and Wiles and Faure (1977). Holman et al (1987) estimated that on the basis of the findings from their study, the proportion of cases in underground miners caused by occupational factors was about 50%. They also concluded that based on duration of employment in underground gold mining, the probability of an occupational cause in individual cases is estimated to vary from 40% to 80%.

Kreiss et al 1989

A cross-sectional, community-based prevalence study of respiratory function in miners and non-miners in a Colorado hard-rock mining town[N] observed a positive exposure-response effect for both dust exposure and smoking (Kreiss et al 1989). The study included 389 males; 281 were molybdenum miners with 236 of these having some dust exposure. The average RCS content of the sampled dust ranged from 10% to 30% for the period 1977 to 1981, with 27% of the personal samples exceeding the current MOL OEL of 0.1 mg/m^3 and 40% exceeding the current OSHA PEL of 0.05 mg/m^3.

Miners had worked underground or at the molybdenum crusher for an average of 9.3 years (range 1 to 43 years). The cumulative RD exposures ranged from zero to 46.5 mg/m^3-years with a mean of 8.2 mg/m^3-years. The only respiratory symptom consistent with lengthy mining exposures was dyspnea (shortness of breath). The smoking adjusted odds ratio for dyspnea in a miner with more than 10 years of underground exposure was 3.31; 95% 1.43-7.67 compared to non-miners with no dust exposure.

Age-adjusted odds ratios for dyspnea were calculated for various combinations of smoking (pack-years) and cumulative dust exposure (mg/m^3 – years). A positive exposure-response was observed for non-smoking miners. An exposure of 5 mg/m^3 – years resulted in a nearly 2-fold increased odds of dyspnea (OR=1.9; 95% CI 0.9-3.8) with the odds increasing to 2.5; 95% CI 0.9-6.7 for miners with a cumulative dust exposure of 30 mg/m^3 – years. Similarly, the odds ratio for dyspnea for a miner with the same

[N] *This was a molybdenum hard-rock mine*

cumulative dust exposure but with a 10 pack-year smoking history increased from 3.4 to 4.4 (all results were statistically significant). The combination of smoking and cumulative dust exposure appeared to have a multiplicative effect on the increased risk of dyspnea.

Multiple regression analysis found that cumulative dust exposure was negatively associated with lung function measurements such as FEV_1, FEF_{25-75} and FEV_1/FVC. Dust exposure alone had similar quantitative effects as smoking alone on these lung function measurements. The combination of smoking and dust exposure was associated with greater airflow limitation than seen in smokers without dust exposure. The spirometry results showed dust-associated airflow limitation in both smokers and non-smokers.

The authors concluded that the finding of an exposure-response relationship between RD exposure and airflow limitation was consistent with the findings of other studies that observed an obstructive pattern of lung function independent of cigarettes (Becklake 1985, Wiles and Faure 1977, Manfreda et al 1982).

Cowie and Mabena (1991)[o]

A cross-sectional study of 1,197 black South African gold miners by Cowie and Mabena (1991) found that chronic bronchitis symptoms which included shortness of breath (dyspnea) and excess sputum production were associated with the intensity of mining dust exposure. Miners exposed to high levels of dust (qualitative assessment) had more than twice the odds of dyspnea (OR = 2.2; 95% CI 1.33-3.60) compared to those in low dust exposures. They also had a nearly 2-fold increased risk of excess sputum production (OR = 1.8; 95% CI 1.19-2.68). Both of these findings were statistically significant; however, excessive smoking classified as >24 pack-years also increased the odds of dyspnea (OR = 1.7; 95% CI 1.20-2.36) and excess sputum production (OR = 3.7; 95% CI 2.62-5.23). Chronic bronchitis was present in 62% of the gold miners who had smoked and in 45% of those who had never smoked.

Reviews

Other researchers have reviewed relevant studies and concluded that there is a relationship between RD or RCS and the development of chronic bronchitis: ATS (1997), NIOSH (2002), Hnizdo and Vallyathan (2003) and OSHA (2016).

The ATS (1997) published an official statement on the adverse effects of RCS exposure that included a section that discussed studies on chronic bronchitis (defined by chronic sputum production). According to the ATS review, chronic bronchitis was found to be common among workers exposed to dusty environments contaminated with RCS. In support of this conclusion, ATS cited studies showing positive

[o] *The first study of respiratory disorders in black South African gold miners who were less frequently studied since their work tended to be more transient – many black miners worked for short periods before returning to their families.*

findings for South African gold miners: Hnizdo et al (1990a) and Australian gold miners: Holman et al (1987).

The 2002 NIOSH Hazard Review on RCS reviewed studies related to RCS exposure and development of chronic bronchitis: NIOSH (2002). NIOSH concluded that, based on the same studies reviewed by OSHA, occupational exposure to RCS is associated with chronic bronchitis, but that some epidemiologic studies suggested that this effect may be less frequent or absent in non-smokers.

Hnizdo and Vallyathan (2003) also reviewed studies addressing COPD due to occupational RCS exposure and concluded that chronic exposure to RCS at levels that were insufficient to cause silicosis may cause chronic bronchitis. They based this conclusion on studies that showed the prevalence of chronic bronchitis increased with intensity of exposure. The cited studies were also considered by OSHA in 2016 when it established a new standard for occupational exposure to RCS [Cowie and Mabena (1991); Holman et al (1987); Kreiss et al (1989); Sluis-Cremer et al (1967) and Wiles and Faure (1977)].

As part of the review of epidemiological evidence to support the lowering of the permissible exposure limit (PEL) from 0.1 mg/m^3 to 0.05 mg/m^3, OSHA concluded that exposure to RCS is associated with minimal risk of chronic bronchitis but that an exposure-response relationship may exist (OSHA 2016). OSHA also concluded that smokers may be at increased risk compared to non-smokers and chronic bronchitis may occur in RCS-exposed workers who do not have silicosis.

Conclusions for Bronchitis

No longitudinal studies designed to investigate the relationship between RD or RCS in hard rock mining and chronic bronchitis were available. However, five cross-sectional studies demonstrated a qualitative or semiquantitative relationship between exposure to RD or RCS and chronic bronchitis among gold miners (Sluis-Cremer et al. 1967, Wiles and Faure, 1977, Holman et al., 1987, Kreiss et al., 1989, Cowie and Mabena 1991). Other researchers have reviewed these and other relevant studies and concluded that there is a relationship between exposure to RD or RCS and the development of chronic bronchitis (ATS 1997, NIOSH 2002, Hnizdo and Vallyathan 2003, OSHA 2016).

Pulmonary Function Impairment

This section reviews studies that were designed to evaluate the relationship between exposure to RD or RCS and pulmonary function loss such as reduction in forced expiratory volume in 1 second (FEV_1) or forced vital capacity (FVC) – the total volume of air exhaled in one breath. FEV_1 and the ratio of FEV_1/FVC are also commonly used to define COPD (GOLD 2018, Oliver 2020).

The studies considered in this section are;

Irwig and Rocks 1978

Manfreda et al 1982
Kreiss et al 1989
Hnizdo et al 1990a
Cowie and Mabena 1991
Hnizdo et al 1992
Cowie 1993
Cowie 1998
Ehrlich et al 2011
Möhner and Nowak 2020
Ulvestad et al 2020

Only two of these studies were included in the WSIB SR document: Kreiss et al (1989) and Cowie and Mabena (1991). With the exception of the Ehrlich et al (2011), Mohner and Nowak (2020) and Ulvestad et al (2020) studies, the remainder of the studies were published within the period 1980-1999 and considered in the preparation of the WSIB COPD SR and AA. However, for unclear reasons none of these was included in either of these documents.

Irwig and Rocks (1978)

An early study of 1,973 South African gold miners by Irwig and Rocks (1978) compared 134 miners with silicosis to the remainder who did not have silicosis. Those with silicosis had a 5% lower average FEV_1 than those miners without silicosis ($p<0.01$). Statistical analysis of independent variables such as height, age, smoking history and exposure to mining dust found that the difference in lung function was almost entirely accounted for by mining dust exposure. The authors concluded that silicosis seemed to have little effect on lung function but that there was a significant exposure-response relationship between exposure to dust in gold mines and lung function test results indicative of COPD.

Manfreda et al. (1982)

Manfreda et al. (1982) studied 95 underground miners from two mining companies in Manitoba, Canada and 382 controls from the general population. Most of the workers were smokers or ex-smokers. Only one miner had a radiographic appearance consistent with silicosis. Although no historical exposure records were available, exposure measurements made shortly after the lung function testing indicated minimal, median, and maximal RD exposure levels of 0.2, 0.5, and 4.8 mg/m^3, respectively. The RD contained 6 to 9 percent RCS. Changes in FVC, FEV_1 and FVC were significantly related to duration of employment ($p < 0.05$).

Kreiss et al (1989)

A cross-sectional, community-based prevalence study of respiratory function in 181 Colorado hard rock molybdenum miners and 108 non-miner residents (discussed above in the Bronchitis section) observed a

reduction in FEV_1 among miners exposed to dust (Kreiss et al 1989)[P]. The analysis found a significant difference in the average FEV_1 for non-smokers with dust exposure compared to that of non-smokers without occupational dust exposure (95% and 101% of predicted FEV_1, respectively, $p<0.05$).

Hnizdo et al (1990a)

A 1990 study by Hnizdo et al reanalyzed the earlier study by Wiles and Faure (1977) of 2,209 white South African gold miners and 483 non-miners described earlier in the Chronic Bronchitis section. The Hnizdo et al (1990a) study examined the individual lung function measurements including FEV_1, FVC and FEF_{25-75} for any exposure-response trend for dust exposure and for the presence of interaction between dust and smoking. This study also estimated the attributable risk for tobacco smoking and underground gold mining dust exposure separately.

The occupations were classified into 11 groups on the basis of dust counts done by Beadle et al (1970) and summarized in Appendix 1 of Page-Shipp and Harris (1972). For each miner the number of shifts worked in each occupation was weighted by the mean RD count for that occupational group. The dust exposure was analyzed in terms of 6 categories of cumulative particle-years; ranging from 0 to ≥ 46 particle-years/1,000.

The miners were also divided into 5 smoking categories: non-smoker, ex-smoker, current smoker with < 20 pack-years, current smoker with 20 to 39 pack-years and current smokers ≥ 40 pack-years. To assess obstructive effects consistent with COPD, the miners were classified into 3 lung function profiles: minimal, moderate and marked obstruction.

In comparison to the reference group of non-smokers, the prevalence of each level of lung function impairment (minimal, moderate and marked obstruction) increased as the cumulative dust exposure and smoking increased for ex-smokers and current smokers. The miners with the marked obstruction profiles were found to have the highest tobacco consumption and the strongest exposure-response trend with dust; yet they did not have the highest dust exposure. The mean cumulative RD exposure for the marked obstruction group was 30.6 particle-years/1000; whereas the mean cumulative dust exposure for the moderate obstruction group was 31.5 particle-years/1000.

The authors did not provide details of how the particle-years were calculated; however, it likely was calculated as follows:

particle-years = (av. RD count in ppcc x av hours per shift x total shifts) ÷ (av 240 shifts per year)

The simplified calculation becomes; particle-years = av. ppcc x av shift length x years of exposure

[P] *discussed in the section on Chronic Bronchitis*

For example, for a miner exposed for 25 years at the average RD count of 180 ppcc, reported in Table II of page-Shipp and Harris (1972) as the average konimeter particle count for all 11 occupational groups as measured in the Beadle (1970) survey, the cumulative particle-years can be estimated as:

180 ppcc x av 6.7 hrs/shift x 25 years = 30,150 particle-years or 30.1 particle-years/1000

This result is very close to the 30.6 particle-years/1000 associated with marked obstruction reported by Hnizdo et al (1990b).

The equivalent cumulative RD exposure may be estimated as 4.25 mg/m^3 - years from the average concentration of 0.17 mg/m^3 for all 11 occupational groups as calculated by Page-Shipp and Harris (see Table 3 above); (25 years x 0.17 mg/m^3).

Applying a correction factor of 2 (4.25 x 2 = 8.50 mg/m^3 – years) or 4 (4.25 x 4 = 17 mg/m^3 – years), to account for underestimates of dust exposure as suggested by Gibbs and DuToit (2002), shows that marked obstruction would have been associated with cumulative RD exposures well below the current WSIB COPD exposure guideline of 40 to 50 mg/m^3 – years.

Hnizdo et al (1990a) concluded that smoking potentiates the effect of dust exposure in underground gold mining and increases the chances for development of obstructive impairment in lung function. The interaction between dust exposure and smoking was found to be closer to multiplicative than additive. They also estimated that in the group with marked obstruction, 48% was attributable to dust exposure, 82% to smoking and 90% to the combined effect of smoking and dust exposure.

Cowie and Mabena (1991)

This cross-sectional study (the first of a working population of black South African gold miners), discussed in the section above on Chronic Bronchitis, also examined pulmonary function. Statistically significant reductions in FEV_1, FEV_1/FVC%, and MMEF (maximal mid-expiratory flow) were found to be associated with the duration of exposure to the underground environment, after controlling for silicosis and smoking. Statistically significant reductions of 200 ml (p=0.006) in FEV_1 and 3.6% (p=0.0001) in the FEV_1/FVC ratio were observed after 25 years of exposure underground after controlling for smoking. The average annual loss of FEV_1 attributable to continued exposure to the dusty mine environment was calculated to be 8 ml per year.

In contrast to the finding for duration of exposure, no reduction in lung function attributable to high intensity dust exposure was observed. Rather, workers in the highest intensity of exposure had modest increases not decreases in some of their lung function parameters. There was clear evidence of strong confounding by a healthy worker effect, with the healthiest workers tending to work in the hardest and dustiest jobs. The effect of dust exposure would therefore be underestimated if the healthiest men were

associated with the most intense dust exposure and the weakest with the lowest dust intensity. Another significant limitation of this study was the lack of specific dust exposure information.

Hnizdo et al (1992)

The purpose of the Hnizdo et al (1992) study was to quantify the loss of lung function attributable to exposure to RD and RCS in South African gold mines. This was a 5-year follow-up of the Hnizdo et al (1990a) study of white miners who had pulmonary function testing during the period 1968 to 1971. Of the 2,209 white miners in the 1990 study, 1,249 had five-year follow-up tests of lung function in for the 1992 study.

All the occupations of the gold mines were categorized into seven occupational categories for which the RD concentrations were estimated in terms of the dust respirable surface area (RSA) as determined by Beadle and Bradley (1970). The measurements of RSA in square micrometers per cubic centimeter (um^2/cc) (obtained after heat and acid treatment) were converted into mass of RD (MRD) in mg/m using the following equation: $MRD = 0.027 \times (RSA)^{0.474}$. The conversion from measurements of RSA to MRD was discussed in more detail in the Exposure Assessment Considerations section of this document.

The formula was applied to the occupational group means. The authors estimated that the error associated with the conversion from RSA over the range 100-1000 um^2/cc was generally less than 0.1 mg/m^3. Hnizdo et al concluded that the estimates of RD mass in mg/m^3 using the RSA conversion equation were comparable to measurements obtained by gravimetric sampling from various surveys done in the gold mines.

The cumulative exposure to RD for each occupational group was calculated up to the 1968-71 examination and for the 5-year follow up period as follows:

gram hours per cubic meter (ghm^3) = (shifts x av. hrs./shift x av. mg/m^3 RD) ÷ 1000

Cumulative exposure to RD up to the 1968 to 1971 examination was a more significant predictor of most indices of lung function, including FEV_1 and FVC than duration of exposure. It was observed that the average duration of exposure for the cohort was 24 years and the average concentration of RD was 0.30 mg/m^3 resulting in a cumulative exposure of 7.2 mg/m^3 – years. Based on the RD containing 30% RCS, the cumulative RCS exposure would have been 2.16 mg/m^3 – years [24 years x (0.30 mg/m^3 x 0.30)].

The authors estimated that at the 1968-1971 examination, the estimated loss of lung function attributable to dust exposure for a 50-year-old miner exposed for 24 years to the average RD concentration of 0.30 mg/m^3 (cumulative dust exposure of exposure of 14.4 gh/m^3 or 7.2 mg/m^3 – years) was 236 ml of FEV_1; 95% CI 135-338 and 217 ml of FVC; 95% CI 110-324. Both decreases in lung function were statistically significant.

This was estimated to have been equivalent to about half of the loss of FEV_1 in a typical U.S. male nonminer who smoked one pack of cigarettes per day for 30 years; FEV_1 loss 552 ml; 95% CI 461-644 (Dockery et al 1988, Hnizdo et al 1992).

For miners in the highest RD exposure category of ~ 0.5 mg/m^3 (cumulative dust exposure of 22.2 gh/m^3 or 11.1 mg/m^3 – years) the estimated loss of lung function attributable to RD was also statistically significant: FEV_1 reduction of 364 ml (95% CI 207-501) and FVC reduction of 335 ml (95% CI 170-500). For a 50-year-old current smoker with a smoking history of 30-pack-years it was estimated that the loss in FEV_1 due to smoking alone would be 552 ml (95% CI 461 ml-644 ml). The multiple linear regression analysis showed that the effects of dust and smoking were additive.

Therefore, the expected combined loss of FEV_1 for a 50-year-old currently smoking miner who worked in the highest RD exposure (cumulative exposure of 22.2 gh/m^3 or 11.1 mg/m^3 – years), and who had a 30 pack-year smoking history, was 916 ml (364 ml contribution from high dust exposure + 552 ml from smoking). This result was statistically significant (95% CI 784 ml – 1042 ml).

The authors concluded that exposure to RD and RCS among South African gold miners was associated with significant loss of lung function and attributed a 10 ml per year loss of FEV_1 to continued RD exposure. Even though the contribution of smoking was greater than that of exposure to dust, the combined effect of RCS exposure and smoking appeared to be additive and had the potential to cause serious pulmonary disability.

As discussed previously, if the correction factor of 2 for the RD concentrations, as determined by Gibbs and DuToit (2002) is used, the respective cumulative exposures are as follows:

In the case described above, the adjusted average RD concentration is 0.60 mg/m^3, resulting in a cumulative exposure 14.4 mg/m^3 – years (0.60 mg/m^3 x 24 years). Similarly, the adjusted value for the cumulative RD exposure in the highest exposure category would be 22.2 mg/m^3–years.

Similar to other South African gold mining studies by Hnizdo et al (1990a) and Hnizdo et al (1991), it may be concluded that even after using the adjusted higher RD values, the cumulative exposure associated with statically significant reduction in lung function (FEV_1), remains below the WSIB exposure guideline of 40-50 mg/m^3-years.

Cowie (1998)

Cowie (1998) studied lung function in 242 gold miners in a follow-up of an earlier case-control study by (Cowie and Mabena (1991) discussed above. Baseline chest x-rays and pulmonary function studies from an initial cohort were compared to those obtained five years later. Before adjustment for age, the annual loss of FEV_1 was 37 ml. in miners without silicosis. A similar loss was noted for FVC. After adjusting expected loss due to aging, an excess annual loss of 13 ml for FEV_1 was estimated for gold miners

without silicosis. This was very similar to the excess 10 ml per year loss for FEV_1 attributed to continued RD exposure in the Hnizdo et al 1992 study.

Results of an earlier study by Cowie et al (1993), suggested that a loss of approximately 8 ml of FEV_1 per year would be expected due to continued exposure to the dusty mine environment, which when added to the expected loss of 24 ml/yr due to aging, would be equivalent to approximately 32 ml/yr. This finding was similar to the annual FEV_1 loss of 37 ml in the Cowie (1998) (above). This loss is equivalent to a 0.35 % loss of predicted FEV_1 annually. Cowie (1998) noted that this was remarkably similar to the loss of 0.38% loss of FEV_1 annually attributed to RCS dust exposure that was reported by Malmberg et al. (1993) in a study of COPD among Swedish granite workers.

Ehrlich et al (2011)

A 2011 study by Ehrlich et al was the first to examine the association between cumulative dust exposure measured gravimetrically and loss of lung function in black South African gold miners. The average duration of employment among the 520 black miners aged > 37 years was 21.8 years, the average RD concentration was 0.37 mg/m^3, and the average RCS concentration was 0.053 mg/m^3. Gravimetric dust measurements were used to calculate cumulative RD and RCS exposures. Concentrations of RD and RCS in mg/m^3 that were measured in 2000/2001 for the purposes of the 2011 study were comparable to gravimetric measurements available from 1992 to 1996.

Increasing exposure to both RD and RCS were associated with increasing excess loss in both FEV_1 and FVC. It was estimated that there was an average of 16.2 ml excess loss in FEV_1 for each 1 mg/m^3-year of cumulative dust exposure. Over a working lifetime of 30 years at an average RD concentration of 0.37 mg/m^3 (cumulative RD exposure 11.1 mg/m^3-years) this would result in an additional 180 ml (95% CI -3 to 362) loss in FEV_1 (11.1 mg/m^3-years x 16.2 ml loss per mg/m^3-years = 180 ml). The equivalent loss in FVC was estimated to be 208 ml (95% CI 3 to 412).

It is unclear why this study found a smaller decrease in FEV_1 of 180 ml among black miners compared to the Hnizdo et al (1992) study that observed a decrease in FEV_1 of 364 ml among white miners with a similar cumulative RD exposure (11.1 mg/m^3-years).[Q]

The Ehrlich et al (2011) study estimated that there was an average of 95.6 ml excess loss in FEV_1 for each 1 mg/m^3-year of cumulative RCS exposure. Over a working lifetime of 30 years at an average RCS concentration of 0.053 mg/m^3 (cumulative RCS exposure 1.59 mg/m^3-years) it may be estimated that this would result in an additional 152 ml loss in FEV_1 (1.59 mg/m^3-years x 95.6 ml loss per mg/m^3-years = 152 ml). The equivalent loss in FVC would be estimated to be about 189 ml.

[Q] *Blacks are generally thought to have smaller lung volumes than whites, and for many prediction equations used in spirometry a correction of 10-15% for blacks and 4-6% for Asians is built in.*

An interesting observation in this study was that smoking did not play a role in lung function loss in this group of miners. This is consistent with a previous study of black miners that failed to show any association between smoking and lung function loss (Cowie et al 1998). The likely reason for this is a low daily consumption of cigarettes. While 51% of this group had ever smoked, only 28% were current smokers. The median number of cigarettes smoked daily was only 5.3 and median pack-years was 4.9.

This study found a substantial loss in lung function in this population of gold miners even though they had been exposed to an average RD concentration below 0.4 mg/m^3 and an average RCS concentration below the South African mining occupational exposure limit of 0.1 mg/m^3. This was the first study of any South African gold miners to use directly obtained gravimetric measurements.

Möhner and Nowak (2020), Ulvestad et al (2020)

As described to this point, many studies have found an association between occupational exposure to RD containing RCS and increased risk of COPD in the absence of silicosis (Hnizdo et al 2003, Malmberg et al 1993, Rushton 2007, Irwig and Rocks 1978, Tsuchiya et al 2017). These studies indirectly estimated the risk of COPD morbidity & mortality from RCS by estimating concentrations of RCS contained as a percentage of RD. However, more recent studies have assessed the association of COPD & occupational exposure to RCS more directly and derived quantitative estimates of the relationship between RCS exposure and COPD (Möhner, Nowak et al 2020, Ulvestad et al 2020).

A 2020 German study proposed a new methodological approach which may be used to derive criterion for the recognition and compensation of RCS-related COPD (Möhner Nowak et al 2020). A sub-cohort of 1,418 miners ranging in age from 18 to 36 years old were selected from the large German uranium miners cohort study (Kreuzer 2010, 2013). Estimates of cumulative RCS exposures for each miner were calculated from a comprehensive job-exposure matrix based on extensive RCS measurements at each site. The mean duration of employment for the cohort was 12.8 years (range 0.87-19.9 years), mean annual RCS concentration was 0.074 mg/m^3 (range 0.0–2.07 mg/m^3) and the mean number of spirometry tests was 5 (range 2-15). COPD was regarded as RCS-induced if RCS exposure caused a decrease in FEV_1/FVC from the expected value to the lower limit of normal (LLN)[R]. The generally accepted GOLD criteria for the definition of COPD are based on post-bronchodilator FEV_1/FVC ratio and COPD is assumed if FEV_1/FVC is less than 0.7. As this study only had pre-bronchodilator data available, the Global Lung Function Initiative (GLFI) spirometry equations were used to defined lower limits of normal lung function (Quanjer et al 2012).

Evidence was found for an exposure-response relationship between cumulative RCS exposure and pre-bronchodilator lung function with a threshold for an RCS exposure concentration of about 0.1 mg/m^3 (0.089 mg/m^3; 95% CI: 0.071-0.101 mg/m^3). It was estimated that the reference dose or cumulative RCS

[R] *Using the GOLD criteria, COPD is assumed if FEV_1/FVC is below 0.7*

exposure needed to reduce lung function (i.e., FEV1/FVC) to the LLN was 2.33, 95% CI:1.86-3.11 mg/m^3-years. The authors concluded that an average RCS cumulative exposure of 2 mg/m^3-years above 1.0 mg/m^3 leads to a decrease in the ratio FEV_1/FVC to the lower level of normal and should be recognized as COPD according to the GOLD definition. This cumulative RCS exposure would equate to 20 years of exposure to RCS greater than 0.1 mg/m^3.

This paper also provided practical examples of how this finding could be applied to different industries or occupations exposed to RCS. A study on Swedish granite workers reported a mean duration of exposure of 22 years and an RCS concentration of 0.32 mg/m^3; or 0.22 mg/m^3 above the estimated concentration threshold of 0.1 mg/m^3 (Malmberg et al 1993). Therefore, if a granite crusher was exposed to an RCS concentration of 0.32 mg/m^3, he would reach the reference dose of 1.0 mg/m^3-years after 9 years and one month (2 mg/m^3-years ÷ 0.22 mg/m^3 = 9.09 years). They suggested that if this worker was diagnosed with COPD at this or a later time, it would have to be recognized as occupational.

Similar findings were reported in a 2020 study of Norwegian rock drillers that observed airways obstruction in those workers in the highest RCS exposure group (n=45) who had an average exposure of 0.08 mg/m^3 for 21.7 years (cumulative exposure 1.7 mg/m^3-years) (Ulvestad et al 2020). This was a shorter duration of exposure and lower concentration of RCS than reported in the previous studies reviewed by Rushton (2007) and Bruske et al (2014).

The study of German uranium miners discussed above (Möhner Nowak et al 2020), suggested that the Ulvestad et al 2020 study could serve as another example of estimating the length of time a worker exposed to RCS concentrations above 0.1 mg/m^3 would need to reach the reference dose of 2.0 mg/m^3-years for COPD. Among the listed job categories, only a rock driller using a feed mounted control panel (0.24 mg/m^3) was exposed above the threshold level of 0.1 mg/m^3. In this case they estimated that he would reach the reference dose after 14 years and 4 months (2 mg/m^3-years ÷ 0.14 mg/m^3) of constant exposure at that level and a potential COPD in his case would be considered occupational from that time on.

If the approach proposed by Möhner Nowak et al (2020) were applied to Ontario hard rock miners, and it could be established that a similar relationship between a cumulative RCS exposure of 2.0 mg/m^3 – years and a decrease in lung function that meets the GOLD criteria for COPD, then it may be concluded that 20 years of exposure to average RCS concentrations greater than 0.1 mg/m^3 results in occupationally related COPD. However, to date, WSIB only considers cumulative RD exposure of 40-50 mg/m^3 as being associated with occupationally related COPD.

A cumulative RCS exposure of 2.0 mg/m^3-years for an Ontario gold miner would be equivalent to 20 mg/m^3-years of RD (assuming an average concentration of 10% RCS in gold mines[S]). Similarly, a

[S] *It is generally accepted that the RCS content in respirable dust for Ontario gold, uranium and nickel mines were 10%, 20% and 5% respectively. This is also stated in Appendix 10 of the WSIB COPD ASMB.*

cumulative RCS exposure of 2.0 mg/m^3-years for an Ontario uranium miner would be equivalent to 10 mg/m^3-years of RD (assuming an average concentration of 20% in uranium mines). These values of cumulative RD exposure are about one half and one quarter, respectively, of that required by the current WSIB COPD exposure guideline.

Conclusions for Pulmonary Function Impairment

Several studies have examined relationship between RD or RCS exposure and pulmonary function impairment.

An important observation from three studies that included quantitative RD and RCS assessments; Hnizdo et al (1990a), Hnizdo et al (1992) and Ehrlich et al (2011), was that significant reductions in lung function occurred at cumulative RD exposures (even after using the adjusted higher RD values) that were well below the WSIB exposure guideline of 40-50 mg/m^3-years.

Three studies directly addressed the question of whether RCS-exposed miners can develop pulmonary function impairment in the absence of silicosis[T] (Irwig and Rocks 1978, Hnizdo et al 1992, Cowie 1998). These studies found that pulmonary function impairment: (1) can occur in RCS-exposed miners in the absence of silicosis, (2) was still evident when silicosis was controlled for in the analysis, and (3) was related to RCS exposure rather than to the presence or severity of silicosis. Many other researchers have concluded that a relationship exists between occupational exposure to RD or RCS and lung function impairment (IARC 1997, ATS 1997, Hnizdo and Vallyathan 2003).

Contradictory findings were observed for the combined effect of exposure to RD and tobacco smoking on lung function. An earlier study by Hnizdo et al (1990a) concluded that the joint effect of dust and tobacco smoking on lung function impairment was more likely multiplicative rather than additive. In contrast a later study by Hnizdo et al (1992) that included a re-analysis of some of the data from the 1990 study, concluded that the combined effect of dust exposure and smoking was more likely additive than multiplicative.

Recent studies have assessed the association of COPD & occupational exposure to RCS more directly and derived quantitative estimates of the relationship between RCS exposure and COPD (Möhner, Nowak 2020, Ulvestad et al 2020). These studies observed an exposure-response relationship between a cumulative RCS exposure of about 2.0 mg/m^3-years and a decrease in lung function (FEV_1/FVC) to the lower limit of normal that meets the GOLD definition of COPD.

Mortality from COPD

This section reviews four studies of South African gold miners that examined the relationships between exposure to RD and RCS and mortality from COPD: Wyndham et al (1986), Hnizdo et al (1990b), Reid-

[T] *Silicosis is considered evidence of exposure to high concentrations of RCS.*

Sluis-Cremer et al (1996), Roscoe et al (1997). These studies were included in the WSIB COPD SR document.

Wyndham et al (1986)

The first part of the Wyndham et al (1986) study compared the mortality from chronic respiratory diseases (primarily bronchitis and emphysema) in a cohort of 3,917 white South African gold miners for the period Jan 1, 1970 to December 31, 1978 to the total white male population of South Africa. A statistically significant excess mortality was observed: SMR = 1.66; 95% CI 1.08-2.42.

This study also included a nested case-control analysis in which each of the of the 26 cases of chronic respiratory diseases was matched with four controls. Two dust exposure indicators were used in the analysis: total years of underground mining and cumulative dust exposure in particle-years. The analysis found that the major risk factor for chronic respiratory disease was smoking; RR= 3.67; 95% CI 1.43-9.40 for an average smoking history of 20 cigarettes per day. It was observed that cumulative dust exposure had a statistically significant additional effect on the relative risk of chronic respiratory disease; RR = 2.48; 95% CI 1.03-6.00 per 10,000 particle-years of exposure (p = 0.03). Cumulative exposure in units of particle-years is calculated by the following formula: particles/cc x shift length x years.[U]

The number of years required to reach a cumulative exposure of 10,000 particle-years may be estimated by using the mean particle concentration of 178 ppcc and average shift length 6.7 hours for the 11 mining occupations from the konimeter as measured by Beadle and Bradley (1970) and reported in Table II and Appendix 1 of Page-Shipp and Harris (1972):

(10,000 particle-years ÷ 178 ppcc) ÷ (av shift length of 6.7 hours) = 8.4 years of exposure.

Therefore, a miner with 8.4 years of exposure to an average dust concentration of 180 ppcc would have a nearly 2 1/2-fold statistically significant increased risk of chronic respiratory disease (primarily bronchitis and emphysema).

Hnizdo et al (1990b)

A 1990 mortality study by Hnizdo et al (1990b) of white South African gold miners included a nested case-control and a cohort analysis to examine the individual and combined effects of exposure to gold mining dust and smoking on mortality from COPD. This was a follow-up to the study by Wiles and Faure (1977) that was discussed in the Chronic Bronchitis section of this review. The study included 945 gold miners who died out of the full cohort of 2,209 miners during the follow-up period from 1968-71 to 1986. Lung function tests collected during the 1968-71 annual medical examinations included FEV_1, FVC and

[U] *Hnizdo E, Sluis-Cremer GK. (1993) Risk of silicosis in a cohort of white South African gold miners. Am J Ind Med; 24:447–57. Leigh J, et al Revised quantitative risk assessment for silicosis and silica related lung cancer in Australia. Ann Occup Hyg: 41, Supplement 1:480-484.*

FEF_{25-75}. Each miner who had died of COPD was matched to a set of 6 controls who were randomly selected from those miners who were born closest to the date of birth of the case and who had survived the case in age.

The occupations were classified on the basis of measured dust counts into 11 groups for which the average RD counts were calculated by Beadle (1971) and summarized by Page-Shipp and Harris (1972). For every miner the number of shifts worked in each occupation was weighted by the average dust count (expressed as RD) for that occupational group. The cumulative dust counts were calculated to the end of the follow-up period and converted into particle-years[v].

The cumulative dust exposures were also calculated for each decade; 1940s, 50s, 60s, and 70s. After adjusting for smoking, exposure to RD during the 1940s was most strongly related to death from COPD and this variable was used to assess the combined effect of RD exposure and smoking. These findings were consistent with the findings of the case-control study of emphysema by Becklake et al (1987). In that study, the number of shifts worked in high dust exposure before 1950 was most strongly related to the presence of emphysema at autopsy in miners aged 50-70 years old.

However, as pointed out in the Hnizdo et al (1990b) study, the assumption that the dust concentrations were higher in the 1940s is contradicted by Beadle and Bradley (1970) who reported that the concentrations in the 1940s were similar those that they measured after the 1940s.

Cigarette smoking was evaluated in terms of cigarettes smoked each day, years of cigarette smoking and pack-years of smoking. Cumulative RD exposure was categorized by particle-years/1000 (< 5, 5-9, 10-14 and ≥ 15)[w] and smoking history by years of cigarette smoking (< 10, 10-19, 20-29 and ≥ 30).

The results of the case-control analysis are summarized below in Table 4 below.

The analysis observed a statistically significant increased trend for RD particle-years and for cigarette smoking when comparing those miners in the three highest exposure or smoking categories with those in the lowest category. The odds ratios were largest and statistically significant for the two highest RD exposures (10-14 particle-years/1000: OR=3.3; 95% CI 1.2-9.4 and ≥ 15 particle-years/1000: OR=5.3; 95% CI 1.8-15.9). The odds ratios were also largest and statistically significant for the two highest smoking categories after adjusting for age (20-29 years: OR=15.8; 95% CI 2.1-118.0 and ≥ 30 years: OR=32.3; 95% CI 4.4-248.2).

Table 4 Odds Ratios for cumulative respirable dust particle-years and years of cigarette smoking (case-control analysis)		
Respirable Dust Exposure:	**Cigarette-years smoked: category (average)**	

[v] Particle-years = total # shifts x average daily (shift) respirable dust concentration ÷ 240 (av. # shifts/year)
[w] These cumulative particle-year exposures would be equivalent to 20 years of exposure to 250 ppcc, 450 ppcc, 500-700 ppcc and ≥750 ppcc respectively. These values were calculated using the formula: (total # shifts in 20 years (4,800) x daily (shift) average dust exposure) ÷ 240 shifts/year. Simplified, the equation becomes: daily (shift) average dust exposure = cumulative dust exposure x 240 (av # shifts/year) ÷ total shifts)

Category (Average particle- years/1000 before 1950)	< 10 (2)	10-19 (16)	20-29 (26)	≥ 30 (34)	OR (95% CI) [X]
< 5 (3.99)	1.0	2.1	3.3	8.5	1.0
5-9 (7.44)	2.7	6.3	6.3	8.8	2.5 (0.9-6.9)
10-14 (10-14)	0.6	5.3	10.3	14.0	**3.3 (1.2-9.4)**
≥ 15 (17.58)	1.3	6.9	12.5	21.0	**5.3 (1.8-15.9)**
OR (95% CI)[Y]	1.0	8.4	15.4	26.2	
OR (95% CI)[Z]	1.0	8.0 (0.9-69.7)	**15.8 (2.1-118.0)**	**32.3 (4.2-248.2)**	

Statistically significant ORs in bold

There was a synergistic effect for RD categories > 10,000 particle-years and for duration of smoking of > 20 years. The combined effect of RD and smoking appeared to be more than additive and likely closer to multiplicative.

The cohort analysis (discussed below) used similar categories of RD and smoking history as the case-control analysis. The risk of death from COPD for miners in the three higher RD exposure categories were compared to that for miners in the lowest RD exposure category. Statistically significant risk estimates are in bold.

Dust Exposure category(particle-years)[AA]	**Relative Risk (RR) mortality from COPD[Y]**
< 5,000	1.0
5,000-9,000	2.49; 95% CI 0.9-6.9
10,000-14,000	**3.12; 95% CI 1.2-8.3**
≥ 15,000	**5.30; 95% CI 2.0-14.4**
	p for trend = 0.003

A statistically significant trend for increased death from COPD and cigarette smoking was also observed:

Years of Cigarette Smoking	**Relative Risk (RR) mortality from COPD[BB]**
< 10	1.0

[X] *ORs adjusted for age*
[Y] *Unadjusted ORs*
[Z] *ORs adjusted for age*
[AA] *These cumulative particle-year exposures would be equivalent to 20 years of exposure to 250 ppcc, 450 ppcc, 500-700 ppcc and ≥750 ppcc respectively. These values were calculated using the formula: (total # shifts in 20 years (4,800) x daily (shift) average dust exposure) ÷ 240 shifts/year. Simplified, the equation becomes: daily (shift) average dust exposure = cumulative dust exposure x 240 (av # shifts/year) ÷ total shifts)*
[BB] *Adjusted for age, year at risk and the other exposure (i.e., dust RR adjusted for smoking and smoking RR adjusted for dust exposure)*

10-19	7.29; 95% CI 0.9-62
20-29	**16.15; 95% CI 2.3-118**
≥ 30	**29.18; 95% CI 4.0-214**
	p for trend = 0.0001

Compared to miners in the lowest dust exposure category (5,000 particle-years), death from COPD was statistically significant among miners in the two highest dust exposure categories of 10,000 to 14,000 particle-years and ≥ 15,000 particle years and borderline statistically significant for the lower exposure category of 5,000 to 9,000 particle-years. Similarly, the largest risk estimates were observed in the two highest smoking categories of 20-29 years and ≥ 30 years of smoking.

The findings of the cohort analysis were in good agreement with those of the case-control analysis. The RR for every 1000 particle-years of cumulative RD exposure, adjusted for cigarette smoking and age estimated from the case-control analysis was 1.11; 95% CI 1.10-1.18. The estimate from the cohort analysis was RR = 1.10; 95% CI 1.04-1.153

The authors estimated the following increased risk of a miner over 50 years of age dying from COPD compared to a miner who was exposed to < 5,000 particle-years;

10,000 particle-years	2.5 times increased risk
17,500 particle-years	5.1 times increased risk
20,000 particle-years	6.4 times increased risk

As shown previously, the number of years required to reach these cumulative exposures may be estimated using the mean daily dust exposure of 178 ppcc and average shift length 6.7 hours for the 11 mining occupations from the konimeter as measured by Beadle et al (1970) and reported in Table II and Appendix 1 of Page-Shipp and Harris (1972) (see Table 2 above): (10,000 particle-years ÷ 178 ppcc) ÷ (av shift length of 6.7 hours) = 8.4 years of exposure. Therefore, a miner with 8.4 years of exposure to an average RD concentration of 180 ppcc would have a nearly 2 1/2-fold statistically significant increased risk of dying from COPD. This finding was similar to that in the Wyndham et al (1986) study.

Using a similar calculation; to achieve a cumulative exposure of 17,500 particle-years a miner would require 14.7 years of exposure to an average RD concentration of 180 ppcc that would have resulted in a greater than 5-fold increased risk of dying from COPD. Similarly, a cumulative exposure of 20,000 particle-years, equivalent to 16.8 years of exposure to 180 ppcc, would have resulted in a greater than 6-fold increased risk of mortality from COPD.

The years of exposure to achieve these cumulative exposures would have been lower for miners who were classified as working in production operations as they were estimated to have an average dust exposure of 208 ppcc (Table 2 above).

10,000 particle years 6.6 years at 208 ppcc 2.5-fold increased risk of mortality from COPD

17,500 particle-years	11.5 years at 208 ppcc	5.1-fold increased risk of mortality from COPD
20,000 particle-years	13.2 years at 208 ppcc	6.4-fold increased risk of mortality from COPD

The observed trend of increased risk with increasing RD and smoking exposure, suggested a synergistic effect between RD and smoking. After analyzing various combinations of dust exposure and smoking variables, the authors concluded that the multiplicative model fit the data significantly better than the additive model, confirming the synergistic effect identified in the case-control analysis.

Reid and Sluis-Cremer (1996)

A 1996 cohort mortality study and a case-control analysis by Reid and Sluis-Cremer (1996) of nearly 5,000 white South African gold miners was one of the largest studies of South African gold miners. This was a follow-up of the Wyndham et al (1986) study that, due to different inclusion criteria, included an additional 955 miners in the cohort and extended the follow-up period by 11 years to December 31, 1989, for a total of 20 years.

Similar to the Wyndham et al (1986) study, the first part of the analysis compared the mortality from chronic respiratory diseases (primarily bronchitis and emphysema) in the cohort of 4,925 white South African gold miners for the period Jan 1, 1970 to December 31, 1989 to the total white male population of South Africa. A statistically significant excess mortality was observed: SMR = 1.89; 95% CI 1.62-2.19 compared to the SMR of 1.66; 95% CI 1.08-2.42 observed in the Wyndham et al (1986) study. The authors noted that although complete mining histories were available, these were not taken into account in this part of the study.

In the case-control analysis, duration (years of mining service) and cumulative dust exposure in mg/m^3 – years were taken into account in examining the risk for COPD (160 deaths). The average number of cigarettes smoked per day in 1960 to 1990 was used as an estimate of smoking history.

Cumulative dust exposure was estimated by classifying the underground mining jobs into six categories according to the degree of dust exposure. The dust exposures were estimated by thermal precipitator count of respirable mass after acid treatment were used to calculate RD concentration over a work shift in units of $h\text{-}mg/m^{3.}$ Cumulative RD exposures were calculated by multiplying the number of shifts worked in any of the 6 exposure categories by the dust concentration in $h\text{-}mg/m^3$ per shift as reported by Beadle and Bradley (1970) and summarized by Page-Shipp and Harris (1972). The cumulative exposures in $h\text{-}mg/m^3$ were divided by 1920 hours/year (average of 240 shifts per year x average 8 hours per shift) to get the cumulative RD exposures expressed in $y\text{-}mg/m^3$ or mg/m^3-years.

To simplify the interpretation of the study findings, the average RD exposure was calculated by dividing the high exposure category of 2.14 $h\text{-}mg/m^3$, that was used a reference for the other exposure categories, by the average shift length of 8 hours (2.14 $h\text{-}mg/m^3 \div 8$ hrs = 0.27 mg/m^3).

Exposure category (occupation)	Time-Weighted Average (TWA)

	RD Exposure[CC]
High (shaft sinker/developer)	0.27 mg/m^3
Medium-high (stoper)	0.18 mg/m^3 (2/3 high dust exposure)
Medium (shift boss, trammer)	0.135 mg/m^3 (1/2 high dust exposure)
Low (artisans, surveyors)	0.09 mg/m^3 (1/3 high dust shift)
Surface	0.07 mg/m^3 (1/4 high dust shift)
Surface (non-dusty)	0.00 mg/m^3

The authors chose an end point of 5 years before death for the calculation of cumulative dust exposure for their case-control study. The 5-year lag period was chosen as the authors concluded that the cases would not accumulate much dust exposure for the period of illness preceding death due to COPD. The case-control analysis found a statistically significant relative risk for COPD associated with cumulative RD exposure up to 5 years before death: RR=1.23; 95% CI 1.0-1.5. Controlling for smoking had a minimal effect and the relative risk for COPD remained statistically significant (RR= 1.20; 95% CI 1.0-1.4).

As this was a broader mortality study, the authors did not provide an exposure-response analysis for each of the cumulative dust exposure categories. However, they reported that the typical underground miner in the study worked for 27 years and was exposed to an average RD concentration of 0.14 mg/m$^{3[DD]}$ resulting in a cumulative RD exposure of 3.7 mg/m^3 – years. The authors concluded that if this miner smoked approximately ½ pack of cigarettes per day (13.5 pack-years) the risk of COPD could be estimated according to the following relationship; RR=1.2 multiplied by the power of the cumulative dust exposure. In the case of the typical underground miner described above, the risk of COPD was calculated as RR=1.2$^{3.7}$ = 1.96.

The authors stated that another interpretation was that the average gold miner with this exposure to RD and cigarette smoke was twice as likely to die of COPD. A miner in the high exposure category such as a shaft sinker or developer who was exposed to an average RD concentration of 0.27 for 27 years (cumulative exposure 7.29 mg/m^3 – years) and who also smoked ½ pack of cigarettes per day, would have a nearly 4-fold increased risk of dying from COPD (RR=1.2$^{7.29}$ = 3.78).

This is another example of statistically significant increased risk of death from COPD at much lower cumulative RD concentrations that the current WSIB COPD exposure guideline.

Roscoe (1997)

An updated retrospective cohort mortality study by Roscoe (1997) followed 3,238 white Colorado Plateau uranium miners from 1960 to 1990. Statistically significant mortality was observed for COPD diseases

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[CC] the time-weighted average (TWA) respirable dust exposure of 0.27 mg/m^3 for the high exposure category was used to estimate the TWA respirable dust exposure for the other exposure (occupation) categories
[DD] [27 years x (av. 240 shifts per year x av 8 hrs per shift)] ÷ 7104 hmg/m^3 = 0.14 mg/m^3

overall: SMR = 2.8; 95% CI 2.2-3.5, and emphysema in particular: SMR = 2.5; 95% CI 1.9-3.2. No RD or RCS data were provided. When analyzed by duration of employment, a 4-fold statistically significant increase in mortality was observed for emphysema after 10-15 years of work in the uranium mines; SMR = 4.0; 95% CI 2.2-6.7. Statistically significant mortality increase was also seen for duration of mining work > 15 years; SMR = 2.8; 95% CI 1.7-4.4; however, there was no significant trend for increased emphysema mortality with increasing duration of employment.

When analyzed by decade, the highest mortality for emphysema occurred from 1970-1979: SMR = 3.8; 95% CI 2.6-5.4. Although no increasing trend was observed over the three decades of the study, significantly elevated mortality for COPD remained in the last decade of follow-up; 1980-1990: SMR = 2.7; 95% CI 2.0-3.5. The authors concluded that although smoking rates were somewhat higher than in the general population, this was not a likely explanation for the elevated mortality from COPD or emphysema. They also concluded that COPD, in addition to lung cancer and silicosis, continued to be the most important long-term mortality risk for uranium miners.

Conclusions for COPD mortality

Wyndham et al. (1986) found a significant excess mortality for chronic respiratory diseases in a cohort of white South African gold miners. Among death certificate data that included those that died from silicosis, the authors found evidence that the most common cause of death was COPD. A case-control analysis found a nearly 2 ½-fold statistically significant increased risk for chronic respiratory disease per 10,000 particle years of dust exposure.

A synergistic effect of smoking and cumulative dust exposure on mortality from COPD was found in a study of white South African gold miners by Hnizdo (1990). Analysis of various combinations of dust exposure and smoking revealed a trend and indicated synergism between dust exposure and smoking. There was a statistically significant increasing trend for dust particle-years and for cigarette-years of smoking and COPD. For cumulative dust exposure, an exposure-response relationship was found, with the analysis estimating that those with exposures of 10,000, 17,500, or 20,000 particle-years exposure had a 2.5, 5.1, and 6.4-times higher mortality risk for COPD, respectively, than those with the lowest dust exposure of less than 5000 particle-years. The authors concluded that dust alone would not lead to increased COPD mortality but that dust and smoking acted synergistically to cause COPD and were the main risk factor for death from COPD in their study.

One of the largest studies of white South African gold miners to date (Reid and Sluis-Cremer 1996) found a statistically significant increased relative risk of COPD mortality of 1.2 after controlling for smoking. The authors concluded that the relative risk of death from COPD for miners who smoked ½ pack of cigarettes per day could be calculated as 1.2 raised to the power of cumulative dust exposure. For example, a typical miner who had a cumulative dust exposure of 3.7 mg/m^3 – years and a smoking history of ½ pack

of cigarettes per day, would have a statistically significant 2- fold increased risk of death from COPD (RR = $1.2^{3.7}$ = 1.96).

McIntyre Aluminum Powder

As documented in the Mining Master File, between 1943 and 1979 at least 27,500 Ontario hard rock miners, mainly underground gold and uranium miners, were required to inhale a finely ground aluminium powder known as “McIntyre Powder” (MP) before every shift in the belief that this would protect the lungs from silicosis caused by RCS.

The MP exposure level recommended by the McIntyre Research Foundation was one gram of MP per 1000 cubic feet of locker room volume (1g/1000 ft^3 = 1 g/28.32 m^3 = 35.3 mg/m^3) with a typical duration of 10 minutes. The time-weighted average (TWA) exposure for an eight-hour workday may be calculated as 0.74 mg/m^3 [(35.3 mg/m^3 x 10 minutes) ÷ (8 hours x 60 minutes) = 0.74 mg/m^3].[EE] For a hypothetical miner who was exposed at the start of every shift for 30 years from 1949 to 1979 the cumulative RD exposure from the MP may be estimated to have been ~ 22 mg/m^3-years (30 years x 0.74 mg/m^3 = 22.2 mg/m^3-years).

The contribution of MP to the cumulative RD exposure for Ontario miners was not included in the WSIB ASMB for COPD and therefore was typically not taken into account when estimating cumulative RD exposures. However, in October 2017, the WSIB acknowledged that exposure to MP routinely resulted in additional exposure to RD in the form of aluminum (WSIB Memo October 5, 2017 “McIntyre Powder Aluminum Prophylaxis – Associated Exposure). The WSIB concluded:

> *“In summary, based on available information, it is reasonable to expect that as a result of McIntyre powder aluminum prophylaxis in the miners’ change room at the onset of each shift, the worker’s equivalent 8-hour Time Weighted Average exposure to aluminum dust would be in the range of 0.5-1.0 mg/m³, during each working day when the practice was in place.”*

Using the WSIB’s estimated range of 0.5 to 1.0 mg/m^3 for the hypothetical miner considered earlier who was exposed to MP for 30 years, would result in a cumulative RD exposure ranging from 15 to 30 mg/m^3 – years that would have contributed from about one third to two thirds of the cumulative RD exposure of 40 to 50 mg/m^3 – years required by the WSIB COPD guideline.

[EE] *The American Conference of Government Industrial Hygienists (ACGIH) and the Ontario Ministry of Labour (MOL) currently recommend a TLV of 1.0 mg/m³ TWA for the respirable fraction of Al metal and insoluble compounds. The Ontario OHSA Regulation 833/90 currently includes excursion criteria for substances that do not have a short term (15 minutes) exposure limit (STEL). The excursion criteria state that if a substance does not have a STEL, which is currently the case for Al, the exposure shall not exceed the following excursion limits: three times the TWA TLV for any 30-minute period or five times the TWA TLV at any time (O. Reg. 833/90).*

Zarnke et al (2019) described the particle size and dose rate of MP in comparison to the TWA for aluminum powder and concluded that the calculated TWA exposure of 0.74 mg/m^3 for MP does not sufficiently consider the biological and physiological responses elicited by exposure to high concentrations of ultrafine particulates and likely underestimates the related health effects.

Diesel Exhaust (DE)

Background

Diesel engines are used in a variety of industries to power vehicles (e.g., trucks, forklifts, buses, railroad engines) and a wide range of heavy-duty equipment (e.g., mining equipment, earth movers, other construction equipment). The use of diesel-powered vehicles for ore haulage underground in Ontario gold mines began in the 1960s (Kabir et al 1993). Exposure to DE is not considered as contributing to Ontario mining exposures associated with COPD and is not included in the SR or AA documents. Only RD and RCS are considered.

Diesel exhaust is a complex mixture of gases and particulates generated by the combustion of diesel fuel (IARC Monograph 105, National Toxicology Program 2006, CAREX Canada 2019). The composition of DE depends on the type of diesel fuel, the type and age of the engine, tuning and maintenance, workload, and the exhaust treatment system. The gas compounds can include water vapour, carbon dioxide, carbon monoxide, nitrogen oxides, and volatile organic compounds, such as benzene and formaldehyde. The particulates consist of elemental and organic carbon, ash, sulfate, and metals. Polycyclic aromatic hydrocarbons (PAHs) and nitroarenes are present in the gas phase, and are also adsorbed onto the surface of the elemental carbon particles. Almost all the particulates in DE are respirable (<10 micrometers in diameter), with the majority having diameters of less than 1.0 micrometer. The particulates can occur individually or can “clump” together into clusters called agglomerates.

Elemental carbon (EC), also referred to as respirable elemental carbon (REC), was chosen by researchers as a surrogate or representative indicator of DE exposure since the early 1990s when it was found that most of the carcinogenic and mutagenic properties of DE were associated with the carbon particles (IARC Vol 105).

The current Ontario Ministry of Labour (MOL) mining regulations (MOL Reg 854) require that the time-weighted average exposure value (TWAEV) for DE in underground mines must not exceed 0.4 mg/m^3 or 400 ug/m^3 measured as total carbon (TC), or that the elemental carbon (EC) multiplied by 1.3 is not more than 0.4 mg/m^3, therefore, the equivalent TWAEV for EC should not exceed 310 ug/m^3.

Table 5 summarizes some representative occupational exposures to DE measured as EC from air sampling done between 1990 and 2007 (IARC Monograph 105, Pronk et al 2009).

There is considerable variation in DE exposures among different occupations and industries (IARC Monograph 105, Pronk et al 2009). Underground production or maintenance miners have some of the highest exposures to DE (≥ 50 ug/m^3), mainly because of the enclosed nature of the workplace and their proximity to the diesel vehicles and equipment. There can also be considerable variation in DE exposures within an occupation or industry. For example, underground miners are typically exposed to DE concentration 10 or more times greater than surface mine workers. There can also be variation within the same occupation due to variations in the diesel equipment or other exposure conditions. The variability in the concentrations of DE makes it difficult to assess the potential for health effects associated with these exposures.

The concentrations of DE in some workplaces have decreased in recent years largely because of stricter standards that required changes in diesel fuel composition (e.g., lower sulphur content) and more efficient exhaust treatment devices (e.g., filters or catalysts). The use of diesel-powered equipment and the resulting exposure to DE can be roughly divided into three periods:

1. *Traditional diesel exhaust (TDE) refers to the period before 1988 when diesel exhaust was essentially unregulated.*
2. *Transitional diesel exhaust refers to the period between about 1988 and 2006 when there were progressively more stringent emission requirements.*
3. *New technology diesel exhaust (NTDE) refers to the period after 2006 when the most recent diesel emission standards were adopted.*

The composition of NTDE is significantly different as it contains about 90% less particulates than TDE (IARC Monograph 105, Pronk et al 2009).

Table 5 Occupational exposure to DE measured as elemental carbon (EC)		
Occupation/Job Title	**Elemental Carbon[FF] (ug/m^3) (personal sampling)**	
	Range	**Average/Mean**
High Exposures > 50 ug/m^3		
tunnel construction	100 to 300	215
underground mining maintenance	53 to 144	141
underground mining production	148 to 637	135
Intermediate Exposures (≥ 10 ug/m^3 to 50 ug/m^3)		
dock workers (lift trucks)	4 to 122	43
diesel mechanics (trucks/buses)	4 to 39	29
railroad maintenance, mechanics	5 to 39	24
loading/unloading ships	6 to 49	11
above ground construction (heavy equipment operators)	4 to 13	10
Low Exposures (< 10 ug/m^3)		
vehicle testing, parking attendant, toll booth worker, transport terminal worker, traffic police officer	generally < 10	
unloading baggage from planes	generally < 10	
firefighters	non-detected to 40	generally < 10
mining- surface production	3.5 to 23	8
railroad train crews	4 to 20	8
truck drivers	1 to 22	2

DE Exposure and COPD

There have been several recent studies that have identified an increased risk of COPD in occupations exposed to DE.

Hnizdo et al (2002)

One of the most comprehensive studies of occupations with exposure to DE and COPD was carried out by Hnizdo et al (2002) based on data from the US Third National Health and Nutrition Examination Study (NHANES III). After adjustment for age, sex, race/ethnicity, body mass index, smoking (status and pack-

[FF] *Elemental carbon (EC), also referred to as respirable elemental carbon (REC), was chosen by researchers as a surrogate or representative indicator of DE exposure since the early 1990s when it was found that most of the carcinogenic and mutagenic properties of DE were associated with the carbon particles (adapted from Tables 1 to 4 in Pronk et al 2009 and IARC Monograph 105 Tables 1.14 to 1.17).*

years), educational level and socioeconomic status, elevated odds ratios (OR) were observed for industries with likely diesel exhaust exposure, such as, construction (OR = 1.3; 95% CI 0.8–2.3) and transportation and trucking (OR = 1.2 95% CI: 0.8–2.0). The odds ratios were higher in never smokers (construction OR = 3.5; 95% CI 0.9–14.0, transportation and trucking OR = 2.0; 95% CI 0.3–15.0). When analyzed by occupation, odds ratios were also elevated for occupations such as vehicle mechanics (OR = 2.0; 95% CI 0.9-4.1), transportation (OR = 1.4; 95% CI 0.6-3.0), construction workers (OR = 1.2; 95% CI 0.6-2.5), and motor vehicle operators (OR = 1.2; 95% CI 0.7-2.1).

Hart et al (2006)

A case-control study of US railroad workers found that after adjustment for smoking and other variables including age, race and healthy worker survivor effect (HWSE), work in DE-exposed jobs such as engineers and conductors was associated with higher risks of COPD mortality compared with work in unexposed jobs (Hart et al 2006). These risks increased with increasing years of work with the greatest risks observed for those individuals with the longest duration of work on operating trains. For example, engineers/conductors with ≥ 16 years of work starting in 1959 had statistically significant increased risk of COPD; multivariate-adjusted OR = 1.61; 95% CI 1.12–2.30.

Weinman et al (2008)

In a large case-control study by Weinman et al (2008), COPD cases and controls completed a questionnaire with information on demographics, family history and a detailed work history. As part of the job history, individuals reported routine (weekly) exposures to DE. In addition, industrial hygienists assessed the potential for DE exposure based on a review of the jobs and industries each individual reported. After adjusting for age and sex, individuals with any DE exposure diesel exhaust had a nearly 2-fold statistically significant increased risk of COPD compared to those with no DE exposure; OR = 1.9; 95% CI 1.3–3.0. The risk was greater than 6-fold among never-smokers; OR 6.4; 95% CI 1.3–31.6 and less than 2-fold for ever-smokers; OR = 1.7; 95% CI 1.1-2.7. However, as noted by the authors, this difference in risk may have been due to the small number of never smokers (n=7) compared to ever-smokers (n=89).

Hart et al (2009)

A retrospective cohort study of US railroad workers also observed that workers in jobs with DE exposure had an increased risk of COPD mortality relative to those in unexposed jobs (Hart et al 2009). Hazard ratios were calculated for those hired after 1945 by the following categories of years of work; 0 to ≤ 5 years, 5 to ≤ 10 years, 10 to ≤ 15 years, 15 to ≤ 20 years and ≥ 20 years. For example, a nearly 2-fold statistically significant increase in the age and HWSE adjusted hazard ratio was observed for those with ≥ 20 years of work in DE-exposed work; HR = 1.85; 95% CI 1.15-3.00. The results were only slightly attenuated and remained statistically significant after controlling for pack-years of smoking and years since quitting; HR = 1.67; 95% CI 1.03-2.71 for those with ≥ 20 years of work in DE-exposed work.

This study found that the association of COPD mortality and years of work as a railroad engineer/brakeman hired after the introduction of diesel locomotives in the 1950s was linear. For each additional year of work, the risk of COPD mortality increased by 2.5%; 95% CI 0.9% to 4.2%. This risk was only slightly reduced to 2.1% increase in COPD mortality risk for each additional year of work in DE exposed jobs after adjustment for smoking history. These findings were like those in the earlier case-control study (Hart et al 2006) that observed a smoking-adjusted odds ratio of 1.02; 95% CI 1.01 to 1.04) for COPD mortality that increased by about 2% per year for each additional year of work as an engineer/conductor.

Ferguson et al (2020)

A recent study by Ferguson et al (2020) provided a detailed examination of COPD mortality associated with DE exposure in the Diesel Exhaust Miners Study (DEMS)[GG]. This was the first study to directly examine the association between quantitative exposure estimates of DE and COPD. Hazard ratios (HRs) were estimated for COPD mortality and cumulative exposure to respirable elemental carbon (REC). Exposures were lagged by 0, 10 and 15 years to allow for a variable latency period between DE exposure and COPD mortality. Time-varying estimates of average and cumulative exposure to REC (µg/m^3 – years) and RD (mg/m^3 – years) were calculated for each year from first exposure during the period 1947 to 1967 to the end of the study follow-up in 1997.

The median average intensity REC was two orders of magnitude higher for ever-underground (92.1 µg/m^3) than surface-only workers (0.94 µg/m^3). The median average intensity of RD was higher among the ever-under-ground workers. However, it was observed that surface-only workers were more likely to die from COPD as well as from all causes combined.

As shown in Table 6, COPD mortality was elevated in both the middle (> 273–828 µg/m^3 - years) and highest tertiles (> 828-10,217 µg/m^3 - years) of cumulative REC exposure across all lag periods[HH] ranging from HR = 1.14; 95% CI 0.62-2.11 for ever-underground workers with cumulative exposures of > 828 - 10,217 µg/m^3 – years (no lag) to HR = 1.49; 95% 0.82-2.72 for ever-underground workers with cumulative exposures of > 273–828 µg/m^3 – years (15-year lag).

The cumulative exposure to > 273 µg/m^3 to 828 µg/m^3 – years for underground production or maintenance miners would be equivalent to a range of > 2 years to about 6 years at an average concentration of RC of approximately 140 ug/m^3 (based on the measurements reported for underground

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[GG] DEMS was conducted jointly by the National Institute of Occupational Safety and Health (NIOSH) and the National Cancer Institute (NCI). The study was initially designed to study lung cancer in eight non-metal U.S. mines (limestone, potash, salt, or trona) (Attfield et al., 2012). These facilities were chosen as study sites because they had extensive exposure to emissions from underground diesel engines and only low-level exposures to known lung carcinogens, radon, asbestos, and silica. (Silverman et al 2012, Attwood et al 2012).

[HH] Lagging refers to the period of time that exposures are not considered as they are thought to not affect the risk of cancer. For example, a 10-year lag period means that the last 10 years of exposure are not included in the calculation of the lung cancer risk.

production or maintenance miners by Pronk et al (2009) in Table 5 above). Surface-only workers had a greater than 2-fold risk of mortality from COPD after a cumulative exposure to > 2 μg/m^3 to 22 μg/m^3 – years. This would be equivalent to a range of > 1 year to < 3 years at an average concentration of RC of approximately 8 ug/m^3 (based on the measurements for mining surface production reported in Table 6).

As shown in Table 6, the highest HRs were observed among surface-only workers: hazard ratios were increased two-fold for the middle tertiles (> 9–22μg/m^3-years) in the unlagged (HR = 2.34; 95% CI 1.11–4.61) and 10-year lagged models (HR = 2.25; 95% CI 1.10–4.64) and were statistically significant. In the 10-year and 15-year lagged models, surface-only workers in the highest cumulative REC category (>22 to 293 μg/m^3-year) were 2.0 (95% CI 0.74-5.93) and 2.01 (95% CI 0.77–5.26) times more likely to die of COPD than those in the lowest category of exposure (0–9 μg/m^3-years), respectively.

The authors suggested that the larger risk of COPD mortality among surface-only workers may have been due to a different underlying susceptibility than the ever-underground workers that resulted in a different exposure-response relationship.

Another possible explanation is that the healthy worker survivor effect (HWSE)[II] is greater for underground workers than for surface-only workers. Bias would be introduced into the analysis by the transfer of sick workers to the surface where exposures are lower, or out of the workforce altogether. (Buckley et al., 2015). Thus ever-underground miners might well be a hybrid group made up of healthier workers able to tolerate higher dust and DE exposures and unhealthy workers unable to tolerate underground working conditions and forced to the surface or out of the job. This may result in a likely underestimation of adverse effects of underground RD and DE exposures and lower hazard ratios for COPD mortality in the ever-underground group.

Ferguson et al (2020) also included a more complex analysis (parametric g-formula) to control for the HWSE effect. This analysis found that COPD mortality among ever-underground miners would have been reduced by 38% if they had never been exposed to REC.

Strengths of this study included a large and highly DE-exposed cohort with detailed work histories and long follow-up, as well as a quantitative assessment of historical DE exposure.

The authors acknowledged the limitation of the small number of COPD cases for the analyses; however, they concluded that although the estimates were not very precise, they were consistent with the published literature showing that reducing DE exposures would reduce the number of COPD deaths (Hart et al 2012, Blanc 2012). Taken together, these findings indicate that DE exposure may be associated with excess COPD mortality among underground miners.

[II] Healthy worker survivor effect (HWSE) is a form of selection bias where healthy workers tend to accumulate the most exposures as those that are unhealthy are likely to leave employment or work in areas of lower exposure (Brown et al 2017, Buckley et al 2015)

The WSIB does not routinely include exposure to DE in the assessment of exposure to respirable particles for COPD claims from Ontario miners.

Table 6 Hazard Ratios (HR) (95% confidence intervals) for COPD mortality by cumulative exposures to respirable elemental carbon (REC) for the Diesel Exhaust and Miners (DEMS) cohort			
All Workers			
Cumulative REC (ug/m^3 - years	**No Lag [# cases]**	**10-year lag [# cases]**	**15-year lag [# cases]**
0-22	1.00 [40]	1.00 [48]	1.00 [52]
> 22-453	0.73 (0.43-1.24) [47]	0.80 (0.46-1.37) [42]	1.28 (0.75-2.21) [50]
>453-10,218	0.88 (0.45-1.71) [53]	1.17 (0.60-2.29) [50]	1.31 (0.66-2.58) [38]
Ever-Underground Workers			
0-273	1.00 [23]	1.00 [27]	1.00 [34]
> 273 - 828	1.35 (0.72-2.52) [27]	1.48 [0.80-2.72) [28]	1.49 (0.82-2.72) [28]
> 828 – 10,217	1.14 (0.62-2.11) [33]	1.25 (0.66-2.39) [28]	1.20 (0.61-2.37) [21]
Surface-Only Workers			
0-9	1.00 [11]	1.00 [15]	1.00 [23]
> 9 - 22	**2.34 (1.11-4.61) [25]**	**2.25 (1.10-4.64) [24]**	1.36 (0.67-2.80) [18]
> 22 - 293	1.22 (0.47-3.21) [21]	2.00 (0.74-5.39) [18]	2.01 (0.77-5.26) [16]

Statistically significant HR in bold; HRs adjusted for exposure to respirable dust, time-varying job location (all workers, & ever-underground workers), year of hire, birth year and race [adapted from Table 2 in Ferguson et al (2020)]

Vapors, Gases, Dusts, and Fumes (VGDF) and COPD

The WSIB guideline for COPD only takes into account occupational exposure to RD; however, Ontario hard rock miners are also exposed to other inhaled vapors, dusts, fumes and gases. These include blasting agents, other metals and contaminants in the rock being mined, oil mist generated during the operation of mining equipment and solvents used for cleaning or maintenance tasks. There is considerable recent literature that suggests exposure to a wide range of VGDF is associated with an increased risk of COPD (Oliver, 2020, Eisner et al 2010, Bergdahl et al 2004, Balmes et al 2003, Trupin et al 2003).

Occupational exposures to VGDF may influence the course of COPD in several ways by:

1. causing COPD;
2. interacting to increase the risk for COPD and/or the severity of COPD;
3. interacting with cigarette smoke and amplifying its effect on risk for COPD (as has been shown);
4. increasing exposure-related disability; and
5. accelerating the rate of decline of respiratory function in persons with established COPD.

Historically, the identification of work-related COPD has been based on the observation of an excess occurrence of COPD among exposed workers in specific industries (Becklake, 1989). Subsequently,

large population-based studies conducted by Zock et al (2001) and Sunyer et al (2005) using data from the European Community Respiratory Health Survey (ECRHS), and by Blanc et al (2004), Blanc et al (2009), and Weinmann et al (2008) using data from cohorts of COPD patients with occupational exposure to VGDF observed increase in COPD risk associated with these exposures. A cross-sectional study of 185 male COPD patients by Rodriguez et al (2008) found significant increase in risk for respiratory symptom of sputum and dyspnea and significant decrease in lung function in association with occupational exposure to mineral dust or any VGDF.

Harber et al (2007) investigated the impact of occupational exposures on lung function change over a 5-year period in persons with early COPD. Controlling for smoking, ongoing occupational exposure to VGDF after onset of COPD was associated with decline in FEV1. The authors concluded that although the average magnitude of this effect on the rate of lung function decline was smaller than that of continued cigarette smoking (0.25% vs. 1.2-1.9% of predicted FEV_1 per year of exposure), the effect was sufficient to cause a significant functional loss over a prolonged period of time, particularly when added to decline due to smoking. Furthermore, for some more sensitive individuals, the loss will exceed average values.

There have been a number of systematic reviews and meta-analyses on the association between occupational exposures to VGDF and COPD. An October 2018 paper reviewed the systematic reviews and meta-analyses published between January 1, 2009 and June 2017 in which COPD was assessed by data on lung function and when work-related exposures to VGDF were described (van der Molen et al 2018). From 23 potentially eligible reviews or meta-analyses, eight met the inclusion criteria. Two were considered to be of high quality (Ryu et al 2015, Bruske et al 2013), four were moderate quality (Alif et al 2016, Bruske et al 2014, Omland 2014 and Borup 2017), and two were of low quality (Baur et al 2012, Fontana et al 2017).

All of the studies found that occupational exposures to VGDF were associated with increased COPD risk. Four adjusted for smoking (Ryu et al 2015; Bruske et al 2013, 2014; and Alif et al 2016). Of the two high quality studies, one (Ryu et al (2015) showed summary odds ratios of 1.4, 95% CI 1.19-1.73, for occupational exposure to VGDF and the other Bruske et al (2013) found a mean decline in FEV1 of 5.7%, 95% CI 2.71 – 8.62, in association with exposure to inorganic dust.

A recent paper by Grahn et al 2021, described the findings from a large population-based cohort study in Stockholm, Sweden. The purpose of the study was to determine which types of airborne particles in occupational settings are associated with an increased risk of developing COPD. Exposures were assessed using a quantitative job exposure matrix (JEM) and exposure levels were based on exposure measurements from Sweden when available or from other Nordic countries. Statistically significant increased risk of COPD was observed among men in the high exposure categories for RCS (mean

concentration 0.052 mg/m^3)[JJ]; HR=1.63; 95% CI 1.17-2.27; and welding fumes (mean concentration 0.805 mg/m^3); HR = 1.57; 95% CI 1.12-2.21.

Conclusions for VGDF

The published scientific literature supports associations between risk for COPD and occupational exposures to a number of VGDF together and individually. Most notable and relevant to exposures experienced by miners in Northern Ontario are RDs, RCS, and DE. An additional and unique exposure for underground miners in Ontario gold and uranium mines is McIntyre Powder. Studies have shown increased COPD risk at cumulative exposures to VGDF (that includes RD, RCS, DE and McIntyre Powder) in far lower than that required by the WSIB COPD guidelines of 40-50 mg/m^3-years for eligibility for compensation for COPD.

A recent independent scientific review conducted on contract to the WSIB has concluded that increasing intensity and/or duration of occupational exposure to VGDF and/or cigarette smoke is associated with greater decrement in lung function and greater COPD risk (WSIB 2021a). Nevertheless, the WSIB has not expanded its COPD guidelines to take into account more recently published scientific data regarding dose-response relationships and the broader classification of VGDF.

[JJ] *The mean RCS concentration was about ½ the current MOL OEL of 0.1 mg/m^3.*

Appendix 1 Konimeter, Thermal Precipitator and Size-Selective Gravimetric Sampler

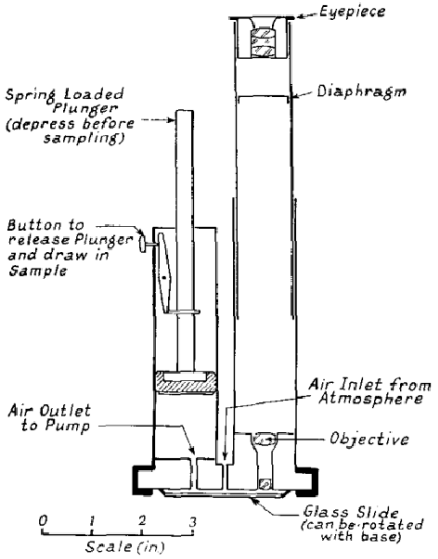
Konimeter

Konimeter Particle Counting

- provides instantaneous “grab samples” of total dust particles (non-selective)
- piston draws in 1 sec of air
- particles deposited on glass slide & counted under microscope units in ppcc

Source: University of Toronto Scientific Instruments Collection
https://utsic.utoronto.ca/wpm_instrument/particle-instrument/

Figure 2 Schematic of a konimeter



Source: Monitoring for Safety and Health. Unwin, Ian. (2018)

Thermal Precipitator

Figure 3 Casella Thermal Precipitator

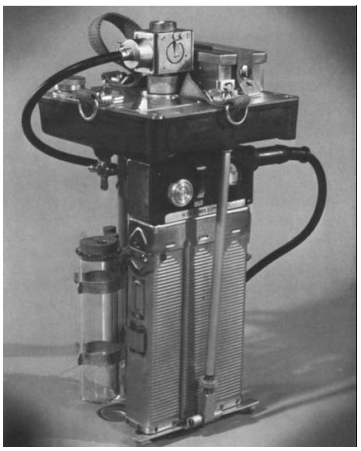
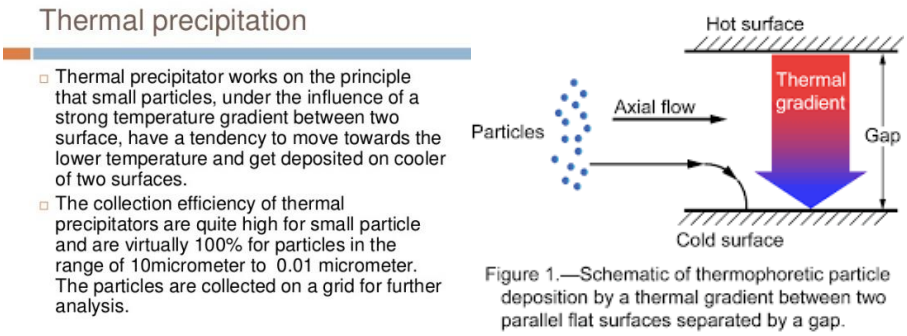
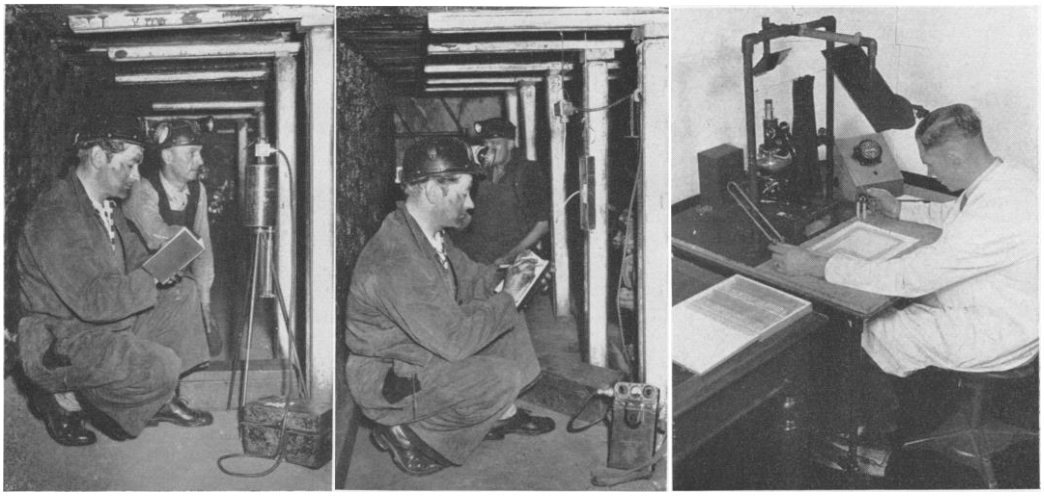


Figure 4 Description of how a thermal precipitator works



Source: Design of a Thermal Precipitator for the Characterization of Smoke Particles from Common Spacecraft Materials. National Aeronautics and Space Administration (NASA) June 2015

Figure 5 Sampling with a Thermal Precipitator



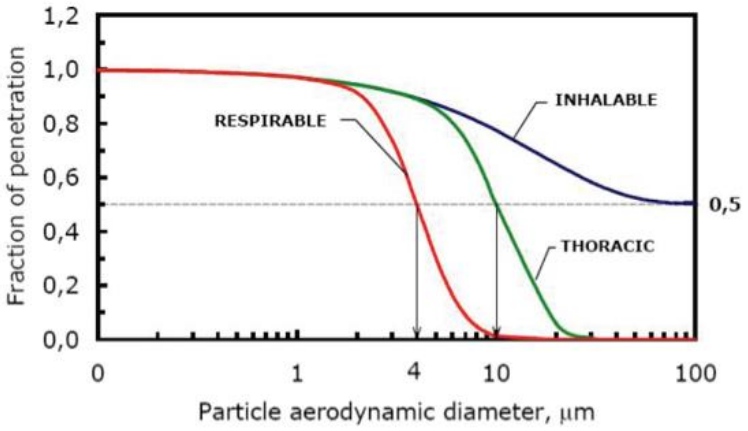
Standard Equipment **Portable Equipment** **Counting the dust particles**

Source: Roach Measuring Dust Exposure with the thermal precipitator in collieries and foundries Br J Ind Med. 1959 Apr; 16(2): 104–122.

Figure 6 Size-Selective Gravimetric Dust Sampler

Gravimetric Sampling

- provides size selective samples of different fractions of dust (inhalable, thoracic & respirable)
- battery powered pump allows partial or full shift sampling
- sampling head clips onto a worker's lapel & provides samples representative of personal exposure
- filter is weighed & by knowing the sampling time & flow rate of the pump the time-weighted average concentration (mg/m^3) of dust can be determined



Source: Dust Sampling Instrumentation and Methods. Jay Colinet Office of Mine Safety and Health Research Senior Scientist, National Institute for Occupational Safety and Health (NIOSH). Silica Dust Control Workshop Elko, Nevada September 28, 2010

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