

**OCCUPATIONAL EXPOSURE TO CHRYSOTILE ASBESTOS IN THE
CHRYSOTILE ASBESTOS CEMENT MANUFACTURING INDUSTRY
IN ZIMBABWE**

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A thesis submitted to the Faculty of Health Sciences, University of the Witwatersrand, in
fulfilment of the requirements for the degree of Doctor Philosophy

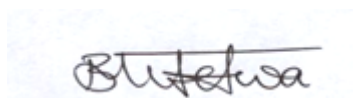
Johannesburg, South Africa, 2023

DECLARATION

This thesis is submitted in the optional format, approved by the Faculty of Health Sciences, of published work with encompassing introduction and conclusion.

I, Benjamin Mutetwa, declare that this thesis is my own work, except where acknowledged as otherwise. It is being submitted for the degree of Doctor of Philosophy in the University of the Witwatersrand, Johannesburg, South Africa. It has not been submitted before for any degree or examination at this or any other university.

Benjamin Mutetwa

A handwritten signature in black ink, appearing to read 'Mutetwa', is written over a light blue rectangular background.

Signed on the 14th day of August 2023

DEDICATION

I would like to dedicate this PhD to my wife and children;

Nyasha Pauline Mutetwa

and

Tinotenda, Anotida and Benjamin Jr

PUBLICATIONS, PAPERS AND PRESENTATIONS ARISING FROM THIS THESIS

Published Papers

1. **Mutetwa B**, Moyo D & Brouwer D (2022). Job Exposure Matrix for Chrysotile Asbestos Fibre in the Asbestos Cement Manufacturing (ACM) Industry in Zimbabwe. *International Journal of Environmental Research and Public Health*, 19(5), 2680. <https://doi.org/10.3390/ijerph19052680>.
2. **Mutetwa B**, Moyo D & Brouwer D (2021). Trends in Airborne Chrysotile Asbestos Fibre Concentrations in Asbestos Cement Manufacturing Factories in Zimbabwe from 1996 to 2016. *International Journal of Environmental Research and Public Health*, 18(20), 10755. <https://doi.org/10.3390/ijerph182010755>.
3. **Mutetwa B**, Moyo D and Derk Brouwer (2022). Prediction of Asbestos-Related Diseases (ARDs) and Chrysotile Asbestos Exposure Concentrations in Asbestos-Cement (AC) Manufacturing Factories in Zimbabwe. *International Journal of Environmental Research and Public Health*, 20(1), 58. <https://doi.org/10.3390/ijerph20010058>.
4. **Mutetwa B**, Moyo D & Brouwer D (2022). Examining approaches to prevention of exposure to chrysotile asbestos and examine some perspectives on the debate on ban of asbestos (Unpublished).

PAPER PRESENTATIONS AND OR POLICY DOCUMENTS DEVELOPED.

1. **Mutetwa B**, Moyo D & Brouwer D (2022). Occupational exposure to chrysotile asbestos in the chrysotile asbestos cement manufacturing industry in Zimbabwe. Policy Briefing to the Zimbabwe Occupational Safety and Health Council (ZOSHC) – a tripartite body that advises the Minister of Public Service, Labour and Social Welfare. 19 May 2022. National Social Security (NSSA) House.
2. **Mutetwa B**. Occupational exposure to chrysotile asbestos in the chrysotile asbestos cement manufacturing industry in Zimbabwe. Interim Seminar as part of the University of Witwatersrand, School of Public Health, Faculty of Health Sciences Interdisciplinary Doctoral Programme, Virtual, 22 July 2022.
3. **Mutetwa B**. Occupational exposure to chrysotile asbestos and prediction of asbestos related diseases in the chrysotile asbestos cement manufacturing industry in Zimbabwe. Safety and Health at Work conference, 3 October 2022. The Kingdom Hotel, Victoria Falls, Zimbabwe.
4. **Mutetwa B**. Developed draft regulation on “Control of Asbestos exposure Regulation, 2022. To be part of the new OSH regulations to come out with the New OSH Act expected in 2023 which is currently in Bill form.
5. **Mutetwa B**, Moyo D & Brouwer D (2023). Submitted an abstract on “Trends in Occupational Personal Chrysotile Asbestos Fibre Concentrations in Asbestos Cement Manufacturing Factories, 1996 to 2016 and Prediction of Asbestos Related Diseases (ARDs) in Zimbabwe” to present a paper at the Wits School of Public Health Research Day and CARTA Conference scheduled for 14-15 September 2023 at Wits School of Public Health.
6. **Mutetwa B** (2023). Submitted an abstract on “Trends in Occupational Personal Chrysotile Asbestos Fibre Concentrations in Asbestos Cement Manufacturing Factories, 1996 to 2016 and Prediction of Asbestos Related Diseases (ARDs) in Zimbabwe” to World Congress on Safety and Health scheduled for 27 -30 November 2023, Sydney, Australia. The Abstract was accepted (Abstract ID 898) and I am scheduled to present a digital poster.

ABSTRACT

Introduction

Asbestos is a generic term for a group of naturally occurring silicates that principally include serpentine variety (white chrysotile asbestos) and the amphibole variety, consisting of crocidolite (blue asbestos), amosite (brown asbestos), anthophyllite, actinolite and tremolite. Asbestos exposure has drawn much international, regional and national attention as it presents significant public and occupational health concerns. All asbestos types are known to cause asbestos related disease.

Objectives

The objectives of this PhD were;

1. To analyse trends in airborne chrysotile asbestos fibre exposure data obtained by the chrysotile asbestos cement manufacturing factories for the period 1996 to about 2016.
2. To establish a job exposure matrix (JEM) to estimate occupational exposure levels in the Zimbabwe chrysotile asbestos industry using available exposure data.
3. To predict asbestos related diseases (ARDs) namely lung cancer, mesothelioma, gastrointestinal cancer and asbestosis in the chrysotile asbestos cement manufacturing industry through exposure levels obtained in the factories.
4. To assess amphibole contaminants in the chrysotile asbestos fibre being used by the factories in the manufacture of asbestos cement (AC) products.
5. To examine approaches for prevention of exposure to chrysotile asbestos fibre and some perspectives on the debate on asbestos ban.

Methodology

A retrospective cross-sectional study using the factories personal chrysotile exposure data was designed to evaluate exposure patterns over time. Analysis involved close to 3000 personal exposure measurements extracted from paper records in the two-asbestos cement (AC) manufacturing factories in Harare and Bulawayo, covering the period 1996-2020. Exposure trends were characterised according to three to four time periods and calendar years to gain insight into exposure trends over time. Operational areas for which personal exposure data were available were saw cutting, fettling table, kollergang, moulded goods, ground hard waste,

laundry room, and pipe making operations in the case of the Bulawayo factory. The standard method of the Asbestos International Association (AIA) Recommended Technical Membrane Filter Reference Method (AIA, 1982) was reported to be used to collect personal chrysotile asbestos fibre in various operational areas over the years. Quantitative personal exposure chrysotile fibre concentration data collected by the two factories over the considered period were used to construct the JEM. Analysis of amphiboles in locally produced and imported raw chrysotile fibre samples used in the manufacturing processes was done using Scanning Electron Microscopy (SEM) and Energy Dispersive Spectroscopy (SEM). Prediction of asbestos related diseases (ARDs) was done by combining the JEM converted to cumulative exposures, with OSHA's linear dose effect model in which asbestos related cancers was derived using linear regression equations established for lung cancer, mesothelioma and gastrointestinal cancer by plotting estimates of cancer mortality cases versus respective cumulative exposures. The linear regression equations were applied to establish estimates of possible cancer mortality while for asbestosis, the linear in cumulative dose equation, $R_a = m(f)(d)$, where R_a – predicted incidence of asbestosis, m – slope of linear regression taken as 0.055, f – asbestos fibre concentration and d – duration of exposure, was used to estimate possible asbestosis cases over the respective duration of exposure at 1, 10, 20 and 25 years. To examine arguments for approaches used for prevention of exposure to chrysotile asbestos and examine some perspectives on the debate on asbestos ban, a literature search was conducted. Literature materials that advocated for the complete ban of all forms of asbestos including chrysotile as the only means of control of exposure and that, which argues for the controlled use approach, were reviewed. Search words used in literature search were chrysotile asbestos exposure, asbestos-cement, ban asbestos, controlled use, asbestos related disease, mesothelioma, lung cancer and asbestosis.

Data analysis was conducted using IBM SPSS version 26. For analysis, monthly averaged personal exposure levels for the factories were used. Mean personal airborne chrysotile fibre concentrations were analysed per operational area per factory and trends in airborne fibre concentrations over the years were displayed graphically. ANOVA was applied with the aim of identifying patterns of exposure variability among the time-periods for various job categories and determine whether there was a statistically significant difference in exposure concentrations between four time-periods for various jobs. Additionally, a Tukey Post Hoc Test (Tukey's Honest Significance Difference test) was run to find out which specific group means of time periods (compared with each other) were different.

Results and Discussion

Trends in airborne chrysotile asbestos fibre concentrations in asbestos cement manufacturing factories in Zimbabwe from 1996 to 2016.

Mean personal exposure chrysotile asbestos fibre concentrations generally showed a downward trend over the years in both factories. Exposure data showed that over the observed period 57% and 50% of mean personal exposure chrysotile asbestos fibre concentrations in the Harare and Bulawayo factories, respectively, were above the Zimbabwean OEL of 0.1 f/mL, with overexposure generally being exhibited before 2008. Overall, personal exposure asbestos fibre concentrations in the factories dropped from 0.15 f/mL in 1996 to 0.05–0.06 f/mL in 2016, a decrease of 60–67%. Statistically significant relationships were observed over time between exposure levels and calendar year and time periods ($p < 0.001$) for all occupational categories other than fettling table operations in Harare. The general decline in exposure over time from 1996 to 2016 suggests good occupational safety and health (OSH) framework being implemented by the two factories over the years, with the years after 2008 showing much lower exposure levels below the OEL particularly for the Bulawayo factory. However, for the period 2018 to 2020 exposures in the Harare factory were much higher than the preceding time period of 2009 to 2016 due to movement of trucks within the factory as they come to load concrete tiles and other products making it possible for residual chrysotile fibre left during manufacture of AC products to become airborne. The company reported no clean-up of asbestos in the factory or wetting of the floors to control dust, hence the possible increased levels of chrysotile asbestos fibre for the period 2009 to 2016. The general decreasing trends in exposure to chrysotile asbestos fibre may also be viewed from the fact that industry was responding to anticipated lowering of chrysotile OEL as a result of increased calls to ban all forms asbestos, triggering the scaling up of exposure controls in the factories.

Job Exposure Matrix for chrysotile asbestos fibre in the asbestos cement manufacturing (ACM) industry in Zimbabwe.

On average, all jobs/occupations in both factories had annual mean personal exposure concentrations exceeding the OEL of 0.1 f/ml, except for the period 2009 to 2016 in the Harare factory and for the time-periods 2009 to 2020 in the Bulawayo factory. Despite Harare factory having no AC manufacturing activity since 2017, personal exposure concentrations showed elevated levels for the period 2018-2020. Amphiboles were detected in almost all presently

collected bulk samples of chrysotile asbestos analysed. The established JEM, which was successfully generated from actual local quantitative exposure measurements, can be used in evaluating historical exposure to chrysotile asbestos fibre, to better understand, inform and predict occurrence of ARDs in future.

Prediction of Asbestos Related Diseases (ARDs) and chrysotile asbestos exposure concentrations in asbestos-cement (AC) manufacturing factories in Zimbabwe.

The results show that more cancer and asbestosis cases were likely to be experienced among those workers exposed before 2008 as exposure levels (0.11-0.19 f/ml) and subsequently cumulative exposures were generally much higher than those experienced after 2008 (0.04-0.10 f/ml). After a possible working exposure period of 25 years, overall cancer cases, i.e., estimates of possible cancer cases in a factory for each respective duration of exposure, predicted in the Harare factory were 325 cases per 100 000 workers while for the Bulawayo factory 347 cancer cases per 100 000 workers exposed may be experienced. Asbestosis cases likely to be detected after 25-years duration of exposure ranged from 50 to 260 cases per 100 000 workers (0.05 to 0.26% incidence of asbestosis) for various jobs. Possible high numbers of ARDs are likely to be associated with specific tasks/job titles, e.g., saw cutting, kollergang, fettling table, ground hard waste and possibly pipe making operations as cumulative exposures though lower than reported in other studies may present higher risk of health impairment.

Examining approaches for prevention of exposure to chrysotile asbestos and some perspectives on the debate on ban of asbestos.

Different perspectives on approaches to the prevention of exposure to asbestos have been presented. One position argues that there exist major differences in health risk between amphiboles and chrysotile asbestos, that low exposure and risk experienced under today's workplace conditions are completely different to high-risk exposures experienced in the past where occupational hygiene conditions were very poor and levels of education, awareness and training in the asbestos industry was low compared to the present situation. It is further argued that there are low levels of exposure below which risk of health impairment becomes insignificant, hence controlled use approach as a measure of exposure control can be successfully applied. However, the other position holds that all forms of asbestos including chrysotile are equipotent, that there is no safe level of exposure, that controlled use is not

practical and that there is no merit in continuing use of chrysotile asbestos in light of safer alternatives available today. Both positions appear plausible. Banning as a form of control measure occupies a high level in the hierarchy of controls with potential to eliminate the hazard and risk; nonetheless, the banning of chrysotile may imply substitution with materials that have been reported to carry health risk of cancer and other health impairments. On balance, banning may possibly not be the panacea of elimination of ARDs, in view of the fact that some other forms of mining such as diamond and gold mining have been associated with exposure to amphibole asbestos. The controlled use approach may provide real possibilities of prevention of exposure to levels that presents minimal risk to health if effectively implemented as applied to a range of occupational hazards with success.

Conclusion

Not much is known about exposure to airborne chrysotile asbestos fibre exposure in Zimbabwe chrysotile asbestos cement (AC) manufacturing industry. This study may constitute the single largest characterisation of personal exposure chrysotile asbestos fibre concentrations data set in Zimbabwe in which about 3000 airborne personal exposure measurements collected from company records spanning a period of about 25 years, were used in assessing exposure trends over time, building a job exposure matrix, and predicting possible ARDs namely lung cancer, mesothelioma, gastrointestinal cancer and asbestosis in Zimbabwe AC manufacturing industry. The study adds considerably to future epidemiological studies, gives insights into possible magnitude of ARDs that may be observed in AC factories and possibly analysis of exposure response relationships that may be linked to exposure episodes in the distant past. The study also gives some insights into possible amphibole contaminants that may be associated with local and imported chrysotile asbestos that is used in the AC manufacturing processes and thus providing support for a more comprehensive investigation into the presence of amphiboles in chrysotile asbestos in Zimbabwe. The study also provides some perspectives on approaches to prevention of exposure to asbestos and some aspects on the call to ban all forms of asbestos including chrysotile.

Personal exposure chrysotile fibre concentration data in the two AC manufacturing factories showed a downward trend over the years, and that overexposure as evaluated against the OEL of 0.1 f/ml were being exhibited largely before 2008.

The job categories with high exposure levels were saw cutting, fettling, ground hard waste, laundry room and multi-cutter operator and such jobs are likely to be associated with high risk of ARDs particularly for exposures happening before 2008. Moulded goods operators were associated with low exposures as process is generally a wet process.

Despite exposure concentrations being high in the earlier time periods of 1996 to 2008, declines over time particularly for Bulawayo factory which has continued to use chrysotile to date, suggests that controlled use approach may yield exposures that may present minimal risk to health of those exposed to chrysotile asbestos. While banning can still be considered as a way to eliminating ARDs, it may not necessarily be the panacea for prevention of ARDs, as controlled use approach may perhaps still present real possibilities of prevention of exposure to levels that may present minimal risk to health impairment if effectively implemented as applied to a range of hazards with some success. Banning would possibly imply substitution by materials reported to be hazardous to health. These results can be used in future epidemiological studies, and in predicting the occurrence of asbestos-related diseases in Zimbabwe.

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LIST OF ABBREVIATIONS

AC	Asbestos cement
ACGIH	American Conference of Governmental Industrial Hygienists
ACM	Asbestos Cement Manufacturing
AIA	Asbestos International Association
ARD	Asbestos related disease
ATSDR	Agency for Toxic Substances and Diseases Registry
DECOS	Dutch Expert Committee of Occupational Safety
ECHA	European Chemicals Agency
EDS	Energy dispersive spectroscopy
JEM	Job exposure matrix
IARC	International Agency for Research on Cancer
IPCS	International Program on Chemical Safety
IOM UK	Institute of Occupational Medicine United Kingdom
HCN	Health Council of the Netherlands
NIOSH	National Institute for Occupational Safety and Health
NIOH	National Institute of Occupational Health
NSSA	National Social Security Authority
OEL	Occupational Exposure Limit
OSH	Occupational Safety and Health
OSHA	Occupational Safety and Health Administration
PEL	Permissible Exposure Limit
SEM	Scanning Electron Microscopy
TEM	Transmission Electron Microscopy
WHO	World Health Organization
ZNCATF	Zimbabwe National Chrysotile Asbestos Task Force

AUTHOR CONTRIBUTIONS

As part of the declaration, contribution of co-authors to each of the published studies are outlined below.

Chapter 6

Mutetwa B, Moyo D & Brouwer D (2021). Trends in Airborne Chrysotile Asbestos Fibre Concentrations in Asbestos Cement Manufacturing Factories in Zimbabwe from 1996 to 2016. *International Journal of Environmental Research and Public Health*, 18(20), 10755. <https://doi.org/10.3390/ijerph182010755>.

The concept and study design was formulated through discussion with Professor Derk Brouwer. I collected and extracted all the personal chrysotile asbestos exposure data from paper records of the occupational hygiene assessment with the assistance of the company safety, health and environmental personnel. Prof Derk Brouwer and Dr Dingani Moyo reviewed and edited the manuscript.

Chapter 7

Mutetwa B, Moyo D & Brouwer D (2022). Job Exposure Matrix for Chrysotile Asbestos Fibre in the Asbestos Cement Manufacturing (ACM) Industry in Zimbabwe. *International Journal of Environmental Research and Public Health*, 19(5), 2680. <https://doi.org/10.3390/ijerph19052680>.

The concept and study design was formulated through discussion with Professor Derk Brouwer. I developed the methodology in consultation with the supervisor, carried out the data analysis on the personal chrysotile asbestos exposure data I extracted from paper records of the occupational hygiene assessments with the assistance of the company safety, health and environmental personnel and wrote the initial draft of the manuscript. Prof Derk Brouwer and Dr Dingani Moyo reviewed and edited the manuscript.

Chapter 8

Mutetwa B, Moyo D & Brouwer D (2022). Prediction of Asbestos-Related Diseases (ARDs) and Chrysotile Asbestos Exposure Concentrations in Asbestos-Cement (AC) Manufacturing Factories in Zimbabwe. International Journal of Environmental Research and Public Health, 20(1), 58. <https://doi.org/10.3390/ijerph20010058>.

The concept and study design was formulated through discussion with Professor Derk Brouwer. I developed the methodology in consultation with the supervisor, carried out the data analysis and evaluation on the personal chrysotile exposure assessment and wrote the initial draft of the manuscript. Prof Derk Brouwer and Dr Dingani Moyo reviewed and edited the manuscript.

Chapter 9

Mutetwa B, Moyo D & Brouwer D (2022). Examining approaches to prevention of exposure to chrysotile asbestos and examine some perspectives on the debate on ban of asbestos.

The concept and study design was formulated through discussion with Professor Derk Brouwer. The methodology was developed and applied through discussion with Professor Brouwer. Prof Derk Brouwer and Dr Dingani Moyo reviewed and edited the manuscript.

As supervisor of the candidate, I confirm that he acted as principal investigator on all four studies.



Professor Derk Brouwer

14/082023

Date

CHAPTER 1

Introduction

Following a brief **introduction**, the chapter gives the **research aims and objectives** of the research, **conceptual framework** and ends with a brief overview of the **structure of the thesis**.

Asbestos is a generic term for a group of naturally occurring silicates that principally include serpentine variety (white chrysotile asbestos) and the amphibole variety, consisting of crocidolite (blue asbestos), amosite (brown asbestos), anthophyllite, actinolite and tremolite (IARC, 2012). Asbestos exposure has drawn much international, regional and national attention as it presents significant public and occupational health concerns. All asbestos types are known to cause asbestos related disease (IPCS, 1998; IARC, 2012; WHO, 2014; ECHA, 2021).

The World Health Organization reports that 125 million people worldwide are exposed to asbestos at the workplace, with 107 000 people succumbing to asbestos-related diseases annually (WHO, 2014). Although amphibole asbestos production has all but ceased worldwide, chrysotile asbestos continues to be produced and used in some countries. While production and use of asbestos in most developed countries has declined in recent years due to health concerns and the subsequent ban of asbestos and asbestos-containing products, there continues to be extensive production, sale and use of chrysotile in South and Central America, Asia, and Africa (ATSDR, 2001; WHO, 2014). Russia is the world-leading producer of chrysotile asbestos; others include China, Kazakhstan, Brazil and India with production at Zimbabwe chrysotile mines stalling in 2010 due to economic challenges. Currently, there are efforts to resuscitate the mining of chrysotile asbestos with tailings dumps being harnessed to extract fibres for the two-chrysotile asbestos cement manufacturing factories in the cities of Harare and Bulawayo.

Zimbabwe has long been one of Africa's major producers of chrysotile asbestos (Virta, 2006; Nelson and teWater Naude, 2015). During the 1970s, production averaged 200 000 metric tonnes per annum, rising to a peak of 259 000 tonnes in 1979. However, production declined to 100 000 per annum for the period 2004 to 2007 and reduced drastically during the hyperinflation period of 2008 such that, by 2010, only 2 400 tonnes were reported to have been produced (McCulloch, 2003, cited in Nelson and teWater Naude, 2015). Important chrysotile products that are produced in Zimbabwe include reinforced chrysotile asbestos roofing sheets and tiles, water pipes, heat resisting or fire resisting insulation materials, and packings and gaskets in the vehicle industry. The two chrysotile asbestos mines, Shabanie and Mashava Mines, had a combined production capacity of 140 000 metric tonnes of chrysotile asbestos in the 1980s and 1990s; 90% of this product was exported with 10% consumed by the local

chrysotile asbestos cement manufacturing industry (Zimbabwe National Chrysotile Task Force, 2014).

From the early 1990s, about 7 000 workers were engaged in mining and milling at two major mines, with about 4 000 engaged in the manufacturing of chrysotile asbestos products and other downstream Asbestos Cement (AC) industrial sectors such as construction (Cullen and Baloyi, 1991). During the same period, it was reported that 40 000 to 45 000 people lived within a few kilometres of the mills and mines, and a large proportion of population lived and worked in buildings with chrysotile asbestos (Cullen and Baloyi, 1991). Zimbabwe has two major factories that manufacture chrysotile asbestos products, and these factories have been the major users of chrysotile asbestos since their establishment in the 1940s and 1950s.

In Zimbabwe, there is no specific legislative instrument that governs the management of asbestos and enforcement of any occupational exposure limit (OEL) for chrysotile asbestos exposure. Management is generally through non-specific regulations, in particular the Statutory Instrument 68 of 1990 on Accident Prevention and Workers Compensation (Government of Zimbabwe, 1990). Moreover, there is also no statutory OEL for chrysotile asbestos fibre save for guidelines on OELs published by the National Social Security Authority– Occupational Safety and Health Division which has set the limit at 0.1 f/ml for all forms of asbestos fibre (National Social Security Authority, 2017). In the absence of specific guidance on the management of chrysotile asbestos exposure, the chrysotile asbestos cement manufacturing industry developed its own occupational exposure-monitoring programme for chrysotile asbestos where personal and static exposure data have been collected since the 1980s and more regularly from the 1990s. Thus, retrospective analysis of such data will provide an understanding of the extent of exposure to chrysotile asbestos fibre in the AC industry over the years and gives insights into prediction of asbestos related diseases in Zimbabwe. Such data also aided in the development of Zimbabwe’s industry specific job exposure matrix for future exposure assessment to chrysotile asbestos fibre in the ambient work environment, hence this PhD study.

1.1 Conceptual Framework

The conceptual framework diagram below shows the relationship between the dependent variable (chrysotile fibre exposure levels) and some independent variables as well as how a JEM influences estimates of exposure levels for tasks/occupations. It attempts to depict factors which can influence levels of exposure to chrysotile fibre asbestos chief among them being task/activities, process, time period and exposure prevention and control. The JEM combines such factors as exposure levels, time period, industry and tasks/occupation. Scientific views on exposure to chrysotile asbestos will have a bearing on possible asbestos management framework for the country. Implications of exposure levels on human health will also be informed by scientific views.

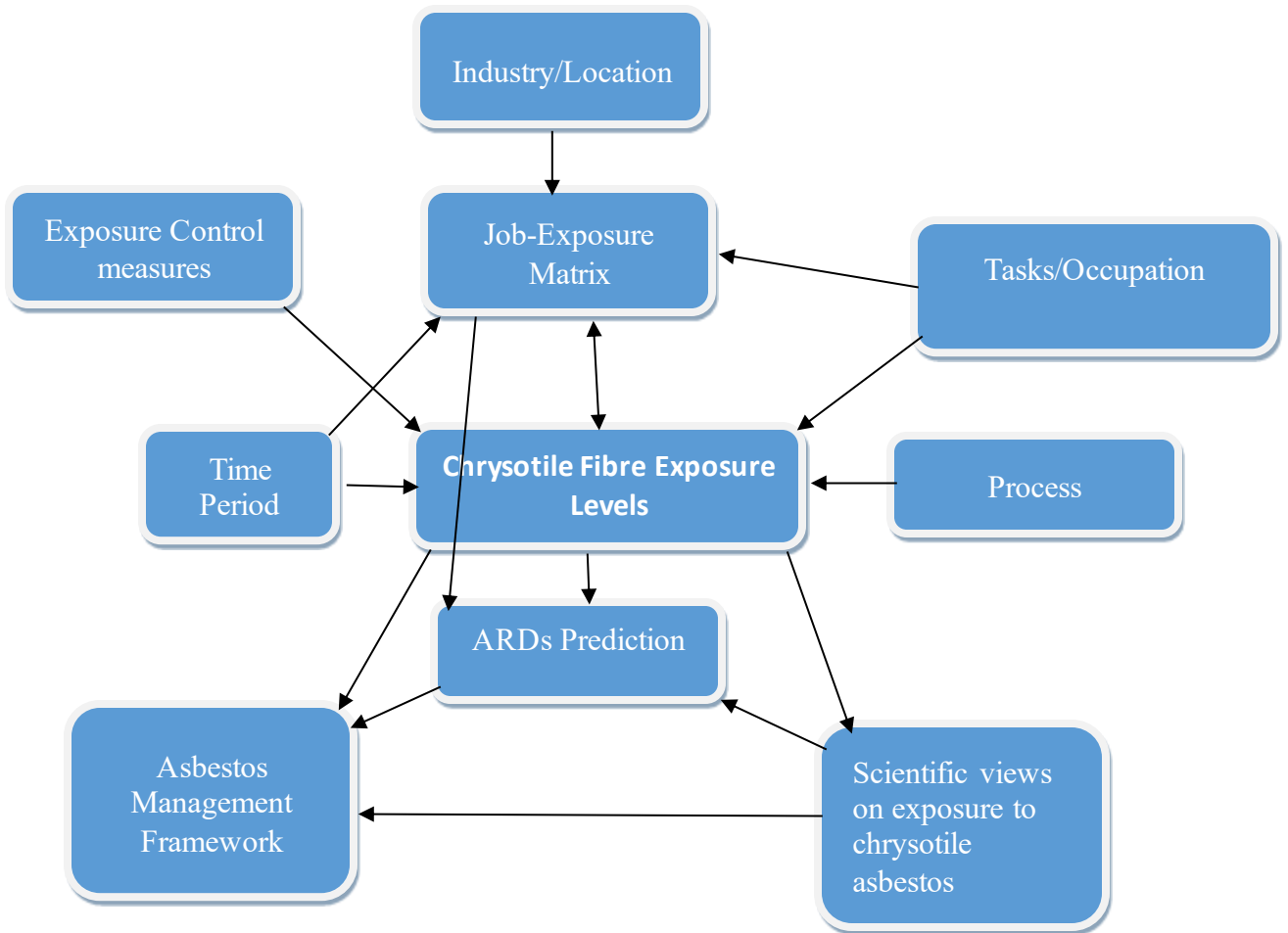


Fig 1: Conceptual Framework for Exposure to Chrysotile Asbestos Fibre (Mutetwa, 2018).

1.2 Research Aim and Objectives

Aim

To evaluate the extent of exposure to chrysotile asbestos fibre and predict asbestos related diseases in the chrysotile asbestos manufacturing industry in Zimbabwe.

Specific Objectives

1. To analyse trends in airborne chrysotile asbestos fibre exposure data obtained by the chrysotile asbestos cement manufacturing factories for the period 1996 to about 2016.
2. To establish a job exposure matrix (JEM) to estimate occupational exposure levels in the Zimbabwe chrysotile asbestos industry using available exposure data.
3. To predict asbestos related diseases (ARDs) in the chrysotile asbestos cement manufacturing industry through exposure levels obtained in the factories.
4. To assess amphibole contaminants in the chrysotile asbestos fibre being used by the factories in manufacturing of AC products.
5. To examine approaches for prevention of exposure to chrysotile fibre and the perspectives on the debate on asbestos ban in Zimbabwe.

1.3 Structure of the thesis

This thesis has 9 chapters. In **Chapter 1** a brief background to the study, aim and objectives of the study is given. In **Chapter 2**, a review of chrysotile asbestos is given highlighting its sources, structure and health effects. Additionally, chrysotile asbestos exposures in the chrysotile asbestos cement manufacturing industry are also given, further providing some aspects on occupational exposure limits associated with airborne asbestos. **Chapter 3** describes methods and materials used in this study. **Chapter 4** summarises the key findings of the study, which are given in more detail in Chapters 6-9. **Chapter 5** presents the results, discussion and conclusions drawn from each of the main four focal areas of the study namely trends in airborne chrysotile asbestos fibre concentrations in asbestos cement manufacturing (ACM) factories in Zimbabwe; development of a job exposure matrix for chrysotile asbestos fibre in the ACM factories in Zimbabwe; the prediction of ARDs with respect to lung cancer, mesothelioma, gastrointestinal cancer and asbestosis using chrysotile concentrations in ACM in Zimbabwe; and examining the approaches for prevention of exposure to asbestos and some perspectives to

the ban asbestos debate ends the thesis. **Chapter 6 – 9** presents details of publication work that essentially form the basis of this thesis. **Chapter 6** presents trends in airborne chrysotile asbestos fibre concentrations in asbestos cement manufacturing (ACM) factories in Zimbabwe, while **Chapter 7** presents the Job exposure matrix for chrysotile asbestos fibre in the ACM industry in Zimbabwe and chapter 8 gives insights into prediction of ARDs and chrysotile concentrations in ACM in Zimbabwe. **Chapter 9** examines the approaches for prevention of exposure to asbestos and some perspectives to the ban asbestos debate.

REFERENCES

Agency for Toxic Substances and Diseases Registry (ATSDR) (2001). Asbestos - Production, Import, Use and Disposal. CDC. USA. Available online at <https://www.atsdr.cdc.gov/toxprofiles/tp61.pdf> . (Accessed 11 July 2018).

Cullen M R and Baloyi SB (1991). Chrysotile asbestos and Health in Zimbabwe: 1 Analysis of Mines and Millers compensated for Asbestos – Related Diseases since Independence (1980). *Am J Ind Med*, 19(2), 161-9. DOI: [10.1002/ajim.4700190205](https://doi.org/10.1002/ajim.4700190205).

Government of Zimbabwe (Government of Zimbabwe) (1990). Statutory Instrument 68 of 1990: Accident Prevention and Workers Compensation Scheme. Government of Zimbabwe.

International Agency for Research on Cancer (IARC) (2012). IARC Monograph on Evaluation of Carcinogenic Risks to Human, Arsenic, Metals, Fibres and Dust: A review of human carcinogens Vol. 100C p.219 – 309. IARC/WHO.

International Programme on Chemical Safety (IPCS) (1998). Chrysotile Asbestos. Environmental Health Criteria 203: Geneva, UNEP, ILO, WHO and IPCS.

McCulloch J (2003). Asbestos mining and occupational disease in Southern Rhodesia/Zimbabwe, 1915 – 98. *Hist Work J*. 56, 131 – 152.

National Social Security Authority (NSSA) (2014, 2017). Guidelines on Occupational Exposure Limits (OELs) for Chemical Substances and Dust. NSSA, Harare.

Nelson G and WaterNaude J (2015). Epidemiology of Malignant Pleural Mesothelioma in Africa. In *Malignant pleural mesothelioma: Present status and future direction*. 1st Ed, UAE, Bentham Science.

Virta RL (2006). Worldwide Asbestos Supply and Consumption Trends from 1900 through 2003: Circular 1298. Available at: <http://pubs.usgs.gov/circ/2006/1298/c1298.pdf>. Accessed 23/4/18.

World Health Organization (WHO) (2014). Chrysotile Asbestos. Available online at;
<https://www.who.int/publications/i/item/9789241564816>. Published 2014. Accessed 9/2/2020.

Zimbabwe National Chrysotile Asbestos Task Force and Government of Zimbabwe (2014).
Zimbabwe Chrysotile Asbestos Position Paper. ZNCTF, Zimbabwe.

CHAPTER 2

Literature Review on Exposure to Chrysotile Asbestos Fibre in the Asbestos Cement (AC) Manufacturing Industry

This chapter reviews literature with respect to structure, chemical and physical properties of chrysotile asbestos. The chapter also provides details of sources of chrysotile asbestos exposures as well as health effects of exposure to chrysotile asbestos. In addition, possible non-occupational exposures to chrysotile asbestos are summarised. Occupational exposure limits relating to chrysotile asbestos are also highlighted.

2.1 Sources of Occupational Exposure to Chrysotile Asbestos.

Chrysotile asbestos is found in many serpentine rock formations and because of natural weathering processes and anthropogenic sources, chrysotile asbestos is found in air, water and landmasses (Nicholson & Pundsack, 1973; IPCS, 1998; IARC, 2012; ECHA, 2021). Globally, major deposits of chrysotile are found in Russia, Canada, South Africa and Zimbabwe with as much as 40 countries registering commercial deposits of chrysotile (IPCS, 1998). It is also interesting to note that as far back as in 1750, airborne chrysotile asbestos was identified in the Greenland ice cap, suggesting its existence long before its commercial exploitation (Bowes et al., 1977; IPCS, 1998), with anecdotal evidence suggesting that asbestos use had begun already 4500 years earlier where it was used to strengthen clay pottery in Finland and by different cultures in crematory shrouds, lamp wicks, incombustible napkins and tablecloths (Virta 2006; ECHA, 2021). Anthropogenic sources include mining and milling, processing of chrysotile into products such as friction materials, asbestos cement pipes and sheets, gaskets and seals, construction and transportation of fibre (IPCS, 1998; IARC, 2012; Eypert-Blaison et al., 2018b; ECHA, 2021). Some studies have also shown that AC factories are important sources of occupational exposure to chrysotile asbestos (Finkelstein, 1987; Kauppinen and Korhonen, 1987; Kimura, 1987; Higashi et al., 1994; Weiner et al., 1994). In mining, chrysotile ore is usually mined in open pit operations where drilling, blasting, and loading and transporting of ore are usually sources of emissions (IPCS, 1998; Schonfeld et al., 2017; Feletto, 2017). In China, for instance as much as 29 f/ml exposure levels were recorded in the ambient air at the largest chrysotile mine (Wang et al., 2012). Maintenance work in the telecommunication works has also been linked to exposure to chrysotile asbestos (Price et al., 1992). Exposure to chrysotile asbestos has also been associated with the petroleum refinery industry and some shipyard workplace settings have recorded exposure to chrysotile asbestos fibre as well (Williams et al., 2007; IARC, 2012). Chrysotile asbestos used today involves mainly production of products where the chrysotile fibre is encapsulated into matrices. The asbestos cement manufacturing industry constitute the largest user of chrysotile fibre accounting for 85% of all use (IPCS, 1998). Most chrysotile cement products contain 10-15% of fibre with the rest being

water and cement (IPCS, 1998). Emission sources of chrysotile asbestos fibre during production of chrysotile cement products includes feeding of asbestos fibre into the mix, blending the mix, and cutting or machining of sheets (IPCS, 1998; IARC, 2012; WHO, 2014). Production of friction products such as brake linings, clutch facings and gaskets have been associated with exposure to chrysotile (IPCS, 1998; Paustenbach et al., 2010; WHO, 2014). In the construction, asbestos friction and asbestos textile industries 0.4 f/ml, 1.7 f/ml and 6.7 f/ml geometric exposures to asbestos respectively were recorded in the Republic of Korea in 1984, however the respective exposure levels declined to 0.14 f/ml, 0.55 f/ml and 1.87 f/ml in 1996 (WHO, 2014).

2.2 Non-occupational exposures to chrysotile asbestos

Non-occupational exposure to asbestos also called environmental exposure can arise because of living with people occupationally exposed to asbestos, air pollution due to industrial processes such as mining and manufacturing (Goldberg and Luce 2009; IARC, 2012; WHO, 2014). Asbestos containing friction materials or naturally occurring asbestos due to weathering may also be a source of environmental exposure (Rees et al., 1999a; White et al., 2008; Phillips et al., 2012; WHO, 2014; Airoidi et al., 2021). South Africa (SA) has reported a rather high burden of environmental mesotheliomas with the highest proportion of environmentally induced mesothelioma at 33% (range 8.8 – 33% from a range of SA studies) being recorded by the South African Asbestos Tumour Reference Panel (Webster, 1973; Phillips et al., 2012). Documented cases of environmental mesothelioma in South Africa have been attributed to exposure to Cape crocidolite with some areas being found to be unfit for human habitation due to amphiboles pollution (Meintjes and Hemanus, 2008; Phillips et al., 2012). Furthermore, cases of ARDs are quite few in studies of non-occupational asbestos exposure. Only seven cases of pleural mesothelioma were reported in women resident in mining areas of Thetford Mines in Canada with population of about 45 000, from 1970 to 1989 (Camus et al., 1998; Phillips et al., 2012). Low levels of asbestos in rural outdoor locations have been reported to be within the range of $10\text{f}/\text{m}^3$ (0.00001 f/ml) with concentrations in urban areas and close to industrial sources and busy traffic intersections being much higher (IARC, 2012; WHO, 2014). Indoor air in schools, homes and other buildings have been reported to have asbestos concentrations ranging from $30\text{--}6000\text{f}/\text{m}^3$ (0.00003-0.006 f/ml) (IARC, 2012; WHO, 2014). In some public buildings holding asbestos, fibre concentrations were also reported to be between 0.0005 and 0.0045 f/ml in Belgium, Canada, Slovak Republic, UK and USA (IPCS, EHC 203, 1998). Additionally, no significant exposures to asbestos in and around houses in Soweto, South Africa, with asbestos

cement roofs, were identified (Phillips et al., 2007). Recently, a study on environmental exposure to asbestos from an AC plant reported that there was an increasing risk of mesothelioma that could possibly be attributed to asbestos materials from the AC plants (Airoldi et al., 2021). However, in Zimbabwe there has not been a single study on environmental exposure to asbestos despite a whole asbestos mining town of Zvishavane being in close proximity to chrysotile asbestos mines.

2.3 Structure and properties of chrysotile asbestos

Chrysotile asbestos, being naturally occurring fibrous hydrated magnesium silicate which belongs to the serpentine category of asbestos minerals generally carries the composition $Mg_3Si_2O_5(OH)_4$ (IPCS, 1998; Pollastri et al., 2016). The composition is largely a sheet of silicates with brucite layers ($Mg(OH)_2$) (IPCS, 1998; Bernstein, et al., 2013). The silicate layer has a tetrahedron embedded in a pseudo hexagonal network (Yarborough, 2006; Bernstein, et al., 2013; Pollastri et al., 2016; Gualtieri et al., 2019). The structure resembles a cylinder which cylinders results in chrysotile fibrils which make up the chrysotile fibre (Bernstein, et al., 2013). The magnesium element is coordinated with the oxygen atom in serpentine silicates (ECHA, 2021). The magnesium element makes a layer on the outside of the cylindrical role and the silicate sheet is 'rolled' around a virtual axis to form a tube known as a fibril (HCN, 2010; ECHA, 2021). Chrysotile asbestos has essentially a silky structure and its micro-fibrils can have a diameter of less than 0.03 μm and the fibrils give the chrysotile fibre its strength and flexibility (ECHA, 2021). It has been reported that chrysotile is highly soluble in aqueous neutral or acidic environment. The magnesium turns to leach from the outside brucite layer at a pH of less than 10 or generally in dilute acids (Atkinson, 1973; Langer and Pooley, 1973; Morgan and Crolley, 1973; Jaurand et al., 1977; IPCS, 1998). It has been argued that the magnesium is readily attacked by the acidic environment of macrophages which have a pH of 4-4.5 and the magnesium disintegrates from the crystalline structure leaving the new unstable silicate sheet which breaks up and decomposes into small particles which are easily cleared from the lungs by macrophages (Oze and Solt, 2010; Bernstein et al., 2013). It has been further suggested that the chemistry of chrysotile result in chrysotile clearing very rapidly from the lungs with $T_{1/2}$ being recorded as from about 0.3 to 11 days compared with amphiboles with $T_{1/2}$ of as much as 500 days to infinity (Bernstein and Hoskins, 2006). The break up and decomposition of chrysotile asbestos in acidic environment of macrophages and subsequently rapid clearance from the lungs by chrysotile has advanced the notion that chrysotile presents

low risk to health than amphiboles that are slow to clear from the lungs (Bernstein and Hoskins, 2006; Bernstein et al., 2013; Bernstein et al., 2020).

However, it has been argued that since chrysotile undergoes fast dissolution in the acidic environment it triggers the release of relatively toxic metal ions in the lung cells and the presence of these toxic ions in the lung environment explains why chrysotile can be considered just as toxic as amphiboles even though chrysotile is less biodurable than amphiboles (Gualtieri et al., 2019). These paradigms are some of the perspectives rendering chrysotile asbestos to remain a matter of significant and open discussion with much research evolving around chrysotile asbestos fibre in comparison with amphiboles asbestiform.

It is important to note that asbestos fibres can be considered as hard or soft depending on fibre flexibility. Generally, amphibole asbestos fibres are considered hard while chrysotile can be considered to be between hard and soft, however most chrysotile fibres are considered soft (Badollet, 1948; IPCS, 1998). The soft nature of chrysotile may necessitate the ease with which it has been reported to dissolve in biological systems and breaks up into smaller pieces (Bernstein et al., 2013).

Chrysotile asbestos ore is generally associated with other mineral contaminants such as iron, brucite, mica, feldspar, talc and carbonate minerals including magnetite, calcite and zeolites (Langer and Nolan, 1994; IPCS, 1998). Some amphibole fibres have also been associated with chrysotile, key among them being tremolite. Tremolite has been found at 0.01% to 0.6% levels with an average of 0.09% tremolite in about 35% of samples analysed (Addison and Davies, 1990; IPCS, 1998). Such trace contaminants are an important factor in disease causation (IPCS, 1998). Fibre type, size, chemical and crystalline composition, and bio-durability are among some critical variables that have been considered to influence the potential toxicity of asbestos fibre (Berman and Crump, 2008; Barlow et al., 2017), and that such determinants may inform measures of control that may be taken to prevent exposure.

2.4 Occupational Exposure Limits (OELs)

Occupational exposure limits (OELs) are standards or threshold limit values of airborne concentrations below which it is assumed that workers can be repeatedly exposed day after day generally over an 8-hour workday, a 40-hour work week and over a working lifetime without suffering any significant adverse health effects (Schenk et al., 2008, 2011; ACGIH, 2014)).

Hence, OELs are used to control exposure to hazardous substances and applied as important regulatory and risk management tools to protect workers' health from adverse health effects of hazardous substances exposure (Schenk, 2011; ACGIH, 2014). OELs have been changing over the years in response to new information on dose-response effects experienced by workers and/or in experimental animals.

OELs can be found as statutory in nature from which workplaces are expected to comply or as guidelines to aid in worker occupational health surveillance programmes. Setting of legally binding OELs is generally influenced by health implications of the hazardous substance as well as the socioeconomics in the respective country and technical feasibility in attaining the standard and cost implications. Hence, statutory OELs usually assume higher levels compared to guidelines usually set based only on the health implications of the hazardous substances on exposed workers.

OELs usually take different forms but principally are expressed in three different forms namely time-weighted average exposure limits (TWA-OEL), short term exposure limit (STEL) and ceiling levels (CL). TWA OELs or threshold limit value is the concentration for a conventional 8-hour workday and a 40-hour work week, to which it is believed that almost all workers may not develop adverse health effects following repeated exposure. Such OELs are intended to ensure that there are no adverse health effects during the entire working life (ACGIH, 2014). STEL is the maximum allowable concentration for a short duration of 15 minutes which must not be exceeded while the ceiling exposure limits are much shorter periods of about 5 minutes (ACGIH, 2014). The most common and widely used type of OEL is the TWA – 8-hour exposure limit. TWA is set based on an 8h- daily exposure in a working life of 40 years.

Setting OELs is part of the risk management decision-making process established by different countries (Schenk and Johnson, 2010). Despite different methods used in setting these OELs by countries, there is general understanding that for a hazardous substance to have an OEL it must be a health hazard to humans which largely paves the way for a limit to be set to control worker exposure to hazardous substances (Hansson and Ruden, 2006; Schenk and Johnson, 2011), in addition to socio-economic and technical feasibility aspects.

The key players influencing the development and setting of OELs are the USA with ACGIH which produces threshold limit values updated annually, NIOSH which recommends exposure limits (RELs), and OSHA which produce legally binding exposure limits referred to as

permissible exposure limits (PELs). Many countries make use of ACGIH TLVs. Other leading jurisdictions with considerable influence in OELs setting include the European Union (EU (Viljoen, 2012), with each country setting its individual OEL but the EU sets a maximum limit value for the EU (EU, 2009). The OEL in EU has evolved over the years until recently in 2022 when the chrysotile exposure limit has now been set at 0.01 f/ml (EC, 2022).

Table 1 below gives different exposure limits for chrysotile asbestos and how they have changed over time as more information on health aspects of exposure have been gathered.

Table 1: Occupational exposure limits (OEL), in f/ml, for various countries over the years

COUNTRY	YEAR						2022
	1969-1971	1972-1976	1983	1986	1994	1995-2018	
USA – OSHA	12	2	0.5	0.2	0.1	0.1	
USA – ACIGH	5 12	5	-	-	0.1	0.1	
USA – NIOSH*							
UK						0.1	
Germany						0.1	
EU			1.0	1.0	0.6	0.1	0.01**
South Africa						0.1	
*Zimbabwe					0.2	0.1	

Source: Schenk, 2011; NSSA, 2017 * Guidelines values.

**Amendment of EC Directive of 2009/148/EC

In Zimbabwe, there is no specific legislative instrument that governs the management and enforcement of any occupational exposure limits (OEL) for chrysotile asbestos exposure. Management is generally through non-specific regulations, in particular the Statutory Instrument 68 of 1990 on Accident Prevention and Workers Compensation (Government of Zimbabwe, 1990). Moreover, there is also no statutory OEL for chrysotile asbestos fibre save for guidelines on OELs published by the National Social Security Authority– Occupational Safety and Health Division which has set the limit at 0.1f/ml for all forms of asbestos fibre (National Social Security Authority, 2017). In the absence of specific guidance on the management of chrysotile asbestos exposure, the chrysotile asbestos cement manufacturing

industry developed its own occupational exposure monitoring programme for chrysotile asbestos where personal and static exposure data have been collected since the mid-1990.

2.5 Health Effects of Chrysotile Asbestos

All forms of asbestos including chrysotile have been associated with an increased risk of pneumoconiosis, lung cancer and mesothelioma in a number of epidemiological studies of exposed employees (IPCS, 1998; Landrigan et al., 1999; Nicholson, 2001; IARC, 2012).

Fig 1 and 2 below shows typical asbestosis and mesothelioma condition of lungs of exposed persons respectively.



Fig 2: Massive Fibrosis

Source: Lung samples preserved at NIOH NHLS, SA, 2022



Fig 3: Mesothelioma

Source: Lung samples preserved at NIOH NHLS, SA, 2022

With regards to chrysotile asbestos, the main concern in the past has been non-malignant lung disease such as asbestosis, which is a disease associated with diffuse interstitial pulmonary fibrosis which was accompanied by varying degrees of pleural involvement (IPCS, 1998;

IARC, 2012). Severe forms of asbestosis were generally associated with the high exposures, however with improved technology; severe asbestosis became less common with focus shifting towards syndromes exhibiting fibrosis of the small and large airways rather than of the large lung parenchyma (Dreesen et al., 1938; IPCS, 1998).

Several studies have also demonstrated changes in radiological and functional changes in workers exposed primarily to chrysotile fibre in mining and asbestos cement industries in which the presence of small opacities and pleural changes were observed with increased cumulative exposure (Rubino et al., 1979; McDemott et al., 1982; Viallat et al., 1982; Enarson et al., 1988; Jones et al., 1989; Cullen and Baloyi, 1991; IPCS, 1998). Additionally, within exposure concentrations of 0.3 and 1.1 f/ml, exposure to chrysotile asbestos has been shown to result in asbestosis (Berry et al., 1979; Huang et al., 1990; IPCS, 1998).

In a study of 1176 Swedish workers who used almost 100% chrysotile, 11 cases of lung cancer were observed compared to 9 expected and no deaths were attributed to mesothelioma. Overall, cumulative exposure was 18 f/ml-year among 10% of workers sampled (Ohlson & Hogstedt, 1985). Six cases of malignant mesothelioma were also identified in Norway from 1970 to 1979 in a chrysotile asbestos cement plant which also to some extent used very small amounts of crocidolite and amosite (Glyseth et al., 1983). In another cohort study of 3057 male workers who were followed from 1953 to 1982 in an asbestos cement plant in which 90% chrysotile and 10 % crocidolite was used in Northern Israel, it was observed that the cohort had an elevated risk of lung cancer, mesothelioma and other malignant neoplasm such as liver, bladder and renal cancers. High ratio of mesothelioma and lung cancer was attributed to high past asbestos exposures and low risk for lung cancer due to possibly early cessation in smoking (Tulchinsky et al., 1999). The incidence of cancer and mortality among workers in essentially chrysotile asbestos cement industry was also studied among 7996 men and 584 women in Denmark between 1928 and 1984 and over 99% were traced. The authors reported that during the initial 25 years exposure levels were generally high ranging from 100 to 1600 times the prevailing Danish exposure limit of 0.5 f/ml with a total of 1346 deaths and 612 cases of cancer observed in the country between 1943 and 1984 and that overall incidence of cancer (Observed/Expected 1.22; 95% CI 1.12-1.32) were significantly increased compared with all Danish men. Other cancers observed in this cohort included mesothelioma and laryngeal (Raffn et al., 1989).

Although it has been established that there is an association between asbestos including chrysotile exposure and lung cancer and mesothelioma, there is still some significant debate on

how risk might vary by exposure to different fibres types and sizes and whether there is risk at low levels (IARC, 2012; Baur and Frank, 2021). While the IARC has concluded that there is sufficient evidence of carcinogenicity in humans for all types of asbestos including chrysotile the question of whether chrysotile asbestos is less potent with respect to lung cancer and mesothelioma causation stems from the observations from studies that chrysotile is less biopersistent in the lungs than the amphiboles (Pierce et al., 2008; Bernstein et al., 2013; Bernstein, 2014; Gilham et., 2015). Pathological case studies of lung tissues of Canadian chrysotile asbestos workers have shown high proportion of amphibole mainly tremolite presenting increased risk of lung cancer and mesothelioma (Liddell et al., 1997; IARC, 2012. WHO, 2014). In a study of 8 Chinese asbestos factories, the mortality from lung cancer was elevated (Zhu et al., 1993). Meta-analysis conducted using 15 cohort studies with quantitative information on the relationship between asbestos exposure and lung cancer mortality to evaluate the relative potency of different fibre types and other fibre characteristics showed that there was no difference in the potency to cause lung cancer between the different types of fibre (Lash et al., 1997; WHO, 2014). Other studies have shown the link between asbestos exposure and lung cancer and mesothelioma, though demonstrating potency difference between fibres types (Hodgson and Darnton, 2000; Berman and Crump, 2008). In South Africa, of the 123 mesothelioma cases identified, 23 were linked to Cape crocidolite, 3 with amosite mine and 3 with both crocidolite and amosite asbestos exposure. However, no cancer was linked to chrysotile asbestos (Rees et al., 1999). Cases of lung cancer and mesothelioma have also been reported among workers in the asbestos mines and mills in Zimbabwe (Cullen and Baloyi, 1991). These studies and many other demonstrate the importance of the role of asbestos including chrysotile in causing malignant

2.6 Trends in Occupational exposure to chrysotile asbestos fibre over the years

A limited number of papers have reported temporal trends in exposure with respect to chrysotile asbestos cement manufacturing plants. In Germany, it was observed that there has been continual decrease in asbestos dust concentration for the period 1950 to 1990 which was linked or attributed to the rapid decline in the use of asbestos since 1980 when more rigorous regulations and bans on the production, use and placement of asbestos on the market was introduced (Hagemeyer et al., 2006); Creely et al., 2007). Furthermore, Coble et al., (2001) reported that there was a 5% decline in asbestos exposures observed during compliance inspections of pulp and paper facilities. In another study of exposure – response relationships for asbestos related diseases, Finkelstein, (1983) reported declines in exposure for the years

1949, 1969 and 1979, where exposure estimates were recorded as 40, 20, 0.2 f/ml for willow operators, 16, 8, 0.5 f/ml for forming machine operators and 8, 4, 0.3 f/ml for lathe operators for the respective years of 1949, 1969 and 1979 respectively. Additionally, asbestos exposure declines were reported in asbestos cement plants in Sweden (Albin et al., 1990), South Africa (Weiner et al., 1994), Japan (Higashi et al., 1994), Yugoslavia, Poland and Latvia (Albin et al., 1999) and USA (Williams et al., 2007a). These studies suggest that exposure during the earlier years were high particularly during the 1970s compared to the period 1990 to 2000s.

2.7 Job exposure matrices

Job exposure matrices (JEMs) have been used as tools for assessing past exposure levels to various hazardous factors. Historical exposure to workplace hazards and indeed chrysotile asbestos is a key factor in the onset of ARDs because chrysotile asbestos has been used in manufacturing asbestos cement products in construction works since the 1920s to 1930s.

The principle of JEMs is based on the construction of a database that associate exposures to various hazardous factors with occupations/jobs or workstations (Pannet et al., 1985; Goldberg and Imbernon, 2002; Kang et al., 2021). Thus, a JEM is a tool through which information on jobs collected in epidemiological studies may be converted into information on possible exposures (Kauppinen et al., 1998). Essentially, the key objective of a JEM is to try to link job/occupation information with workplace hazardous exposure information. The idea of a JEM dates back to the time when Ramazzini tried to link diseases in 52 occupations to which the occupations were exposed to the respective hazards. In 1941, the first JEM to be developed consisted of a cross tabulation of occupations list with that of a list of hazards (Carnevale et al., eds. 2009). Hence, the concept of JEM is that it is essentially a table in which one axis comprise of occupations/jobs while the other axis comprises of workplace hazards. For a given job/occupation, each cell of the matrix can contain qualitative or quantitative exposure indicators (Goldberg et al., 1993). The JEMs may be constituted by four axes namely job/occupation, agent of exposure, time or time-period and place/location (Goldberg et al., 1993; Kauppinen et al., 1998). Exposure can vary with respect to occupations/jobs and workplaces and hence jobs can be categorised into homogenous groups to reflect similar exposures. Hence, workers exposed to a particular agent under similar or same conditions should correspond to the same entry of the matrix (Goldberg et al., 1993). Furthermore, JEMs for application in retrospective studies should consider changes in exposures over time to aid in assigning health outcomes at a point in time in future. In this respect, a time variable must

be introduced when exposure has changed over time (Goldberg et al., 1993; Kauppinen et al., 1998). It is also important to include the place/location variable in JEMs since exposure may vary across different plants or factory situated in different locations (Kauppinen et al., 1998).

Information sources for which exposure estimates for a JEM can be obtained include actual measurements collected over time in workplace plants or factories of interest, by company occupational hygienists, scientific literature and exposure data banks (Goldberg et al., 1993; Nills and Steineck, 1993). It is insightful to note that in this study, data collected spanning about two and half decades provided a good resource to obtain exposure estimates upon which the JEM was built.

2.8 Prediction of ARDs

Prediction of ARDs can be done through models that use direct or indirect estimates of asbestos including chrysotile exposure (Reid, 2016). Direct estimates make use of exposed persons where airborne asbestos fibre levels were measured over time while indirect estimates use information about total or fibre specific imports (Reid, 2016).

Exposure to asbestos has been reported to generally follow a linear in dose model wherein relative risk of asbestos disease was linear in dose (OSHA, 1986; Stayner et al., 1997; Berman & Crump, 2004). The US Occupational Safety and Health Administration (OSHA) selected a linear in dose model to describe the relationship between the excess relative risk of lung cancer and asbestos exposure (OSHA, 1986). Evidence of the linear in dose-response relationship for lung cancer has been observed for several studies with respect to cumulative asbestos exposure in the workplace (Liddell et al., 1977; Henderson and Enterline, 1979; Dement et al., 1982; OSHA, 1986; Selikoff, 1991; MSHA, 2005, 2008). In a study by Finkelstein, it was observed that the rates of death from mesotheliomas were proportional to the magnitude of cumulative asbestos exposure and that the exposure response data set was approximately linear in nature (Finkelstein, 1983). Furthermore, Berry et al., and Finkelstein have demonstrated an approximate linear relationship between asbestosis incidences and cumulative exposure (Berry et al., 1979; Finkelstein, 1982). These studies and others upon which OSHA based its risk assessment models to predict asbestos related cancers demonstrated linear relationships over a range of observations. In the present study, the direct method was applied to predict chrysotile asbestos related mortality in two AC manufacturing factories in Zimbabwe as airborne chrysotile asbestos fibre concentrations over the period of 1996 to 2020 were available.

REFERENCES

Addison J & Davies LS (1990). Analysis of amphibole asbestos in chrysotile and other minerals. *The Annals of Occupational Hygiene*, 34(2), 159–175. <https://doi.org/10.1093/annhyg/34.2.159>.

Airoldi C, Magnani C and Lazzarato F et al., (2021). Environmental asbestos exposure and clustering of malignant mesothelioma in community: a spatial analysis in a population-based case–control study. *Environ Health*, **20**, 103 <https://doi.org/10.1186/s12940-021-00790-3>.

Albin M, Jakobsson K, Attewell R, Johansson L & Welinder H (1990). Mortality and cancer morbidity in cohorts of asbestos cement workers and referents. *British Journal of Industrial Medicine*, 47(9), 602–610. <https://doi.org/10.1136/oem.47.9.602>.

Albin M, Magnani C, Krstev S, Rapiti E & Shefer I (1999). Asbestos and cancer: An overview of current trends in Europe. *Environmental Health Perspectives*, *107 Suppl 2* (Suppl 2), 289–298. <https://doi.org/10.1289/ehp.99107s2289>.

American Conference of Governmental Industrial Hygienist (ACGIH) (2014). Threshold limit values for chemical substances, physical agents, and biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

Badollet (1948). Research on asbestos fibre. *Can Min Metal Trans*. 51, 48-51.

Baur X. & Frank AL (2021). Ongoing downplaying of the carcinogenicity of chrysotile asbestos by vested interests. *Journal of Occupational Medicine and Toxicology* (London, England), 16(1), 6. <https://doi.org/10.1186/s12995-021-00295-2>.

Berman DW & Crump KS (2008). Update of potency factors for asbestos-related lung cancer and mesothelioma. *Critical Reviews in Toxicology*, 38 Suppl 1, 1–47. <https://doi.org/10.1080/10408440802276167>.

Bernstein DM (2014). The health risk of chrysotile asbestos. *Current Opinion in Pulmonary medicine*, 20(4), 366–370. <https://doi.org/10.1097/MCP.0000000000000064>.

Bernstein D, Dunnigan J, Hesterberg T, Brown R, Velasco JA, Barrera R, Hoskins J & Gibbs A (2013). Health risk of chrysotile revisited. *Critical Reviews in Toxicology*, 43(2), 154–183. <https://doi.org/10.3109/10408444.2012.756454>.

Bernstein DM & Hoskins JA (2006). The health effects of chrysotile: current perspective based upon recent data. *Regulatory Toxicology and Pharmacology: RTP*, 45(3), 252–264. <https://doi.org/10.1016/j.yrtph.2006.04.008>.

Bernstein DM, Toth B, Rogers RA, Kling DE, Kunzendorf P, Phillips JI & Ernst H (2020). Evaluation of the dose-response and fate in the lung and pleura of chrysotile-containing brake dust compared to TiO₂, chrysotile, crocidolite or amosite asbestos in a 90-day quantitative inhalation toxicology study - Interim results Part 2: Histopathological examination, Confocal microscopy and collagen quantification of the lung and pleural cavity. *Toxicology and Applied Pharmacology*, 387, 114847. <https://doi.org/10.1016/j.taap.2019.114847>.

Bowes DR, Langer AM and Rohl AN (1977). Nature and range of mineral dusts in the environment *Philosophical Transactions of the Royal Society of London. Series A, Mathematical and Physical Sciences* 286593–610. <http://doi.org/10.1098/rsta.1977.0134>.

Camus M., Siemiatycki J & Meek, B (1998). Nonoccupational exposure to chrysotile asbestos and the risk of lung cancer. *The New England Journal of Medicine*, 338(22), 1565–1571. <https://doi.org/10.1056/NEJM199805283382201>.

Creely KS, Cowie H, Van Tongeren M, Kromhout H, Tickner J & Cherrie JW (2007). Trends in inhalation exposure--a review of the data in the published scientific literature. *The Annals of Occupational Hygiene*, 51(8), 665–678. <https://doi.org/10.1093/annhyg/mem050>.

Coble JB, Lees PS & Matanoski G (2001). Time trends in exposure measurements from OSHA compliance inspections of the pulp and paper industry. *Applied Occupational and Environmental Hygiene*, 16(2), 263–270. <https://doi.org/10.1080/10473220120763>.

Cullen MR & Baloyi RS (1991). Chrysotile asbestos and health in Zimbabwe: I. Analysis of miners and millers compensated for asbestos-related diseases since independence (1980). *American Journal of Industrial Medicine*, 19(2), 161–169. <https://doi.org/10.1002/ajim.4700190204>.

Dressen WC, Dalavalle JW, Edwards TI, Miller JW and Sayers RR (1938). A study of asbestos in the asbestos textile industry. Washington, DC, US Public Health Service (Public Health Bulletin No. 241.

Gilham C, Rake C, Burdett G, Nicholson AG, Davison L, Franchini A, Carpenter J, Hodgson, J, Darnton A, & Peto J (2016). Pleural mesothelioma and lung cancer risks in relation to occupational history and asbestos lung burden. *Occupational and Environmental Medicine*, 73(5), 290–299. <https://doi.org/10.1136/oemed-2015-103074>.

Health Council of the Netherlands - Dutch Expert Committee on Occupational Safety (HCN-DECOS) (2010). Asbestos: Risks of environmental and occupational exposure, The Hague: Health Council of the Netherlands; publication no. 2010/10E.

Enarson DA., Embree V, MacLean L & Grzybowski S (1988). Respiratory health in chrysotile asbestos miners in British Columbia: a longitudinal study. *British Journal of Industrial Medicine*, 45(7), 459–463. <https://doi.org/10.1136/oem.45.7.459>.

European Chemicals Agency (ECHA) (2021). ECHA Scientific report for evaluation of limit values for asbestos at the workplace. Helsinki, Finland, ECHA. Available online at [4605fc92-18a2-ae48-f977-4dffdecfec11 \(europa.eu\)](https://doi.org/10.28388/4605fc92-18a2-ae48-f977-4dffdecfec11). Accessed on 18 July 2023.

European Commission (EC) (2022). Directive 2009/148/EC - exposure to asbestos at work. Brussels, EC. Available online at <https://osha.europa.eu/en/legislation/directives/2009-148-ec-exposure-to-asbestos-at-work>. Accessed on 18 July 2023.

Eypert-Blaison C, Romero-Hariot A, Clerc F & Vincent R (2018). Assessment of occupational exposure to asbestos fibres: Contribution of analytical transmission electron microscopy analysis and comparison with phase-contrast microscopy. *Journal of occupational and environmental hygiene*, 15(3), 263–274. <https://doi.org/10.1080/15459624.2017.1412583>.

Felletto E, Schonfeld SJ, Kovalevskiy EV, Bukhtiyarov IV, Kashanskiy SV, Moissonnier M, Straif K, Schüz J & Kromhout H (2017). A comparison of parallel dust and fibre measurements of airborne chrysotile asbestos in a large mine and processing factories in the Russian Federation. *International Journal of Hygiene and Environmental Health*, 220(5), 857–868. <https://doi.org/10.1016/j.ijheh.2017.04.001>,

Finkelstein MM (1983). Mortality among long-term employees of an Ontario asbestos-cement factory. *British Journal of Industrial Medicine*, 40(2), 138–144. <https://doi.org/10.1136/oem.40.2.138>.

Gualtieri AF, Lusvardi G, Zoboli A, Di Giuseppe D & Gualtieri LM (2019). Biodurability and release of metals during the dissolution of chrysotile, crocidolite and fibrous erionite. *Environmental Research*, 171, 550–557. <https://doi.org/10.1016/j.envres.2019.01.011>.

Gylseth B, Mowé G & Wannag A (1983). Fibre type and concentration in the lungs of workers in an asbestos cement factory. *British journal of industrial medicine*, 40(4), 375–379. <https://doi.org/10.1136/oem.40.4.375>.

Goldberg M & Luce D (2009). The health impact of nonoccupational exposure to asbestos: what do we know? *European journal of cancer prevention: the official journal of the European Cancer Prevention Organisation (ECP)*, 18(6), 489–503. <https://doi.org/10.1097/CEJ.0b013e32832f9bee>.

Govt of Zimbabwe (1990). Statutory Instrument 68 of 1990: Accident Prevention and Workers Compensation Scheme. 3rd Schedule.

Hagemeyer O, Otten H & Kraus T (2006). Asbestos consumption, asbestos exposure and asbestos-related occupational diseases in Germany. *International Archives of Occupational and Environmental Health*, 79(8), 613–620. <https://doi.org/10.1007/s00420-006-0091-x>.

Higashi T, Hori H, Sakurai H, Omae K, Tsuda T, Tanaka I, Satoh T & Hoshi H (1994). Work environment of plants manufacturing asbestos-containing products in Japan. *The Annals of Occupational Hygiene*, 38(4), 489–409. <https://doi.org/10.1093/annhyg/38.4.489>.

Hodgson JT & Darnton A (2000). The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. *The Annals of Occupational Hygiene*, 44(8), 565–601.

International Agency for Research on Cancer (IARC) (2012). IARC Monograph on Evaluation of Carcinogenic Risks to Human, Arsenic, Metals, Fibres and Dust: A Review of Human Carcinogens Vol. 100C p.219 – 309. IARC/WHO.

International Programme on Chemical Safety (IPCS) (1998). Chrysotile Asbestos. Environmental Health Criteria 203: Geneva, UNEP, ILO, WHO and IPCS.

Jaurand MC, Bignon J, Sebastien P & Goni J (1977). Leaching of chrysotile asbestos in human lungs. Correlation with in vitro studies using rabbit alveolar macrophages. Environmental Research, 14(2), 245–254. [https://doi.org/10.1016/0013-9351\(77\)90036-6](https://doi.org/10.1016/0013-9351(77)90036-6).

Kauppinen T & Korhonen K (1987). Exposure to asbestos during brake maintenance of automotive vehicles by different methods. American Industrial Hygiene Association journal, 48(5), 499–504. <https://doi.org/10.1080/15298668791385101>.

Kimura K (1987). Asbestos and environment. Dig Science Laboratory. 42, 4-13.

Landrigan PJ, Nicholson WJ, Suzuki Y & LaDou J (1999). The hazards of chrysotile asbestos: a critical review. Industrial Health, 37(3), 271–280. <https://doi.org/10.2486/indhealth.37.271>.

Langer AM & Nolan RP (1994). Chrysotile: its occurrence and properties as variables controlling biological effects. The Annals of Occupational Hygiene, 38(4), 427–451. <https://doi.org/10.1093/annhyg/38.4.427>.

Lash TL, Crouch EA & Green LC (1997). A meta-analysis of the relation between cumulative exposure to asbestos and relative risk of lung cancer. Occupational and Environmental Medicine, 54(4), 254–263. <https://doi.org/10.1136/oem.54.4.254>.

Meintjes S, Hermanus M (2008). "Unfit for human habitation: The Penge report". In: Phiroshaw C, editor. National Asbestos Conference Report, ISBN: 978-0-620-46691-2 ed. Johannesburg: KRT and ART. p 55-59.

National Social Security Authority (2017). Guidelines on Occupational Exposure Limits (OELs) for Chemical Substances and Dust. NSSA, Harare.

Nicholson WJ (2001). The carcinogenicity of chrysotile asbestos--a review. Industrial Health, 39(2), 57–64. <https://doi.org/10.2486/indhealth.39.57>.

Nicholson WJ and Pundsack FL (1973). Asbestos in the Environment. In: Biological effects of asbestos. Lyon, IARC, pp 126-130.

Ohlson CG & Hogstedt C (1985). Lung cancer among asbestos cement workers. A Swedish cohort study and a review. *British Journal of Industrial Medicine*, 42(6), 397–402. <https://doi.org/10.1136/oem.42.6.397>.

Oze C and Solt K (2010). Bio-durability of chrysotile and tremolite asbestos in simulated lung and gastric fluids. *Am Mineral*, 95, 825-831. Available online at http://www.minsocam.org/msa/ammin/toc/Abstracts/2010_Abstracts/MJ10_Abstracts/Oze_p825_10.pdf. (Accessed 10 August 2021).

Paustenbach DJ, Finley BL, Lu ET, Brorby G & Sheehan, PJ (2004). Environmental and Occupational Health Hazards Associated With the Presence of Asbestos in Brake Linings and Pads (1900 to Present): A “State-of-the-Art” Review, *Journal of Toxicology and Environmental Health, Part B*, 7:1, 25-80, DOI: [10.1080/10937400490231494](https://doi.org/10.1080/10937400490231494).

Pierce JS, McKinley MA, Paustenbach DJ & Finley BL (2008). An evaluation of reported no-effect chrysotile asbestos exposures for lung cancer and mesothelioma. *Critical Reviews in Toxicology*, 38(3), 191–214. <https://doi.org/10.1080/10408440701845609>.

Phillips JI, Renton K, Murray J, Garton E, Tylee BE, Rees D (2007). Asbestos in and around Soweto dwellings with asbestos roofs. *Occupational Health Southern Africa*, 13:3 – 7.

Phillips JI, Rees D, Murray J & Davies JC (2012). Mineralogy and Malignant Mesothelioma: The South African Experience. In C. Belli, & S. Anand (Eds.), *Malignant Mesothelioma*. IntechOpen. <https://doi.org/10.5772/47974>.

Price B, Crump KS & Baird EC., 3rd (1992). Airborne asbestos levels in buildings: maintenance worker and occupant exposures. *Journal of exposure analysis and environmental epidemiology*, 2(3), 357–374.

Pollastri S, Perchiazzi N, Lezzerini M et al. (2016). The crystal structure of mineral fibres I. Chrysotile. *Periodico di Mineralogia*. 85, 249-259. Available online at

https://www.researchgate.net/publication/311425221_The_crystal_structure_of_mineral_fibres_1_Chrysotile. (Accessed 3 January 2021).

Raffn E, Lyng E, Juel K et al. (1989). Incidence of cancer and mortality among employees in the asbestos cement industry in Denmark. *Br J Ind Med*. 46, 90-96. Available online; <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1009733/pdf/brjindmed00130-0018.pdf>. Accessed 20 Jan 2021.

Rees D, Goodman K, Fourie E, Chapman R, Blignaut C, Bachmann MO & Myers J. (1999). Asbestos exposure and mesothelioma in South Africa. *South African Medical Journal = Suid-Afrikaanse tydskrif vir geneeskunde*, 89(6), 627–634.

Schenk L, Hansson SO, Rudén C & Gilek M (2008). Occupational exposure limits a comparative study. *Regulatory Toxicology and Pharmacology: RTP*, 50(2), 261–270. <https://doi.org/10.1016/j.yrtph.2007.12.004>.

Schenk L (2011). Setting occupational exposure limits Practices and outcomes of toxicological risk assessment. Thesis. Royal Institute of Technology (KTH), SE-100 44 Stockholm, Sweden. Royal Institute of Technology (KTH), SE-100 44 Stockholm, Sweden. Available online <http://www.diva-portal.org/smash/get/diva2:397699/SUMMARY01>. (Accessed 19 January 2019).

Schonfeld SJ, Kovalevskiy EV, Feletto E, Bukhtiyarov IV, Kashanskiy SV, Moissonier M, Straif K, McCormack VA, Schüz J, Kromhout H. Temporal Trends in Airborne Dust Concentrations at a Large Chrysotile Mine and its Asbestos-enrichment Factories in the Russian Federation During 1951-2001 (2017). *Ann Work Expo Health* 1, 61(7), 797-808. doi: 10.1093/annweh/wxx051. PMID: 28810689; PMCID: PMC6005011.

Stayner L, Smith R, Bailer J, Gilbert S, Steenland K, Dement J, Brown D & Lemen R (1997). Exposure-response analysis of risk of respiratory disease associated with occupational exposure to chrysotile asbestos. *Occupational and environmental medicine*, 54(9), 646–652. <https://doi.org/10.1136/oem.54.9.646>.

Tulchinsky TH, Ginsberg GM, Iscovich J, Shihab S, Fischbein A & Richter ED (1999). Cancer in ex-asbestos cement workers in Israel, 1953-1992. *American Journal of Industrial Medicine*, 35(1), 1–8. [https://doi.org/10.1002/\(sici\)1097-0274\(199901\)35:1<1::aid-ajim1>3.0.co;2-5](https://doi.org/10.1002/(sici)1097-0274(199901)35:1<1::aid-ajim1>3.0.co;2-5). (Accessed 13 November 2020).

Virta R (2006). *Worldwide Asbestos Supply and Consumption Trends from 1900 through 2003*, Reston, VA, U.S. Department of the Interior, U.S. Geological Survey. Circular 1298.

Wang X., Lin S, Yano E, Qiu H, Yu IT, Tse L, Lan Y & Wang M (2012). Mortality in a Chinese chrysotile miner cohort. *International Archives of Occupational and Environmental Health*, 85(4), 405–412. <https://doi.org/10.1007/s00420-011-0685-9>.

Williams PR, Phelka AD & Paustenbach DJ (2007). A review of historical exposures to asbestos among skilled craftsmen (1940-2006). *Journal of toxicology and environmental health. Part B, Critical reviews*, 10(5), 319–377. <https://doi.org/10.1080/10937400601034191>.

Webster I (1973). Asbestos and malignancy. *S Afr Med J* 47:165-171. Available online at https://journals.co.za/doi/pdf/10.10520/AJA20785135_30216. (Accessed 26 July 2022).

Weiner R, Rees D, Lunga FJ & Felix MA (1994). Third wave of asbestos-related disease from secondary use of asbestos. A case report from industry. *South African medical journal = Suid-Afrikaanse tydskrif vir geneeskunde*, 84(3), 158–160.

White N, Nelson G & Murray J (2008). South African experience with asbestos related environmental mesothelioma: is asbestos fiber type important? *Regulatory Toxicology and Pharmacology: RTP*, 52(1Suppl), S92–S96. <https://doi.org/10.1016/j.yrtph.2007.09.013>. (Accessed 20 February 2021).

William P, Paustenbach D, Balzer JL & Mangold C (2007). Retrospective exposure assessment of airborne asbestos related to skilled craftsmen at a petroleum refinery in Beaumont, Texas (1940-2006). *Journal of toxicology and environmental health. Part A*, 70(13), 1076–1107. <https://doi.org/10.1080/15287390701208305>. Accessed 15 April 2021.

World Health Organization (WHO) (2014). Chrysotile Asbestos. Available online at; http://www.who.int/ipcs/assessment/public_health/chemicals_phc. Accessed 9/2/2020.

Yarborough CM (2006). Chrysotile as a cause of mesothelioma: an assessment based on epidemiology. *Critical Reviews in Toxicology*, 36(2), 165–187.
<https://doi.org/10.1080/10408440500534248>.

Zhu H & Wang Z (1993). Study of occupational lung cancer in asbestos factories in China. *British Journal of Industrial Medicine*, 50(11), 1039–1042.
<https://doi.org/10.1136/oem.50.11.1039>.

CHAPTER 3

Methodology

This chapter describes settings where the study was done, methods used for measuring chrysotile asbestos fibre, approaches used in trend analysis, development of job exposure matrix and prediction of asbestos related diseases (ARDs). Quality assurance aspects are also given as well as ethical approval of the study. The methods and materials are described in detail in the four manuscripts in chapters 6-9.

3.1. Study locations

The study was carried out in two major chrysotile-manufacturing factories situated in Harare and Bulawayo. Harare is the capital city of Zimbabwe, in the northern part of the country, 387 km from Zvishavane town, while Bulawayo is the second largest city and is situated in the southern part of the country, 184 km from the Zvishavane town where chrysotile asbestos mines Shabanie and Mashava are located. Activities in the two factories include manufacture of chrysotile cement sheets, pipes and fascia boards.

3.2. Study design

Secondary data was used to evaluate extent of exposure to chrysotile asbestos fibre in the chrysotile cement factories, build a job exposure matrix and predict ARDs. A comprehensive retrospective review of chrysotile fibre levels (data set for the period 1996 to 2020) in various work areas of manufacturing factories for chrysotile products was done in order to link such exposure levels to possible health outcomes. Primary data was also generated with respect to assessment of possible amphiboles contaminants in chrysotile asbestos fibre being used in the manufacturing of AC products in the factories.

3.3. Collection of measurements

Operational areas for which personal exposure data were available were saw cutting, fettling table, kollergang, moulded goods, ground hard waste, laundry room, lathe machining of pipes, and multi-cutter operations (Table 1 in Chapter 6). Generally, exposure data were collected once every month, though in some years measurements depended on the availability of plant operating, sampling equipment and consumables. Description of tasks used in characterising personal chrysotile fibre concentrations in various operational areas are given in chapter 6.

3.4. Method of chrysotile asbestos fibre measurements

The method on file in the factories showed that measurements of airborne asbestos fibre concentrations followed the Asbestos International Association (AIA) Recommended Technical Membrane Filter Reference Method (AIA, 1982). As part of following AIA technical reference method, field blank filter samples were reported used as controls as part of the quality control programme. In summary, a personal sampling pump set at 1 L/min flowrate was connected to a sampling train, consisting of plastic tubing and a sample holder (cowl) with a 25 mm membrane filter. The whole sampling train of the pump, tubing, sample holder and filter was hooked to a worker. The pump was then switched on and sampling took place over a period of about four hours, after which the filters were removed, placed at the appropriate labelled slides and treated with acetone vapour to make the filters optically transparent. Using a hypodermic syringe, a drop of triacetin was placed onto the acetone-cleared filters and covered with a cover slip. The treated filters on the slides were stored for 24 hours, after which counting of the fibres took place using a phase contrast microscope. The limit of detection (LoD) for the method was 0.02 f/ml.

3.5 Data description and classification of measurements

Approximately 3 000 personal exposure measurements were collected in the operational areas over the 25-year period 1996 to 2020, in the two factories. Personal sampling data points were classified into six production areas for both Harare and Bulawayo factories; a further classification was made for the pipe section of the Bulawayo factory. For the pipe section, personal sampling data were classified into three broad areas, namely (a) pipe plant operations – lathe machining asbestos pipe joints, (b) pipe plant – lathe machining of full-length asbestos sewer and water pipes and (c) multi-cutter machine where cutting of full-length pipes into collars for coupling of pipes was done. For the saw cutting operations, measurements were done over 4 hours period for 4 to 6 times per month. Personal sampling data for each broad operational area was averaged for each month. The tasks indicated above were considered to have potential for highest exposure to airborne chrysotile asbestos fibre.

The period of 1996 to 2016 was divided into three time periods: 1996–2000, 2001–2008, 2009–2016 and another time period added of 2017 -2020 as more personal exposure data became available. During 1996 – 2000, the chrysotile asbestos cement manufacturing industry was in a self-regulatory mode with respect to safety and health standards and monitoring of exposure

considering the call to phase out the use of chrysotile asbestos and industry setting its own exposure limits. During these early years of the 1990s, the asbestos industry set its own chrysotile exposure limit of 0.2 f/ml and an action limit of 0.15 f/ml in the absence of a national statutory exposure limit on chrysotile asbestos. From 2001 to 2008, the chrysotile exposure monitoring program continued, however there was a sharp decline in economic activity nationally. Monitoring of exposure continued for the period 2009 to 2016 against the backdrop of improved retooling of the industry and change from the use of locally produced asbestos to largely imported fibre.

3.6 Materials and methods for various aspects of the study

3.6.1 Trends in Airborne Chrysotile asbestos fibre concentrations in the asbestos cement manufacturing (ACM) industry in Zimbabwe

For analysis, monthly averaged personal exposure levels for the factories were used. Mean personal airborne chrysotile fibre concentrations were analysed per operational area, per time period, per factory and trends over the years were displayed graphically using the Excel chart functionally. The arithmetic mean was used as a representative value for analysis of the measurements as this was considered as the best summary measure of exposure in epidemiological studies of chronic diseases when adopting a linear exposure response model (Seixas et al., 1988; Seixas et al., 1988; Choi et al., 2017).

3.6.2 Job Exposure Matrix for chrysotile asbestos fibre in the asbestos cement manufacturing (ACM) industry in Zimbabwe.

Personal exposure data measured for the period 1996 to 2020 extracted from paper records of the two manufacturing factories in Harare and Bulawayo cities were used to build the JEM. The data comprised of all personal exposure measurements collected by the company for close to 25 years in various operational areas examined in the two AC manufacturing factories. The data collected was examined to assess the chrysotile asbestos exposure for each combination of job, time period, place and mean personal exposure levels and possible amphibole contamination in the chrysotile asbestos fibre being used in the manufacturing processes. The industry is the chrysotile asbestos cement manufacturing. The jobs were classified into 9 broad categories assuming similar or homogenous exposure, namely saws cutting, fettling, moulded goods, kollergang, ground hard waste, laundry, pipe joints and multi-cutter operators. Measurement of

airborne chrysotile asbestos followed the standard method of the Asbestos International Association (AIA) Reference method for the determination of airborne asbestos fibre concentrations at workplaces (AIA, 1982).

Detection of amphiboles in the chrysotile asbestos used for manufacturing AC products in the Bulawayo factory was done by the National Institute of Occupational Health (NIOH) – National Health Laboratory Service (NHLS), South Africa, using Scanning Electron Microscopy (SEM) and Energy Dispersive Spectroscopy (EDS).

3.6.3 Prediction of Asbestos Related Diseases (ARDs) and chrysotile asbestos exposure concentrations in asbestos-cement (AC) manufacturing factories in Zimbabwe.

Prediction of ARDs was done by combining the job-exposure matrix for airborne chrysotile asbestos fibre concentrations adjusted to cumulative exposures and OSHA's linear dose effect model. In this model ARDs were estimated through linear regression equations established for lung cancer, mesothelioma and gastrointestinal cancer, while for asbestosis the linear cumulative dose equation, $R_a = m(f)(d)$, where R_a – predicted incidence of asbestosis, m – slope of linear regression taken as 0.055, f – asbestos fibre concentration and d – duration of exposure (MSHA, 2005; 2008). ARDs were predicted at exposure duration of 1, 10, 20 and 25 years in 2020, assuming an entry to work dated back from 2020.

Hence, the OSHA's risk assessment on asbestos related diseases used Tables 1 in (Chapter 8) to establish linear regression equations that were applied to derive possible asbestos related cancers at various cumulative exposures and duration of exposures of 1, 10, 20 and 25 years. Additionally, Table 2 (Chapter 8) adapted from the study by Mutetwa et al., (2022) was also used to predict ARDs by drawing exposure concentrations from various time periods for various jobs in the two AC manufacturing factories to calculate cumulative exposures.

3.6.4 Examining approaches for prevention of exposure to chrysotile asbestos and some perspectives on the debate on ban of asbestos.

Literature materials that advocate for the complete ban of all forms of asbestos including chrysotile as the only means of control of exposure and that which argues for the controlled use approach were reviewed. Search words used in literature search were chrysotile asbestos

exposure, chrysotile, asbestos-cement, ban asbestos, controlled use, asbestos related disease, mesothelioma, lung cancer and asbestosis.

3.7 Data Analysis

Summary descriptive statistics and statistical testing for chrysotile asbestos fibre exposure concentrations were done using IBM SPSS version 26. Trends in exposure over time were analysed graphically using the Excel chart functionality. ANOVA was applied with the aim of identifying patterns of exposure variability among the time-period for various job categories and also determine whether there was a statistically significant difference in exposure concentration between time-periods for the various jobs. A Tukey Post Hoc Test (Tukey's Honest Significance Difference test) was run to find out which specific group means of time-periods for various jobs/occupations (compared with each other) were different.

3.8. Quality assurance and reliability of the chrysotile asbestos fibre exposure data.

From the early 1990s to 2011, correspondences at the two factories showed that the factories participated in an inter-laboratory quality assurance and control fibre counting programme, which involved a laboratory at two chrysotile mines in Zimbabwe, another chrysotile asbestos cement plant laboratory in Zambia, the Department of Minerals and Energy in South Africa, and a French laboratory in Paris, with a view to improve the quality and reliability of exposure measurements. Additionally, as part of an oversight programme on quality control, in 2008, the Institute of Occupational Medicine (IOM), UK, was invited to conduct an independent evaluation of levels of chrysotile asbestos fibre in the ambient air of various work processes (Jones and Clark, 2008). The independent evaluation of levels of chrysotile asbestos fibre in the two factories provided a good measure of reliability and assurance to the exposure values generated by the company over the years, and subsequently used in this study. The IOM reported that personal and static samplers were being correctly mounted on the workers with proper positioning of sample holders in the workers breathing zones. They further reported that the company analytical laboratory was adequately equipped for collection and measurement of airborne chrysotile asbestos fibres and that there was good consistency between the IOM and the company's calibration equipment for calibration flows of the sampling pumps (Jones and Clark, 2008). These efforts demonstrated that the data used in this study provided a measure of reliability of exposure values obtaining in the factories over the years.

3.9. Ethics

The University of the Witwatersrand Human Research Ethics Committee (clearance certificate number M181157) and the Medical Research Council of Zimbabwe (MRCZ) (approval number MRCZ/A/2445) approved the study.

REFERENCES

Asbestos International Association (AIA) (1982). Reference Method for the Determination of Airborne Asbestos Fibre Concentrations at Workplaces by Light Microscopy (Membrane Filter Method).

Choi S, Kang D, Park D et al. (2017). Developing Asbestos Job Exposure Matrix Using Occupation and Industry Specific Exposure Data (1984-2008) in Republic of Korea. *Safety and Health at Work*, 8(1), 105–115. <https://doi.org/10.1016/j.shaw.2016.09.002>. (Accessed 20 June 2021).

Jones AD and Clark R (2008). Evaluation of asbestos dust measurement and control at a Factory in Harare, Zimbabwe. Strategic Consulting Report: 629-00224. Institute of Occupational Medicine (IOM), UK.

Seixas NS, Robins TG & Moulton LH (1988). The use of geometric and arithmetic mean exposures in occupational epidemiology. *American Journal of Industrial Medicine*, 14(4), 465–477. <https://doi.org/10.1002/ajim.4700140410>. Accessed 15 April 2022.

Occupational Safety and Health Administration, US Department of Labor (1986). 51 FR 22 612 - Occupational exposure to asbestos, tremolite, anthophyllite and actinolite. Final Rule. Available online at: <https://www.grsm.com/Templates/media/files/pdf/51%20fr%2022612.pdf>. (Accessed on 20 February 2022).

Mine Safety and Health Administration (MSHA) USA Department of Labour (2008). Asbestos Exposure Limit; Final Rule. Department of Labor, Mine Safety and Health Administration. Available online at: <https://www.govinfo.gov/content/pkg/FR-2008-02-29/pdf/E8-3828.pdf#page=22>. (Accessed 18 February 2022).

Mine Safety and Health Administration (MSHA) USA Department of Labour (2005). Asbestos Exposure Limit; Final Rule. Department of Labor, Mine Safety and Health Administration. Available online at:

<https://www.govinfo.gov/content/pkg/FR-2005-07-29/pdf/05-14510.pdf>. (Accessed 18 February 2022)

Mutetwa B, Moyo D & Brouwer D (2022). Job Exposure Matrix for Chrysotile Asbestos Fibre in the Asbestos Cement Manufacturing (ACM) Industry in Zimbabwe. *International journal of environmental research and public health*, 19(5), 2680. <https://doi.org/10.3390/ijerph19052680>.

CHAPTER 4

Results

Detailed results are reported in chapters 6 to 9 where full manuscripts for various aspects of the thesis are provided. In this chapter a summary of key findings of the study are briefly given.

4.1. Trends in airborne chrysotile asbestos fibre concentrations in asbestos cement manufacturing factories in Zimbabwe from 1996 to 2016.

There were 2 890 personal samples collected over the 21-year period 1996 to 2016 in the different operational areas of the two chrysotile cement manufacturing factories (Appendix 1, Table S1 page 161, in the Supplementary materials for **Chapter 6**), from which 1 663 monthly averaged personal chrysotile asbestos fibre concentrations were drawn from (Appendix 1, Table S2, page 162 for **Chapter 6**). A description of jobs considered in this study are also given in Table 1 of Chapter 6. The Harare factory had the greater proportion (63.9%) of monthly averaged concentrations. Tables 2 and 3 of **chapter 6** show the summary statistics of the personal chrysotile asbestos fibre concentrations for both factories with factories showing overall exposure concentration around 0.11 f/ml. Overall, 60.3% and 58.6% of personal exposure measurements in the Harare and Bulawayo factories respectively exceeded the OEL of 0.1 f/ml. Figure 1 and 2 in **Chapter 6** show that mean personal exposure chrysotile asbestos fibre concentrations at the Harare and Bulawayo factories respectively have generally been declining from 1996 to 2016 and for the Bulawayo factory where all manufacturing of AC products is taking place since 2017. Declines in exposure concentrations were further recorded for the time period of 2018 to 2020 for Bulawayo factory, while for the Harare factory exposure concentrations for the period 2018 to 2020 were elevated despite AC manufacturing processes having ceased since 2017 (**Table 1 in Chapter 7**). Additionally, personal exposure concentrations were generally high, above the OEL of 0.1 f/ml, before 2008 in both factories. Overall, during the period 1996 – 2000 exposure levels ranged from 0.11-0.18 f/ml compared to 0.04-0.12 f/ml personal chrysotile concentrations recorded for the period 2009 to 2016 for the Harare factory. Similarly, for the Bulawayo factory, personal chrysotile fibre concentrations ranged from 0.09-0.22 f/ml during the earlier years of 1996 – 2000 compared to 0.03-0.10 f/ml recorded for the period 2009 -2016. The one odd value recorded in 2013 as 0.01 f/ml could have just been recorded as less than the limit of detection (<LOD).

4.2. Job Exposure Matrix (JEM) for chrysotile asbestos fibre in the asbestos cement manufacturing (ACM) industry in Zimbabwe

Chapter 7 describes the architecture of the JEM which captured chrysotile asbestos exposure concentration up to 2020. Three thousand and sixty six (3066) airborne chrysotile personal measurements were collected from company records spanning a period of about 25 years. In total 1788 annual mean personal exposure, concentrations were used to build the job exposure matrix (JEM) for chrysotile asbestos fibre in the AC manufacturing factories. For the JEM, jobs selected had the most data and were in most common operational areas even up to 2020. The jobs involved are as outlined in Table 1 of **Chapter 7**.

The JEM presented in **Chapter 7** illustrates job categories with their description, factory location, mean and range, period and the possible amphiboles contaminants identified in chrysotile asbestos materials used in the manufacturing process. There was a statistically significant difference ($p < 0.001$) in the annual mean personal chrysotile exposure concentrations among the different time-periods for various job categories as determined by one-way ANOVA in both factory locations except for fettling table operator in Harare (Table 2 in **Chapter 7**). More specifically, in the Harare factory, the exposure concentrations during the period 2009 to 2016 was statistically significantly lower than exposure concentrations during the periods 1996-2000, 2001-2008 and 2018-2020. For the Bulawayo factory, the post hoc test (Table 2 **Chapter 7**) shows that exposure concentrations during the period 1996 to 2000 was statistically significantly higher than exposure concentration during the periods 2009-2016 and 2017-2019. The saw cutting, kollergang and pipe joints operators exposure concentrations were also statistically significantly higher during the period 1996-2000 than during the period 2001 to 2008 ($p < 0.05$).

Following bulk chrysotile sample analysis using SEM of locally and imported chrysotile fibre, fibres with aspect ratio greater than 3:1 morphologically resembling asbestos were observed in all the samples. For the 6 locally produced chrysotile samples analysed, 4 had amphiboles detected, namely tremolite and anthophyllite as shown in table 3 of **Chapter 7**, while for the imported chrysotile asbestos, amphiboles namely tremolite, crocidolite and actinolite were detected in 5 out of 6 samples analysed.

4.3 Prediction of Asbestos Related Diseases (ARDs) and chrysotile asbestos exposure concentrations in asbestos-cement (AC) manufacturing factories in Zimbabwe.

Following establishment of linear regression equations from the OSHA cancer risk assessment Table 1 (in **Chapter 8**) from which respective asbestos related cancers were derived at various cumulative exposures and respective duration of exposure of 1, 10, 20 and 25 years, Table 3 (**Chapter 8**) show summary of overall predicted cancer mortality cases (lung cancer, mesothelioma and gastrointestinal cancer). The table also show incidence rates, which may be experienced after being exposed at the mean exposure levels, associated with each respective time period for each respective job.

Overall summary estimates of cancer mortality cases by factory and duration of exposure suggests that 15, 135, 278 and 347 cases per 100 000 workers (saw cutting, fettling table, ground hard waste and laundry operators), of asbestos related cancers may be experienced after 1, 10, 20 and 25 years of exposure respectively in the Harare factory at the respective exposure periods (Appendix 3, Tables S7 to S10, pages 177-180 for Chapter 8). The Bulawayo factory with similar or same order of magnitude of cumulative exposure levels to those obtaining in the Harare factory also displayed a similar trend wherein same or similarly number of cancer cases per 100 000 workers exposed are likely to be experienced at saw cutting operators, fettling table operators as well as at pipe section after 1, 10, 20 and 25 years of exposure.

Estimates of possible asbestosis incidences likely to be observed at various cumulative exposures after a possible duration of exposure of 25-years duration of exposure and by job and time period are shown in table 4 of **Chapter 8**. Cumulative exposure levels ranged from 1.0 to 4.8 f/ml-years, with highest cumulative exposures being exhibited at the saw cutting operations in both factories, followed by ground hard waste operations particularly before 2008. Asbestosis cases likely to be detected after 25-years duration of exposure range from 50 to 260 cases per 100 000 workers (0.05 to 0.26% incidence of asbestosis) exposed for various jobs. On average in both factories, asbestosis cases likely to be detected were within the range of 150 – 160 per 100 000 workers exposed (or 0.15% to 0.16% incidence) of asbestosis.

4.4. Examining approaches to prevention of exposure to chrysotile asbestos and some perspectives on the debate on ban of asbestos.

Control of exposure to asbestos has been a contentious issue wherein banning asbestos is advocated whereas others argue that it is possible to properly use chrysotile in a manner which is safe since chrysotile in its modern-day high-density applications does not present unreasonable risks to exposed workers and the public. The measure centres on controlled use approach, which approach entails that industry, government and workers have a responsibility in the prevention of exposure (ILO, 1984; ILO, 1986; ICA, 2019). Controlled use approach means the development of asbestos regulations, implementation of good work practices, engineering controls, work environment and medical surveillance, education and training of all levels of employees on various aspects of asbestos and provision of appropriate and suitable personal protective equipment and clothing. The controlled use approach has been applied to known hazards and risks in many countries and is generally at the centre of prevention of exposure to many hazardous substances. The aim in the case of chrysotile asbestos like in many hazardous factors, is to ensure that exposure is below the occupational exposure limits of airborne chrysotile fibre, below which it is expected that risk of health impairment becomes minimal. Controlled use approach has been linked with low degrees of exposure, which suggest minimal or insignificant excess risk with respect to mortality and other asbestos related diseases (Weill et al., 1979; Thomas et al., 1982; Berry & Newhouse, 1983; Ohlson & Hogstedt, 1985; Gardner et al., 1986; Newhouse and Sullivan, 1989; Liddell et al., 1997; Sichletidis et al., 2008; Gibbs & Berry, 2008; Paustenbach et al., 2021). Exposure to chrysotile asbestos has been recently reported to decrease over time in more than two decades from 1996 to 2020 in Zimbabwe chrysotile asbestos cement manufacturing factories (Mutetwa et al., 2021; 2022). In particular one of the factories located in Bulawayo which has continued with AC products manufacturing to date has shown that exposures continued to decrease, with decreases being linked to improved occupational safety and health practices over the years and a good occupational safety and health system in place (Mutetwa et al., 2021; 2022).

It has however been argued that the controlled use approach is not practical as chrysotile asbestos cannot be used or handled safely (Smith & Wright, 1996; Tossavainen, 1997; Lemen et al., 2004b; LaDou et al., 2010; WHO, 2014). It has further been submitted that there is no safe dose and any exposure from any source is deemed to cause cancer (LaDou et al., 2010; IARC, 2012; WHO, 2014; Markowitz, 2015). These arguments are premised on the linear no threshold model of carcinogenesis that contents that every exposure to a carcinogen contributes

to a cumulative linear increase in risk of developing cancer even at very low exposure levels (Frank, 2016; Golden et al., 2019). Hence it is opined that the only plausible approach to control exposure is to ban all forms of asbestos including chrysotile (LaDou et al., 2010; WHO, 2014), adding that safer substitutes for asbestos exists and must replace chrysotile in all its uses (LaDou et al., 2010; Park, 2018).

REFERENCES

Berry G & Newhouse ML (1983). Mortality of workers manufacturing friction materials using asbestos. *British journal of industrial medicine*, 40(1), 1–7. <https://doi.org/10.1136/oem.40.1.1>.

Gardner MJ, Winter PD, Pannett B & Powell CA (1986). Follow up study of workers manufacturing chrysotile asbestos cement products. *British Journal of Industrial Medicine*, 43(11), 726–732. <https://doi.org/10.1136/oem.43.11.726>.

Gibbs GW & Berry G (2008). Mesothelioma and asbestos. *Regulatory toxicology and pharmacology: RTP*, 52(1 Suppl), S223–S231. <https://doi.org/10.1016/j.yrtph.2007.10.003>.

Golden R, Bus J & Calabrese E (2019). An examination of the linear no-threshold hypothesis of cancer risk assessment: Introduction to a series of reviews documenting the lack of biological plausibility of LNT. *Chemico-biological interactions*, 301, 2–5. <https://doi.org/10.1016/j.cbi.2019.01.038>.

International Agency for Research on Cancer (IARC) (2012). *IARC Monograph on Evaluation of Carcinogenic Risks to Human, Arsenic, Metals, Fibres and Dust: A review of human carcinogens Vol. 100C* p.219 – 309. IARC/WHO.

International Chrysotile Association (ICA) (2019). *Safety in the use of Chrysotile – Requirements and Achievements*. Available online at https://chrysotileassociation.com/data/ICA_Chrysotile_Safety_Requirements_Achievements_v5F_LR.pdf. (Accessed 13 April 2022).

International Labour Organisation (1984). *Safety in the use of asbestos: An ILO Code of practice*, ILO, Geneva.

International Labour Organization (ILO) (1986). *Convention No. 162 Convention - Safety in the Use of Asbestos*, ILO, Geneva, Switzerland.

LaDou J, Castleman B, Frank A, Gochfeld M, Greenberg M, Huff J, Joshi TK, Landrigan PJ, Lemen R, Myers J, Soffritti M, Soskolne CL, Takahashi K, Teitelbaum D, Terracini B, &

Watterson A (2010). The case for a global ban on asbestos. *Environmental Health Perspectives*, 118(7), 897–901. <https://doi.org/10.1289/ehp.1002285>.

Lemen RA (2004). Chrysotile asbestos as a cause of mesothelioma: application of the Hill causation model. *International Journal of Occupational and Environmental health*, 10(2), 233–239. <https://doi.org/10.1179/oeh.2004.10.2.233>.

Markowitz S (2015). Asbestos-related lung cancer and malignant mesothelioma of the pleura: selected current issues. *Seminars in Respiratory and Critical Care Medicine*, 36(3), 334–346. <https://doi.org/10.1055/s-0035-1549449>.

Mutetwa B, Moyo D & Brouwer D (2021). Trends in Airborne Chrysotile Asbestos Fibre Concentrations in Asbestos Cement Manufacturing Factories in Zimbabwe from 1996 to 2016. *International journal of environmental research and public health*. 18(20), 10755. <https://doi.org/10.3390/ijerph182010755>.

Mutetwa B, Moyo D & Brouwer D (2022). Job Exposure Matrix for Chrysotile Asbestos Fibre in the Asbestos Cement Manufacturing (ACM) Industry in Zimbabwe. *International journal of environmental research and public health*, 19(5), 2680. <https://doi.org/10.3390/ijerph19052680>.

Ohlson CG & Hogstedt C (1985). Lung cancer among asbestos cement workers. A Swedish cohort study and a review. *British Journal of Industrial Medicine*, 42(6), 397–402. <https://doi.org/10.1136/oem.42.6.397>.

Sichletidis L, Chloros D, Spyrtos D, Haidich AB, Fourkiotou I, Kakoura M & Patakas D (2009). Mortality from occupational exposure to relatively pure chrysotile: a 39-year study. *Respiration; international review of thoracic diseases*, 78(1), 63–68. <https://doi.org/10.1159/000163443>.

Smith AH & Wright CC (1996). Chrysotile asbestos is the main cause of pleural mesothelioma. *American Journal of Industrial Medicine*, 30(3), 252–266. [https://doi.org/10.1002/\(SICI\)1097-0274\(199609\)30:3<252::AID-AJIM2>3.0.CO;2-0](https://doi.org/10.1002/(SICI)1097-0274(199609)30:3<252::AID-AJIM2>3.0.CO;2-0).

Tossavainen A (1997). Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution. (1997). *Scandinavian Journal of Work, Environment & Health*, 23(4), 311–316. [doi:10.5271/sjweh.226](https://doi.org/10.5271/sjweh.226).

Thomas HF, Benjamin IT, Elwood PC & Sweetnam PM (1982). Further follow-up study of workers from an asbestos cement factory. *British Journal of Industrial Medicine*, 39(3), 273–276. <https://doi.org/10.1136/oem.39.3.273>.

World Health Organization (WHO) (2014). Chrysotile Asbestos. Available online at; http://www.who.int/ipcs/assessment/public_health/chemicals_phc. (Accessed February 9 2020).

Weill H, Hughes J & Waggenpack C (1979). Influence of dose and fiber type on respiratory malignancy risk in asbestos cement manufacturing. *The American Review of Respiratory Disease*, 120(2), 345–354. <https://doi.org/10.1164/arrd.1979.120.2.345>.

CHAPTER 5

Discussion and Conclusion

5.1 DISCUSSION

This chapter discusses methodological issues associated with various aspects of the study. It discusses limited chrysotile measurements and quality assurance issues. Challenges to occupational health research are also discussed. The chapter also provides possible policy, operational and research recommendations. An overall conclusion sums up the chapter.

Exposure to chrysotile asbestos fibre has been ongoing in Zimbabwe for many decades since the mines were opened around 1910 and manufacturing began around 1943. Despite such exposures, and general decline in the use of chrysotile asbestos and even a complete ban on the same in some other countries, Zimbabwe among other countries continues to mine, process, and use chrysotile asbestos in the manufacture of various AC products. Occupational exposure data has been collected by the chrysotile asbestos manufacturing factories in Zimbabwe over the past decades, however, such data has not been examined much to inform extent of exposure to chrysotile fibre and or applied in the development of such tools as the job-exposure matrix. Therefore, the key objectives of the study were to examine trends in airborne chrysotile asbestos fibre data obtained by the chrysotile asbestos cement manufacturing factories for the period 1996 to about 2020; develop a job exposure matrix (JEM); predict asbestos related diseases namely lung cancer, mesothelioma, gastrointestinal cancer and asbestosis in the AC manufacturing industry through the exposure levels obtained in the AC factories; and assess possible amphiboles contamination in the chrysotile asbestos fibre being used in the manufacturing processes. The study also sought to examine approaches for prevention of exposure to chrysotile asbestos and provide perspectives on the debate around the banning of all forms of asbestos including chrysotile.

The study constitutes the single largest chrysotile asbestos exposure dataset in Zimbabwe and perhaps Africa, spanning a period of about two and a half decades. It is also the first study that attempts to build an occupational JEM specific to chrysotile asbestos as well as give insights into possible ARDs that may be observed following exposure at various cumulative exposures associated with some time periods in Zimbabwe. The study also considerably provides an opportunity to expand and deepen research in the ARDs particularly cancer in Zimbabwe. The data set thus provides a basis for epidemiological research in AC manufacturing industries in Zimbabwe, an area that has very limited evidence based occupational and public health information materials specific to Zimbabwe workplace settings.

Methodological issues

Trends in airborne chrysotile fibre concentrations in the AC manufacturing factories.

Although the AC manufacturing factories had considerable measurements taken through static sampling, only personal exposure measurements were included in this study since this sampling strategy directly measures an individual's exposure as it occurs. Personal chrysotile measurements after 2009 generally became fewer and hence may have reduced the accuracy of mean exposure values for various operational areas. Generally, and statistically, collection of as much data values increases accuracy of mean values. Some operational areas had too few measurements and these were laundry (28 values) and sheeting plant mixer (30 values) collected for the entire two and half a decade period for the Bulawayo factory, hence these were not considered for analysis.

Another methodological issue arising from the study is that no accurate data on a potential exposure determinant or modifier, i.e. production volumes over the years, was available limiting appropriate interpretation of the exposure data before and after 2008 based on productivity.

As can be observed from Appendix 1 Tables S1 and S2, page 161, the number of personal samples during the latter years from about 2008 to 2020, decreased largely due limited availability of consumables required for sampling of chrysotile asbestos fibre in various operational areas. This may have diminished somewhat the accuracy of the monthly mean personal exposures and subsequently annual mean personal chrysotile fibre exposures for various operational areas.

For analysis, personal sampling points were classified into similar or homogeneous operational areas from which monthly averaged concentration per operational area were derived. The number of monthly averaged personal chrysotile concentrations per operational area were used to derive annual average chrysotile exposure concentration. Although personal sampling points from which chrysotile exposure data was obtained was considered to be similar or homogenous, some variations in exposure concentrations may exists within similar or homogenous operational areas due to possible differences in work practices or performance of equipment being used by the operators or the way in which the operators carryout their work.

It is also insightful to note that occupational health research with respect to asbestos related diseases has been lacking in Zimbabwe largely because there has not been any attempt to build a database on chrysotile asbestos exposure measurements, which measurements could inform dose-response relationships as well as provide overall picture of ARDs in the chrysotile AC manufacturing industry.

Reliability and sustainability of chrysotile asbestos measurements over the years

From about 2013 to 2020, participation of the factories in quality assurance programmes has been limited largely as a result of lack of resources induced by general economic challenges affecting industries, thus compromising the validity and reliability of results in the latter years. Going forward the factories will be encouraged to resuscitate the quality assurance and quality control programme that used to obtain during the period 1990s to about 2011 in order to restore confidence in the quality of chrysotile asbestos fibre measurements. The company used widely recognised method for asbestos fibre measurements namely the Asbestos International Association Recommended Technical Membrane Filter Reference Method, thus giving some measure of reliability on personal chrysotile fibre data produced by the factories. Observations also made from sampling and sample treatment provided insights into the correctness by the factories, in following the correct methodology including use of blanks, further providing confidence in the quality of data produced. The OSH arm of National Social Security Authority (NSSA) will be better placed to encourage and promote quality assurance and quality control programme for chrysotile asbestos fibre measurements in the AC manufacturing industries. This will help the nation to have a database of exposure measurements that are valid and reliable.

Phase contrast microscopy

One of the limitations of phase contrast microscopy (PCM) is that it does not differentiate between asbestos and non-asbestos fibres (AIA, 1982; NIOSH, 1994). Hence, it may be possible that non-asbestos fibres were counted thereby overestimating the airborne fibre concentrations. Transmission electron microscopy (TEM) and scanning electron microscopy (SEM) methods can be used to detect thinner and shorter fibres than PCM and also fibre type can be identified with additional analysers based on elemental composition and crystal structure (ISO, 2019, 2019a, 2019b; ECHA, 2021). Asbestos by TEM is used to determine asbestos fibres in the optically visible range and is intended to complement the PCM method (NIOSH, 1994b).

TEM also offers adequate resolution to allow detection of small fibres and is a technique capable of unequivocal identification of the majority of individual fibres of asbestos and generally has been adopted as an international standard for asbestos measurement (ISO, 2019b). In Europe most laboratories use SEM as it is considered better in terms of minimum detection limit, measurement uncertainty and the likelihood of contamination (Health Council of the Netherlands, 2010). The SEM method is also intended to be used as a complement of the PCM methods for samples where:

- different types of inorganic fibres are present, which have to be distinguished from each other and from organic fibres;
- the limit of detection of the phase-contrast microscopic method is not sufficient to monitor the compliance of a given threshold limit values and trigger thresholds (ECHA, 2021).

SEM also counts fibres thinner than that observable using PCM, with detection limits as little as 0.01 f/ml being able to be detected (Health Council of the Netherlands, 2010). Despite superior performance of TEM and SEM, the use of electron microscopy is more expensive, instrumental techniques quite complex requiring the involvement of specially trained personnel, is not readily available in Zimbabwe and the region in general, hence the PCM method naturally became method of choice for asbestos fibre measurement. As stated earlier on, PCM is not able to determine fibre type; however, the factory assumed that since the factories were essentially using chrysotile asbestos, exposure concentrations were those for chrysotile asbestos fibres.

Prediction of asbestos related diseases

Prediction of possible cases of ARDs have been undertaken across many countries with a view to inform government policy as well as public and occupational health needs of the country (Reid, 2016). Estimates of the future burden of diseases can thus help to evaluate what preventive programmes are required and what extend of compensation claims maybe anticipated from persons possibly afflicted by ARDs. A number of approaches for cancer risk assessment have been published and include the French Agency for Environmental and Occupational Health & Safety (Afsset, 2009b; Yamani, 2012; ECHA, 2021). This model considered among other issues the protective level associated with TWA exposure limit of 0.1 f/ml, and being the exposure level then obtaining in Europe. It was concluded that since all commercial varieties of asbestos had the potential to induce cancer in humans through

inhalation, there was no need to differentiate them when setting an OEL (ECHA, 2021). In addition, it was pointed out that carcinogenicity of asbestos fibre presented itself in a manner that does not have a threshold and that available data can derive a dose-effect relationship at low exposures and obtain an excess risk that takes into account lung cancer and mesothelioma. The French model took the approach of the INSERM model (INSERM, 1997), a model that was based on the EPA, (1986) linear in dose model. The application of the INSERM model was also motivated by the fact that the model used French mortality data and hence it was considered to give more relevant cancer risk level consistent with the French population. Following modelling of the data it was concluded that even though 0.01 f/ml exposure level could constitute a significant step towards risk reduction due to asbestos exposure, it possibly was better to retain an even lower exposure level of 0.00003 f/ml corresponding to a risk level of 1×10^{-6} (i.e. 1 cancer case per 10^6 exposed), this taken in the context of presumed no threshold mechanism associated with the occurrence of cancer due to asbestos in humans (Afsset, 2009b; ECHA, 2021).

In Germany, the Committee on Hazardous Substances (AGS), (AGS, 2008), applied the EPA model (EPA, 1986) to derive workplace air concentrations that correspond to what was considered as nominal risk (4×10^{-3} and tolerable risk (4×10^{-4}) or 4×10^{-5} as of 2018. These risk levels were derived based on 40 years of work life of about 240 workdays per year and 8 hours a day. This led to the following risk level – exposure concentration scenarios;

- 4×10^{-3} for exposure concentration of 0.1 f/ml
- 4×10^{-4} for exposure concentration of 0.01 f/ml
- 4×10^{-5} for exposure concentration of 0.001 f/ml

However, the concentration values were based on SEM method.

The Health Council in the Netherlands (HCN) – Dutch Expert Committee on Occupational Safety (DECOS), derived excess risk for lung cancer and mesothelioma through meta-analysis of cohort studies they considered had the highest quality of data that included the South Carolina textile factory and the UK asbestos chrysotile textile factory (HCN, 2010). The HCN-DECOS (HCN, 2010), derived occupational exposure reference levels corresponding to the nationally established benchmark excess risk levels of 4×10^{-3} and 4×10^{-5} . These reference levels were calculated combining excess risk of lung cancer and excess risk of mesothelioma. Exposure concentration of various types of asbestos based on TEM were used to derive excess risk. For chrysotile, excess risk associated with exposure levels of 0.2 f/ml was 4×10^{-3} , while that for 0.002 f/ml it was 4×10^{-5} . HCN-DECOS (HCN, 2010) considered that the asbestos-

related risk is much higher for lung cancer and mesothelioma than for the other cancers, including ovarian and laryngeal cancer and therefore lung cancer and mesothelioma risks were used to define the exposure standards.

Comparatively, AGS and Afsset used the EPA (1986) model data applied to the national population and consequently have close to identical fibre levels. On the other hand, HCN-DECOS used meta-analysis to derive excess risk of cancer. These models provides insights into possible approaches that can be applied to gain insights into asbestos risk levels associated with certain exposure levels to asbestos.

The EPA, (1986) model used in this study to predict ARDs has been a model adapted in the past to do cancer risk assessment in some countries as shown in the case of Germany and France above. Furthermore, the Germany (AGS) model also used linear extrapolation to different cumulative exposures to derive cancer risk at various exposure levels (ECHA, 2021), an approach similarly applied in this study. Prediction of ARDs was based on cumulative exposure, a matrix frequently used in exposure-response analysis in epidemiologic studies. However, the imposed symmetry between duration and intensity of exposure has been considered as a potential problem with this measure, wherein the relationship between cumulative and dose was not necessarily linear particularly at higher exposure levels (Finkelstein, 1983, 1995; Smith, 1992; MSHA, 2005). However, the OSHA risk assessment model generally relied on a risk model that was linear in cumulative dose in computation of estimates of ARDs namely lung cancer, mesothelioma, gastrointestinal and asbestosis. Cumulative exposure matrices have also been noted to obscure the interplay of exposure intensity and duration of exposure (Vocht et al., 2015). Other limitations with respect to the OSHA risk assessment models for asbestos related cancer suggest that the epidemiological data used in developing linear in dose relationship for human exposure to asbestos are limited because of the fact that current health effects are largely as a result of past exposures, assuming that in the past exposure control were inadequate. Dose-response data with respect to gastrointestinal cancer is limited compared to that for lung cancer and mesothelioma. It has been reported that quality of the cohort studies as a criterion in evaluating exposure response were not usually considered in several meta-analysis and that the range of uncertainty in risks of lung cancer and mesothelioma could be wide (HCN, 2010; Lenters et al., 2011), thus further pointing towards some limitation in applicability of the OSHA Risk Assessment model. Despite these limitations the personal exposure data used in this study and applied in the context of the

OSHA risk assessment linear in dose relationship, provides some possible estimates of asbestos disease in the chrysotile asbestos cement manufacturing factories in Zimbabwe

The limited or lack of information regarding the number of workers employed as per job title over the years, limits the prediction of ARD with respect to lung cancer, mesothelioma gastrointestinal cancer and asbestosis. It could only be estimated at a level of incidence rate per 100 000 workers, rather than a more precise estimate of the expected number of cases in the work population of both factories.

Discussion around various topical aspects of the thesis.

The dataset for chrysotile personal exposure concentrations used in this study is the largest in Zimbabwe to the best of our knowledge and provides a good basis for a reference point for future epidemiology studies that may be undertaken. The observed decline in mean monthly measured exposure concentrations from 1996 to 2016 suggests application of good occupational safety and health (OSH) framework that was being implemented by the factories coupled with the desire to ensure that exposure levels were below the OEL of asbestos as much as possible. Exposure concentrations were generally well above the OEL before 2008 possibly as a result of significant economic activity which entailed increased demand for chrysotile asbestos products and also weak OSH standards which were still at an early stage of development. Nonetheless, lack of accurate data on production volumes over the years made interpretation of the exposure data before and after 2008 limited.

The downward trend observed in exposure concentrations over time is not unique to Zimbabwe AC manufacturing entities as Zilout et al., (2020) reported that there was a decline in exposure in respirable dust and quartz concentration in European countries over a 15-year period. Additionally, it has been reported that after 1950s occupational hygiene controls improved progressively in factories resulting in a steady decline in chrysotile asbestos fibre concentrations to about 0.5 to 1 f/ml from 1970s onwards. Decreases in exposure were attributed to good occupational hygiene controls. The downward trend in exposure concentrations were also consistent with patterns observed in other AC manufacturing facilities where enforcement of legislation and good occupational hygiene practices were implemented. Decreases in and banning the use of asbestos were also cited as factors contributing to declines in exposure over time (Yoshizumi et al., 2001; Hagemeyer et al., 2006; Creely et al., 2007; Park et al., 2008; Pira et al., 2018).

The study also sought to establish a job exposure matrix for chrysotile asbestos fibre in the AC manufacturing factories. The personal chrysotile exposure levels were related to work characteristics of jobs which were broadly classified into homogenous exposure groups. The job categories with high exposures levels were saw cutting, fettling table, ground hard waste, laundry room and multi-cutter operators and as such, workers in these jobs were observed to be at increased risk of developing ARDs. Although there was a general decline in exposure levels over the years particularly for the Bulawayo factory for which manufacturing has continued to date, exposure concentrations in the Harare factory for the period 2018 to 2020 were much higher than the preceding time-period of 2009 to 2016 despite ceasing manufacturing of AC products end of 2016. Elevated levels in the Harare factory for the period 2018 to 2020 may be attributed to lack of dust suppression methods in the current activities of concrete products manufacturing, wherein suspension of chrysotile fibre from the floors which accumulated chrysotile fibre in the past could be responsible for higher exposure levels for the time period 2018 to 2020. The company continued to monitor exposure to chrysotile asbestos as it was its considered view that movement of trucks within the factory as they come load concrete tiles and other products makes much of the possible residual chrysotile fibre to become airborne. The company also did not report any wet cleaning of floors following switching to concrete products manufacturing, hence any frequent movement of trucks into and out of the factory yard may have contributed to increased dust in the factory environment. Sometimes, though not so often, it was observed and reported that the factory continued to use two saw cutting machines to cut AC sheets manufactured in the Bulawayo factory to size and in accordance with some customer specifications, hence the saw cutting process may also be contributing to elevated exposure levels after 2018. On the other hand, continued decline in exposure levels in the Bulawayo factory for the period 2017 to 2020 compared to the preceding time period suggest continued adherence to good work practices and effective implementation of occupational safety and health (OSH) standards. While decline in exposure may be attributed to implementation of good OSH practices, it may also be possible that decrease in exposure may possibly be attributed reduced production as the company reported that in some cases the factory was operating at 60-70% plant capacity utilisation due limited materials for production.

The JEM established in this study also show that there may be risk of exposure to asbestiform amphiboles during manufacture of AC products. Analysis of amphibole contaminants has shown that tremolite and anthophyllite may be associated with locally produced chrysotile, while tremolite, crocidolite and actinolite may be associated with imported chrysotile (Mutetwa et al., 2022). A study on tremolite in Southern Africa chrysotile including chrysotile from

Zvishavane (Shabanie and Mashava Chrysotile Mines) seem to suggest that a small amount tremolite were found in the Southern African chrysotile (Rees et al., 1992), while other reports suggest that Zimbabwe chrysotile contains anthophyllite (Kohyama et al., 1996). Anecdotal information through a discussion with a long time expert geologist who worked for Shabanie and Mashava chrysotile asbestos mines suggested that his experience with the chrysotile asbestos in these mines was that there was no amphiboles associated with Zvishavane chrysotile asbestos mines. The presence of amphiboles as a contaminant in chrysotile asbestos is not uncommon.

Chrysotile asbestos has been found to contain asbestiform amphiboles (Pooley, 1976; Rowlands et al., 1982; Addison and Davies, 1990; Liddell et al., 1997; McDonald et al., 1997; Hein et al., 2007; IARC, 2012). In addition, chrysotile bulk samples originating from six Chinese chrysotile mines, amphibole asbestos largely tremolite was identified within the range 0.002 and 0.310 w-% (Tossavainen et al., 2001). Although the presence of amphiboles asbestos such as tremolite in chrysotile fibre has been suggested to be the main cause of lung cancer and mesothelioma, it has been argued that separating tremolite from the chrysotile would not make a difference to the disease outcome. Hence, chrysotile can be viewed as equally potent as the amphiboles (WHO, 2014). Additionally, reports of cancer cases among asbestos workers in settings considered amphibole free such as Zimbabwe mines and Balangero, Italy may suggest the presence of amphiboles associated with chrysotile asbestos mined or used (Piolatto et al., 1990; Cullen and Baloyi, 1991; Mirabelli et al., 2008). The revelation of amphiboles contamination in locally and imported chrysotile asbestos, in this study, though not quantitatively evaluated, may suggest increased risk of cancer among workers exposed due the presence of amphiboles in the chrysotile asbestos being used in the manufacturing processes.

The results in this study provide support for the need of more detailed investigation into the presence of possible amphiboles in locally produced and imported chrysotile. Such observations may thus call for the need to monitor regularly through analysis, the quality of chrysotile fibre being used in the AC manufacturing factories, as amphiboles have been associated with increased risk of ARDs (Hodgson and Darnton, 2000; IARC, 2012; WHO, 2014).

The study also attempted to predict possible ARDs in the AC manufacturing factories by applying the OSHA linear in dose response relationship and establishing linear regression equations for lung cancer, mesothelioma, and gastrointestinal tract cancer in order to derive

estimates of asbestos related cancers and asbestosis in combination with chrysotile asbestos exposure levels collated for the period 1996 to 2020. The results suggest that saw cutting operators followed by kollergang and ground hard waste operators in both factories may be at increased risk of developing ARDs. The results (Tables 3 page 110 and Table 4 page 113, of Chapter 8) show high number of predicted cancer mortality cases and asbestosis cases especially if workers were to be exposed before 2008 time periods. Exposure concentrations and subsequently cumulative exposures before 2008 were high compared to exposure concentrations for the time period of 2009 to 2020, when duration of exposure of 20 years or 25 years is applied.

A study carried out in Iran on prediction of asbestos cancer mortality in an AC factory through use of chrysotile asbestos fibre concentrations and application of the OSHA risk assessment model (OSHA, 1986), revealed that there was increased risk of ARDs particularly in the milling and cutting operations of the factory. The predicted ARDs in the current study were however lower than those reported by Jafari et al. (2010). The study in Iran reported 499 cases per 100 000 workers after 1 year of exposure and 6965 cases per 100 000 workers after 20 years of exposure, following application of the OSHA risk assessment model (Jafari et al., 2010), compared to about 14 and 255 cancer mortality cases after 1- and 20-years exposure respectively in the case of Harare factory. Bulawayo factory follows a similar pattern to that of Harare in which cancer mortality cases that may be experienced after 1- and 20-years exposure were also much lower than those reported by Jafari et al., (2010).

ARDs cases derived from this study although they appear low may possibly constitute a significant risk if evaluated against the 1 case per 1000 workers measure considered as a significant risk (OSHA, 1986; MSHA 2005, 2008). Overall cancer mortality cases ranged from 2.89 to 3.47 in the Harare factory while for the Bulawayo factory, cases ranged from 2.89 to 4.6 per 1000 workers. These predicted overall cancer mortality cases appear elevated because of high chrysotile exposures experienced in the earlier time periods of 1996 to 2008. However, if consideration is given to other risk levels such as 4 cases per 1000 workers exposed (Netherlands and Germany), and then risk range obtained above for this study may not necessarily be significant. Risk of ARDs may be reduced by reducing exposure levels as shown in the Bulawayo factory in which exposure levels over the years 1996 to 2020 have been progressively on a downward trend to the extent that exposures within the range of 0.04 to 0.07 f/ml were being realised during the period 2018 to 2020. This may in turn suggest that cancer mortality cases may possibly approach 1 or less per 1000 workers exposed as exposure levels

declined due to strict adherence to and implementation of measures of control that included engineering controls, good occupational hygiene practices, and use of appropriate respiratory protective equipment of which such measures should be premised on a sustainable occupational safety and health management system.

Despite cancer mortality cases seemingly suggesting an approach to less than 1 as discussed above, it is insightful to note that recent cancer risk assessment models applied to determine excess risk at various exposure levels demonstrate that even at very low exposure levels, risk of asbestos related cancer remains (ECHA, 2021). Hence, the argument that asbestos is a non-threshold carcinogen. Subsequently, no health-based OEL has been identified and an exposure-risk relationship (ERR) expressing the excess risk for lung cancer and mesothelioma mortality (combined) in relation to ambient air concentration was derived for Europe. The ERR was calculated for all asbestos, combining all studies regardless of the asbestos fibre type the population was exposed to. The ERR focuses on air concentrations at and below the current OEL of 0.1 f/ml (ECHA, 2021) and without taking into consideration that the AC industry in Zimbabwe essentially uses chrysotile, the ERR matrix presented by ECHA would possibly suggest increased risk of ARD since exposure levels in the AC manufacturing industry had considerable exposure levels above 0.1f/ml especially before 2008. Both factories recorded about 60% of exposures above the OEL (Mutetwa et al., 2021). Notwithstanding this, Zimbabwe as a developing nation may consider to adopt a phased approach to implement a much more protective exposure limit taking into account its technological and economic development over perhaps a 5 to 10 year period.

Asbestos measurement method that is readily available in Zimbabwe is the PCM while in developed nations like Europe TEM and SEM are increasingly becoming the methods of choice for asbestos measurements. Examination of a fibre samples by either TEM or SEM allows the detection of much smaller fibres than PCM, and so much thorough data can be collected on fibre length and diameter distribution (ECHA, 2021). Much of the exposure data used in some various models for cancer risk assessment used TEM and SEM as a basis of cancer risk estimates and thus risk estimates derived may potentially differ with those established using the EPA 1986 model whose exposure levels were based on PCM and much of the data used in this model involved exposure data of the past. Europe has proposed an OEL of 0.01 f/ml in 2022 and such an exposure concentration correspond to excess life time cancer risk of 12 per 100 000 exposed or 0.12 per 1000 exposed (ECHA, 2021; EC, 2022). Such an exposure limit may be difficult to attain in the context of Zimbabwe technological and economic development

hence a phased approach in establishing a plausible exposure limit and benchmark excess cancer risk values can be considered by Zimbabwe.

Discussion around exposure to asbestos and in particular chrysotile has evoked panic, anxiety and fear across a broad range of societies. Opinions on health effects around various types of asbestos namely chrysotile and amphiboles have differed sharply in which one view holds that asbestos is very toxic and cancerous in all its forms including chrysotile, that there is no safe level of exposure and that it is not possible to control exposure. Hence, the only means of preventing exposure is taken as a ban. This view is further amplified by arguing that safer alternatives to chrysotile are readily available and thus continued use of chrysotile is not justified (Harrison et al., 1999).

On the other hand, one perspective holds the view that chrysotile asbestos is a less hazardous substance compared to amphiboles. Furthermore, the view highlights that if properly controlled, chrysotile presents minimal health risk to workers and the public. The view argues that like any other hazardous material, exposure to chrysotile can be managed through a tripartite approach in which government, industry and workers have a responsibility in preventing exposure (ILO, 1984; ILO, 1986, ICA, 2019). Prevention of exposure means among other aspects, effective implementation of engineering controls, good work practices, monitoring of the work environment with respect to asbestos exposure, medical surveillance, education, training and awareness raising of workers and employers, and provision of appropriate respiratory protective equipment (ILO, 1984; ILO, 1986; HSE, 2012; ICA, 2019; SA Govern, 2020; Paustenbach, 2021). It has been further been submitted that controlled use approach has been applied to many known hazards in many countries to which exposure levels have been observed to gradually decline over the years with exposure levels below 0.1 f/ml under today's work conditions not being uncommon (Paustenbach, 2021). In addition, application of controlled use approach has been linked with low degrees of exposures which have been reported to be associated with minimal or insignificant excess risk of ARDs (Weill et al., 1979; Thomas et al., 1982; Ohlson & Hogstedt, 1985; Gardner et al., 1986; Newhouse and Sullivan, 1989; Liddell et al., 1997; Sichelidis et al., 2008; Gibbs & Berry, 2008; Mutetwa et al., 2022).

It has further been argued that chrysotile substitutes are no safer than chrysotile asbestos itself (Infante et al., 1994; Siemiatycki, 2001) and since some manmade chrysotile substitute fibres such as PVA, cellulose fibre and p-aramid are subject to controlled use approach, chrysotile

fibre should also be subjected to same preventive measures as the substitutes (Camus, 2001; Harrison 1999). It has also been submitted that chrysotile has been studied extensively with health risk being overestimated because of heavy exposures of the past; hence, a comparative risk-based approach under today's exposure conditions may better inform a basis for banning chrysotile asbestos use (Camus, 2001).

5.2 Recommendations

Some recommendations arise from this study. These recommendations can be classified as policy, operational or research related recommendations.

Policy related recommendations.

Following the ratification of ILO convention 162 on safety in the use of asbestos, Zimbabwe should develop a regulatory framework to anchor asbestos management in the country. Such a regulation will strengthen the already existing policy on controlled use approach that government has adopted as reflected in the National Chrysotile Task Force (ZNCTF) policy document (ZNCATF, 2014) as well industry's own efforts in implementing sustainable OSH systems. Among other provisions, the regulation should compel AC manufacturing entities to report periodically, records of ARDs surveillance programmes and chrysotile asbestos fibre exposure levels obtaining in the factories. The chrysotile exposure measurements must be underpinned by robust and credible quality assurance and control programmes. Additionally, locally produced and imported chrysotile should be subject to periodic assessment for amphibole contaminants. Results from this study suggest that there is a possibility that chrysotile asbestos used in the AC manufacturing processes, whether produced locally or imported may be contaminated with amphiboles such as tremolite, crocidolite or anthophyllite (Mutetwa et al., 2022).

Any alternatives/ substitutes to chrysotile asbestos should be appropriately evaluated before being introduced to the market. Chrysotile is a mineral that has remained under the spotlight and its exploitation and trade may prove challenging in view of a strong worldwide call to ban all forms asbestos including chrysotile. To date about 60 countries have banned use of chrysotile (Kazan Allen, 2022). While chrysotile asbestos may be argued and considered safer than amphiboles, this area remains largely contested as others argue that the only safe way to handle chrysotile is to ban its use.

Operational recommendations

AC manufacturing factories and the chrysotile asbestos industry must have strong OSH systems, resourced and well capacitated to establish health surveillance programmes that include post-employment medical surveillance in light of long latency period associated with ARDs. Furthermore, factories should have robust work environment monitoring and quality assurance programmes for chrysotile asbestos exposure in order to produce validated data on chrysotile asbestos exposure. While the current Pneumoconiosis Act Chapter 15:08, 1996, compels dusty occupations of which AC manufacturing factories are classified as such, to have their workers go through health surveillance with respect to being taken X-rays to check for pneumoconiosis, the Act is limited in scope as far as prevention aspects are concerned. Hence AC manufacturing entities should step up their preventive programmes so that the downward trend with respect to chrysotile exposure continue to be sustained ensuring that wet methods in which water is generously used during manipulation of the ACM, including cleaning and sweeping are used to minimise the release of chrysotile fibre in the workplace air.

Government through the National Social Security Authority (NSSA) Division of Occupational Safety and Health (OSH) should build capacity to monitor chrysotile asbestos exposure in the AC manufacturing industry, the NSSA OSH Division itself and chrysotile asbestos industry in general including the mining sector. Such capacity should include among others issues, training of regulatory aspects of chrysotile asbestos, sampling and measurements, infrastructure development and provision of adequate equipment and accessories for monitoring chrysotile asbestos fibre exposure in applicable work environment settings. Specialist occupational medicine practitioners should also be trained to correctly diagnose ARDs particularly cancers which in the case of mesothelioma has been reported to be misdiagnosed and difficult to recognise it.

Additionally, government through the NSSA's OSH Division or through an institution of higher learning such as the University of Zimbabwe establish a state-of-the-art facility for the science of exposure measurements that include setting up of SEM, TEM and other related analytical instrumentation to strengthen capacity for occupational health, occupational hygiene and occupational medicine programmes in the country. The setting up of the state of art facility to also cater for advance occupational health programmes will also strengthen the national OSH

system. The manufacturing processes should also be improved by introduction of innovative technology that minimise interface between people and processing machinery and equipment.

Research related recommendations.

Possible ARDs have been predicted; however, more research is required to examine actual ARDs and in particular asbestos related cancers in the AC manufacturing industry. Additionally, AC manufacturing should record and maintain employment histories in order to build a basis for cohort studies. Research on occurrence of ARDs can also initially start by identifying workers who worked in high-risk operations such as saw cutting, kollergang, fettling and ground hard waste operations and follow-up such workers to assess whether they have not been affected by any ARD. Such a study will also provide insights into effectiveness of control measures being instituted by the factories.

Further research is required to explore perhaps over a period of 1-2 years, amphibole contaminants in chrysotile produced locally and that which is imported. Results from this study though being a snapshot cross sectional in nature has shown that locally produced and imported chrysotile asbestos may contain amphiboles contaminants. A more elaborate and comprehensive assessment of the chrysotile fibre being used will also inform policy on importation of chrysotile into the country as well as mining approaches that may minimise exposures to chrysotile. Collaborative research with countries that are still producing and exporting chrysotile asbestos are necessary to examine the presence of possible amphiboles in chrysotile asbestos being used in AC manufacturing industry.

It has also been argued that other forms of mining such as diamond and platinum mining presents risk of exposure to asbestos (Nelson et al., 2012). Against this background, it is important that research on exposure assessment to asbestos be extended to other forms of mining in order to generate, a body of knowledge on extend of asbestos exposures in the mining sector in Zimbabwe. Zimbabwe is very active in diamond, platinum and iron mining and hence a collaborative approach on asbestos exposure assessment is necessary to generate asbestos exposure profiles in other mining sectors.

Efficacy related research on control measures could also be important to consider particularly focussing on high-risk operations such as saw cutting, kollergang and ground hard waste. In addition to wet related methods of dust suppression methods, technologically driven control

measures at the cited operational areas should be developed and assessed to further reduce significantly exposures to chrysotile asbestos fibre.

5.3 Conclusion

The personal exposure chrysotile asbestos fibre measurements collected over two and half decades from 1996 to 2020 in key operational areas of the factories aided in a comprehensive analysis of trends in personal exposure chrysotile asbestos fibre concentrations in the asbestos cement manufacturing industry in Zimbabwe. The study has shown that personal exposure chrysotile fibre concentration data in the two factories has been on a downward trend over the years, with high concentrations being exhibited in or before 2008, thus showing that ACM presents a health risk. Chrysotile exposure decreases over the years was linked to implementation of aspects of the controlled use approach. The JEM generated in this study has provided quantitative estimates of personal chrysotile asbestos exposure concentrations for workers operating in ACM plants, based on jobs held and factory locations worked in and may give estimates of latency based on estimates of time period of exposure used.

The study has also concluded that saw cutting, kollergang and ground hard waste operators in both factories may be at increased risk of developing ARDs. The high number of predicted cancer mortality cases and asbestosis cases especially if workers were exposed before 2008, where exposure concentrations and subsequently cumulative exposure were high compared to exposure concentrations for the time period of 2009 to 2020, if duration of exposure was 20 years or 25 years suggest increased risk of ARDs among workers in jobs indicated above.

Asbestos mortality/morbidity studies are required with the aim of a comprehensive comparable analysis of predicted and actual mortality cases that may be obtaining in the AC manufacturing factories in Zimbabwe.

Asbestos management in Zimbabwe needs to be strengthened by specific regulatory provisions, effective implementation of occupational safety and health management systems that include a strong component on occupational health to oversee chrysotile asbestos fibre monitoring and early detection of ARDs with a view to sustain further reduction in exposure and risk of ARDs. Research is required to comprehensively assess or characterize amphiboles contaminants in locally produced and imported chrysotile asbestos being used in the AC manufacturing factories. The current conditions under which asbestos substitutes are produced and used are

not comparable to conditions under which chrysotile and other forms of asbestos were used in the past, hence a comparative approach in applying similar exposure controls, procedures and rules to chrysotile and its substitutes may be warranted.

On balance, banning can still be considered as a way to eliminate ARDs, but may not necessarily be the panacea for prevention of ARDs, in view of the fact that some other forms of mining such as diamond and gold mining have been associated with exposure to amphibole asbestos and that banning may imply use of substitutes reported to carry a health risk. Controlled use approach may perhaps still present possibilities of prevention of exposure to levels that may result in minimal risk to health impairment if effectively implemented as applied to a range of hazards with some success.

REFERENCES

Addison J & Davies LS (1990). Analysis of amphibole asbestos in chrysotile and other minerals. *The Annals of Occupational Hygiene*, 34(2), 159–175. <https://doi.org/10.1093/annhyg/34.2.159>.

AFSSET (2009b). Opinion of the French Agency for Environmental and Occupational Health Safety Relating to the proposed Occupational Exposure Limits of chemicals in the workplace. Asbestos fibres: assessment of the health effects and methods used to measure exposure levels in the workplace, Maisons-Alfort, France. Available online at <https://www.anses.fr/en/system/files/VLEP2005et9900RaEN.pdf>. Accessed July 20 2023.

Asbestos International Association (AIA). (1982). Reference Method for the Determination of Airborne Asbestos Fibre Concentrations at Workplaces by Light Microscopy (Membrane Filter Method).

Berry G & Newhouse ML (1983). Mortality of workers manufacturing friction materials using asbestos. *British Journal of Industrial Medicine*, 40(1), 1–7. <https://doi.org/10.1136/oem.40.1.1>.

Camus M (2001). A ban on asbestos must be based on a comparative risk assessment. *CMAJ: Canadian Medical Association journal = journal de l'Association medicale canadienne*, 164(4), 491–494. Available online at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC80777/>. (Accessed on 10 April 2022).

Creely KS, Cowie H, Van Tongeren M, Kromhout H, Tickner J & Cherrie JW (2007). Trends in inhalation exposure--a review of the data in the published scientific literature. *The Annals of Occupational Hygiene*, 51(8), 665–678. <https://doi.org/10.1093/annhyg/mem050>.

Cullen MR & Baloyi RS (1991). Chrysotile asbestos and health in Zimbabwe: I. Analysis of miners and millers compensated for asbestos-related diseases since independence (1980). *American Journal of Industrial Medicine*, 19(2), 161–169. <https://doi.org/10.1002/ajim.4700190204>.

Finkelstein MM (1983). Mortality among long-term employees of an Ontario asbestos-cement factory. *British Journal of Industrial Medicine*, 40(2), 138–144. <https://doi.org/10.1136/oem.40.2.138>.

Finkelstein MM (1995). Potential pitfall in using cumulative exposure in exposure-response relationships: demonstration and discussion. *American Journal of Industrial Medicine*, 28(1), 41–47. <https://doi.org/10.1002/ajim.4700280104>.

Gardner MJ, Winter PD, Pannett B & Powell CA (1986). Follow up study of workers manufacturing chrysotile asbestos cement products. *British Journal of Industrial Medicine*, 43(11), 726–732. <https://doi.org/10.1136/oem.43.11.726>.

Gibbs GW & Berry G (2008). Mesothelioma and asbestos. *Regulatory Toxicology and Pharmacology: RTP*, 52 (1 Suppl), S223–S231. <https://doi.org/10.1016/j.yrtph.2007.10.003>.

Government of South Africa (2020). Asbestos abatement Regulations. Dept of Labour, South Africa.

Hein M. J, Stayner LT, Lehman E & Dement JM (2007). Follow-up study of chrysotile textile workers: cohort mortality and exposure-response. *Occupational and Environmental Medicine*, 64(9), 616–625. <https://doi.org/10.1136/oem.2006.031005>.

Hagemeyer O, Otten H & Kraus T (2006). Asbestos consumption, asbestos exposure and asbestos-related occupational diseases in Germany. *International Archives of Occupational and Environmental Health*, 79(8), 613–620. <https://doi.org/10.1007/s00420-006-0091-x>.

Harrison PT, Levy LS, Patrick G, Pigott GH & Smith LL (1999). Comparative hazards of chrysotile asbestos and its substitutes: A European perspective. *Environmental Health Perspectives*, 107(8), 607–611. <https://doi.org/10.1289/ehp.99107607>.

HSE (2012). *The Control of Asbestos Regulations*, HSE, UK.

Hodgson JT & Darnton A (2000). The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. *The Annals of Occupational Hygiene*, 44(8), 565–601.

Infante PF, Schuman LD, Dement J & Huff J (1994). Fibrous glass and cancer. American Journal of Industrial Medicine, 26(4), 559–584. <https://doi.org/10.1002/ajim.4700260413>.

International Agency for Research on Cancer (IARC) (2012). IARC Monograph on Evaluation of Carcinogenic Risks to Human, Arsenic, Metals, Fibres and Dust: A review of human carcinogens Vol. 100C p.219 – 309. IARC/WHO.

International Chrysotile Association (ICA) (2019). Safety in the use of Chrysotile – Requirements and Achievements. Available online at https://chrysotileassociation.com/data/ICA_Chrysotile_Safety_Requirements_Achievements_v5F_LR.pdf. (Accessed 13 April 2022).

International Labour Organization (ILO) (1986). Convention No. 162 Convention - Safety in the Use of Asbestos, ILO, Geneva, Switzerland.

International Labour Organization (ILO)(1986). R172 – Asbestos Recommendation, 1986 (No. 172), ILO, Geneva, Switzerland.

International Labour Organization (ILO) (2006). Resolution concerning asbestos, 2006. Available online at https://www.ilo.org/safework/info/standards-and-instruments/WCMS_108556/lang--en/index.htm. Accessed 10 Feb 2021.

International Labour Organisation (1984). Safety in the use of asbestos: An ILO Code of practice, ILO, Geneva.

ISO (2019). Ambient air — Determination of asbestos fibres — Direct transfer transmission electron microscopy method. Available online at <https://cdn.standards.iteh.ai/samples/75577/014822d1b9c6479a83a594881b7ae7de/ISO-10312-2019.pdf>. Accessed July 19 2023.

ISO (2019a, ISO 13794:2019). Ambient air — Determination of asbestos fibres — Indirect-transfer transmission electron microscopy method.

ISO (2019b, ISO 14966:2019). Ambient air — Determination of numerical concentration of inorganic fibrous particles — Scanning electron microscopy method.

Jafari MJ, Karimi A, Mohammad Bardshahi A (2010). Prediction of cancer mortality by evaluation of asbestos fibers concentrations in an asbestos-cement products factory. Iran J. Environ. Health. Sci. Eng. Accessed November 20 2021.

https://www.researchgate.net/publication/47734184_Prediction_of_cancer_mortalityby_evaluation_of_asbestos_fibers_concentrations_in_an_asbestocement_products_factory/citation/download.

Kazan-Allen L (2022). Current Asbestos Bans. International Ban Asbestos Secretariat. Available online at http://ibasecretariat.org/alpha_ban_list.php. Accessed 21 July 2023.

Kohyama N, Shinohara Y & Suzuki Y (1996). Mineral phases and some re-examined characteristics of the International Union against Cancer standard asbestos samples. American Journal of Industrial Medicine, 30(5), 515–528. [https://doi.org/10.1002/\(SICI\)1097-0274\(199611\)30:5<515::AID-AJIM1>3.0.CO;2-S](https://doi.org/10.1002/(SICI)1097-0274(199611)30:5<515::AID-AJIM1>3.0.CO;2-S).

Liddell FD, McDonald AD & McDonald JC (1997). The 1891-1920 birth cohort of Quebec chrysotile miners and millers: development from 1904 and mortality to 1992. The Annals of Occupational Hygiene, 41(1), 13–36. [https://doi.org/10.1016/S0003-4878\(96\)00044-0](https://doi.org/10.1016/S0003-4878(96)00044-0).

McDonald AD, Case BW, Churg, A., Dufresne, A, Gibbs GW, Sébastien P & McDonald JC (1997). Mesothelioma in Quebec chrysotile miners and millers: epidemiology and aetiology. The Annals of Occupational Hgiene, 41(6), 707–719. [https://doi.org/10.1016/S0003-4878\(97\)00020-3.8](https://doi.org/10.1016/S0003-4878(97)00020-3.8)

Mirabelli D, Calisti R, Barone-Adesi F, Fornero E, Merletti F & Magnani C (2008). Excess of mesotheliomas after exposure to chrysotile in Balangero, Italy. Occupational and Environmental Medicine, 65(12), 815–819. <https://doi.org/10.1136/oem.2007.037689>.

Mine Safety and Health Administration (MSHA) USA Department of Labour (2005). Asbestos Exposure Limit; Final Rule. Department of Labor, Mine Safety and Health Administration. Available online at: <https://www.govinfo.gov/content/pkg/FR-2005-07-29/pdf/05-14510.pdf>. (Accessed 18 February 2022).

Mutetwa B, Moyo D & Brouwer D (2021). Trends in Airborne Chrysotile Asbestos Fibre Concentrations in Asbestos Cement Manufacturing Factories in Zimbabwe from 1996 to 2016. *International Journal of Environmental Research and Public Health*. 18 (20), 10755. <https://doi.org/10.3390/ijerph182010755>.

Mutetwa B, Moyo D & Brouwer D (2022). Job Exposure Matrix for Chrysotile Asbestos Fibre in the Asbestos Cement Manufacturing (ACM) Industry in Zimbabwe. *International Journal of Environmental Research and Public Health*, 19(5), 2680. <https://doi.org/10.3390/ijerph19052680>.

Newhouse ML & Sullivan KR (1989). A Mortality Study of Workers Manufacturing Friction Materials: 1941-86. *British Journal of Industrial Medicine*, 46(3), 176–179. <http://www.jstor.org/stable/27726756>.

NIOSH Manual of Analytical Methods (NMAM), 4th Edition (1994). Asbestos and other fibres by PCM. NMAM 7400. Issue 2.

NIOSH (1994b). Publication 7402. ASBESTOS by TEM. National Institute for Occupational Safety and Health. Available online at <https://www.cdc.gov/niosh/docs/2003-154/pdfs/7402.pdf>. Accessed July 19 2023.

Occupational Safety and Health Administration, US Department of Labor (1986). 51 FR 22 612 - Occupational exposure to asbestos, tremolite, anthophyllite and Actinolite. Final Rule. Available online at: <https://www.grsm.com/Templates/media/files/pdf/51%20fr%2022612.pdf>. (Accessed on 20 February 2022).

Ohlson CG & Hogstedt C (1985). Lung cancer among asbestos cement workers. A Swedish cohort study and a review. *British journal of Industrial Medicine*, 42(6), 397–402. <https://doi.org/10.1136/oem.42.6.397>.

Park D, Choi S, Ryu K, Park J & Paik N (2008). Trends in occupational asbestos exposure and asbestos consumption over recent decades in Korea. *International Journal of Occupational and Environmental health*, 14(1), 18–24. <https://doi.org/10.1179/oeh.2008.14.1.18>.

Piolatto G, Negri E, La Vecchia C, Pira E, Decarli A & Peto J (1990). An update of cancer mortality among chrysotile asbestos miners in Balangero, northern Italy. *British Journal of Industrial Medicine*, 47(12), 810–814. <https://doi.org/10.1136/oem.47.12.810>.

Pooley FD (1976). An examination of the fibrous mineral content of asbestos lung tissue from the Canadian chrysotile mining industry. *Environmental Research*, 12(3), 281–298. [https://doi.org/10.1016/0013-9351\(76\)90038-4](https://doi.org/10.1016/0013-9351(76)90038-4).

Paustenbach DJ, Finley BL, Lu ET, Brorby GP & Sheehan PJ (2004). Environmental and occupational health hazards associated with the presence of asbestos in brake linings and pads (1900 to present: A "state-of-the-art" review. *Journal of Toxicology and Environmental Health. Part B, Critical reviews*, 7(1), 25–80. <https://doi.org/10.1080/10937400490231494>.

Pira E, Donato F, Maida L & Discalzi G (2018). Exposure to asbestos: past, present and future. *Journal of Thoracic Disease*, 10 (Suppl 2), S237–S245. <https://doi.org/10.21037/jtd.2017.10.126>.

Reid A (2016). Review of the effectiveness of predictive models for mesothelioma to identify lessons for asbestos – related policy. Accessed March 16, 2018. <https://www.anzso.gov.au/resource-library/research/evidence-base-journal>. The Australia and New Zealand School of Government.

Rowlands N., Gibbs GW & McDonald AD (1982). Asbestos fibres in the lungs of chrysotile miners and millers--a preliminary report. *The Annals of Occupational Hygiene*, 26(1-4), 411–415.

Sichletidis L, Chloros D, Spyrtos D, Haidich AB, Fourkiotou I, Kakoura M & Patakas D (2009). Mortality from occupational exposure to relatively pure chrysotile: a 39-year study. *Respiration; International Review of Thoracic Diseases*, 78(1), 63–68. <https://doi.org/10.1159/000163443>.

Siemiatycki J. Should Canadian health care professionals support the call for a worldwide ban on asbestos? [editorial]. (2001) *CMAJ*; 164(4), 495-7. Available online at <https://www.cmaj.ca/content/cmaj/164/4/495.full.pdf>. (Accessed on 10 April 2022).

Smith TJ (1992). Occupational exposure and dose over time: limitations of cumulative exposure. *American Journal of Industrial Medicine*, 21(1), 35–51. <https://doi.org/10.1002/ajim.4700210107>.

Thomas HF, Benjamin IT, Elwood PC & Sweetnam PM (1982). Further follow-up study of workers from an asbestos cement factory. *British Journal of Industrial Medicine*, 39(3), 273–276. <https://doi.org/10.1136/oem.39.3.273>.

Tossavainen A, Kotilainen M, Takahashi K, Pan, G & Vanhala E (2001). Amphibole fibres in Chinese chrysotile asbestos. *The Annals of Occupational Hygiene*, 45(2), 145–152.

World Health Organization (WHO) (2014). Chrysotile Asbestos. Available online at; http://www.who.int/ipcs/assessment/public_health/chemicals_phc. (Accessed February 9 2020).

Weill H, Hughes J & Waggenpack C (1979). Influence of dose and fiber type on respiratory malignancy risk in asbestos cement manufacturing. *The American Review of Respiratory Disease*, 120(2), 345–354. <https://doi.org/10.1164/arrd.1979.120.2.345>.

Yamani ME, Boulanger G, Catelinois E and Paillat A (2012). Revision of French Occupational Exposure Limits of Asbestos and Recommendation of Measurement Method: Can the Dimensional Characteristics of the Asbestos Fibers (Long, Thin, Short) Be Taken Into Account? *Critical Reviews in Environmental Science and Technology*, 42, 1441-1484. Available online <https://www.researchgate.net/publication/254217084>. Accessed July 2023.

Yoshizumi K, Hori H, Satoh T & Higashi T (2001). The trend in airborne asbestos concentrations at plants manufacturing asbestos-containing products in Japan. *Industrial Health*, 39(2), 127–131. <https://doi.org/10.2486/indhealth.39.127>.

Zilaout H, Houba R & Kromhout H (2020). Temporal trends in respirable dust and respirable quartz concentrations within the European industrial minerals sector over a 15-year period (2002-2016). *Occupational and Environmental Medicine*, 77(4), 268–275. <https://doi.org/10.1136/oemed-2019-106074>.

CHAPTER 6

**Trends in Airborne Chrysotile Asbestos
Fibre Concentrations in Asbestos
Cement Manufacturing Factories in
Zimbabwe from 1996 to 2016.**



Article

Trends in Airborne Chrysotile Asbestos Fibre Concentrations in Asbestos Cement Manufacturing Factories in Zimbabwe from 1996 to 2016

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Abstract: Zimbabwe has two major factories that have been manufacturing chrysotile asbestos cement products since the 1940s. Exposure monitoring of airborne fibres has been ongoing since the early 1990s. This study examines trends in personal exposure chrysotile asbestos fibre concentrations for the period 1996–2016. Close to 3000 historical personal exposure measurements extracted from paper records in the two factories were analysed for trends in exposure. Exposure over time was characterised according to three time periods and calendar years. Mean personal exposure chrysotile asbestos fibre concentrations generally showed a downward trend over the years in both factories. Exposure data showed that over the observed period 57% and 50% of mean personal exposure chrysotile asbestos fibre concentrations in the Harare and Bulawayo factories, respectively, were above the OEL, with overexposure being exhibited before 2008. Overall, personal exposure asbestos fibre concentrations in the factories dropped from 0.15 f/mL in 1996 to 0.05–0.06 f/mL in 2016—a decrease of 60–67%. These results can be used in future epidemiological studies, and in predicting the occurrence of asbestos-related diseases in Zimbabwe.

Keywords: exposure; chrysotile asbestos; trends; personal exposure; airborne asbestos fibre concentration



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1. Introduction

Asbestos is a generic term for a group of naturally occurring silicates that principally include the serpentine variety (white chrysotile asbestos) and the amphibole variety, consisting of crocidolite (blue asbestos) and amosite (brown asbestos) [1]. Asbestos exposure has drawn much international, regional, and national attention, as it presents significant public and occupational health concerns. All asbestos types are known to cause asbestos-related disease [1–3].

The World Health Organization reports that 125 million people worldwide are exposed to asbestos at the workplace, with 107,000 people succumbing to asbestos-related diseases annually [2]. Although amphibole production has all but ceased worldwide, chrysotile asbestos continues to be produced and used in some countries. While the production and use of asbestos in most developed countries has declined in recent years due to health concerns, and the subsequent ban of asbestos-containing products, there continues to be extensive production, sale, and use of chrysotile in South and Central America, Asia, and Africa [2,4]. Russia is the world's leading producer of chrysotile asbestos; others include China, Kazakhstan, Brazil, and India, with production at Zimbabwe's chrysotile mines stalling in 2010 due to economic challenges. Currently, there are efforts to resuscitate the mining of chrysotile asbestos, with tailings dumps being harnessed to extract fibres

for the two chrysotile asbestos cement manufacturing factories in the cities of Harare and Bulawayo.

Zimbabwe has long been one of Africa's major producers of chrysotile asbestos [5,6]. During the 1970s, production averaged 200,000 metric tonnes per annum, rising to a peak of 259,000 tonnes in 1979. However, production declined to 100,000 tonnes per annum for the period 2004–2007, and reduced drastically during the hyperinflation period of 2008 such that, by 2010, only 2400 tonnes were reported to have been produced [5]. Important chrysotile products that are produced in Zimbabwe include reinforced chrysotile asbestos roofing sheets and tiles, water pipes, heat-resistant or fire-resistant insulation materials, and packings and gaskets in the vehicles industry. The two chrysotile asbestos mines—the Shabanie and Mashava mines—had a combined production capacity of 140,000 metric tonnes of chrysotile asbestos in the 1980s and 1990s; 90% of this product was exported, with 10% consumed by the local chrysotile asbestos cement manufacturing industry [7].

From the early 1990s, ~7000 workers were engaged in mining and milling at the two major mines, with ~4000 engaged in the manufacturing of chrysotile asbestos products [8]. During the same period, it was reported that 40,000–45,000 people lived within a few kilometres of the mills and mines, and a large proportion of the population lived and worked in buildings with chrysotile asbestos [8]. Zimbabwe has two major factories that manufacture chrysotile asbestos products, and which have been the main users of chrysotile asbestos since their establishment in the 1940s and 1950s.

A limited number of papers have reported temporal trends in personal exposure chrysotile asbestos fibre concentrations in chrysotile asbestos cement manufacturing plants. In Germany, it was observed that there was a decrease in asbestos dust concentrations for the period 1950 to 1990, which was attributed to the rapid decline in the use of asbestos since 1980, when regulations and bans on the production, use, and placement of asbestos on the market were introduced [9,10]. Furthermore, Coble et al. reported that there was a 5% decline in asbestos exposure observed during compliance inspections of pulp and paper facilities [11]. In another study of exposure–response relationships for asbestos-related diseases, Finkelstein reported declines in exposure for the years 1949, 1969, and 1979, with estimates recorded as 40 f/mL, 20 f/mL, and 0.2 f/mL for willow operators, 16 f/mL, 8 f/mL, and 0.5 f/mL for forming machine operators, and 8 f/mL, 4 f/mL, and 0.3 f/mL for lathe operators for 1949, 1969, and 1979, respectively [12]. Additionally, declines in asbestos exposure were reported in asbestos cement plants in Sweden [13], South Africa [14], Japan [15], Yugoslavia, Poland, and Latvia [16], and the USA [17]. These studies show that exposure during the earlier years was high—particularly during the 1970s—compared to the period 1990 to 2000s.

To control and reduce exposure, various regulatory agencies dealing with occupational safety and health (OSH) have established occupational exposure limits (OELs) or threshold limit values (TLVs) for airborne asbestos fibres. It is expected that workers exposed repeatedly to levels at or below the OEL are at low risk of developing adverse health effects [18,19].

OELs have been declining over the years in response to new information on exposure–response effects experienced by workers and/or experimental animals, thereby influencing the exposure levels observed in various asbestos workplace settings. In the USA, OELs have moved from as high as 12 f/mL in the early 1970s, to 2 f/mL in the mid-1970s, 0.2 f/mL in the mid-1980s and, from 1995 to date, the OEL has been set at 0.1 f/mL. Today, most countries have aligned their asbestos OEL to that of the USA's OSHA or ACGIH, which has been set at 0.1 f/mL [18,20].

In Zimbabwe, there is no specific legislative instrument that governs the management and enforcement of an OEL for chrysotile asbestos. Management is generally through non-specific regulations, such as the Statutory Instrument 68 of 1990 on Accident Prevention and Workers Compensation [21]. Moreover, there is no statutory OEL for chrysotile asbestos fibres, save for guidelines on OELs published by the National Social Security Authority (NSSA)—Occupational Safety and Health Division, which has set the limit at

0.1 f/mL for all forms of asbestos fibres [22]. Hence, the current OEL for chrysotile asbestos fibres in Zimbabwe is 0.1 f/mL. However, this OEL is not a statutory limit but, rather, a recommended limit, which is expected to be part of an envisaged asbestos regulation. In the absence of specific guidance on the management of chrysotile asbestos exposure, the chrysotile asbestos cement manufacturing industry has developed its own occupational exposure monitoring programme, where personal and static exposure sampling data have been collected since the 1980s, and more structured in the 1990s to the mid-2000s through to 2016. These data provide an opportunity to understand the extent of exposure to asbestos fibres in the Zimbabwean chrysotile asbestos cement manufacturing industry over the years.

2. Materials and Methods

2.1. Study Design

This secondary data analysis study was carried out in the two chrysotile asbestos cement manufacturing (ACM) factories situated in Harare and Bulawayo. The original data of personal exposure chrysotile asbestos fibre measurements were provided from the company factories to the authors following the company agreeing to access the records of the personal exposure measurements data. The data were extracted from the paper records of personal chrysotile asbestos fibre exposure measurements taken in the factories by company personnel. Data recorded from 1996 to 2016 by the two chrysotile asbestos cement factories were analysed for trends.

2.2. Collection of Measurements

Operational areas for which personal exposure data were available were cutting saws, fettling table, kollergang, moulded goods, ground hard waste, laundry room, sheeting planter mixer, lathe machining of pipes, and multi-cutter operations (Table 1). Generally, exposure data were collected once every month, though in some years, measurements depended on the availability of plant operations, sampling equipment, and consumables. Table S1 (Supplementary Materials) shows the number of measurements collected in the various operational areas.

2.3. Method of Chrysotile Asbestos Fibre Measurements

The written asbestos method on file in the factories showed that measurements of airborne asbestos fibre concentrations followed the standard method of the Asbestos International Association (AIA) Recommended Technical Membrane Filter Reference Method (AIA, 1982) [23]. As part of adherence to the AIA technical reference method, field blank filter samples were reported and used as controls as part of the quality control programme. In summary, a personal sampling pump set at a 1 L/min flowrate was connected to a sampling train, consisting of plastic tubing and a sample holder (cowl) with a 25 mm membrane filter. The whole sampling train of the pump, tubing, sample holder, and filter was hooked to a worker. The pump was then switched on, and sampling took place over a period of around four hours, after which the filters were removed, placed at the appropriate labelled slides, and treated with acetone vapour to clear. Using a hypodermic syringe, a drop of triacetin was placed onto the acetone-cleared filters and covered with a cover slip. The treated filters on the slides were stored for 24 h, after which counting of the fibres took place using a phase-contrast microscope. The limit of detection (LoD) for the method was 0.02 f/mL.

Table 1. Description of tasks and operational areas used in analysing personal chrysotile fibre measurements in the Harare and Bulawayo factories.

Task/Operational Area	Description of Task
Saw Cutting operations	Operator cuts chrysotile asbestos cement sheets and fascia boards to size.
Fettling table operations	Scrapping of unwanted chrysotile asbestos cement matter on finished moulded goods such as ridges, garden ware, and polishing using sandpaper by operators.
Kollergang operations	Operator opens ~50 kg chrysotile asbestos bags using a knife and loads the fibre into the process machine.
Moulded goods table operations	Operators mould various goods under wet conditions, such as ridges, or garden ware goods such as flower vessels.
Ground hard waste operations	Operator feeds chrysotile asbestos cement waste material into grinder machine for recycling back into process.
Pipe section—lathe machining of chrysotile asbestos pipe joints	Operators operate lathe machines such as sewer lathe, Lang lathe, broad bend lathe, and Geminis lathe machines by machining joints so that they are ready for coupling pipes.
Pipe section—lathe machining of full-length chrysotile asbestos sewer/water pipes.	Operator operates lathe machines—namely, Faben, Voith, and O&S lathe machines—to prepare full-length pipe for a joint, and polish joint with sandpaper.
Pipe section—multi-cutter operations	Cutting full-length pipes into collars used for coupling pipes using a multi-cutter machine.

These tasks were considered to have potential for highest exposure to airborne chrysotile asbestos.

The period of 1996 to 2016 was divided into three time periods: 1996–2000, 2001–2008, and 2009–2016. During 1996–2000, the chrysotile asbestos cement manufacturing industry was in a self-regulatory mode with respect to safety and health standards, and the ACM manufacturing industry had an active exposure monitoring programme in light of the call to phase out the use of chrysotile asbestos. During these early years of the 1990s, the asbestos industry set its own chrysotile exposure limit of 0.2 f/mL and an action limit of 0.15 f/mL in the absence of a national statutory exposure limit on chrysotile asbestos. From 2001 to 2008, the chrysotile exposure monitoring program continued; however, there was a sharp decline in economic activity nationally. Monitoring of exposure continued for the period 2009 to 2016 against the backdrop of improved retooling of the industry and change from the use of locally produced asbestos to largely imported fibre.

2.4. Quality Assurance and Reliability of the Chrysotile Asbestos Fibre Exposure Data

From the early 1990s to 2011, correspondences at the two factories showed that the factories participated in an inter-laboratory quality assurance and control fibre-counting programme, which involved laboratories at the two chrysotile mines in Zimbabwe, another chrysotile asbestos cement plant laboratory in Zambia, the Department of Minerals and Energy in South Africa, and a French laboratory in Paris, with a view to improving the quality and reliability of exposure measurements. Additionally, as part of an oversight programme on quality control, in 2008, the Institute of Occupational Medicine (IOM), UK, was invited to conduct an independent evaluation of levels of chrysotile asbestos fibres in the ambient air around various work processes [24]. The independent evaluation of levels of chrysotile asbestos fibres in the two factories provided a good measure of reliability and assurance to the personal exposure chrysotile fibre concentrations generated by the company over the years, and subsequently used in the study described in this paper. The IOM reported that personal and static samplers were being correctly mounted on the workers, with proper positioning of sample holders in the workers' breathing zones. They

further reported that the company's analytical laboratory was adequately equipped for the collection and measurement of airborne chrysotile asbestos fibres, and that there was good consistency between the IOM and the company's calibration equipment for calibration flows of the sampling pumps [24]. These efforts demonstrated that the data used in this study provided a measure of reliability of the exposure values obtained in the factories over the years.

2.5. Data Description and Classification of Measurements

Approximately 3000 personal exposure measurements were collected in the operational areas (Table S1 in the Supplementary Materials) over the 21-year period in the two factories. Personal sampling points were classified into six production areas for both the Harare and Bulawayo factories; a further subclassification was made for the pipe section of the Bulawayo factory. For the two main ACM factories, personal sampling data were classified as described in Table 1. However, for laundry room (28 values) and sheeting plant mixer operations (30 values), actual measured values for the Bulawayo factory for the period 1996 to 2016 were too few and, thus, were not considered in the analysis. Additionally, for the pipe section, personal sampling data were classified into three broad areas—namely, (a) pipe plant operations—lathe machining asbestos pipe joints, (b) pipe plant—lathe machining of full-length asbestos sewer and water pipes, and (c) multi-cutter, where cutting of full-length pipes into collars for coupling of pipes was carried out. For the cutting saw operations, measurements were taken at ~4–6 saws per month. Personal sampling data for each broad operational area were averaged for each month (Table S2 of the Supplementary Materials).

These tasks were considered to have the highest potential for exposure to airborne chrysotile asbestos.

2.6. Statistical Analysis

Data analysis was conducted using IBM SPSS version 26. For analysis, monthly averaged personal exposure levels for the factories were used. Mean personal airborne chrysotile fibre concentrations were analysed per operational area per factory, and trends in airborne fibre concentrations over the years were displayed graphically.

2.7. Ethics

This study was approved by the University of the Witwatersrand Human Research Ethics Committee (clearance certificate number M181157) and the Medical Research Council of Zimbabwe (MRCZ) (approval number MRCZ/A/2445).

3. Results

There were 2890 personal samples collected over the 21-year period in the different operational areas of the two chrysotile cement manufacturing factories (Table S1 in the Supplementary Materials), and 1663 monthly averaged personal chrysotile asbestos fibre concentrations (Table S2). The Harare factory had the greater proportion (63.9%) of monthly averaged concentrations. Tables 2 and 3 show the summary statistics of the personal chrysotile asbestos fibre concentrations for the Harare and Bulawayo factories, respectively. Small variations in airborne chrysotile asbestos fibre concentrations were recorded at the fettling table operations ($SD \pm 0.02$) for the Harare factory. The other operational areas in both factories showed variation in airborne chrysotile asbestos fibre concentrations, ranging from 0.01 to 0.30 f/mL. Fettling table operations in both factories (Harare 76.2%, Bulawayo 84.3%) and multi-cutter operations in Bulawayo (81.3%) had the highest proportion of airborne concentrations above the OEL. Overall, 60.3% and 58.6% of measurements in the Harare and Bulawayo factories, respectively, exceeded the OEL.

Table 2. Mean airborne personal chrysotile asbestos fibre concentrations (f/mL) for various operations/tasks in the Harare chrysotile cement manufacturing factory.

Job/Task	N	Mean	SD	Range		% >0.1 f/mL *
				Min	Max	
Cutting saw operator	(225)	0.12	0.05	0.03	0.24	60.9
Fettling table operator	(126)	0.12	0.02	0.05	0.19	76.2
Moulded goods operator	(192)	0.10	0.04	0.03	0.20	46.4
Kollergang operator	(203)	0.10	0.04	0.04	0.20	54.2
Ground hard waste operator	(168)	0.12	0.04	0.02	0.22	63.7
Laundry room operator	(149)	0.12	0.04	0.03	0.21	73.8
Overall factory	(1063)	0.11	0.04	0.04	0.18	60.3

N: actual number of personal samples, 1996–2016; *: % measurements greater than OEL of 0.1 f/mL.

Table 3. Mean airborne personal chrysotile fibre concentrations (f/mL) for various operations/tasks in the Bulawayo chrysotile cement manufacturing factory.

Job/Task	N	Mean	SD	Range		% >0.1 f/mL *
				Min	Max	
Cutting saw operator	(113)	0.13	0.04	0.01	0.24	75.2
Fettling table operator	(51)	0.16	0.06	0.06	0.30	84.3
Kollergang operator	(111)	0.11	0.04	0.03	0.24	60.4
Ground hard waste operator	(64)	0.12	0.04	0.04	0.24	54.7
Pipe joints operator	(99)	0.12	0.03	0.04	0.30	69.7
Full length pipe operator	(97)	0.12	0.03	0.04	0.30	67.0
Multi-cutter operator	(64)	0.12	0.03	0.05	0.20	81.3
Overall factory	(600)	0.11	0.04	0.03	0.22	58.6

N: actual number of personal chrysotile asbestos samples, 1996–2016; *: % measurements greater than OEL of 0.1 f/mL.

Figures 1 and 2 show changes in the mean personal exposure chrysotile asbestos fibre concentrations at the Harare and Bulawayo factories, respectively, from 1996 to 2016. Annual personal mean exposure levels generally showed a downward trend over the years, with high levels recorded from 1996 to 2001 for the Harare factory, and from 1996 to 2007 for both factories, in almost all operational areas. Personal exposure concentrations below the OEL began after 2008, except for fettling table operations in the Harare factory and multi-cutter operations in the Bulawayo factory. For the Harare factory, cutting saw operations, ground hard waste operations, laundry rooms, and kollergang operations exhibited high levels of personal exposure chrysotile fibre concentrations in or before 2007, with a considerable decline thereafter. The Bulawayo factory also showed a similar trend in all operational areas in which high personal exposure concentrations were observed in or before 2008.

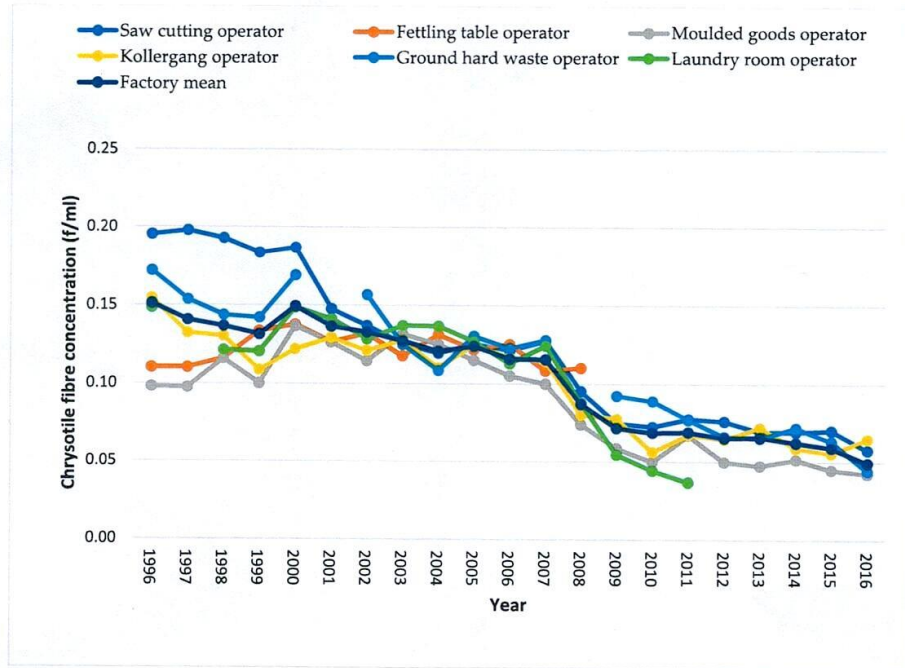


Figure 1. Changes in personal exposure chrysotile asbestos fibre concentrations from 1996 to 2016 for the Harare factory.

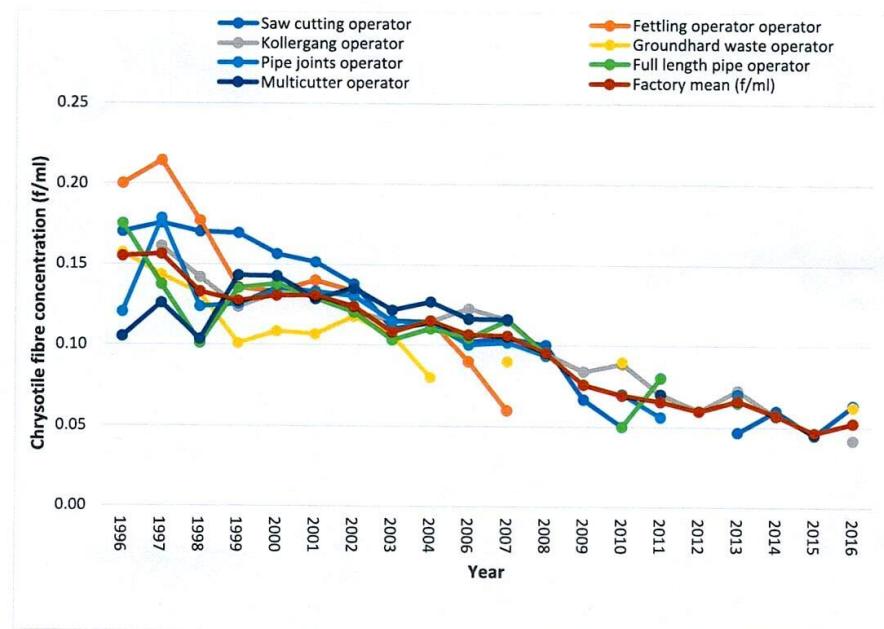


Figure 2. Changes in personal exposure chrysotile asbestos fibre concentrations from 1996 to 2016 for the Bulawayo factory.

Tables 4 and 5 show the mean personal exposure chrysotile fibre concentrations by time- period for the Harare and Bulawayo factories, respectively.

Table 4. Mean personal exposure chrysotile asbestos fibre concentrations (f/mL) by period between 1996 and 2016: Harare factory.

Job/Task	Time Period	N	Mean	SD	95% CI		Range	
					LB	UB	Min	Max
Saw cutting operator	1996–2000	60	0.19	0.01	0.19	0.19	0.16	0.24
	2001–2008	88	0.13	0.02	0.12	0.13	0.08	0.18
	2009–2016	77	0.07	0.02	0.07	0.08	0.03	0.11
Fettling table operator	1996–2000	53	0.12	0.04	0.11	0.13	0.05	0.19
	2001–2008	73	0.12	0.02	0.12	0.13	0.04	0.20
	2009–2016	nil						
Moulded goods operator	1996–2000	58	0.11	0.04	0.10	0.12	0.04	0.20
	2001–2008	82	0.11	0.04	0.11	0.12	0.03	0.18
	2009–2016	52	0.05	0.01	0.05	0.06	0.03	0.08
Kollergang operator	1996–2000	58	0.13	0.03	0.12	0.14	0.05	0.20
	2001–2008	81	0.12	0.02	0.11	0.12	0.04	0.16
	2009–2016	64	0.07	0.02	0.06	0.07	0.04	0.11
Ground hard waste operator	1996–2000	57	0.16	0.03	0.15	0.16	0.08	0.22
	2001–2008	56	0.13	0.03	0.14	0.13	0.03	0.20
	2009–2016	55	0.07	0.02	0.07	0.08	0.02	0.17
Laundry room operator	1996–2000	47	0.13	0.03	0.12	0.14	0.06	0.20
	2001–2008	87	0.13	0.02	0.12	0.13	0.06	0.21
	2009–2016	15	0.05	0.01	0.04	0.05	0.03	0.07
Overall factory	1996–2000	60	0.14	0.02	0.14	0.15	0.11	0.18
	2001–2008	92	0.12	0.02	0.12	0.12	0.07	0.18
	2009–2016	80	0.06	0.01	0.06	0.07	0.04	0.12

N: number of personal chrysotile asbestos fibre samples; SD: standard deviation; 95% CI: 95% confidence interval; LB: lower bound; UB: upper bound; Min: minimum; Max: maximum.

Table 5. Mean personal exposure chrysotile asbestos fibre concentrations (f/mL) by period between 1996 and 2016: Bulawayo factory.

Job/Task	Time Period	N	Mean	SD	95% CI		Range	
					LB	UB	Min	Max
Cutting saw operator	1996–2000	50	0.17	0.02	0.16	0.18	0.12	0.24
	2001–2008	49	0.12	0.02	0.11	0.12	0.09	0.16
	2009–2016	14	0.06	0.02	0.05	0.07	0.01	0.08
Fettling table operator	1996–2000	40	0.17	0.06	0.16	0.19	0.07	0.30
	2001–2008	11	0.12	0.03	0.10	0.14	0.06	0.15
	2009–2016							
Kollergang	1996–2000	36	0.14	0.03	0.13	0.15	0.08	0.24
	2001–2008	42	0.12	0.01	0.11	0.12	0.08	0.14
	2009–2016	33	0.07	0.03	0.06	0.08	0.03	0.18
Ground hard waste	1996–2000	44	0.13	0.04	0.11	0.14	0.07	0.24
	2001–2008	15	0.11	0.04	0.10	0.11	0.08	0.13
	2009–2016	5	0.07	0.02	0.05	0.09	0.04	0.09
Pipe joints	1996–2000	44	0.13	0.04	0.12	0.14	0.06	0.30
	2001–2008	46	0.11	0.01	0.11	0.12	0.08	0.15
	2009–2016	9	0.06	0.01	0.05	0.07	0.04	0.08
Full length pipe operator	1996–2000	43	0.13	0.04	0.12	0.14	0.06	0.27
	2001–2008	45	0.11	0.01	0.11	0.11	0.07	0.14
	2009–2016	9	0.07	0.02	0.05	0.08	0.04	0.09
Multi-cutter operator	1996–2000	26	0.13	0.04	0.11	0.14	0.05	0.20
	2001–2008	36	0.12	0.01	0.12	0.13	0.10	0.14
	2009–2016	2	0.07	0.03	0.02	0.32	0.05	0.20
Overall factory	1996–2000	51	0.14	0.03	0.13	0.15	0.09	0.22
	2001–2008	50	0.11	0.01	0.12	0.11	0.09	0.15
	2009–2016	45	0.02	0.01	0.06	0.07	0.03	0.10

N: number of personal chrysotile asbestos fibre samples; SD: standard deviation; 95% CI: 95% confidence interval; LB: lower bound; UB: upper bound; Min: minimum; Max: maximum.

For the Harare factory, the overall percentage decline over the time periods was 14.3% from the time period 1996–2000 to 2001–2008, and a 50% decline was registered from the period 2001–2008 to 2009–2016. The Bulawayo factory showed a generally similar pattern to that of the Harare factory, with an overall factory exposure decline of 21.4% between the periods 1996–2000 and 2001–2008, while a 45.5% decline in personal exposure chrysotile fibre concentrations was registered between the time periods 2001–2008 and 2009–2016. Overall, during the period 1996–2000, exposure levels ranged from 0.11–0.18 f/mL, compared to 0.04–0.12 f/mL personal exposure chrysotile fibre concentrations recorded for the period 2009–2016 for the Harare factory. Similarly, for the Bulawayo factory, personal exposure chrysotile fibre concentrations ranged from 0.09–0.22 f/mL during the earlier years of 1996–2000, compared to 0.03–0.10 f/mL recorded for the period 2009–2016.

Observations

Observations made during site visits and during data gathering at the factories noted that manufacturing equipment and ventilation systems were generally in good condition. Respiratory protective equipment such as dust masks was provided, and cleaning of floors and other operations was carried out under wet conditions. These observations were made to check the state of the manufacturing equipment, and whether good work practices were being followed.

Furthermore, personnel in the factories indicated that the equipment in use has been in operation since the 1990s and the 2000s, and that such equipment was subject to regular maintenance.

Essentially, personal sampling during the period 1996–2016 took place—and still takes place—in areas where raw chrysotile fibres or asbestos products are processed and handled.

Raw fibre is supplied in plastic-wrapped bags and stored on site. The bags are moved to the fibre preparation (or fibre treatment) area, where they are loaded into fibre preparation machines called kollergang. The fibre is manually tipped into the kollergang machine following opening of the bags with a knife. This area provides much scope for fibre release in the workplace when bags are opened and tipped into the kollergang machines and, hence, may explain the rather elevated personal exposure fibre concentrations observed at the kollergang operational area. However, some form of ventilation is provided that produces a positive or inward draft into the kollergang machines at the fibre entry point.

A slurry of fibre cement is processed through a continuous flow process to form chrysotile asbestos cement sheets, and this is performed through a controlled computerized system while an operator is in the control cabin. The corrugated chrysotile cement sheets are then lifted from the production line by an automated machine, stacked on pallets, and taken by forklifts to areas such as the sawing/cutting areas.

Other ancillary operations include the sawing or cutting of chrysotile cement sheets and facia boards to size using powered saws equipped with local exhaust ventilation. The moulded goods section, where various goods are moulded under wet conditions, is generally labour intensive. Furthermore, in Bulawayo, lathe machines are used to cut asbestos pipes, prepare joints and couplings, and polish products. Discussions with personnel at the two factories indicated that workers had always been—and were still being—provided with masks, and that they were monitored to check whether they followed the good work practices set by the factories.

It was also noted that the ACM factories have followed international best practices in manufacturing, occupational safety and health, and environmental management systems throughout the period 1996–2016. This has led to ACM factories being accredited to ISO 9001, ISO14001, and OSHAS 18001 (now ISO 45001). Such accreditations suggests that the ACM factories endeavour to provide a safe work environment. The factories have been certified to the international standards indicated above since 2001. The period 1996–2000 was characterized by a build-up towards certification as reported by the company personnel in the ACM entities; hence, because of pursuing such standards, this may have contributed towards the downward trends in chrysotile asbestos fibre concentrations over

the years. While there was a general decline in industrial production across the country over the period 2001–2008—and, in particular, an accelerated decline from 2006 to 2008, due to hyperinflation—the good occupational safety and health framework may also have been a contributing factor to the downward trend as the ACM factories strived for continuous improvement in their business processes.

4. Discussion

This study constitutes the single largest personal exposure chrysotile asbestos fibre concentration dataset in Zimbabwe. The general decline in exposure over time from 1996 to 2016 suggests good occupational safety and health (OSH) framework implementation by the two factories over the years, with the years after 2008 showing much lower concentration levels below the OEL. Decreasing trends in personal exposure chrysotile asbestos fibre concentrations may also be viewed from the perspective that industry was responding to the anticipated lowering of the airborne chrysotile fibre OEL as a result of increased calls to ban all forms of asbestos, triggering the scaling up of exposure controls in the factories. However, at cutting saw operational areas, personal exposure chrysotile fibre concentration levels suggest high-risk activity in both factories during the earlier years of 1996–2008, perhaps due to weak controls, as fibre concentrations considerably exceeded the OEL.

During the period 1996–2000, economic activity was generally high, and this may have contributed to the high personal exposure chrysotile asbestos fibre concentrations observed during these early years. Economic instability, however, set in during the period 2001–2008, which resulted in a significant decline in industrial production across all industries, which may also have contributed to decreases in personal exposure chrysotile asbestos fibre concentrations. Although production rates were not available from the asbestos cement manufacturing factories, it is widely known that production across all industries—including the ACM factories—in Zimbabwe was seriously affected by hyperinflation during the period 2001–2008, such that in 2008 there was almost an economic standstill situation in the country, which could thus have contributed to the observed decline in personal exposure chrysotile asbestos fibre concentrations. Zilaout et al. (2020), in a study on trends in respirable dust and respirable quartz concentrations in the European industrial minerals sector over a 15-year period, cited macroeconomic developments as affecting trends, and postulated that recession may have contributed to the downward trends observed [25]. From 2009 to 2016, there was general stability in the economy, with most companies back to optimal operation. Retooling of operations and systems was made easier as the country adopted a multicurrency system, with the US dollar being the main currency of use. This may have contributed to improved OSH programmes which, in turn, could also possibly have contributed to further decline in personal exposure chrysotile asbestos fibre concentrations during this period.

Despite the overall decline in occupational personal exposure chrysotile asbestos fibre concentrations based on an occupational exposure limit of 0.1 f/mL, descriptive statistics for both factories suggest that there was overexposure among those exposed—especially during the period 1996–2000. The Harare factory shows that 60.3% of personal exposure chrysotile asbestos fibre concentrations exceeded the OEL, while for the Bulawayo factory, 58.6% of personal exposure chrysotile asbestos fibre concentrations exceeded the OEL (Tables 2 and 3, respectively). The exposure limit of 0.2 f/mL adopted by the chrysotile asbestos industry in Zimbabwe was consistent with the threshold limit value (TLV) set by the American Conference of Governmental Industrial Hygienists (ACGIH), which adopted a TLV of 0.2 f/mL during the early-to-mid-1990s. During the 1990s, mean personal exposure chrysotile asbestos fibre concentrations at various operational areas did not exceed this TLV and, as such, the industry was of the view that they were generally making the necessary efforts to prevent overexposure. However, with new information and knowledge on the risk profile for chrysotile asbestos exposure since the mid-1990s to date, personal exposure chrysotile asbestos fibre concentrations could be considered as presenting a risk of disease if evaluated against the OEL of 0.1 f/mL.

Although observations made during site visits showed that the general state of the factories was good, the Bulawayo factory appeared to show better housekeeping than the Harare factory, and it may be assumed that the Bulawayo factory may have been maintaining such good housekeeping, thus possibly contributing to fewer chrysotile asbestos fibre concentrations above the OEL compared to the Harare factory. Additionally, the Harare factory had always had a greater proportion of workers over the years compared to the Bulawayo factory, suggesting more activity and handling of chrysotile asbestos and associated products, which could thus have also contributed to more personal chrysotile asbestos fibre concentrations above the OEL compared to the Bulawayo factory.

The overall downward trend in personal exposure fibre concentrations observed over the years 1996–2016 is consistent with patterns observed in other places where chrysotile asbestos cement products were being produced [26,27]. In a study where 2089 asbestos exposure datasets were put together from 1995 to 2006, asbestos exposure levels were shown to decrease from 0.92 f/mL in 1996 to 0.60 f/mL in 1997, to 0.19 f/mL in 1998, and to 0.06 f/mL in 1999, and this decrease was considered as possibly being due to enforcement of legislation and the banning of the use of amosite and crocidolite. The mean asbestos fibre concentration in the asbestos cement plants was recorded as 0.31 f/mL [25], whereas in the Zimbabwe factories, the overall mean personal exposure chrysotile asbestos fibre concentration was 0.11 f/mL. However, specific exposure patterns in the chrysotile asbestos cement plants worldwide became limited after 2000, as most countries banned the use, handling, and production of chrysotile asbestos [20]. Nevertheless, in Germany, there was a steady decline in asbestos exposure between 1950 and 1990 in textile, cement brake pads, and drilling/sawing operations [1,2,9].

In this study, personal exposure chrysotile asbestos fibre concentrations in the chrysotile cement asbestos pipe manufacturing industry ranged from 0.03 to 0.30 f/mL. In Thailand, breathing zone asbestos concentrations in cement pipe production ranged from 0.12–2.13 f/mL between 1987 and 1988 [2,28]. Thus, the declining pattern in personal exposure asbestos fibre concentration estimates over the years in Thailand is similar to the declines in concentrations observed in this study. Creely et al., in a review on trends in inhalation exposure, also reported decreases in respirable fibre levels in various workplace settings involving possible exposure to asbestos, where asbestos fibre concentrations were reported to decline by as much as 32% per annum. Regulatory intervention, good occupational hygiene practices, and improved ventilation were factors cited as contributing to decreasing temporal trends [10].

In another study by Albin et al. in a Swedish asbestos cement factory, the authors reported asbestos fibre concentrations declining from 1.5–6.3 f/mL in 1956 to 0.3–5 f/mL in 1969 and 0.5–1.7 f/mL in 1975 [13]. Higashi et al. evaluated personal exposure at two Japanese manufacturing and processing plants producing asbestos-containing products such as roofing sheets, and reported that asbestos fibre concentrations ranged from 0.05 to 0.78 f/mL [15]. In this study, overall, during the earlier years of 1996–2000, personal exposure chrysotile asbestos fibre concentrations ranged from 0.11 to 0.18 f/mL and 0.09 to 0.22 f/mL in the Harare and Bulawayo factories, respectively. Additionally, declines in asbestos fibre concentrations over the years in various workplace settings have been reported [29,30]. These decreasing trends in asbestos fibre concentrations over time, as observed and reported in the various studies mentioned above, are also consistent with the declines observed in personal exposure chrysotile fibre concentrations in the chrysotile asbestos cement manufacturing industry in Zimbabwe.

Strengths and Limitations of the Study

The considerable large amount of airborne chrysotile fibre concentration data collected over a long period of time—spanning two decades—using recognized standard asbestos methods and equipment, and being unique in Zimbabwe, offers a key point of strength to this study. Additionally, a collection of such chrysotile exposure data could be used as a basis for future epidemiological studies. However, within each factory, there were

years for which measurements were not available in various operational areas in each calendar year, resulting in fibre concentration data gaps in some years and, thus, affecting fibre concentration patterns over the years. Furthermore, production rates—which may have provided further insights in personal exposure fibre concentration patterns for the period 1996–2016—were not available from the factories. However, notwithstanding these limitations, the considerable amount of data provides insights into changes in personal exposure chrysotile asbestos fibre concentrations over time, serving as input data for future research.

5. Conclusions

The personal exposure chrysotile asbestos fibre measurements collected over two decades—from 1996 to 2016—in key operational areas of the factories aided in a comprehensive analysis of trends in personal exposure chrysotile asbestos fibre concentrations in the asbestos cement manufacturing industry in Zimbabwe. Personal exposure chrysotile fibre concentration data in the two factories show a downward trend over the years, with high concentrations being exhibited in or before 2008. These findings are consistent with the downwards trends over time observed in other studies. The Harare factory showed more overexposure than the Bulawayo factory. Wet processes should continue to be applied in order to continuously sustain reductions in levels of exposure to airborne chrysotile asbestos fibres in the workplace. These results can aid in future epidemiological studies, serve as a basis for the establishment of a job-exposure matrix for similar workplace settings, and assist in predicting the possible occurrence of asbestos-related diseases in Zimbabwe.

Supplementary Materials: The following are available online at <https://www.mdpi.com/article/10.3390/ijerph182010755/s1>: (a) One-way ANOVA output for various operational areas for the Harare and Bulawayo factories; (b) Table S1: Number of chrysotile fibre personal sampling point measurements per operational area per year in the chrysotile cement manufacturing factories: 1996–2016; (c) Table S2: Number of monthly mean chrysotile fibre personal concentrations by factory, operational area, and year in the chrysotile asbestos cement manufacturing factories: 1996–2016; (d) Table S3a–c: Linear and multiple regression modelling of personal exposure experience by operators at various operational areas, by year and time period: Harare factory; (e) Table S4a–d: Linear and multiple regression modelling of personal exposure experience by operators at various operational areas, by year and time period: Bulawayo factory; (f) Tables S4a and S5b: Logistic regression modelling to examine whether year and time period have any effect on personal exposure exceeding the OEL limit of 0.1 f/mL: Harare factory.

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References

1. International Agency for Research on Cancer (IARC). *IARC Monograph on Evaluation of Carcinogenic Risks to Human, Arsenic, Metals, Fibres and Dust: A Review of Human Carcinogens*; IARC: Lyon, France; WHO: Geneva, Switzerland, 2012; Volume 100C, pp. 219–309.
2. World Health Organization. Chrysotile Asbestos. 2014. Available online: http://www.who.int/ipcs/assessment/public_health/chemicals_phc (accessed on 9 February 2020).
3. World Health Organization. *International Programme on Chemical Safety. Chrysotile Asbestos*; Environmental Health Criteria 203. UNEP, ILO, WHO and IPCS; World Health Organization: Geneva, Switzerland, 1998.
4. Agency for Toxic Substances and Diseases Registry (ATSDR). Asbestos—Production, Import, Use and Disposal. CDC. 2001. Available online: <https://www.atsdr.cdc.gov/toxprofiles/TP.asp?id=30&tid=4> (accessed on 11 July 2018).
5. Nelson, G.; WaterNaude, J. Epidemiology of Malignant Pleural Mesothelioma in Africa. In *Malignant Pleural Mesothelioma: Present Status and Future Direction*, 1st ed.; Mineo, T.C., Ed.; Bentham Science Publishers: Sharjah, United Arab Emirates, 2016; pp. 95–113.
6. Virta, R.L. Worldwide Asbestos Supply and Consumption Trends from 1900 through 2003: Circular 1298. 2006. Available online: <http://pubs.usgs.gov/circ/2006/1298/c1298.pdf> (accessed on 23 April 2018).
7. Zimbabwe National Chrysotile Asbestos Task Force, Government of Zimbabwe. *Zimbabwe Chrysotile Asbestos Position Paper*; ZNCTF: Harare, Zimbabwe, 2014.
8. Cullen, M.R.; Baloyi, S.B. Chrysotile asbestos and Health in Zimbabwe: Analysis of Mines and Millers compensated for Asbestos—Related Diseases since Independence (1980). *Am. J. Ind. Med.* **1991**, *19*, 161–169. [[CrossRef](#)] [[PubMed](#)]
9. Hagemeyer, O.; Otten, H.; Kraus, T. Asbestos consumption, asbestos exposure and related occupational diseases in Germany. *Int. Arch. Occup. Environ. Health* **2006**, *79*, 613–620. [[CrossRef](#)] [[PubMed](#)]
10. Creely, K.S.; Cowie, H.; Tongeren, M.V.; Kromhout, H.; Tickner, J.; Cherie, J.W. Trends in Inhalation Exposure—A review of the data in published scientific literature. *Ann. Occup. Hyg.* **2007**, *51*, 665–678. Available online: <https://academic.oup.com/annweh/article/51/8/665/204886> (accessed on 20 June 2020). [[PubMed](#)]
11. Coble, J.B.; Lees, P.S.; Matanoski, G. Time trends in exposure measurements from OSHA compliance inspections of the pulp and paper industry. *Appl. Occup. Environ. Hyg.* **2001**, *16*, 263–270. [[CrossRef](#)] [[PubMed](#)]
12. Finkelstein, M.M. Mortality among long term employees of an Ontario asbestos-cement factory. *Br. J. Ind. Med.* **1983**, *40*, 138–144. [[CrossRef](#)] [[PubMed](#)]
13. Albin, M.; Jakobsson, K.; Attewell, R.; Johansson, L.; Welinder, H. Mortality and cancer morbidity in cohorts of asbestos cement workers and referents. *Br. J. Ind. Med.* **1990**, *47*, 602–610. Available online: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1035245/pdf/brjindmed00045-0026.pdf> (accessed on 13 November 2020). [[CrossRef](#)] [[PubMed](#)]
14. Weiner, R.; Rees, D.; Lunga, F.J.P.; Felix, M.A. Third wave of asbestos related disease from secondary use of asbestos. A case report from industry. *S. Afr. Med. J.* **1994**, *84*, 158–160. [[PubMed](#)]
15. Higashi, T.; Hori, H.; Sakurai, H.; Omae, K.; Tsuda, T.; Tanaka, I.; Satoh, T.; Hoshi, H. Work environment of plants manufacturing asbestos-containing products in Japan. *Ann. Occup. Hyg.* **1994**, *38*, 489–494. [[PubMed](#)]
16. Albin, M.; Magnani, C.; Krstev, S.; Rapiti, E.; Shefer, I. Asbestos and cancer: An overview of current trends in Europe. *Environ. Health Perspect.* **1999**, *107*, 289–298. Available online: <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1566265/pdf/envhper00519-0069.pdf> (accessed on 27 October 2020). [[PubMed](#)]
17. Williams, P.; Paustenbach, D.; Balzer, J.L.; Mangold, C. Retrospective exposure assessment of airborne asbestos related to skilled craftsmen at a petroleum refinery in Beaumont, Texas (1940–2006). *J. Toxicol. Environ. Health* **2007**, *70*, 1076–1107. [[CrossRef](#)] [[PubMed](#)]
18. American Conference of Governmental Industrial Hygienist (ACGIH). *Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices*, 2014 ed.; ACGIH Publications: Cincinnati, OH, USA, 2014; pp. 3–4.
19. Schenk, L. Setting Occupational Exposure Limits: Practices and Outcomes of Toxicological Risk Assessment. Ph.D. Thesis, Division of Philosophy, Department of Philosophy and the History of Technology, Royal Institute of Technology (KTH), Stockholm, Sweden. Available online: https://www.researchgate.net/publication/265044801_Setting_occupational_exposure_limits_Practices_and_outcomes_of_toxicological_risk_assessment/link/545350040cf26d5090a3a902/download (accessed on 24 November 2020).
20. Pira, E.; Donato, F.; Maida, L.; Discalzi, G. Exposure to asbestos: Past, present and future. *J. Thorac. Dis.* **2018**, *10*, S237–S245. [[CrossRef](#)] [[PubMed](#)]
21. Government of Zimbabwe. *Statutory Instrument 68 of 1990: Accident Prevention and Workers Compensation Scheme*; Government of Zimbabwe: Harare, Zimbabwe, 1990.
22. National Social Security Authority. *Guidelines on Occupational Exposure Limits (OELs) for Chemical Substances and Dust*; National Social Security Authority: Harare, Zimbabwe, 2017.
23. Asbestos International Association. *Reference Method for the Determination of Airborne Asbestos Fibre Concentrations at Workplaces by Light Microscopy (Membrane Filter Method)*; Asbestos International Association: Ontario, ON, Canada, 1982.
24. Jones, A.D.; Clark, R. *Evaluation of Asbestos Dust Measurement and Control at a Factory in Harare, Zimbabwe*; Strategic Consulting Report: 629-00224; Institute of Occupational Medicine: Edinburgh, UK, 2008.
25. Zilaout, H.; Houba, R.; Kromhout, H. Temporal trends in respirable dust and respirable quartz concentrations within the European industrial minerals sector over a 15-year period (2002–2016). *Occup. Environ. Med.* **2020**, *77*, 268–275. [[CrossRef](#)] [[PubMed](#)]

26. Park, D.; Choi, S.; Ryu, K.; Park, J.; Paik, N. Trends in occupational asbestos exposure and asbestos consumption over recent decades in Korea. *Int. J. Occup. Environ. Health* **2008**, *14*, 18–24. Available online: https://www.researchgate.net/publication/5531085_Trends_in_Occupational_Asbestos_Exposure_and_Asbestos_Consumption_over_Recent_Decades_in_Korea/link/56b8d7e908ae0a89c12f8eb6/download (accessed on 12 June 2019). [CrossRef] [PubMed]
27. Yoshizumi, K.; Hori, H.; Satoh, T.; Higashi, T. The trends in Airborne Asbestos Concentrations at Plants Manufacturing Asbestos-Containing Products in Japan. *Ind. Health* **2001**, *39*, 127–131. Available online: <https://www.jstage.jst.go.jp/article/indhealth1963/39/2/392127/article> (accessed on 5 December 2019). [CrossRef] [PubMed]
28. Siriruttanapruk, S.; Taptagaporn, S. Asbestos in Thailand: Country Report. Available online: http://www.thaihealthconsumer.org/noasbestos/article/siriruttanapruk_en.pdf (accessed on 9 November 2020).
29. Peters, S.; Vermeulen, R.; Portengen, L.; Olsson, A.; Kendzia, B.; Vincent, R.; Savary, B.; Lavoue, J.; Cavallo, D.; Cattaneo, A.; et al. SYN-JEM: A Quantitative Job Exposure Matrix for Five Lung Carcinogens. *Ann. Occup. Hyg.* **2016**, *60*, 795–811. Available online: <https://academic.oup.com/annweh/article-abstract/60/7/795/2196/93> (accessed on 14 February 2018). [CrossRef] [PubMed]
30. Schonfeld, S.J.; Kovalevskiy, E.V.; Feletto, E.; Bukhtiyarov, I.V.; Kashanskiy, S.V.; Moissonier, M.; Straif, K.; McCormack, V.A.; Shutz, J.; Kromhout, H. Temporal Trends in Airborne Dust Concentrations at a Large Chrysotile Mine and its Asbestos-enrichment Factories in the Russian Federation during 1951–2001. *Ann. Work Expo. Health* **2017**, *61*, 797–808. [CrossRef] [PubMed]

CHAPTER 7

Job Exposure Matrix for Chrysotile Asbestos Fibre in the Asbestos Cement Manufacturing (ACM) Industry in Zimbabwe.



Article

Job Exposure Matrix for Chrysotile Asbestos Fibre in the Asbestos Cement Manufacturing (ACM) Industry in Zimbabwe

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Abstract: Occupational chrysotile asbestos exposure data in Zimbabwe is limited. The aim of this study was therefore to develop a job exposure matrix (JEM) specific to the chrysotile asbestos cement manufacturing industry using the available personal exposure concentration data. Quantitative personal exposure chrysotile fibre concentration data collected by the two factories from 1996 to 2020 were used to construct the JEM. Exposure groups from which data was extracted were classified based on the Zimbabwe Standard Classification of Occupations (ZSCO), 2009–2019. Analysis of amphiboles in raw chrysotile was done by scanning electron microscopy (SEM) and energy dispersive spectroscopy (EDS). Descriptive statistics, namely mean, standard deviation and range were computed for the main variable, job/occupation. All jobs/occupations in both factories had annual mean personal exposure concentrations exceeding the OEL of 0.1 f/mL, except for the period from 2009 to 2016 in the Harare factory and the period from 2009 to 2020 in the Bulawayo factory. Despite the Harare factory having no AC manufacturing activity since 2017, personal exposure concentrations showed elevated levels for the period 2018–2020. Amphiboles were detected in almost all bulk samples of chrysotile asbestos analysed. The established JEM, which has been generated from actual local quantitative exposure measurements, can be used in evaluating historical exposure to chrysotile asbestos fibre, to better understand and predict occurrence of ARDs in future.

Keywords: job exposure matrix; chrysotile asbestos; asbestos-related disease; occupational exposure



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1. Introduction

Asbestos is a group of naturally occurring fibrous silicate minerals that include chrysotile, crocidolite, amosite, anthophyllite, tremolite and actinolite [1–4]. These fibrous materials are resistant to heat, fire and corrosion, extremely durable and because of such properties, they have found widespread use in industry [2–5]. Today, Russia, China, Brazil and Kazakhstan are leading producers of chrysotile asbestos. Zimbabwe has been a major producer of chrysotile asbestos; however, full-scale mining of the mineral ceased in 2010. The major consumer of chrysotile asbestos was the asbestos cement manufacturing (ACM) industry, taking up about 10% of the produced chrysotile asbestos, while 90% was exported. Since 2010, chrysotile mainly used in the ACM industry in Zimbabwe has been largely imported from Russia. Currently the chrysotile mines are harnessing chrysotile from chrysotile dumps, and there are efforts by government to resume full-scale mining of chrysotile, making Zimbabwe the only country in Africa to still be producing and using chrysotile asbestos.

In Africa, major producers of asbestos were South Africa, Swaziland, and Zimbabwe [6]. Production of chrysotile asbestos was about 17,000 metric tonnes (mt), rising to about 50,800 mt in 1940 and reaching a peak of 250,949 mt in 1980. By 2010, production dropped to 2400 mt.

Occupational exposure to all forms of asbestos, including chrysotile, have been associated with risk of asbestos-related diseases (ARDs), such as lung cancer, mesothelioma and cancer of the larynx and ovary [1,2,4,7]. ARDs have been observed to have a dose response relationship with a long latency period between exposure and onset of disease. The minimum latency period generally associated with onset of most ARDs is 10 years depending on levels of exposure. Hence the estimation of past exposures before occurrence of ARD is crucial to elucidate the association between occupational exposure and onset of disease [3,4,8]. Occurrence of ARDs generally can be determined by the historical exposure to asbestos of the individual affected by the ARD [1,9].

Job exposure matrices (JEMs) have been used as tools for assessing past exposure levels to various hazardous factors. Historical exposure to workplace hazards and indeed chrysotile asbestos is a key factor in the onset of ARDs in Zimbabwe because chrysotile asbestos has been used in manufacturing asbestos cement (AC) products in construction works since the 1940s.

The principle of JEMs is based on the construction of a database that associate exposures to various hazardous factors with occupations/jobs or workstations [4,10,11]. Thus, a JEM is a tool through which information on jobs collected in epidemiological studies may be converted into information on possible exposures [12]. Essentially, the key objective of a JEM is to try and link job/occupation information with workplace hazardous exposure information. The idea of a JEM dates back to the time when Ramazzini tried to link diseases in 52 occupations to which the occupations were exposed to the respective hazards. In 1941, the first JEM to be developed consisted of a cross tabulation of an occupations list with that of a list of hazards [13]. Hence, the concept of JEM is that it is essentially a table in which one axis is comprised of occupations/jobs, while the other axis is comprised of workplace hazards. Additionally, for a given job/occupation each cell of the matrix can contain qualitative or quantitative exposure indicators. The JEMs may be constituted by four axes namely job/occupation, agent of exposure, time or time-period and place/location [12,14]. Exposure can vary with respect to occupations/jobs and workplaces and thus jobs can be categorised into homogenous groups to reflect similar exposures. Hence, workers exposed to a particular agent under similar or same conditions should correspond to the same entry of the matrix [14]. Furthermore, JEMs for application in retrospective studies should consider changes in exposure over time to aid in assigning health outcomes at a point in time in future. In this respect a time variable must be introduced when exposure has changed over time [12,14]. It is also important to include the place/location variable in JEMs since exposure may vary across different plants or factory situated in different locations [12].

Quantitative exposure measurements have often been considered as best estimates of actual dose [15,16]. Hence the JEM provides possible dose estimates for use in dose-response relationship studies. Where measurement data was available, it has been used in the development of JEM in workplace settings [12,17].

Information sources for which exposure estimates for a JEM can be obtained include actual measurements collected over time in workplace plants or factories of interest, company occupational hygienists, scientific literature and exposure data banks [14,18]. It is important to note that, in this study, data collected spanning almost two and half decades provided a good resource to obtain exposure estimates upon which the JEM was built.

JEMs have some limitations, among them being that variability of exposure within occupational or job classes in different workplaces, countries or over time are usually not considered in applying the JEM, leading to possible exposure misclassifications [19]. Despite some limitations, the JEM approach has advantages that can be used in situations in which traditional methods for occupational exposure assessment may be difficult or impossible to implement [10]. Additionally, JEM have become favoured approaches for occupational exposure assessment in industrial cohort studies of cancer. They are also commonly used as common occupational hygiene tools applied for accident prevention in the workplace. JEMs have also been used extensively in industry-specific studies for

various study designs to aid in the retrospective evaluation of occupational exposure in employees whose exposure history may not be readily available [14].

A chrysotile asbestos JEM built using historical exposure is important to aid in the prevention and prediction of occupational cancers with long latency periods. Nonetheless, to the best of our knowledge, there are no JEMs developed for various workplace hazards in Zimbabwe industry sectors and in particular a JEM focusing on chrysotile ACM industry; thus, this is the first one of its kind in a Zimbabwe workplace setting. A JEM specific for chrysotile asbestos in ACM industries in Zimbabwe will be useful in future epidemiology studies rather than using or extrapolating exposure estimates from international studies which may not be suitable for Zimbabwe workplaces settings. Hence, this study aimed to construct a JEM using quantitative occupational exposure data produced by the ACM industry over a period of about two and half decades and qualitative information on possible amphibole presence in the chrysotile asbestos being used in the manufacture of AC products.

2. Materials and Methods

Personal exposure data measured for the period 1996 to 2020 extracted from paper records of the two main manufacturing factories in Harare and Bulawayo cities were used to build the JEM. Harare is the capital city of Zimbabwe, in the northern part of the country, 387 km from the town of Zvishavane, while Bulawayo is the second largest city and is situated in the southern part of the country, 184 km from the town of Zvishavane, where chrysotile asbestos mines Shabanie and Mashava are located. The data was comprised of all personal exposure measurements collected by the company for close to 25 years in various operational areas examined in the two AC manufacturing factories. The data collected was examined to assess the chrysotile asbestos exposure for each combination of job, time period, place and mean personal exposure level and possible amphibole contamination. The industry is the chrysotile asbestos cement manufacturing. The jobs were classified into 9 broad similar or homogenous categories, namely saws cutting, fettling, moulded goods, koller gang, ground hard waste, laundry, pipe joints and multi-cutter operators.

Measurement of airborne chrysotile asbestos followed the standard method of the Asbestos International Association (AIA) Reference method for the determination of airborne asbestos fibre concentrations at workplaces by light microscopy as previously described by Mutetwa et al. (2021) [20]. Briefly, the chrysotile fibres were sampled on 25 mm membrane filters of 1.2- μm pore size with printed grids and then counted by means of a phase contrast microscope (PCM). The fibres counted were generally longer than 5 μm with a width of less than 3 μm and length-to-width ratio of more than 3:1.

Detection of amphiboles in the chrysotile asbestos being used for manufacturing AC products in the Bulawayo factory was done by National Institute of Occupational Health (NIOH)—National Health Laboratory Service (NHLS), South Africa, using scanning electron microscopy (SEM) and energy dispersive spectroscopy (EDS).

2.1. Data Analysis

Data analysis was conducted using IBM SPSS version 26. Monthly averaged personal exposure concentrations for the factories were used. Mean personal exposure concentrations were analysed per operator working in a particular location per factory.

ANOVA was applied with the aim of identifying patterns of exposure variability among the time-period for various job categories and determining whether there was a statistically significant difference in exposure concentration between the four time-periods for various jobs. A Tukey post hoc test (Tukey's honest significance difference test) was run to find out which specific group means of time-periods for various jobs/occupations (compared with each other) were different.

The arithmetic mean was used as a representative value for analysis of the measurements as this is normally taken as the best summary measure of exposure in epidemiological studies of chronic diseases when adopting a linear exposure response model [7,21].

2.2. Ethics

The study was approved by the University of the Witwatersrand Human Research Ethics Committee (clearance certificate number M181157) and the Medical Research Council of Zimbabwe (MRCZ) (approval number MRCZ/A/2445).

3. Results

A total of 3066 airborne chrysotile personal measurements collected from company records, spanning a period of about 25 years from which 1788 annual mean personal exposure concentrations were drawn from, were used to build the job exposure matrix (JEM) for chrysotile asbestos fibre in the AC manufacturing factories. For the purpose of the JEM, jobs selected had the most data and were in most common operational areas even up to 2020. The jobs involved are as outlined in Table 1, and their description is briefly given. Additionally, the jobs were coded with respect to the Zimbabwe Standard Classification of Occupations (ZSCO) [22].

Table 1 presents the JEM with jobs categorised with their description, factory location, mean and range, period and the possible amphiboles identified in chrysotile asbestos materials used in the manufacturing process. Table 2 shows statistical significance in variability in exposure concentrations for time periods for various job categories. Supplementary information Tables S1 and S2 further show post hoc output showing statistical significance in exposure concentrations for various time periods for each job category.

Table 3 shows results of type of amphiboles detected in the bulk chrysotile asbestos samples collected in bags of asbestos used in the manufacturing process in the Bulawayo factory.

Annual mean personal exposure concentration for saw cutting, fettling, ground hard waste operators (Harare and Bulawayo factory), laundry room operator, moulded goods operator (Harare factory), and pipe section operators (Bulawayo factory) showed high levels exceeding the OEL of 0.1 f/mL for the time-period 1996 to 2008. As reported by Mutetwa et al. (2021), high exposure levels above the OEL were exhibited in the 1990s and 2000s compared to the period 2009 to 2016 for both factory locations with saw cutting, kollergang and ground hard waste operators in both locations generally exposed to high levels of airborne chrysotile fibre in the years 1996 to 2000 [20]. It is insightful to note that the results for the Harare factory for the years 2018–2020 for all operational areas had exposure concentrations exceeding the OEL, even though manufacturing of AC products during this period had ceased; however, exposure concentrations were also comparable to those reported previously by Mutetwa et al. (2021), for the period 1996 to 2008. The results for the measurement period of 2017–2019 for the Bulawayo factory were all below the OEL for all key operational areas examined, although they were lower than the exposure concentrations reported by Mutetwa et al. (2021) [20], for the period 2009 to 2016, though the order of magnitude is about the same.

The factories reported that importation of chrysotile asbestos started in 2008 following marked decline of mining operations at the two chrysotile mines of Shabanie and Mashava and eventual ceasing of mining operations in 2010. This would suggest that for the Harare factory manufacturing of AC products continued for 8 years with the use of imported fibre, while for the Bulawayo factory, manufacturing of AC products using imported fibre has been ongoing for 12 years up to 2020. However, manufacturing at the Bulawayo factory continues to this day, using largely imported fibre and a small component from locally produced fibre harnessed from chrysotile dumps.

Table 1. Job exposure matrix based on airborne chrysotile asbestos fibre occupational exposure data for ACM industry factories for the period 1996–2020. The codes in brackets in the Job column is the ZSCO code.

Job	Job Description	Time Period	Hazare Factory (Tr, Anth, Cr, Act)				Bulawayo Factory (Tr, Anth, Cr, Act)				p-Value	Range Min Max	p-Value
			N	Mean ± SD (f/mL)	95%CI LB UB	Range Min Max	N	Mean ± SD (f/mL)	95%CI LB UB	Range Min Max			
Saw cutting operator (1023)	Cutting by saw asbestos sheets and fascia boards to size	1996–2000	60	0.19 ± 0.01	0.19 0.19	0.16 0.24	50	0.17 ± 0.02	0.16 0.18	0.12 0.24	<0.001	0.09 0.16 0.01 0.08 0.05 0.07	<0.001
		2001–2008	88	0.13 ± 0.02	0.12 0.13	0.08 0.18	49	0.12 ± 0.02	0.11 0.12	0.09 0.16			
		2009–2016	77	0.07 ± 0.02	0.07 0.08	0.03 0.11	14	0.06 ± 0.02	0.05 0.07	0.01 0.08			
		2018–2020*	29	0.10 ± 0.02	0.09 0.11	0.06 0.15	24	0.05 ± 0.01	0.05 0.06	0.05 0.07			
Fettling table operator (1023)	Scrapping/polishing AC moulded goods	1996–2000	53	0.12 ± 0.04	0.11 0.13	0.05 0.18	40	0.17 ± 0.06	0.16 0.19	0.07 0.30	0.561	0.06 0.15	<0.001
		2001–2008	73	0.12 ± 0.02	0.12 0.13	0.05 0.19	11	0.12 ± 0.03	0.10 0.14	0.06 0.15			
		2009–2016	Nil	-	-	-	-	-	-	-			
2018–2020*	4	0.11 ± 0.03	0.05 0.16	0.06 0.14	-	-	-	-					
Moulded goods operator (1024)	Moulding of AC goods under wet conditions	1996–2000	58	0.11 ± 0.04	0.10 0.12	0.04 0.20	-	-	-	-	<0.001	-	-
		2001–2008	82	0.11 ± 0.04	0.11 0.12	0.03 0.18	-	-	-	-			
		2009–2016	52	0.05 ± 0.01	0.05 0.06	0.03 0.08	-	-	-	-			
		2018–2020*	5	0.11 ± 0.02	0.08 0.13	0.08 0.13	-	-	-	-			
Kollergang operator (1021)	Opening of & loading chrysotile bags into process machine and operate machine	1996–2000	58	0.13 ± 0.04	0.12 0.14	0.05 0.20	36	0.14 ± 0.03	0.13 0.15	0.08 0.24	<0.001	0.03 0.18 0.03 0.09	<0.001
		2001–2008	81	0.12 ± 0.02	0.11 0.12	0.04 0.16	42	0.12 ± 0.01	0.11 0.12	0.08 0.14			
		2009–2016	64	0.07 ± 0.02	0.06 0.07	0.04 0.11	33	0.07 ± 0.03	0.06 0.08	0.03 0.18			
		2018–2020*	9	0.12 ± 0.01	0.11 0.13	0.10 0.13	15	0.06 ± 0.01	0.05 0.07	0.03 0.09			
Ground hard waste operator (1021)	Feeding AC waste materials into grinder machine	1996–2000	57	0.16 ± 0.03	0.15 0.16	0.08 0.22	44	0.13 ± 0.04	0.11 0.14	0.07 0.24	<0.001	0.04 0.09 0.04 0.08	<0.001
		2001–2008	56	0.13 ± 0.03	0.12 0.14	0.03 0.20	15	0.11 ± 0.04	0.10 0.11	0.08 0.13			
		2009–2016	55	0.07 ± 0.02	0.06 0.08	0.02 0.17	5	0.07 ± 0.02	0.05 0.09	0.04 0.09			
		2018–2020*	8	0.12 ± 0.01	0.12 0.13	0.11 0.13	12	0.06 ± 0.02	0.05 0.06	0.04 0.08			
Laundry room operator (1024)	Laundering of PPC using wash machine	1996–2000	47	0.13 ± 0.03	0.12 0.14	0.06 0.20	-	-	-	-	<0.001	-	-
		2001–2008	87	0.13 ± 0.02	0.12 0.13	0.06 0.21	-	-	-	-			
		2009–2016	15	0.05 ± 0.01	0.04 0.05	0.03 0.07	-	-	-	-			
		2018–2020*	14	0.11 ± 0.02	0.10 0.12	0.07 0.14	-	-	-	-			
Pipe joints operator (1023)	Lathe machining of AC joints pipes	1996–2000	-	-	-	-	44	0.13 ± 0.04	0.12 0.14	0.06 0.30	-	0.08 0.15 0.04 0.08 0.02 0.08	<0.001
		2001–2008	-	-	-	46	0.11 ± 0.01	0.11 0.12	0.08 0.15				
		2009–2016	9	0.05 ± 0.02	0.05 0.07	0.04 0.08	4	0.05 ± 0.02	0.02 0.08	0.02 0.08			
2018–2020	-	-	-	-	-	-	-	-					

Table 1. Cont.

Job	Job Description	Time Period	Harare Factory (Tr, Anth, Cr, Act)			Bulawayo Factory (Tr, Anth, Cr, Act)			p-Value	
			N	Mean ± SD (f/mL)	95%CI LB UB	Range Min Max	N	Mean ± SD (f/mL)		95%CI LB UB
Full length pipe operator (1023)	Lathe machining & polishing of full-length AC pipe joints	1996–2000	-	-	-	43	0.13 ± 0.04	0.12 0.14	0.06 0.27	<0.001
		2001–2008	-	-	-	45	0.11 ± 0.01	0.11 0.11	0.07 0.14	
		2009–2016 2018–2020	-	-	-	9	0.07 ± 0.02	0.05 0.08	0.04 0.08	
Multi-cutter operator (1023)	Cutting full length pipes into collars for coupling pipes	1996–2000	-	-	-	26	0.13 ± 0.04	0.11 0.14	0.05 0.20	<0.001
		2001–2008	-	-	-	36	0.12 ± 0.01	0.12 0.13	0.10 0.14	
		2009–2016 2018–2020	-	-	-	2	0.07 ± 0.03 0.04 ± 0.01	0.12 0.13 0.00 0.11	0.05 0.20 0.04 0.05	

ACM—asbestos cement manufacturing, AC—asbestos cement, PPC—personal protective clothing, SD—standard deviation, Min—minimum, Max—maximum, N—number of monthly-averaged personal chrysotile fibre concentrations, 1996–2020, LB—lower bound and UB—upper bound values for the 95% confidence intervals of the mean. () Bracketed number refers to Zimbabwe Standard Classification of Occupations code (ZSCO) (NSSA, 2009–2019), Tr—tremolite, Anth—anthophyllite, Cr—crocidolite, Act—actinolite. * Care and maintenance of equipment and cleaning—Harare factory when manufacturing of AC products no longer takes place. ** Possible exposure to amphiboles in both factories could have started in 2010 following major shift in use of imported fibre.

Table 2. Analysis of variability of mean personal exposure concentrations between time period categories for various jobs.

Operator	Harare Factory			Bulawayo Factory		
	df	F	p-Value	df	F	p-Value
Saw cutting operator	2, 250	519.6	<0.001	3, 134	236.8	<0.001
Fettling table operator	2, 127	0.6	0.561	1, 49	5.2	<0.001
Moulded goods operator	3, 193	59.2	<0.001	-	-	-
Kollrgang operator	3, 208	83.3	<0.001	3, 123	68.9	<0.001
Ground hard waste	3, 172	96.9	<0.001	3, 73	18.2	<0.001
Laundry	3, 169	48.0	<0.001	2, 26	5.0	<0.001
Pipe joints	-	-	-	3, 100	24.8	<0.001
Full-length	-	-	-	2, 26	20.6	<0.001
Multicutter	-	-	-	3, 62	9.3	<0.001

Df—degrees of freedom. F—F test statistic.

Table 3. Amphiboles in chrysotile samples collected from bags of raw chrysotile material used for manufacturing AC products.

Chrysotile Sample	EDS Result
Local 01	Straight and curved fibres exhibited peaks of magnesium and silicon. Chrysotile and tremolite detected in the sample
Local 02	Straight and curved fibres exhibited peaks of magnesium and silicon. Chrysotile, tremolite and anthophyllite detected in the sample
Local 03	Straight and curved fibres exhibited peaks of magnesium and silicon. Chrysotile, tremolite and anthophyllite detected in the sample
Local 04	Curved fibres exhibited peaks of magnesium and silicon. Chrysotile only detected in the sample
Local 05	Curved fibres exhibited peaks of magnesium and silicon. Chrysotile only detected in the sample
Local 06	Curved fibres exhibited peaks of magnesium and silicon. Chrysotile and tremolite detected in the sample
Imported 01	Curved fibres exhibited peaks of magnesium and silicon. Chrysotile and tremolite detected in the sample
Imported 02	Curved and straight fibres exhibited peaks of magnesium and silicon. Chrysotile, crocidolite and tremolite detected in the sample.
Imported 03	Curved and straight fibres exhibited peaks of magnesium and silicon. Chrysotile, crocidolite, tremolite and actinolite detected in the sample
Imported 04	Curved and straight fibres exhibited peaks of magnesium and silicon. Chrysotile, tremolite and actinolite detected in the sample
Imported 05	Curved fibres exhibited peaks of magnesium and silicon. Chrysotile only detected in the sample
Imported 06	Curved fibres exhibited peaks of magnesium and silicon. Chrysotile and tremolite detected in the sample

For the Harare factory, during the period from 2017 to 2020, operations in areas where personal samples were collected, the jobs involved essentially care, maintenance and general cleaning of equipment, except for the saws cutting where cutting of AC sheets from the Bulawayo factory continued as what used to happen during the time manufacturing was ongoing for the period 1996 to 2016.

For the 6 locally produced chrysotile samples analysed, 4 had amphiboles detected, namely tremolite and anthophyllite as shown in Table 3. Furthermore, for the imported chrysotile asbestos, amphiboles, namely tremolite, crocidolite and actinolite were also detected in 5 out of 6 samples analysed (Table 3).

Variability of mean personal exposure concentrations between time periods for each job category was also tested using ANOVA. There was a statistically significant difference in the annual mean personal chrysotile exposure concentrations among the different time periods for various job categories as determined by one-way ANOVA in both factory locations except for the fettling table operator in Harare (Table 2).

Furthermore, for the Harare factory after 2016, jobs in the areas examined essentially had similar activities of care and maintenance of equipment and cleaning in the respective areas which involved AC manufacturing and handling, and despite no manufacturing of AC products taking place, exposure concentrations remained elevated above the OEL of 0.1 f/mL, except for saw cutting operator, in which exposure concentration was same as the OEL. Additionally, the exposure concentrations for the time period 2018 to 2020 were statistically significantly higher compared to the prior period of 2009 to 2016 ($p < 0.001$).

For almost all jobs in the Harare factory, the post hoc tests show that exposure concentrations during the period 2009 to 2016 was statistically significantly lower than exposure concentrations during the periods 1996–2000, 2001–2008 and 2018–2020. Additionally, for the laundry operator, post hoc test further reveals that exposure concentrations during the period 2018–2020, although elevated above the OEL of 0.1 f/mL, was statistically significantly lower and higher than exposure concentrations during the periods, 1996–2000 and 2009–2016, respectively. There was no statistically significant difference in exposure concentrations between the time periods 1996–2000 and 2018–2020 for all jobs ($p > 0.05$) except for the saw cutting operator ($p < 0.05$) in the Harare factory (Supplementary material Table S1).

For the Bulawayo factory, the post hoc test (Table S2) shows that exposure concentrations during the period 1996 to 2000 was statistically significantly higher than exposure concentration during the periods 2009–2016 and 2017–2019, with saw cutting, kollergang and pipe joints operators exposure concentrations also being statistically significantly higher during the period 1996–2000 than during the period 2001 to 2008 ($p < 0.05$). There was no statistically significant difference in exposure concentrations between the time periods 2009–2016 and 2017–2019 for all jobs ($p > 0.05$).

Analysis of Presence of Amphiboles in Samples Collected from Bags of Raw Chrysotile Materials Used for Manufacturing AC Products in the Bulawayo Factory

Six (6) local bulk chrysotile samples and 6 imported chrysotile samples were randomly collected from bags ready to be processed at the holding bay and at the kollergang area. Fibres with aspect ratio greater than 3:1 were observed in all the samples morphologically resembling asbestos using SEM.

4. Discussion

The focus of this study was on the construction of a JEM using a large number of personal chrysotile asbestos fibre measurements relevant to the Zimbabwean AC industry and collected over a period of about 25 years. The data used were related to work characteristics of jobs outlined under the results section, which jobs are the most common jobs in the ACM industry in Zimbabwe and the matrix arising therein provides a tool for exposure assessment in future studies.

The job categories with high exposure levels were saw cutting, fettling, ground hard waste, laundry room and multi-cutter operator and such levels of exposure may present increased risk of ARDs. As reported by Mutetwa et al. (2021) [20], exposure concentrations declined over time for both factories for the period 1996 to 2016, and further declines in exposure concentrations for all jobs in the Bulawayo factory was observed for the period 2017 to 2019. Exposure concentrations in the Harare factory, for the period 2018 to 2020 were, however, much higher than those reported by Mutetwa et al. (2021) [20], for the

preceding time period 2009 to 2016, during which concentrations ranged from 0.05 to 0.07 f/mL in various operations examined compared to 0.10 to 0.12 f/mL for the period of 2018 to 2020, even though manufacturing had ceased. The Harare factory reported no clean-up before the current activities of manufacturing concrete tiles and other concrete products started, so resuspension of chrysotile fibre from the floors which accumulated chrysotile fibre in the past could be responsible for the elevated levels for the time-period 2018 to 2020. The work practices deployed during the period for manufacturing of AC products, e.g., wet dust suppression methods may no longer be practiced.

On the other hand, the lower exposure concentration values exhibited in the Bulawayo factory for the period 2017 to 2019 compared to 2009 to 2016 fibre concentration levels reported by Mutetwa et al. (2001) [20] may suggest continued adherence to good work practices and continued implementation of occupational safety and health management systems which the factory has been subscribing to over the years in its AC manufacturing processes. Although the factories were of the same company, it can be viewed that since the Harare factory was now producing concrete products, exposure to chrysotile asbestos fibre could have been considered not much of a threat to health, hence the low OSH standards at the Harare factory compared to the Bulawayo factory.

During the period 1996 to 2000, the chrysotile ACM industry developed and used its own occupational exposure standard of 0.2 f/mL in the absence of a national statutory limit. This may mean that exposure concentrations up to this limit were deemed as presenting insignificant health risk to workers exposed, hence control measures were then possibly designed to contain airborne chrysotile levels to be within 0.2 f/mL. However, exposure at a level of 0.2 f/mL does present some health risk of ARDs, in light of the fact that the occupational exposure limit in many countries, including Zimbabwe, has been set at 0.1 f/mL [23–25], as an attempt to minimise health risks associated with exposure to asbestos.

The categorisation of exposure measurements into measurement periods reflecting ACM industry occupational hygiene practices as well as general economic status provides insights into variation of exposure estimates over time [16]. Post hoc test data further demonstrate that the earlier time periods of 1996–2000 had statistically significantly higher exposure concentrations than exposure concentrations during the period 2001–2008 and 2009–2016 in both factories ($p < 0.05$). Such high exposure concentrations, as reported by Mutetwa et al., 2021 [20], during the 1990s and early to mid-2000s as well as the industry's own belief that maintaining exposure to below 0.2 f/mL may be the reason for higher exposure levels exhibited in the 1990s and early 2000s compared to periods 2009–2016.

The detection of amphiboles in the chrysotile asbestos being used in the manufacture of AC products further heighten the possible risk of ARD occurrence among workers exposed. Anthophyllite fibres have also been detected in the past by XRD and TEM in Zimbabwe chrysotile [26,27]. Amphiboles, particularly crocidolite have significantly been associated with the development ARDs, such as mesothelioma and lung cancer [1,2,28]. Significant importation of chrysotile asbestos has been ongoing since 2008 and the detection of amphiboles and notably crocidolite in some of the samples taken from the chrysotile bags further suggests a serious risk of ARD occurrence in the form of mesothelioma as crocidolite is one the most dangerous form of asbestos [28]. The Bulawayo factory reported that about 5200 tonnes per annum of chrysotile is imported and 550 tonnes annually of local chrysotile is used for manufacturing AC products. This translates to about 69,000 tonnes and 6600 tonnes of imported and local chrysotile asbestos, respectively, being consumed for the 12-year period to 2019 from the time significant imports started being used in 2008. Assuming that the Harare factory also used similar quantities of asbestos per annum, for the 8-year period to 2016 before AC production ceased, a considerable amount of chrysotile asbestos amounting to 46,000 tonnes (41,600 imported plus 4400 tonnes local) was used up to the time AC manufacturing ceased. While chrysotile asbestos is dangerous to human health, the possible presence of amphiboles in the chrysotile being used could further amplify the health risk presented by exposure to asbestos in these factories.

Kollergang operators who handle raw chrysotile asbestos, ground hard waste operators who are involved in a hazardous process that generate considerable dust in both factories particularly during the 1996 to 2000 and 2001 to 2008 time periods were possibly at high risk of exposure to amphiboles contaminants in chrysotile asbestos being used in manufacturing of AC products. Additionally, higher exposure concentrations in lathe machines operators in the Bulawayo factory may also suggest elevated risk of exposure to amphiboles during the earlier time periods of 1996 to 2008 and thus possible increased risk of ARDs for operators working with these machines.

Strength and Limitations

The strength of the JEM is that the data used in its construction is purely from the local ACM industry workplace settings. It can also be useful in the evaluation of the contribution of asbestos exposure on ARD occurrence, taking into account the job profile of exposed workers and time period. Fewer measurements data for the period 2016 to 2020 diminished the accuracy of the mean exposure concentration for this time period. "Additionally, while phase contrast microscopy (PCM) provides relatively quick and cost-effective analysis of asbestos samples, the PCM is not able to distinguish whether fibres observed are chrysotile or amphiboles fibres. Nonetheless, in this study, it was considered that fibres were generally chrysotile as the factories have been using chrysotile asbestos in their manufacturing processes since their establishment in the 1940s". In view of limited resources, the factories were not able to participate in interlaboratory quality assurance and control fibre counting programmes, particularly for the later years of 2013 to 2020; hence, this could have presented a limitation in the results. The factories, however, continued to consistently apply the standard method for asbestos measurements as previously reported by Mutetwa et al., (2021), making sure that use of blank samples was always part of the methodology.

5. Conclusions

The JEM generated in this study provides quantitative estimates of personal chrysotile asbestos exposure concentrations for workers operating in ACM plants, based on jobs held and factory locations worked in and may give estimates of latency based on estimates of time period of exposure used. Furthermore, the JEM may provide an opportunity for prediction of occurrence of ARDs and possible analysis of exposure response relationships that may be linked to exposure episodes of measurement period in the distant past. The JEM also gives a perspective on the possible amphiboles associated with the local and imported chrysotile asbestos used in the manufacturing processes.

Supplementary Materials: The following are available online at <https://www.mdpi.com/article/10.3390/ijerph19052680/s1>, Table S1: Harare Factory Post Hoc Test: Multiple comparisons showing mean differences between time periods for various job categories that reaches significance ($p < 0.05$). Table S2: Bulawayo Factory Post Hoc Test: Multiple comparisons showing mean differences between time periods for various job categories that reaches significance ($p < 0.05$).

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References

- International Agency for Research on Cancer (IARC). *IARC Monograph on Evaluation of Carcinogenic Risks to Human, Arsenic, Metals, Fibres and Dust: A Review of Human Carcinogens*; IARC/WHO: Lyon, France, 2012; Volume 100C, pp. 219–309.
- World Health Organization. Chrysotile Asbestos. WHO, 2014. Available online: http://www.who.int/ipcs/assessment/public_health/chemicals_phc. (accessed on 9 January 2020).
- Scarselli, A.; Marinaccio, A.; Corfiati, M.; Di Marzio, D.; Iavicoli, S. Occupational asbestos exposure after the ban: A job exposure matrix developed in Italy. *Eur. J. Public Health* **2020**, *30*, 936–941. [[CrossRef](#)]
- Kang, D.; Jung, S.; Kim, Y.J.; Kim, J.; Choi, S.; Kim, S.Y.; Kim, Y. Reconstruction of the Korean Asbestos Job Exposure Matrix. *Saf. Health Work* **2021**, *12*, 74–95. [[CrossRef](#)] [[PubMed](#)]
- Bernstein, D.; Dunnigan, J.; Hesterberg, T.; Brown, R.; Velasco, J.A.; Barrera, R.; Hoskins, J.; Gibbs, A. Health risk of chrysotile revisited. *Crit. Rev. Toxicol.* **2013**, *43*, 154–183. [[CrossRef](#)]
- Nelson, G.; WaterNaude, J. Epidemiology of Malignant Pleural Mesothelioma in Africa. In *Malignant Pleural Mesothelioma: Present Status and Future Direction*, 1st ed.; Mineo, T.C., Ed.; Bentham Science Publishers: Al Sharjah, United Arab Emirates, 2016; pp. 95–113.
- Choi, S.; Kang, D.; Park, D.; Lee, H.; Choi, B. Developing Asbestos Job Exposure Matrix Using Occupation and Industry Specific Exposure Data (1984–2008) in Republic of Korea. *Saf. Health Work* **2017**, *8*, 105–115. [[CrossRef](#)] [[PubMed](#)]
- Olsson, A.C.; Vermeulen, R.; Schüz, J.; Kromhout, H.; Pesch, B.; Peters, S.; Behrens, T.; Portengen, L.; Mirabelli, D.; Gustavsson, P.; et al. Exposure-Response Analyses of Asbestos and Lung Cancer Subtypes in a Pooled Analysis of Case-Control Studies. *Epidemiology (Camb. Mass)* **2017**, *28*, 288–299. [[CrossRef](#)] [[PubMed](#)]
- Brims, F.J. Asbestos—A legacy and a persistent problem. *J. R. Nav. Med. Serv.* **2009**, *95*, 4–11. [[CrossRef](#)] [[PubMed](#)]
- Goldberg, M.; Imbernon, E. The use of job exposure matrices for cancer epidemiology research and surveillance. *Arch Public Health* **2002**, *60*, 173–185. Available online: https://www.wiv-isp.be/APH/pdf/aphfull60_173_185.pdf (accessed on 5 August 2021).
- Pannett, B.; Coggon, D.; Acheson, E.D. A job-exposure matrix for use in population-based studies in England and Wales. *Br. J. Ind. Med.* **1985**, *42*, 777–783. [[CrossRef](#)]
- Kauppinen, T.; Toikkanen, J.; Pukkala, E. From cross-tabulations to multipurpose exposure information systems: A new job-exposure matrix. *Am. J. Ind. Med.* **1998**, *33*, 409–417. [[CrossRef](#)] [[PubMed](#)]
- Carnevale, F.; Mendini, M.; Moriani, G. *Bernardino Ramazzini Works*, 1st ed.; Cierre Edizioni: Verona, Italy, 2009.
- Goldberg, M.; Kromhout, H.; Guénel, P.; Fletcher, A.C.; Gérin, M.; Glass, D.C.; Heederik, D.; Kauppinen, T.; Ponti, A. Job exposure matrices in industry. *Int. J. Epidemiol.* **1993**, *22* (Suppl. 2), S10–S15. [[CrossRef](#)] [[PubMed](#)]
- Teschke, K.; Olshan, A.F.; Daniels, J.L.; De Roos, A.J.; Parks, C.G.; Schulz, M.; Vaughan, T.L. Occupational exposure assessment in case-control studies: Opportunities for improvement. *Occup. Environ. Med.* **2002**, *59*, 575–594. [[CrossRef](#)] [[PubMed](#)]
- van Oyen, S.C.; Peters, S.; Alfonso, H.; Fritsch, L.; de Klerk, N.H.; Reid, A.; Franklin, P.; Gordon, L.; Benke, G.; Musk, A.W. Development of a Job-Exposure Matrix (AsbJEM) to Estimate Occupational Exposure to Asbestos in Australia. *Ann. Occup. Hyg.* **2015**, *59*, 737–748. [[CrossRef](#)] [[PubMed](#)]
- Offermans, N.S.; Vermeulen, R.; Burdorf, A.; Goldbohm, R.A.; Kauppinen, T.; Kromhout, H.; van den Brandt, P.A. Occupational asbestos exposure and risk of pleural mesothelioma, lung cancer, and laryngeal cancer in the prospective Netherlands cohort study. *J. Occup. Environ. Med.* **2014**, *56*, 6–19. [[CrossRef](#)] [[PubMed](#)]
- Plato, N.; Steineck, G. Methodology and utility of a job-exposure matrix. *Am. J. Ind. Med.* **1993**, *23*, 491–502. [[CrossRef](#)]
- Kauppinen, T.P.; Mutanen, P.O.; Seitsamo, J.T. Magnitude of misclassification bias when using a job-exposure matrix. *Scand. J. Work Environ. Health* **1992**, *18*, 105–112. [[CrossRef](#)] [[PubMed](#)]
- Mutetwa, B.; Moyo, D.; Brouwer, D. Trends in Airborne Chrysotile Asbestos Fibre Concentrations in Asbestos Cement Manufacturing Factories in Zimbabwe from 1996 to 2016. *Int. J. Environ. Res. Public Health* **2021**, *18*, 10755. [[CrossRef](#)]
- Seixas, N.S.; Robins, T.G.; Moulton, L.H. The use of geometric and arithmetic mean exposures in occupational epidemiology. *Am. J. Ind. Med.* **1988**, *14*, 465–477. [[CrossRef](#)] [[PubMed](#)]
- National Social Security Authority (NSSA). *Annual Statistical Reports. 2009–2019*; NSSA: Harare, Zimbabwe, 2019.
- American Conference of Governmental Industrial Hygienist (ACGIH). *Threshold Limit Values for Chemical Substances and Physical Agents and Biological Exposure Indices*; 2014 ed.; ACGIH Publications: Cincinnati, OH, USA, 2014; pp. 3–4.
- Government of South Africa. *Asbestos Abatement Regulations*; No.R.1196; Government of South Africa, Department of Labour: Pretoria, South Africa, 2020.
- NSSA. *National Social Security Authority Guidelines on Occupational Exposure Limits (OELs) for Chemical Substances and Dust*; NSSA: Harare, Zimbabwe, 2017.

26. Kohyama, N.; Shinohara, Y.; Suzuki, Y. Mineral phases and some reexamined characteristics of the International Union Against Cancer standard asbestos samples. *Am. J. Ind. Med.* **1996**, *30*, 515–528. [CrossRef]
27. Tossavainen, A.; Kotilainen, M.; Takahashi, K.; Pan, G.; Vanhala, E. Amphibole fibres in Chinese chrysotile asbestos. *Ann. Occup. Hyg.* **2001**, *45*, 145–152 doiorg/101016/S0003. [CrossRef]
28. Hodgson, J.T.; Darnton, A. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. *Ann. Occup. Hyg.* **2000**, *44*, 565–601. Available online: <https://pubmed.ncbi.nlm.nih.gov/11108782/> (accessed on 20 December 2021). [CrossRef]

CHAPTER 8

Prediction of Asbestos Related Diseases (ARDs) and Chrysotile Asbestos Exposure Concentrations in Asbestos-Cement (AC) Manufacturing Factories in Zimbabwe.



Article

Prediction of Asbestos-Related Diseases (ARDs) and Chrysotile Asbestos Exposure Concentrations in Asbestos-Cement (AC) Manufacturing Factories in Zimbabwe

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Abstract: The use of historical asbestos measurement data in occupational exposure assessment is essential as it allows more quantitative analysis of possible exposure response relationships in asbestos-related disease (ARD) occurrence. The aim of this study was to predict possible ARDs, namely lung cancer, mesothelioma, gastrointestinal cancer, and asbestosis, in two chrysotile asbestos cement (AC) manufacturing factories. Prediction of ARDs was done using a specific designed job exposure matrix for airborne chrysotile asbestos fibre concentrations obtained from the Harare and Bulawayo AC factories and through application of OSHA's linear dose effect model in which ARDs were estimated through extrapolation at 1, 10, 20, and 25 years of exposure. The results show that more cancer and asbestosis cases are likely to be experienced among those exposed before 2008 as exposure levels and subsequently cumulative exposure were generally much higher than those experienced after 2008. After a possible exposure period of 25 years, overall cancer cases predicted in the Harare factory were 325 cases per 100,000 workers, while for the Bulawayo factory, 347 cancer cases per 100,000 workers exposed may be experienced. Possible high numbers of ARDs are likely to be associated with specific tasks/job titles, e.g., saw cutting, kollergang, fettling table, ground hard waste, and possibly pipe-making operations, as cumulative exposures, though lower than reported in other studies, may present higher risk of health impairment. The study gives insights into possible ARDs, namely lung cancer, mesothelioma, gastrointestinal cancer, and asbestosis, that may be anticipated at various cumulative exposures over 1, 10, 20, and 25 years of exposure in AC manufacturing factories in Zimbabwe. Additionally, results from the study can also form a basis for more in-depth assessment of asbestos cancer morbidity studies in the AC manufacturing industries.

Keywords: prediction; asbestos related diseases; exposure concentration; cumulative exposure; chrysotile; Zimbabwe

1. Introduction

Asbestos in all its forms (serpentine group—chrysotile, amphibole group—crocidolite, amosite, tremolite, actinolite, and anthophyllite) is an important occupational carcinogen causing about half of cancer-related deaths [1,2]. There is sufficient evidence in humans for the carcinogenicity of all forms of asbestos from which the International Agency on Research on Cancer (IARC) concluded that asbestos causes mesothelioma and cancer of the lungs, larynx, and ovaries and that asbestos is a Group 1 carcinogen [2]. Additionally, a positive association between exposure to all forms of asbestos and cancer of the pharynx, stomach, and colorectum has been established. However, with respect to the colorectum, the IARC Working Group was evenly divided to conclude that evidence for asbestos was sufficient to be considered as causing cancer. Furthermore, experimental animal studies have demonstrated that there is sufficient evidence showing carcinogenicity of all forms of asbestos [2]. Gastrointestinal tract cancers have also been reported in groups of persons occupationally exposed to amosite, chrysotile, or mixed fibres containing chrysotile [2]. All forms of asbestos cause asbestosis [1–3].

The latency period associated with these diseases can range from 10 to 40 years from exposure [1,2,4]. However, short intense exposures to asbestos, lasting from several months to 1 year or more, can be sufficient to cause asbestosis [4]. The health risks of exposure to all forms of asbestos are largely associated with inhalation [2]. Studies of workers exposed to chrysotile asbestos fibre in different sectors have broadly demonstrated exposure–response or exposure–effect relationships for chrysotile-induced asbestosis, as levels of exposure have increased and hence, such exposures have been reported to result in increased incidence and severity of disease [3]. Asbestosis stage changes have been noted to be common following prolonged exposure of 5 to 20 f/mL [1].

Use of measurement data in occupational exposure assessment is essential as it allows more quantitative analysis of possible exposure response relationships [5]. Furthermore, historical exposure to asbestos is an important factor in asbestos-related disease occurrence because asbestos materials have been used for the past few decades in many workplace settings [6,7]. In Zimbabwe, exposure to chrysotile asbestos has been ongoing for many decades since the mines were opened around 1910 and manufacturing began around 1943 [8]. In occupational exposure assessment studies, cumulative exposure is often used as an exposure metric in quantitative epidemiologic evaluation studies [9–17]. Cumulative exposure is normally defined in terms of fibre/millilitre years (f/mL-years) and the definition is based on the level of exposure in the workplace, measured as the number of fibres found

in each ml of air, in the air which a worker breathes at work and multiplied by the number of years or fraction of a year worked at that level [18–21].

Early studies have reported that a worker exposed to 100 f/mL-years (for example, 50 years of exposure at 2 f/mL or 25 years at 4 f/mL or 10 years at 10 f/mL) are likely to have a 1% chance of developing asbestosis [22]. Thus, the correlation of exposure concentrations with disease occurrence among persons exposed provided a basis for setting an occupational exposure limit for asbestos fibres in the ambient air [18,19]. Since accurate quantitative exposure concentration data are sometimes difficult to obtain, cumulative exposure expressed as fibre/mL-years is usually used as a common metric in exposure assessments reported in epidemiological studies [18,19,23]. Additionally, it has been noted that the variable cumulative exposure assumes that the duration of exposure and exposure concentration carry the same weight [18,19]. Furthermore, exposure at low concentration levels over a long period of time may mathematically be equivalent to exposure at high concentration for short periods; however, the biological effects may be different. Hence, duration or concentration levels maybe more important than their product in predicting occurrence of disease. In the case of mesothelioma, duration of exposure has been reported to appear as the most important factor in occurrence of mesothelioma [16,18,19].

In a study on quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure, it has been reported that a cumulative exposure of 1 f/mL-years for crocidolite yields a lifetime risk for mesothelioma of 650/100,000 of exposed persons. Similarly, the estimates for amosite and for chrysotile were 90/100,000 and 5/100,000, respectively [14]. The authors concluded that at exposure levels seen in occupational cohorts, the exposure specific risk of mesothelioma from the three principal commercial asbestos types is largely in the ratio of 500:100:1 for crocidolite, amosite, and chrysotile, respectively, and that the risk differential between chrysotile and amphiboles (crocidolite and amosite) with respect to lung cancer was somewhere between 1:10 and 1:50 [14]. However, the study by Hodgson and Darnton was observed to have limitations in that the meta-analysis carried out was based on studies for which the quality of the cohort studies used were not considered and that the range of uncertainty in risks of lung cancer and mesothelioma was wide [24,25]. In a study of textile workers from four plants in North Carolina, USA, in which workers were primarily exposed to chrysotile, Loomis and co-authors reported an overall excess of lung cancer, mesothelioma, and pleural cancer amongst the workers, pointing to the potency of chrysotile asbestos [26,27].

A reduction of exposure to 0.2 f/mL of chrysotile asbestos fibre concentration was reported to result in a lifetime incidence of asbestosis of about 0.5%. Furthermore, by reducing the occupational exposure limit from 2 f/mL to 0.1 f/mL, the risk of cancer mortality was observed to be reduced by 95% from estimated cases, as high as 6411 to 336 deaths per 100,000 workers [18], over a 45-year period of exposure. On the other hand, the incidence of asbestosis was calculated to be 250 cases per 100,000 workers after exposure to asbestos at 0.1 f/mL [18].

Prediction of ARDs can be done through models that use direct or indirect estimates of asbestos exposure [28]. Direct estimates make use of exposed persons where airborne asbestos fibre levels are measured over time while indirect estimates use information about total or fibre-specific imports [28].

The risk associated with exposure to asbestos has been reported to generally follow a linear model wherein relative risk of asbestos disease is linear in dose [23,29–31]. The US Occupational Safety and Health Administration (OSHA) selected a linear model to describe the relationship between the excess relative risk of lung cancer and asbestos exposure [22]. Evidence of the linear dose-response relationship for lung cancer has been observed for several studies with respect to cumulative asbestos exposure in the workplace [18,19,23,32–35]. Additionally, a study by Finkelstein observed that the rate of deaths from mesotheliomas were proportional to the magnitude of cumulative asbestos exposure and that the exposure response data set was linear in dose. Furthermore, Berry et al. and Finkelstein have demonstrated an approximate linear relationship between asbestosis incidences and cumulative exposure [22,36]. These studies and many others upon which OSHA based its risk assessment models to predict asbestos-related cancers demonstrated linear relationships over a range of observations.

The approach using the OSHA Risk Assessment Model as initially conceived with respect to the objective of the study was used with the assumption that a linear relationship possibly exists between

exposure and effect. Furthermore, use of the linear design in the dose-response relationship has also been assumed and applied in this study as it presented some advantages, namely:

- Point estimates (average exposures) can be made without knowledge of the individual exposures in the groups, suggesting that excess mortality of an entire exposed group can be related to the average exposure of the group [30].
- Extrapolation to various exposure circumstances can be made easily.
- It is likely to be a conservative extrapolation in the context of human health [30].

In this study, the direct method was applied to predict asbestos-related mortality cases, namely lung cancer, mesothelioma, and gastrointestinal cancer as well as asbestosis in two AC manufacturing factories in Zimbabwe, as airborne chrysotile asbestos fibre concentrations over the period of 1996 to 2020, reported in a previous study by Mutetwa et al. [37] and captured in a job-exposure matrix, were available [38].

2. Materials and Methods

2.1. The Factories

The study involved two major AC manufacturing factories situated in Harare and Bulawayo. Activities in the two factories included the manufacture of chrysotile cement sheets, facia boards, garden ware, and AC pipes in the case of the Bulawayo factory. The factories were built in the 1940s and 1950s and have been using chrysotile asbestos in the manufacture of AC products since their establishment. Estimates from the factories suggest that between 1996 and 2020, the estimated number of workers ranged from about 390 to 420, with the Harare factory possibly having 156 to 220 workers and Bulawayo factory having 190 to 204 during this period. Within the operational areas studied, approximately more than 100 and 150 workers in Harare and Bulawayo factories, respectively, were in various jobs as indicated in Table S12 (Supplementary Materials). The jobs used in predicting ARDs have been described previously by Mutetwa et al. [37,38]. The main raw materials used in the manufacture of asbestos cement products were cement, water, and chrysotile asbestos. Generally, the AC products contain 10–20% asbestos and 80–90% cement and water.

2.2. Measurement of Airborne Chrysotile Fibre

Measurement of airborne chrysotile asbestos fibres was done by the company's safety and health department using the standard method of the Asbestos International Association (AIA) Reference method for the determination of airborne asbestos fibre concentrations at workplaces by phase contrast light microscopy. In summary, a personal sampling pump set at 1 L/min flowrate was connected to a sampling train, consisting of plastic tubing and a sample holder (cowl) with a 25 mm membrane filter. The whole sampling train of the pump, tubing, sample holder, and filter was hooked to a worker. The pump was then switched on and sampling took place over a period of about four hours, after which the filters were removed, placed at the appropriate labelled slides, and treated with acetone vapour to clear. Using a hypodermic syringe, a drop of triacetin was placed onto the acetone-cleared filters and covered with a cover slip. The treated filters on the slides were stored for 24 h, after which counting of the fibres took place using a phase contrast microscope. The fibres counted were generally longer than 5 μm with a width of less than 3 μm and length-to-width ratio of more than 3:1 [37].

2.3. Chrysotile Exposure Concentration Data

Personal exposure chrysotile concentration data measured for the period of 1996 to 2020 extracted from paper records of the two main AC manufacturing factories in Harare and Bulawayo cities were used to assess possible estimates of ARDs, namely lung cancer, mesothelioma, gastrointestinal cancer, and asbestosis in the AC manufacturing factories. As reported by Mutetwa et al. [38], a total of 3066 personal airborne chrysotile measurements collected from company records, spanning a period of about 25 years from which 1788 monthly mean chrysotile personal exposure concentrations were drawn, constituted the data set. In combination with the US Occupational Safety and Health Administration (OSHA) linear

dose–response relationship model, chrysotile personal exposure concentrations were used to generate estimates of cancer mortality cases and asbestosis cases.

2.4. Application of OSHA’s Risk Assessment Models

Table 1 below, reproduced from OSHA linear dose risk assessment models, provides insight into estimates of asbestos cancer mortality per 100,000 exposed workers [18,19,22].

Table 1. Estimates of asbestos-related cancer mortality per 100,000 exposed workers by number of years exposed and exposure level.

Asbestos Fibre Concentration (f/mL)	Cancer Mortality per 100,000 Exposed Workers			
	Lung	Mesothelioma	Gastrointestinal	Total
1-year exposure				
0.1	7.2	6.9	0.7	14.8
0.2	14.4	13.8	1.4	29.6
0.5	36.1	34.6	3.6	74.3
2.0	144	138	14.4	296.4
4.0	288	275	28.8	591.8
5.0	360	344	36.0	740.0
10.0	715	684	71.5	1470.5
20-year exposure				
0.1	139	73	13.9	225.9
0.2	278	146	27.8	451.8
0.5	692	362	69.2	1123.2
2.0	2713	1408	271.3	4392.3
4.0	5278	2706	527.8	8511.8
5.0	6509	3317	650.9	10,476.9
10.0	12,177	6024	1217.7	13,996.7
45-year exposure				
0.1	231	82	23.1	336.1
0.2	460	164	46.0	670.0
0.5	1143	407	114.3	1664.3
2.0	4416	1554	441.6	6411.6
4.0	8441	2924	844.1	12,209.1
5.0	10,318	3547	1031.8	14,896.8
10.0	18,515	6141	1851.5	26,507.5

Source: OSHA, 1986; MSHA, 2005 and 2008.

For lung cancer, mesothelioma, gastrointestinal cancer, and asbestosis, OSHA generally relied on a relative risk model that was linear in cumulative exposure/dose. Linear regression equations established for lung cancer, mesothelioma, and gastrointestinal cancer by plotting

estimates of cancer mortality cases versus respective cumulative exposures (Figures 1–3) were applied to estimate possible cancer mortality cases that may arise due to exposure levels depicted in Table 2.

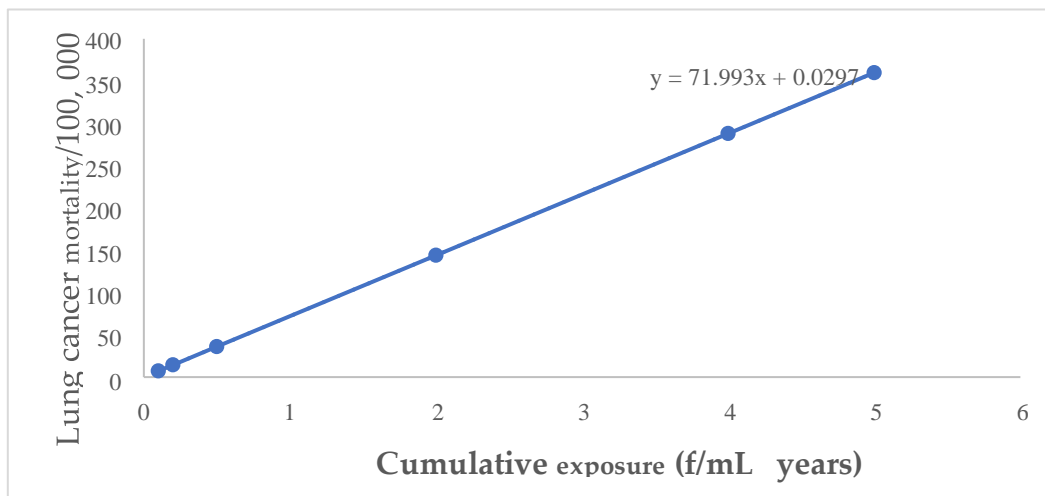


Figure 1. Lung cancer mortality by cumulative exposure linear regression equation.

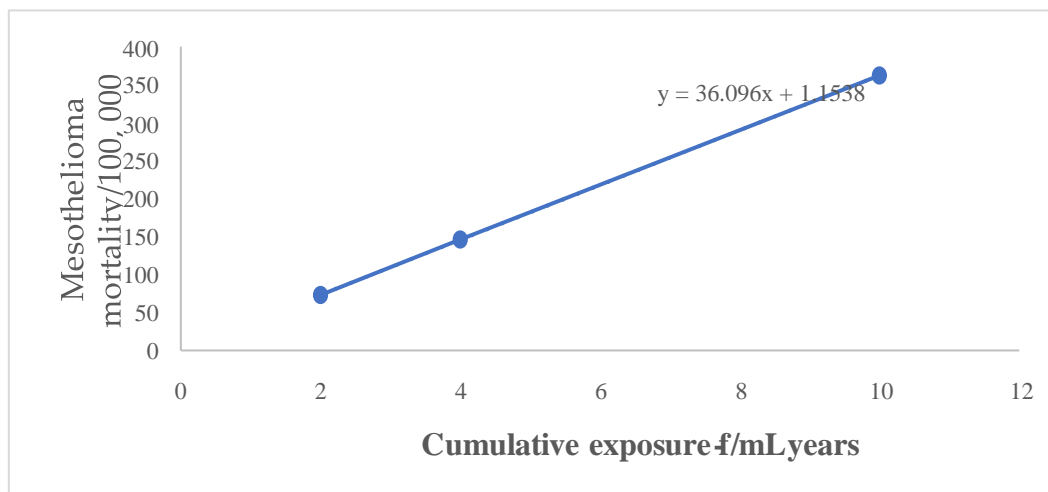


Figure 2. Mesothelioma mortality by cumulative exposure linear regression equation.

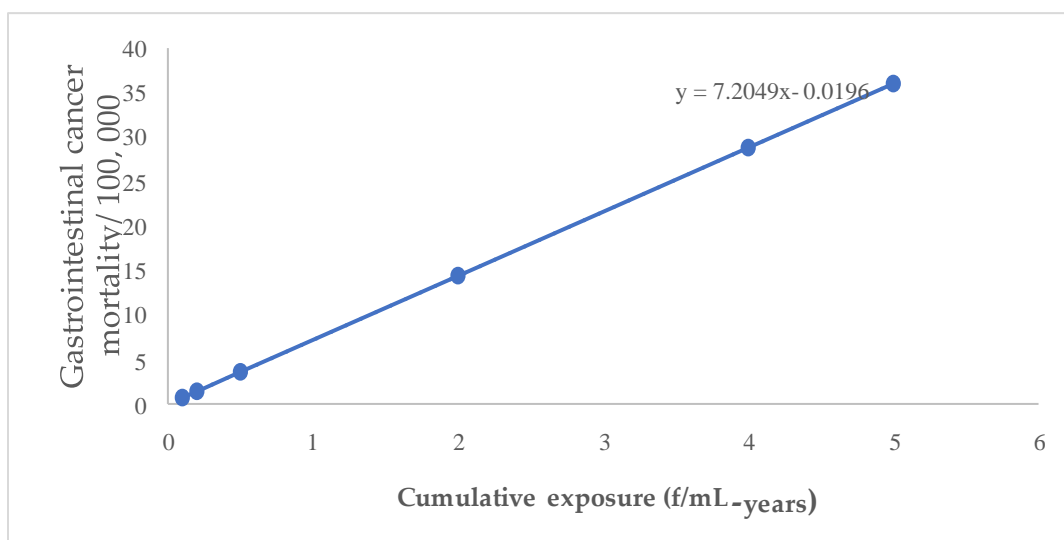


Figure 3. Gastrointestinal cancer mortality by cumulative exposure linear regression equation.

For asbestosis, the linear cumulative dose equation, $R_a = m(f)(d)$, where R_a -predicted incidence of asbestosis, m -slope of linear regression taken as 0.055, f -asbestos fibre concentration, and d -duration of exposure [18,19], was used to estimate possible asbestosis cases over the respective duration of exposure.

Additionally, it has been assumed that at the average exposure concentrations indicated for respective time periods in Table 2 and further assuming that such average exposures may be experienced for durations of 1, 10, 20, and 25 years, cancer mortality cases might be observed after such duration of exposures. Exposure data collected spanned about 25 years, hence the exposure duration capped at 25 years. Furthermore, Table 2 below, adapted from the study by Mutetwa et. [38], was also used to predict ARDs by drawing exposure concentrations from various time periods for various jobs in the two AC manufacturing factories.

2.5. Data Analysis

Data analysis was conducted using IBM SPSS version 26. Mean personal exposure concentrations per time period and by key jobs in a particular factory location as described in Table 1 by Mutetwa et al. [38] were used to derive cancer mortality cases after exposure duration periods of 1, 10, 20, and 25 years as well as possible estimates of asbestosis cases after workers were exposed for a period of 25 years. The arithmetic mean was used as a representative value for analysis of the measurements as this is normally taken as the best summary measure of exposure in epidemiological studies of chronic diseases when adopting a linear exposure response model [6,39].

Table 2. Abridged job-exposure matrix based on mean airborne chrysotile exposure data per time period and place for the period of 1996–2020.

Job	Job Description	Time Period	Harare	Bulawayo
			Factory	Factory
			Mean (f/mL)	Mean (f/mL)
Saw cutting operator (1023)	Cutting by saw asbestos sheets and fascia boards to size	1996–2000	0.19 ± 0.01	0.17 ± 0.02
		2001–2008	0.13 ± 0.02	0.12 ± 0.02
		2009–2016	0.07 ± 0.02	0.06 ± 0.02
		2018–2020 *	0.10 ± 0.02	0.05 ± 0.01
Fettling table operator	Scrapping off small protrusions and polishing AC moulded goods to make them smooth	1996–2000	0.12 ± 0.04	0.17 ± 0.06
		2001–2008	0.12 ± 0.02	0.12 ± 0.03
		2009–2016	-	-
		2018–2020 *	0.11 ± 0.03	-
Moulded goods operator	Moulding of AC goods under wet conditions	1996–2000	0.11 ± 0.04	-
		2001–2008	0.11 ± 0.04	-
		2009–2016	0.05 ± 0.01	-
		2018–2020 *	0.11 ± 0.02	-
Kollergang operator	Opening of and loading chrysotile bags into mouth of process machine and operating machine to move chrysotile into production line	1996–2000	0.13 ± 0.04	0.14 ± 0.03
		2001–2008	0.12 ± 0.02	0.12 ± 0.01
		2009–2016	0.07 ± 0.02	0.07 ± 0.03
		2018–2020 *	0.12 ± 0.01	0.06 ± 0.01
Ground hard waste operator	Feeding AC waste materials into grinder machine	1996–2000	0.16 ± 0.03	0.13 ± 0.04
		2001–2008	0.13 ± 0.03	0.11 ± 0.04
		2009–2016	0.07 ± 0.02	0.07 ± 0.02
		2018–2020 *	0.12 ± 0.01	0.06 ± 0.02
Laundry room operator	Laundering of PPC using wash machine	1996–2000	0.13 ± 0.03	-
		2001–2008	0.13 ± 0.02	-
		2009–2016	0.05 ± 0.01	-
		2018–2020 *	0.11 ± 0.02	-
Pipe joints operator	Lathe machining of AC joints pipes	1996–2000	-	0.13 ± 0.04
		2001–2008	-	0.11 ± 0.01
		2009–2016	-	0.05 ± 0.02
		2018–2020	-	0.05 ± 0.02
Full-length pipe operator	Lathe machining and polishing of full-length AC pipe joints	1996–2000	-	0.13 ± 0.04
		2001–2008	-	0.11 ± 0.01
		2009–2016	-	0.07 ± 0.02
		2018–2020	-	-
Multi-cutter operator	Cutting full-length pipes into collars for coupling pipes	1996–2000	-	0.13 ± 0.04
		2001–2008	-	0.12 ± 0.01
		2009–2016	-	0.07 ± 0.03
		2018–2020	-	0.04 ± 0.01

Source: Mutetwa et al., 2022 [33]; * Care and maintenance of equipment and cleaning.

4 Results

Following the application of the linear regression equations derived from Table 1 as a result of the OSHA cancer risk assessment model for asbestos occupational exposure to the ambient chrysotile asbestos concentration data and cumulative exposures therein gathered from the AC manufacturing factories, Table 3 shows a summary of overall predicted cancer mortality cases (lung cancer, mesothelioma, and gastrointestinal cancer) as well as incidence rates which may be experienced after being exposed at the mean exposure levels associated with each respective time period for each respective job. In the Supplementary Materials, Tables S1 and S2 (lung cancer), Tables S3 and S4 (mesothelioma), and Tables S5 and S6 (gastrointestinal cancer) show the estimates for the specific asbestos-related cancer mortality cases predicted after 1, 10, 20, and 25 years of exposure, respectively, and by time period. **Table 3.** Estimates of the summary of total cancer mortality cases by job and time period.

Table 3. Estimates of the summary of total cancer mortality cases by job and time period.

Job	Time Period	Mean (f/mL)	Harare Factory					Bulawayo Factory				
			Total Cancer Cases per 100,000 Exposed/(% Incidence)					Total Cancer Cases per 100,000 Exposed/(% Incidence)				
			1 Year	10 Years	20 Years	25 Years	Mean (f/mL)	1 Year	10 Years	20 Years	25 Years	
Saw cutting operator	1996–2000	0.19	23 (0.02)	219 (0.22)	439 (0.44)	555 (0.56)	0.17	21 (0.02)	197 (0.20)	394 (0.39)	497 (0.50)	
	2001–2008	0.13	16 (0.02)	151 (0.15)	301 (0.30)	382 (0.38)	0.12	15 (0.02)	137 (0.14)	277 (0.28)	347 (0.35)	
	2009–2016	0.07	9 (0.01)	81 (0.08)	163 (0.16)	209 (0.21)	0.06	8 (0.01)	63 (0.06)	137 (0.14)	174 (0.17)	
	2018–2020	0.10	13 (0.01)	116 (0.12)	231 (0.23)	289 (0.29)	0.05	7 (0.01)	59 (0.06)	116 (0.12)	151 (0.15)	
Fettling table operator	1996–2000	0.12	15 (0.02)	137 (0.14)	277 (0.28)	347 (0.35)	0.17	21 (0.02)	197 (0.20)	394 (0.39)	497 (0.56)	
	2001–2008	0.12	15 (0.02)	137 (0.14)	277 (0.28)	347 (0.35)	0.12	15 (0.02)	137 (0.14)	277 (0.28)	347 (0.40)	
	2009–2016	-	-	-	-	-	-	-	-	-	-	
	2018–2020	0.11	14 (0.01)	128 (0.13)	255 (0.26)	324 (0.32)	-	-	-	-	-	
Moulded goods operator	1996–2000	0.11	14 (0.01)	128 (0.13)	255 (0.26)	324 (0.32)	-	-	-	-	-	
	2001–2008	0.11	14 (0.01)	128 (0.13)	255 (0.26)	324 (0.32)	-	-	-	-	-	
	2009–2016	0.05	7 (0.01)	59 (0.06)	116 (0.12)	151 (0.15)	-	-	-	-	-	
	2018–2020	0.11	14 (0.01)	128 (0.13)	255 (0.26)	324 (0.32)	-	-	-	-	-	
Kollergang operator	1996–2000	0.13	16 (0.02)	151 (0.15)	301 (0.30)	381 (0.38)	0.14	17 (0.02)	163 (0.16)	324 (0.32)	404 (0.40)	
	2001–2008	0.12	15 (0.02)	137 (0.14)	277 (0.28)	347 (0.35)	0.12	15 (0.02)	137 (0.14)	277 (0.28)	347 (0.35)	
	2009–2016	0.07	9 (0.01)	81 (0.08)	163 (0.16)	209 (0.21)	0.07	9 (0.01)	81 (0.08)	163 (0.16)	209 (0.21)	
	2018–2020	0.12	15 (0.02)	137 (0.14)	277 (0.28)	347 (0.35)	0.06	8 (0.01)	63 (0.06)	137 (0.14)	174 (0.17)	
Ground hard waste operator	1996–2000	0.16	20 (0.02)	186 (0.19)	370 (0.37)	463 (0.46)	0.13	16 (0.02)	151 (0.15)	301 (0.30)	381 (0.38)	
	2001–2008	0.13	16 (0.02)	151 (0.15)	301 (0.30)	381 (0.38)	0.11	14 (0.01)	128 (0.13)	255 (0.26)	324 (0.32)	
	2009–2016	0.07	9 (0.01)	81 (0.08)	163 (0.16)	209 (0.21)	0.07	9 (0.01)	81 (0.08)	163 (0.16)	209 (0.21)	
	2018–2020	0.12	15 (0.02)	137 (0.14)	277 (0.28)	347 (0.35)	0.06	8 (0.01)	63 (0.06)	137 (0.14)	174 (0.17)	
Laundry room operator	1996–2000	0.13	16 (0.02)	151 (0.15)	301 (0.30)	382 (0.38)	-	-	-	-	-	
	2001–2008	0.13	16 (0.02)	151 (0.15)	301 (0.30)	382 (0.38)	-	-	-	-	-	
	2009–2016	0.05	7 (0.01)	59 (0.06)	116 (0.12)	151 (0.15)	-	-	-	-	-	
	2018–2020	0.11	14 (0.01)	128 (0.13)	255 (0.26)	324 (0.32)	-	-	-	-	-	
Pipe joints operators	1996–2000	-	-	-	-	-	0.13	16 (0.02)	151 (0.15)	301 (0.30)	381 (0.38)	
	2001–2008	-	-	-	-	-	0.11	14 (0.01)	128 (0.13)	255 (0.26)	324 (0.32)	
	2009–2016	-	-	-	-	-	0.05	7 (0.01)	59 (0.06)	116 (0.12)	151 (0.15)	
	2018–2020	-	-	-	-	-	0.05	7 (0.01)	59 (0.06)	116 (0.12)	151 (0.15)	

Table 3. Cont.

Job	Time Period	Mean (f/mL)	Harare Factory					Bulawayo Factory				
			Total Cancer Cases per 100,000 Exposed/(% Incidence)	1 Year	10 Years	20 Years	25 Years	Mean (f/mL)	Total Cancer Cases per 100,000 Exposed/(% Incidence)	1 Year	10 Years	20 Years
Full-length pipe operator	1996–2000	-	-	-	-	-	0.13	16 (0.02)	151 (0.15)	301 (0.30)	381 (0.38)	
	2001–2008	-	-	-	-	-	0.11	14 (0.01)	128 (0.13)	255 (0.26)	324 (0.32)	
	2009–2016	-	-	-	-	-	0.07	9 (0.01)	81 (0.08)	163 (0.16)	209 (0.21)	
Multi-cutter operator	1996–2000	-	-	-	-	-	0.13	16 (0.02)	151 (0.15)	301 (0.30)	381 (0.38)	
	2001–2008	-	-	-	-	-	0.12	15 (0.02)	137 (0.14)	277 (0.28)	347 (0.34)	
	2009–2016	-	-	-	-	-	0.07	9 (0.01)	81 (0.08)	163 (0.16)	209 (0.21)	
Overall factory		0.11	14 (0.01)	128 (0.13)	255 (0.26)	324 (0.32)	0.12	15 (0.02)	137 (0.14)	277 (0.28)	347 (0.35)	

The predicted cancer cases highlighted in Table 3 are based on Tables S1–S6 of Supplementary Materials. For instance, assuming that exposures associated with the time period of 1996 to 2000 for saw cutting operator in the Harare factory on average persists for 1 year, Table S1 (lung cancer) shows 13.7 cases per 100,000, Table S3 (mesothelioma) shows 8 cases, and Table S5 (gastrointestinal cancer) shows 1.3 cases per 100,000 workers. These cases added, i.e., 13.7 + 8 + 1.3, give 23 cases per 100,000. This process was repeated for all operators across all exposure periods of 1, 10, 20, and 25 years to come up with the summary for Table 3.

The predicted cancer cases highlighted in Table 3 are based on Tables S1–S6 of Supplementary Materials. For instance, assuming that exposures associated with the time period of 1996 to 2000 for saw cutting operator in the Harare factory on average persists for 1 year, Table S1 (lung cancer) shows 13.7 cases per 100,000, Table S3 (mesothelioma) shows 8 cases, and Table S5 (gastrointestinal cancer) shows 1.3 cases per 100,000 workers. These

cases added, i.e., $13.7 + 8 + 1.3$, give 23 cases per 100,000. This process was repeated for all operators across all exposure periods of 1, 10, 20, and 25 years to come up with the summary for Table Tables S1–S6 (Supplementary Materials) show that the incidence of lung cancer, mesothelioma, and gastrointestinal cancer after 1 year of exposure is very low, ranging from as low as 0.0003 to 0.01% in almost all jobs if it is assumed that workers were to be exposed at various exposure levels associated with particular time periods. Furthermore, in tandem with increased cumulative exposure after 20 years of exposure, predicted cancer mortality cases show increased occurrence of asbestos-related cancers if it is assumed that exposed workers were to work for 20 years or more at levels obtained at each respective time period. As reported by Mutetwa et al. [37,38], for the Harare factory, exposure levels show a decreasing trend from 1996 to 2016; however, from 2018 to 2020, exposure levels show an upward trend, with the results that predicted cancer cases for the period of 2018 to 2020 in the Harare factory being higher compared to the preceding time period of 1996 to 2016.

For the Bulawayo factory, nonetheless with exposure concentrations showing a downward trend from 1996 to 2020, there are decreasing levels shown of predicted cancer (i.e., lung cancer, mesothelioma, and gastrointestinal cancer) mortality cases if exposed workers in various jobs work at the obtaining exposure levels associated with the respective time periods.

The results in Table 3 show that more cancer cases are likely to be experienced among those exposed before 2008, as exposure levels and subsequently cumulative exposure were generally much higher than those experienced after 2008.

Table 4 shows estimates of possible asbestosis incidences likely to be observed at various cumulative exposures after a 25-year duration of exposure and by job and time period. Cumulative exposure levels range from 1.0 to 4.8 f/mL-years, with the highest cumulative exposures being exhibited at the saw cutting operations in both factories, followed by ground hard waste operations particularly before 2008. Kollergang operations in both factories, laundry operations (Harare factory), and pipe sections operations before 2008 also exhibit relatively high cumulative exposures with concomitant increased predicted asbestosis cases. Asbestosis cases likely to be detected after a 25-year duration of exposure range from 50 to 260 cases per 100,000 (0.05% to 0.26% incidence of asbestosis) exposed workers for various jobs. Overall, on average in both factories, asbestosis cases likely to be detected were within the range of 150–170 per 100,000 workers exposed (0.15% to 0.17% incidence) to asbestosis. Tables S7–S10 (Supplementary Materials), showing overall summary estimates of cancer mortality cases by factory and duration of exposure, suggest that 15, 139, 278, and 347 cases per 100,000 workers (saw cutting, fettling table, ground hard waste, and laundry operators, respectively) of asbestos-related cancers may be experienced after 1, 10, 20, and 25 years of exposure, respectively, in the Harare factory. The Bulawayo factory with similar or the same order of magnitude of cumulative exposure levels to those obtained in the Harare factory also displays a similar trend wherein the same or similar number of cancer cases per 100,000 workers exposed are likely to be experienced at saw cutting operators, fettling table operators, as well as at pipe section after 1, 10, 20, and 25 years of exposure.

Table 4. Cumulative exposure and asbestosis incidence by time period and after 25 years of exposure.

Job	Time Period	Harare Factory				Bulawayo Factory			
		Mean Concentration (f/mL)	CE	% Incid	Case/100 × 10 ³	Mean Concentration (f/mL)	CE	% Incid	Case/100 × 10 ³
Saw cutting operator	1996–2000	0.19	4.8	0.26	260	0.17	4.3	0.24	240
	2001–2008	0.13	3.3	0.18	180	0.12	3.0	0.17	170
	2009–2016	0.07	1.8	0.10	100	0.06	1.5	0.08	80
	2018–2020	0.10	2.5	0.14	140	0.05	1.3	0.07	70
Fettling table operator	1996–2000	0.12	3.0	0.17	170	0.17	4.3	0.24	240
	2001–2008	0.12	3.0	0.17	170	0.12	3.0	0.17	170
	2009–2016	-	-	-	-	-	-	-	-
	2018–2020	0.11	2.8	0.15	150	-	-	-	-
Moulded goods operator	1996–2000	0.11	2.8	0.15	150	-	-	-	-
	2001–2008	0.11	2.8	0.15	150	-	-	-	-
	2009–2016	0.05	1.3	0.07	70	-	-	-	-
	2018–2020	0.11	2.8	0.15	150	-	-	-	-
Kollergang operator	1996–2000	0.13	3.3	0.18	180	0.14	3.5	0.19	190
	2001–2008	0.12	3.0	0.17	180	0.12	3.0	0.17	170
	2009–2016	0.07	1.8	0.10	100	0.07	1.8	0.10	100
	2018–2020	0.12	3.0	0.17	170	0.06	1.5	0.08	80
Groundhard waste operator	1996–2000	0.16	4.0	0.22	220	0.13	3.3	0.18	180
	2001–2008	0.13	3.3	0.18	180	0.11	2.8	0.15	150
	2009–2016	0.07	1.8	0.10	100	0.07	1.8	0.10	100
	2018–2020	0.12	3.0	0.17	170	0.06	1.5	0.08	80
Laundry room operator	1996–2000	0.13	3.3	0.18	180	-	-	-	-
	2001–2008	0.13	3.3	0.18	180 70	-	-	-	-
	2009–2016	0.05	1.3	0.07	150	-	-	-	-
	2018–2020	0.11	2.8	0.15	-	-	-	-	-
Pipe joints operator	1996–2000	-	-	-	-	0.13	3.3	0.18	180
	2001–2008	-	-	-	-	0.11	2.8	0.15	150
	2009–2016	-	-	-	-	0.05	1.3	0.07	70
	2018–2020	-	-	-	-	0.05	1.3	0.07	70
Full-length pipe operator	1996–2000	-	-	-	-	0.13	3.3	0.18	180
	2001–2008	-	-	-	-	0.11	2.8	0.15	150
	2009–2016	-	-	-	-	0.07	1.8	0.10	100
	2018–2020	-	-	-	-	-	-	-	-
Multicutter operator	1996–2000	-	-	-	-	0.13	3.3	0.18	180
	2001–2008	-	-	-	-	0.12	3.0	0.17	300
	2009–2016	-	-	-	-	0.07	1.8	0.10	100
	2018–2020	-	-	-	-	0.04	1.0	0.05	50
Overall factory		0.11	2.8	0.15	150	0.12	3.0	0.17	170

% Incid—percentage incidence; CE—cumulative exposure in f/mL-years.

4. Discussion

The study attempts to predict possible ARDs in the chrysotile AC manufacturing factories in Zimbabwe by applying the OSHA linear dose–response relationship (OSHA cancer risk assessment model) for lung cancer, mesothelioma, gastrointestinal cancer, and asbestosis. The predicted ARDs drawn through the application of OSHA’s cancer risk assessment tool [18,19,22] provided possible estimates of cancer mortality cases for workers exposed to chrysotile asbestos fibres at various cumulative exposures if exposed at various exposure concentrations obtained at various time periods.

The results suggest that saw cutting operators followed by kollergang and ground hard waste operators in both factories may be at an increased risk of developing ARDs, as results (Tables 3 and 4) show a high number of predicted cancer mortality cases and asbestosis cases especially if workers were to be exposed before 2008, where exposure concentrations and subsequently cumulative exposures were high compared to exposure concentrations for the time period of 2009 to 2020, if duration of exposure of 20 years or 25 years is applied.

The results also suggest that over a possible exposure period of 25 years, high exposure concentrations experienced before 2008 and, subsequently, cumulative exposures may possibly yield high asbestos-related cancers and asbestosis as reflected in Tables S1–S6 (Supplementary Materials).

The predicted cancer cases reported in this study for 1-, 10-, 20- and 25-year durations of exposure are much lower compared to those reported by Jafari et al. [40], who also applied the OSHA linear dose response, in which there were 499 cases per 100,000 workers after 1 year of exposure and 6965 cases per 100,000 workers after 20 years of exposure [35]; and are also lower than those reported by Magnani and Leporati [41]. The high cancer cases as reported by Jafari et al. reflect high cumulative exposures at 1- and 20-year durations of exposure compared to those obtained in this study. It is insightful to note that the cumulative exposures in this study are quite low compared to cumulative exposures recorded in the 1970s where exposures as high as 200–300 f/mL-years were recorded [37], compared to maximum cumulative exposures of 4.3 to 4.8 f/mL-years for an exposure duration as high as 25 years (Table S10, Supplementary Materials) recorded in our study. Such high cumulative exposures in the past would suggest high levels of asbestos-related cancers and asbestosis compared to those predicted in this study.

Cases of ARDs in Zimbabwe have been reported mainly emanating from the mines and mills. Twenty-seven (27) had ARDs, 21 individuals had evidence of asbestosis with one related to asbestos cement manufacturing, while 3 had possible mesothelioma, and 3 possibly had lung cancer/non-malignant pleural diseases. The authors further indicated that the results presented some limitations as they reported that there was under-recognition bias introduced by looking at workers’ compensated derived cases, with the health system having no capacity to follow-up workers who quit or retired from the chrysotile asbestos industry [8]. The levels of ARDs predicted in the study by Cullen and Baloyi may be difficult to infer with respect to our study as the cases were largely from the mines and mills.

On average, over a 25-year duration of exposure, 150 and 170 cases (0.15–0.17% incidence) of asbestosis per 100,000 workers in the Harare and Bulawayo factory, respectively, may develop asbestosis at overall cumulative exposures of 2.8 f/mL-years for Harare and 3.0 f/mL-years for the Bulawayo factory.

For the time period of 1996 to 2000, the period that presents the highest exposure concentrations and subsequently high cumulative exposures, saw cutting, kollergang, fettling table, ground hard waste, and possibly pipe making operational areas may experience the highest cancer incidence rates after 20 years and possibly after a period of 25 years in both factories. Indeed, based on the estimated number of workers in the various jobs as illustrated in Table S12 (Supplementary Materials), and in particular considering just the jobs with the possible highest exposures, cancer incidence cases which may be experienced following exposures associated with the time period of 1996 to 2000, may be 0.24% (saw cutting operators), 0.04% (kollergang operators), and 0.02% (ground hard waste) for the Harare factory after 20 years of exposure. After 25 years of exposure, the same operational areas of saw cutting, kollergang, and ground hard waste, based on the estimated number of workers in the various jobs, also exhibited high cancer incidence rates of 0.30%, 0.05, and 0.03%, respectively, compared to other operational areas. The Bulawayo factory follows a similar pattern for the three operational areas, with saw cutting recording a high incidence rate of 0.27% compared to other operational areas in this factory

after 20 years of exposure. Additionally, as a result of high exposure concentrations and, in turn, high cumulative exposure after 25 years, the three operational areas may experience 0.14%, 0.02%, and 0.01% asbestosis cases among saw cutting, kollergang, and ground hard waste operators, respectively, in the Harare and Bulawayo factories. Given the estimated maximum total number of workers with the job title of saw cutting over both the factories (Table S12) and exposure levels experienced by saw cutting operators, it cannot be excluded that cancer cases may have occurred.

Asbestos-related cancers derived from this study may possibly need to be taken in the context of what may constitute a significant risk to health. OSHA [23] has considered that the risk of cancer mortality of more than 1 case per 1000 workers from occupational causes presents a significant risk. Hence, from Table S10 (Supplementary Materials), which depicts overall possible cancer cases over a 25-year duration of exposure, it may be deduced that the cumulative exposures in both factories, though seemingly low compared to past exposures reported elsewhere [19,42], may possibly present some significant health risk if evaluated against the OSHA significant risk criteria, as overall cancer mortality cases ranged from 2.89 to 3.47 cases in the case of Harare factory operational areas while for the Bulawayo factory, risk of cancer mortality ranged from 2.89 to 4.63 cases per 1000 workers exposed. However, if consideration is given to other risk levels such as four cases per 1000 workers exposed (The Netherlands and Germany) [43,44], then the risk range obtained above for this study may not necessarily be significant. The cancer cases in this study, per 1000 appear elevated as a result of high chrysotile exposures experienced in the earlier time period of 1996 to 2008. Moreover, as elaborated by the OSHA, while some significant risk remains at the OEL of 0.1 f/mL, health risk may also be significantly reduced at or below the OEL of 0.1 f/mL compared to exposures obtaining in the earlier years of the 1960s, 1970s, and 1980s and early 1990s in which exposures could range from 0.2–0.5 f/mL or even higher. Additionally, the risk of asbestos-related cancer may also be further reduced by reducing exposure levels as indicted in Tables S1–S6 (Supplementary Materials), particularly for the Bulawayo factory, in which exposure concentrations over the years from 1996 to 2020 have been on a downward trend to the extent that exposures within the range of 0.04 to 0.07 f/mL were being realised during the time period of 2018 to 2020. This may suggest that cancer cases may possibly approach one case or less per 1000 workers as exposure concentrations decrease and if all possible control measures are implemented to the fullest extent possible. Moreover, studies on cancer mortality have demonstrated that mortality estimates from asbestos-related cancers of any type decrease significantly when exposure is reduced [18,19]. For instance, during the time period of 2009 to 2016 in the Harare factory, at exposure concentrations of 0.05 to 0.07 f/mL depending on the job, 0.9–1.3 lung cancer cases, 0.5–0.7 mesothelioma cases, and 0.09–0.13 gastrointestinal cancers per 1000 workers exposed may be experienced. Similarly for the period of 2018 to 2020, in the case of the Bulawayo factory in which exposure concentrations ranged from 0.04 to 0.06 f/mL over an exposure period of 25 years, 0.7 to 1.1 lung cancer cases, 0.4 to 0.6 mesothelioma cases, and about 0.07 to 0.11 gastrointestinal cases per 1000 workers may also be realised, taking into account exposure concentrations and cumulative exposures reflected in Tables S1–S6 (Supplementary Materials) for the time period of 2009 to 2020. These risk rates may possibly be low if evaluated against the four cases in 1000 workers exposed taken with respect to Netherlands or Germany as acceptable excess cancer risk levels. Zimbabwe does not have a benchmark or reference point for excess cancer risk and might as well consider the four cases in 1000 workers as starting point to consider as part of occupational cancer management policy. Furthermore, health risks presented by exposure concentrations reflected in this study may be minimised further or reduced by effective use of engineering controls, good occupational hygiene practices, and use of appropriate respiratory protective equipment and overall sustained implementation of an occupational safety and health management system across all areas of AC manufacturing processes.

ARDs predicted in this study are based on a fairly large personal airborne chrysotile fibre concentration data set spanning a period of about 25 years and thus provides a reasonable measure of cumulative exposures and possible relationship with diseases which may be associated with various jobs. Cumulative asbestos exposures obtained in this study may be considered as low and hence may provide a good basis for further studies on chrysotile exposure–response relationships relating to asbestos cancer morbidity or mortality cases in Zimbabwe. The study further provides unique insights into the possible estimates of ARDs as it relates to lung cancer if a chrysotile exposure mortality and or

morbidity study is carried out in the future, more so that cumulative exposures may generally give robust answers to the existence of an association, regardless of the underlying true mechanism of disease. Although the OSHA risk assessment tool or dose response relationship model shows that excess risk is linear in dose, excess cancer mortality or morbidity cases may not really be linear especially at high exposure levels. Another limitation of the study was that the possible number of ARDs depicted in this study may also not really reflect the true ARDs cases, as the OSHA risk assessment was also based on studies that included a significant amount of amphibole asbestos, which are generally considered to have a higher potency than chrysotile asbestos [14,29], while chrysotile asbestos used in the AC manufacturing industries in Zimbabwe has been mainly chrysotile, and thus the disease experience which is exhibited in the AC manufacturing factories in Zimbabwe may be different from workplace settings associated with studies used in the development of the OSHA risk assessment model. Furthermore, the epidemiological data used in developing a linear dose relationship for human exposure to asbestos are limited as a result of the fact that current health effects are largely a result of past exposures when exposure controls were inadequate and exposure measurements were imperfect, assuming a number of scenarios were in some cases conversion factors with uncertainties that were applied in expressing asbestos fibre concentrations in f/mL. The dose response data with respect to gastrointestinal cancer have also been limited compared to those for lung cancer and mesothelioma.

Limitations of the OSHA risk assessment approach as applied in this study also include uncertainty in risk by extrapolation from high occupational exposure levels to much lower levels, mass to fibre conversion factors used in modelling the linear in dose response models, and variability in exposure estimates that are built into the linear dose models [30]. Despite these limitations, the personal exposure data used in this study and applied in the context of the OSHA risk assessment linear dose relationship, provide some possible estimates of asbestos disease under the exposure circumstances that have been obtained over the years in the chrysotile asbestos cement manufacturing factories in Zimbabwe.

5 Conclusions

The results suggest that saw cutting operators, followed by kollergang and ground hard waste operators, in both factories may be at increased risk of developing ARDs, as a high number of predicted cancer mortality cases and asbestosis cases, especially if workers were exposed before 2008, where exposure concentrations and subsequently cumulative exposure, would be high compared to exposure concentrations for the time period of 2009 to 2020, assuming that workers continued to be exposed for more than 20 years from around the 1990s, as regular chrysotile exposure data began to be available in the early to mid-1990s. A duration of 20 years or more is commonly associated with a long latency period of ARDs [1,2].

Furthermore, ARDs predicted in this study, although they were generally lower than reported in other studies that used the OSHA risk assessment model, underline the need for asbestos mortality and or morbidity studies in Zimbabwe with the aim of a comprehensive inquiry of a comparative analysis of predicted and actual mortality cases that may be obtained in the AC manufacturing industry in Zimbabwe. It is essential that mortality and/or morbidity epidemiology studies be further carried out to establish the possible actual disease burden associated with exposure to chrysotile asbestos in the AC manufacturing factories in Zimbabwe.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/ijerph20010058/s1>. Table S1: Cumulative exposure on lung cancer risk by job, time period, exposure duration of 1, 10, 20, and 25 years: Harare factory; Table S2: Cumulative exposure on lung cancer risk by job, time period, exposure duration of 1, 10, 20, and 25 years: Bulawayo factory; Table S3: Cumulative exposure on mesothelioma risk by job, time period, exposure duration of 1, 10, 20, and 25 years: Harare factory; Table S4: Cumulative exposure on mesothelioma risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Bulawayo factory; Table S5: Cumulative exposure on gastrointestinal cancer risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Harare factory; Table S6: Cumulative exposure on gastrointestinal cancer risk by job, time period, exposure

duration of 1, 10, 20 and 25 years: Bulawayo factory; Table S7: Overall summary estimates of cancer mortality cases by factory, job and duration of exposure of 1 year.; 8. Table S8: Overall summary estimates of cancer mortality cases by factory, job and duration of exposure of 10 years.; Table S9: Overall summary estimates of cancer mortality cases by factory, job and duration of exposure of 20 years.; Table S10: Overall summary estimates of cancer mortality cases by factory, job and duration of exposure of 25 years.; Table S11: Estimates of possible asbestosis incidence after 25 years of exposure; Table S12: Estimated number of workers working at various jobs in the chrysotile asbestos cement manufacturing factories

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Data Availability Statement: The dataset used in this study are available from the corresponding author on reasonable request. The datasets are not publicly available to maintain confidentiality of the factories used in the study.

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References

1. World Health Organization (WHO). Chrysotile Asbestos. 2014. Available online: <https://www.who.int/publications/i/item/9789241564816>. (accessed on 1 November 2022).
2. International Agency for Research on Cancer (IARC). *IARC Monograph on Evaluation of Carcinogenic Risks to Human, Arsenic, Metals, Fibres and Dust: A Review of Human Carcinogens*; IARC/WHO: Lyon, France, 2012; Volume 100, pp. 219–309.
3. International Programme on Chemical Safety (IPCS). *Chrysotile Asbestos. Environmental Health Criteria 203*; UNEP: Geneva, Switzerland; ILO: Geneva, Switzerland; WHO and IPCS: Geneva, Switzerland, 1998.
4. an Oyen, S.C.; Peters, S.; Alfonso, H.; Fritschi, L.; De Klerk, N.H.; Reid, A.; Franklin, P.; Gordon, L.; Benke, G.; Musk, A.W. Development of a Job-Exposure Matrix (AsbJEM) to Estimate Occupational Exposure to Asbestos in Australia. *Ann. Occup. Hyg.* **2015**, *59*, 737–748. [[CrossRef](#)] [[PubMed](#)]
5. Peters, S.; Vermeulen, R.; Portengen, L.; Olsson, A.; Kendzia, B.; Vincent, R.; Savary, B.; Lavoué, J.; Cavallo, D.; Cattaneo, A.; et al. SYN-JEM: A Quantitative Job Exposure Matrix for Five Lung Carcinogens. *Ann. Occup. Hyg.* **2016**, *60*, 795–811. Available online:

- <https://academic.oup.com/annweh/article-abstract/60/7/795/2196/93> (accessed on 14 February 2018). [CrossRef] [PubMed]
6. Choi, S.; Kang, D.; Park, D.; Lee, H.; Choi, B. Developing Asbestos Job Exposure Matrix Using Occupation and Industry Specific Exposure Data (1984–2008) in Republic of Korea. *Saf. Health Work.* **2017**, *8*, 105–115. [CrossRef] [PubMed]
 7. Hyland, R.A.; Yates, D.H.; Benke, G.; Sim, M.; Johnson, A.R. Occupational exposure to asbestos in New South Wales, Australia (1970–1989): Development of an asbestos task exposure matrix. *Occup. Environ. Med.* **2010**, *67*, 201–2016. Available online: <http://oem.bmj.com/> (accessed on 8 March 2018). [CrossRef]
 8. Cullen, M.R.; Baloyi, S.B. Chrysotile asbestos and Health in Zimbabwe: Analysis of Mines and Millers compensated for Asbestos–Related Diseases since Independence (1980). *Am. J. Ind. Med.* **1991**, *19*, 161–169. [CrossRef]
 9. Luberto, F.; Ferrante, D.; Silvestri, S.; Angelini, A.; Cuccaro, F.; Nannavecchia, A.M.; Oddone, E.; Vicentini, M.; Barone-Adesi, F.; Magnani, C.; et al. Cumulative asbestos exposure and mortality from asbestos related diseases in a pooled analysis of 21 asbestos cement cohorts in Italy. *Environ. Health* **2019**, *18*, 71. [CrossRef]
 10. Pierce, J.S.; Ruestow, P.S.; Finley, B.L. An updated evaluation of reported no-observed adverse effect levels for chrysotile asbestos for lung cancer and mesothelioma. *Crit. Rev. Toxicol.* **2016**, *46*, 561–586. [CrossRef]
 11. van Der Bij, S.; Koffijberg, H.; Lenters, V.; Portengen, L.; Moons, K.G.M.; Heederik, D.; Vermeulen, R.C.H. Lung cancer risk at low cumulative asbestos exposure: Meta-regression of the exposure-response relationship. *Cancer Causes Control.* **2013**, *24*, 1–12. [CrossRef]
 12. Pierce, J.S.; McKinley, M.A.; Paustenbach, D.J.; Finley, B.L. An evaluation of reported no-effect chrysotile asbestos exposures for lung cancer and mesothelioma. *Crit. Rev. Toxicol.* **2008**, *38*, 191–214. [CrossRef]
 13. Gasparrini, A.; Pizzo, A.M.; Gorini, G.; Costantini, A.S.; Silvestri, S.; Ciapini, C.; Innocenti, A.; Berry, G. Prediction of mesothelioma and lung cancer in a cohort of asbestos exposed workers. *Eur. J. Epidemiol.* **2008**, *23*, 541–546. [CrossRef]
 14. Hodgson, J.T.; Darnton, A. The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. *Ann. Occup. Hyg.* **2000**, *44*, 565–601. [CrossRef] [PubMed]
 15. Albin, M.; Jakobsson, K.; Attewell, R.; Johansson, L.; Welinder, H. Mortality and cancer morbidity in cohorts of asbestos cement workers and referents. *Br. J. Ind. Med.* **1990**, *47*, 602–610. [CrossRef] [PubMed]
 16. Finkelstein, M.M. Potential pitfall in using cumulative exposure in exposure-response relationships: Demonstration and discussion. *Am. J. Ind. Med.* **1995**, *28*, 41–47. [CrossRef]
 17. Berry, G. Prediction of mesothelioma, lung cancer, and asbestosis in former Wittenoom asbestos workers. *Br. J. Ind. Med.* **1991**, *48*, 793–802. [CrossRef]
 18. Mine Safety and Health Administration (MSHA); USA Department of Labour, Mine Safety and Health Administration. Asbestos Exposure Limit; Final Rule. 2008. Available online: <https://www.govinfo.gov/content/pkg/FR-2008-02-29/pdf/E8-3828.pdf#page=22> (accessed on 18 February 2022).
 19. Mine Safety and Health Administration (MSHA); USA Department of Labour, Mine Safety and Health Administration. Asbestos Exposure Limit; Final Rule. 2005. Available online: <https://www.govinfo.gov/content/pkg/FR-2005-07-29/pdf/05-14510.pdf> (accessed on 18 February 2022).
 20. Browne, K. A threshold for asbestos related lung cancer. *Br. J. Ind. Med.* **1986**, *43*, 556–558. [CrossRef]

21. Browne, K. Chrysotile: Threshold of risk. Paper Presented at an International Seminar on Safety in the Use of Chrysotile Asbestos: Basis for Scientifically Based Regulatory Action, Havana, Cuba, 12–13 September 2000.
22. Berry, G.; Gilson, J.C.; Holmes, S.; Lewinsohn, H.C.; Roach, S.A. Asbestosis: A study of dose-response relationships in an asbestos textile factory. *Br. J. Ind. Med.* **1979**, *36*, 98–112. [[CrossRef](#)] [[PubMed](#)]
23. Occupational Safety and Health Administration; US Department of Labor. 51 FR 22 612—Occupational Exposure to Asbestos, Tremolite, Anthophyllite and Actinolite. Final Rule. 1986. Available online: <https://www.grsm.com/Templates/media/files/pdf/51%20fr%2022612.pdf> (accessed on 20 February 2022).
24. Health Council of the Netherlands (HCN). *Asbestos: Risks of Environmental and Occupational Exposure*; Publication no. 2010/10E; Health Council of the Netherlands: The Hague, The Netherlands, 2010; Available online: <https://www.healthcouncil.nl/documents/advisory-reports/2010/06/03/asbestos-risks-of-environmental-and-occupational-exposure> (accessed on 3 August 2022).
25. Lenters, V.; Vermeulen, R.; Dogger, S.; Stayner, L.; Portengen, L.; Burdorf, A.; Heederik, D. A meta-analysis of asbestos and lung cancer: Is better quality exposure assessment associated with steeper slopes of the exposure-response relationships? *Environ. Health Perspect.* **2011**, *119*, 1547–1555. [[CrossRef](#)]
26. Loomis, D.; Dement, J.M.; Wolf, S.H.; Richardson, D.B. Lung cancer mortality and fibre exposures among North Carolina asbestos textile workers. *Occup. Environ. Med.* **2014**, *71*, 228, Correction on *Occup. Environ. Med.* **2009**, *66*, 535–542. [[CrossRef](#)]
27. Smith, A.H.; Wright, C.C. Chrysotile asbestos is the main cause of pleural mesothelioma. *Am. J. Ind. Med.* **1996**, *30*, 252–266. [[CrossRef](#)]
28. Reid, A. Review of the Effectiveness of Predictive Models for Mesothelioma to Identify Lessons for Asbestos-Related Policy. The Australia and New Zealand School of Government. 2016. Available online: <https://www.anzsog.edu.au/resource-library/research/evidence-base-journal> (accessed on 16 March 2018).
29. Berman, D.W.; Crump, K. Technical Support Document for a Protocol to Assess Asbestos Related Risk. Environmental Protection Agency (EPA), USA. 2004. Available online: <https://nepis.epa.gov/Exe/ZyPDF.cgi/901D0S00.PDF?Dockey=901D0S00.PDF> (accessed on 20 March 2022).
30. Environmental Protection Agency (EPA). *Airborne Asbestos Health Assessment Update*; EPA 600/8-84/003F; Environmental Criteria and Assessment Office: Research Triangle Park, NC, USA, 1986. Available online: <https://nepis.epa.gov/Exe/ZyNET.exe/20009EBT.TXT?ZyActionD=ZyDocument&Client=EPA&Index=1981+Thru+1985&Docs=&Query=&Time=&EndTime=&SearchMethod=1&TocRestrict=n&Toc=&TocEntry=&QField=&QFieldYear=&QFieldMonth=&QFieldDay=&IntQFieldOp=0&ExtQFieldOp=0&XmlQuery=&File=D%3A%5Czyfiles%5CIndex%20Data%5C81thru85%5CTxt%5C00000002%5C20009EBT.txt&User=ANONYMOUS&Password=anonymous&SortMethod=h%7C-&MaximumDocuments=1&FuzzyDegree=0&ImageQuality=r75g8/r75g8/x150y150g16/i425&Display=hpfr&DefSeekPage=x&SearchBack=ZyActionL&Back=ZyActionS&BackDesc=Results%20page&MaximumPages=1&ZyEntry=1&SeekPage=x&ZyPURL> (accessed on 10 August 2022).
31. Stayner, L.; Smith, R.; Bailer, J.; Gilbert, S.; Steenland, K.; Dement, J.; Brown, D.; Lemen, R. Exposure-response analysis of risk of respiratory disease associated with occupational exposure to chrysotile asbestos. *Occup. Environ. Med.* **1997**, *54*, 646–652. [[CrossRef](#)]

32. Selikoff, I.J. Asbestos disease-1990-2020: The risks of asbestos risk assessment. *Toxicol. Ind. Health* **1991**, *7*, 117–127. [[CrossRef](#)] [[PubMed](#)]
33. Dement, J.M.; Harrism, R.L., Jr.; Symons, M.J.; Shy, C. Estimates of dose-response for respiratory cancer among chrysotile asbestos textile workers. *Ann. Occup. Hyg.* **1982**, *26*, 869–887.
34. Henderson, V.L.; Enterline, P.E. Asbestos exposure: Factors associated with excess cancer and respiratory disease mortality. *Ann. N. Y. Acad. Sci.* **1979**, *330*, 117–126. [[CrossRef](#)] [[PubMed](#)]
35. Liddell, F.D.K.; McDonald, J.C.; Thomas, D.C.; Cunliffe, S.V. Methods of Cohort Analysis: Appraisal by Application to Asbestos Mining. *J. R. Stat. Society. Ser. A (Gen.)* **1977**, *140*, 469–491. [[CrossRef](#)]
36. Finkelstein, M.M. Asbestosis in long-term employees of an Ontario asbestos-cement factory. *Am. Rev. Respir Dis.* **1982**, *125*, 496–501. [[CrossRef](#)] [[PubMed](#)]
37. Mutetwa, B.; Moyo, D.; Brouwer, D. Trends in Airborne Chrysotile Asbestos Fibre Concentrations in Asbestos Cement Manufacturing Factories in Zimbabwe from 1996 to 2016. *Int. J. Environ. Res. Public Health* **2021**, *18*, 10755. [[CrossRef](#)] [[PubMed](#)]
38. Mutetwa, B.; Moyo, D.; Brouwer, D. Job Exposure Matrix for Chrysotile Asbestos Fibre in the Asbestos Cement Manufacturing (ACM) Industry in Zimbabwe. *Int. J. Environ. Res. Public Health* **2022**, *19*, 2680. [[CrossRef](#)]
39. Seixas, N.S.; Robins, T.G.; Moulton, L.H. The use of geometric and arithmetic mean exposures in occupational epidemiology. *Am. J. Ind. Med.* **1988**, *14*, 465–477. [[CrossRef](#)]
40. Jafari, M.J.; Karimi, A.; Mohammad, A.B. Prediction of cancer mortality by evaluation of asbestos fibers concentrations in an asbestos-cement products factory. *Iran J. Environ. Health Sci. Eng.* **2010**. Available online: https://www.researchgate.net/publication/47734184_Prediction_of_cancer_mortalityby_evaluation_of_asbestos_fibers_concentrations_in_an_asbestoscement_products_factory/citation/download (accessed on 20 November 2021).
41. Magnani, C.; Leporati, M. Mortality from lung cancer and population risk attributable to asbestos in an asbestos cement manufacturing town in Italy. *Occup. Environ. Med.* **1998**, *55*, 111–114. [[CrossRef](#)]
42. Peto, J. The incidence of pleural mesothelioma in chrysotile asbestos textile workers. *IARC Sci. Publ.* **1980**, *30*, 703–707.
43. Whittaker, C.; Dankovic, D.A. What is an Acceptable Risk of Cancer due to Occupational Exposure to a Carcinogen? 2015. Available online: <https://www.cdc.gov/niosh/nioshtic-2/20045880.html> (accessed on 4 August 2022).
44. Whittaker, C. The NIOSH Chemical Carcinogen Policy: Classifying Chemical Carcinogens and Establishing a Target Risk Level. 2019. Available online: <https://www.dir.ca.gov/dosh/DoshReg/5155-Meetings/NIOSH-Chemical-Carcinogen-Policy.pdf> (accessed on 29 August 2022).

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CHAPTER 9

Examining approaches to prevention of exposure to chrysotile asbestos and the debate on ban of asbestos.

Examining approaches for prevention of exposure to chrysotile asbestos and perspectives on the debate on ban of asbestos.

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Abstract

Background

Asbestos exposure being a risk factor for asbestosis, lung cancer and mesothelioma causation evokes fear, anxiety and panic in many workplace settings around the world. This paper examines approaches for prevention of exposure to asbestos and presents some perspectives on banning all forms of asbestos including chrysotile use.

Methods and Materials

Literature materials that advocate for the complete ban of all forms of asbestos including chrysotile as the only means of control of exposure and that which argues for the controlled use approach were reviewed. Words used in literature search were chrysotile asbestos exposure, asbestos-cement, ban asbestos, controlled use, asbestos related disease, mesothelioma, lung cancer and asbestosis.

Discussion

Different perspectives have been presented in which one position argues that there exist major differences in health risk between amphiboles and chrysotile asbestos. It is further argued that low exposure and risk experienced under today's workplace conditions are completely different to high-risk exposures experienced in the past where occupational hygiene conditions were very poor and levels of education, awareness and training in the asbestos industry was low and that there is low levels of exposure below which risk of health impairment becomes insignificant,

hence controlled use approach as a measure of exposure control can be successfully applied. However, the other position holds that all forms of asbestos including chrysotile are equipotent, that there is no safe level of exposure, that controlled use is not practical and that there is no merit in continuing use of chrysotile asbestos in light of safer alternatives available today. Both positions appear plausible. Banning as a form of control measure occupies a high level in the hierarchy of controls with potential to eliminate the hazard and risk, nonetheless the banning of chrysotile asbestos has been substituted with materials that have been reported to carry health risk of cancer. On balance, banning may possibly not be the panacea of elimination of ARDs, more so that some other forms of mining such as diamond and gold mining have been associated with exposure to amphibole asbestos. On the other hand, the controlled use approach may well provide real possibilities of prevention of exposure to levels that presents minimal risk to health if effectively implemented as applied to a range of occupational hazards with success.

Conclusion

While consideration may be given to banning chrysotile asbestos use, controlled use approach may possibly remain a choice for a measure to prevent exposure to chrysotile asbestos to levels that may not present an unreasonable risk.

1. Background

Asbestos is a generic term for a group of naturally occurring silicates that principally include serpentine variety (white chrysotile asbestos), the amphiboles variety consisting of crocidolite (blue asbestos) amosite (brown asbestos), anthophyllite, actinolite and tremolite. (IARC, 2012).

The World Health Organization reports that 125 million people worldwide are exposed to asbestos at the workplace with 107 000 people succumbing to asbestos related diseases annually (WHO, 2014). Despite this being of occupational, public and environmental health concern, chrysotile, one of the three major forms of asbestos continues to be produced and used in some countries. While production and use of asbestos in most developed countries notably Europe has significantly declined in recent years as a result of health concerns and bans on many of its uses, there continues to be extensive production, sale and use of chrysotile asbestos in South and Central America, Asia and Africa (ATSDR, 2001; WHO, 2014). Russia, in the Ural Mountains, is the world leading producer of chrysotile asbestos among others that include China, Kazakhstan, Brazil, India and Zimbabwe though production at Zimbabwe chrysotile mines stalled in 2010 due to economic challenges. Currently there are efforts to resuscitate the mining of chrysotile asbestos with chrysotile mining dumps being used to harness and produce chrysotile asbestos fibre for the two-chrysotile asbestos cement manufacturing factories in Zimbabwe.

1.1 Sources of Occupational Exposure to Chrysotile Asbestos.

Chrysotile asbestos is found in many serpentine rock formations and because of natural weathering processes and anthropogenic sources, chrysotile asbestos is found in air, water and land masses (IPCS, 1998; IARC, 2012). Anthropogenic sources include mining and milling, processing of chrysotile into products such as friction materials, asbestos cement pipes and sheets, gaskets and seals, construction and transportation of fibre (IPCS, 1998; IARC, 2012). Chrysotile used today involves mainly production of products where the chrysotile is encapsulated into matrices. Most chrysotile cement products contain 10-15% of fibre with the rest being water and cement (IPCS, 1998). Emission sources of chrysotile asbestos fibre during production of chrysotile cement products includes feeding of asbestos fibre into the mix, blending the mix, and cutting or machining of sheets (IPCS, 1998; IARC, 2012; WHO, 2014).

1.2 Structure and properties of chrysotile asbestos

Chrysotile asbestos, being naturally occurring fibrous hydrated magnesium silicate which belongs to the serpentine category of asbestos minerals generally carries the composition $\text{Mg}_3\text{Si}_2\text{O}_5(\text{OH})_4$ (IPCS, 1998, Pollastri et al., 2016). The composition is largely a sheet of silicates with brucite layers ($\text{Mg}(\text{OH})_2$) (IPCS, 1998; Bernstein, et al, 2013). Chrysotile asbestos is a rolled sheet or concentric rings of silicate centred tetrahedra with the magnesium on the outside of the sheet and the silica on the inside (Bernstein et al., 2013). The silicate layer has a tetrahedron embedded in a pseudo hexagonal network (Pollastri et al., 2016; Bernstein, et al., 2013; Yarborough, 2006). Additionally, the structure resembles a cylinder which cylinders results in chrysotile fibrils which make up the chrysotile fibre. The magnesium element makes a layer on the outside of the cylindrical role and dissolves in biological systems. Additionally, it has been reported that chrysotile is highly soluble in aqueous neutral or acidic environment. The magnesium turns to leach from the outside brucite layer at a pH of less than 7 or generally in dilute acids (Atkinson, 1973; Langer and Pooley, 1973; Morgan and Crolley, 1973; Jaurand et al., 1977; IPCS, 1998) while amphiboles are far more resistant to leaching and hence have a much longer residence time in the lung and pleura (Hesterberg et al., 1998; Korchevskiy, 2019; Paustenbach et al., 2021). It has also been argued that the magnesium is readily attacked by the acidic environment of macrophages which have a pH of 4-4.5 and the magnesium disintegrates from the crystalline structure leaving the new unstable silicate sheet which breaks up and decomposes into small particles which are easily cleared from the lungs by macrophages (Oze and Solt, 2010; Bernstein et al., 2013). The biological half-life of chrysotile fibres has been observed to be only days to weeks or months while that for the amphiboles the half-life has been observed to be in the range of years to decades (deKlerk et al., 1996; Finkelstein and Dufresne, 1999; Bernstein and Hoskins, 2006; Paustenbach et al., 2021; ECHA, 2021). As a result of these and other physiochemical differences, the risk of developing ARDs due to exposure to chrysotile is generally considered to be much lower than being exposed to amphiboles (McDonald and McDonald, 1980; (Paustenbach et al., 2021).

Recently it has however been argued that asbestos fibres including chrysotile remain in the lungs where their sustained presence causes lung disease pointing out that continuous irritation of the tissue results in chronic inflammation to which the tissue responds by scar formation (Feber et al., 2016). It was further pointed out that it is not only the amphibole fibres that can be identified in lung tissue over many years, but also chrysotile fibres, as is typical of pneumoconiosis due to asbestos fibres. Despite chrysotile asbestos being substance that remain

under intense discussion, the study reported that high bio persistence was not only associated with amphibole but also chrysotile asbestos in the human lung and thus gives mechanistic explanations for the toxicity of the fibre and the long latency period of asbestos related diseases (Feber et. al., 2016). In a study on the kinetics of release of metals during the acellular *in vitro* dissolution of chrysotile, crocidolite and fibrous erionite, it was established that since chrysotile undergoes fast dissolution with respect to crocidolite and fibrous erionite in the lungs it behaves like a carrier that releases its metals' ion cargo in the lung environment, mimicking the phenomenon that explains the toxicity of nanoparticles. Hence, the toxicity of chrysotile should also take into account the release of toxic metals in the intracellular/extracellular medium during the rapid dissolution process (Gualtieri et al., 2019).

It is important to note that asbestos fibres can be considered as hash or soft depending on fibre flexibility. Generally, amphibole asbestos fibres are considered hash while chrysotile can be considered to be between hash and soft, however most chrysotile fibres are considered soft (Badollet, 1948; IPCS, 1998). The soft nature of chrysotile may necessitate the easy with which it dissolves in biological systems and breaks up into smaller pieces (Bernstein et al., 2013).

Chrysotile asbestos ore is generally associated with other mineral contaminants such as iron, brucite, mica, feldspar, talc and carbonate minerals including magnetite, calcite and zeolites (Langer and Nolan, 1994; IPCS, 1998). Some amphibole fibres have also been associated with chrysotile, key among them being tremolite. Tremolite has been found at 0.01% to 0.6% levels with an average of 0.09% tremolite in about 35% of samples analysed (Addison and Davies, 1990; IPCS, 1998). Such trace contaminants are an important factor in disease causation (IPCS, 1998). Fibre type, size, chemical and crystalline composition, and bio-durability are among some critical variables that have been considered to influence the potential toxicity of asbestos fibre (Berman and Crump, 2008; Barlow et.al., 2017), and that such determinants may inform measures of control that may be taken to prevent exposure.

1.3 Health Effects of Chrysotile Asbestos

All forms of asbestos including chrysotile have been associated with an increased risk of pneumoconiosis, lung cancer and mesothelioma in several epidemiological studies of exposed employees (IPCS, 1998; Landrigan et al., 1999; Nicholson, 2001; IARC, 2012).

Invariably, the main concern in the past has been non-malignant lung disease such as asbestosis, which is a disease associated with diffuse interstitial pulmonary fibrosis which was accompanied by varying degrees of pleural involvement (IPCS, 1998; IARC, 2012). Severe forms of asbestosis were generally associated with the high exposures, however with improved technology; severe asbestosis became less common with focus shifting towards syndromes exhibiting fibrosis of the small and large airways rather than of the large lung parenchyma (Dreesen et al., 1938; IPCS, 1998).

Several studies have also demonstrated changes in radiological and functional changes in workers exposed primarily to chrysotile fibre in mining and asbestos cement industries in which the presence of small opacities and pleural changes were observed with increased cumulative exposure (Rubino et al., 1979; McDemott et al., 1982; Viallat et al., 1982; Enarson et al., 1988; Jones et al., 1989; Cullen and Baloyi, 1991; IPCS, 1998). Within exposure concentration range of 0.3 and 1.1 f/ml, exposure to chrysotile asbestos over 40 years has been shown to result in asbestosis (Berry et al., 1979; Huang et al., 1990; IPCS, 1998).

In a study of 1176 Swedish workers who used almost 100% chrysotile, 11 cases of lung cancer were observed compared to 9 expected and no deaths were attributed to mesothelioma. Overall, cumulative exposure was 18 f/ml-year among 10% workers sampled (Ohlson & Hogstedt, 1985). Six cases of malignant mesothelioma were also identified in Norway from 1970 to 1979 in a chrysotile asbestos cement plant which also to some extent used very small amounts of crocidolite and amosite (Glyseth et al., 1983). In another cohort study of 3057 male workers who were followed from 1953 to 1982 in an asbestos cement plant in which 90% chrysotile and 10 % crocidolite was used in Northern Israel, it was observed that the cohort had an elevated risk of lung cancer, mesothelioma and other malignant neoplasm such as liver, bladder and renal cancers. High ratio of mesothelioma and lung cancer was attributed to high past asbestos exposures and low risk for lung cancer due to possibly early cessation in smoking (Tulchinsky et al., 1999). The incidence of cancer and mortality among workers in essentially chrysotile asbestos cement industry was also studied among 7996 men and 584 women in Denmark between 1928 and 1984 and over 99% were traced. The authors reported during the initial 25 years that exposure levels were generally high ranging from 100 to 1600 times the prevailing Danish exposure limit of 0.5f/ml with a total of 1346 deaths and 612 cases of cancer observed in the country between 1943 and 1984 and that overall incidence of cancer (Observed/Expected 1.22; 95% CI 1.12-1.32) were significantly increased compared with all Danish men. Other cancers observed in this cohort included mesothelioma and laryngeal (Raffn et al., 1989).

2. Examining approaches for prevention of exposure to chrysotile asbestos

2.1 Controlled use approach and question of low exposures

Control of exposure to asbestos has been a contentious issue wherein others argue that it is possible to properly use chrysotile in a manner which is safe since chrysotile in its modern-day high-density applications does not present unreasonable risks to exposed workers and the public. The measure centres on controlled use approach. The approach entails that employers, government and workers have a responsibility in the prevention of exposure (ICA, 2019; ILO, 1984; ILO, 1986). The aforementioned agencies state that employers, government and workers should make effort to reach consensus and understanding on various standards and regulations that assist in the prevention of exposure to asbestos fibre. A Competent Authority (CA) reporting to government should have the determination to support the objectives of safe and responsible approach necessary for the safety of workers as well as establishment of exposure limits, establishment of measurement methods, collect and keep records of measurement results of various entities dealing with chrysotile asbestos (ILO, 1984; ICA, 2019). Additionally, controlled use and safety in the use of chrysotile asbestos fibre implies development and implementation of regulatory frameworks which the CA enforces for the protection of workers against exposure to asbestos and implementation of good work practices. Control of exposure at source using engineering methods wherein mechanical handling, ventilation and redesign of the process to eliminate, contain or collect asbestos dust emissions by such means as

- (a) process separation, automation or enclosure;
- (b) bonding asbestos fibres with other materials to prevent dust generation;
- (c) general ventilation of the working areas with clean air;
- (d) local exhaust ventilation of processes, operations, equipment, and tools for the prevention of dust dissemination.
- (d) separate workplaces processes (ICA, 1984); should be a priority in preventing exposure to chrysotile asbestos fibre.

Furthermore, appropriate good work practices should include: (a) requirements to use and maintain process machinery, installations, equipment, tools, local exhausts and ventilation systems in accordance with instructions.

- (b) damping using water, where appropriate of asbestos products and materials at workplaces before processing, handling, using, machining, cleaning, stripping or removing;
- (c) regular cleaning of machinery and work areas by appropriate wet methods.

(d) proper use of personal protective equipment (ICA, 1984).

Medical surveillance (MS) should also be a necessary programme to inform effective exposure prevention. Medical surveillance should include among others;

- (a) Periodic medical examinations of workers before, during and after retiring from employment,
- (b) Medical examinations done in accordance with internationally recognised standards that of X-rays, lung function tests etc.);
- (c) Informing workers of the results of medical examinations;
- (d) Ensuring that records of workers' medical surveillance are confidentially kept by the occupational health practitioner or physician (ICA, ILO, 1984).

Monitoring of the work environment with respect to exposure levels against set exposure limits for chrysotile asbestos should also be an important part of the controlled use approach. Monitoring of the work environment should be done by properly trained industrial hygienists who apply recognised methods of sampling and counting and ensuring that such monitoring is done regularly (ICA 2019; ILO). 1984 Workers should also receive education and training on hazards and risks associated with chrysotile asbestos, proper use of appropriate respiratory protective equipment, chrysotile asbestos warning signs and other prevention measures thereof (ILO, 1984; ILO, 1986; HSE, 2012; SA Gov, 2020; ICA, 2019; Paustenbach et al., 2021).

The controlled use approach can be expanded to many other areas where chrysotile asbestos exposure is possible. Such other areas include mining and milling of asbestos ore, textile production, asbestos disposal, construction, demolition and alteration works, friction materials production, handling and transportation of asbestos in ports and container terminals and in acoustic insulation (ILO, 1984). The principles of controlled use approach as described above are quite applicable in such circumstances. For instance in packaging and transportation of asbestos material it is important that asbestos fibre should always be packed in impermeable bags with all bags being printed with an approved label identifying the contents as asbestos and carrying a health warning. In open cast, mining dust generated by drilling operations should be controlled by extraction equipment mounted on the drills while dust emissions from blasting should be minimised by the use, wherever practicable, of multiple small blasts rather than one large blast. Roadways should be regularly wetted in order to reduce to a minimum the creation of asbestos dust (ILO, 2014). During underground mining, mining should be carried out only when an adequate water supply is available and workplaces should be kept wet continuously

during work shifts. The floor of every main travelling road underground should be kept wet while hanging and foot walls should be regularly washed down to prevent the accumulation of dust. These among other preventive measures as outlined in the ILO Code of Practice in the Use Asbestos (ILO, 1984). Demolition and alteration work are major sources of exposure if asbestos is present, hence identification of asbestos containing buildings should always be identified first before works are done. Competent persons with full understanding of the controlled use approach (ILO, 1984) should do the demolition works.

The controlled use approach has been applied invariably to known hazards and risks in many countries and is generally at the centre of prevention of exposure to many hazardous substances. The aim in the case of chrysotile asbestos is to ensure that exposure is below occupational exposure limits of airborne chrysotile fibre, below which it is expected that risk of health impairment becomes minimal. Indeed, exposure trends to airborne hazardous factors including asbestos have been shown to decrease over the years from the 1960's when the measurements began to take a much stronger foothold until to date when exposure levels below 0.1 f/ml are quite common in many occupational settings associated with chrysotile asbestos use. Controlled use approach has been linked with low degrees of exposure that suggest minimal or insignificant excess risk with respect to mortality and other asbestos related diseases (Weill et al., 1979; Thomas et al., 1982; Berry & Newhouse, 1983; Ohlson & Hogstedt, 1985; Gardner et al., 1986; Newhouse and Sullivan, 1989; Liddell et al., 1997; Sichletidis et al., 2008; Gibbs & Berry, 2008;). Indeed, in a study on mortality of workers manufacturing friction products it was reported that after 1950, hygienic controls were improved progressively in factories such that from 1970 exposure levels were no more than 0.5-1.0 f/ml. The authors concluded that with good environmental controls, chrysotile asbestos maybe used in the manufacture of AC products without causing excess mortality (Newhouse and Sullivan, 1989). While progress was registered with reduction in exposure levels, it is insightful to noted that recent evaluations demonstrate that exposures as low as 0.1 f/ml, 0.05 f/ml and 0.01 f/ml can still carry considerable excess risk of cancer, with the evaluation indicating that as much as 125, 62 and 25 cancer cases (mesothelioma and lung cancer) per 100 000 exposed persons can be realised respectively (ECHA, 2021). Hence, the application of the controlled use has to be followed and in particular use of respiratory protective clothing must be enforced to minimise risk of ill impairment due exposure to asbestos.

Recently exposure to chrysotile asbestos has been shown to decrease over time in more than two decades from 1996 to 2020 in Zimbabwe chrysotile asbestos cement manufacturing

factories, with decreases being linked to improved occupational safety and health practices over the years (Mutetwa et al., 2021; 2022). Thus, the practical application of the requirements of the controlled use approach suggests that compliance with mandated low occupational exposure limits and the observance of safe work practices is a practical approach that can minimise risks associated with not only to chrysotile asbestos fibre but to other products, substances and fibres which may potentially pose a health risk. Today ILO convention 162 together with its recommendation 172 on safety in the use of asbestos is an international instrument that promotes the controlled use approach and applied together with ILO code of practice on safety in the use of asbestos sets out the preventive and protective measures required for a safe and responsible use of asbestos and has been in force since 1986 (ILO, 1984; ILO, 1986). To understand the full scope of the convention, it is important that it be considered in its entirety. Occupational hygiene standards were quite poor during the early years of asbestos mining, use and handling in the 1930 through 1950 and even in the 1960's (Davis & McDonald 1988). Airborne dust and asbestos fibre were highly uncontrolled with levels as high as 100-6000 f/ml being recorded in some workplaces (Browne, 1986; Davis & McDonalds, 1988). Since the 1950's and 1960's strict exposure limits began to be imposed by law in many countries in responds to the health effects associated with exposure to asbestos in general. Today's exposure limit for chrysotile asbestos in most countries including Zimbabwe is now set at 0.1 f/ml (NSSA, 2017; Pira et al., 2018; Mutetwa et al., 2021). Application of such occupational exposure limits as part of the controlled use approach has also contributed significantly to reduction in exposure to airborne asbestos over the years.

It has been argued that all asbestos related diseases are dose-related implying that the higher the concentration and duration of exposure, the higher the occurrence of morbidity and mortality and that there is an asbestos exposure threshold particularly for chrysotile below which risk of disease or death from exposure is insignificant (Valic, 2002). The possibility of a threshold and or no observed adverse effects level (NOAEL) (highest exposure levels at which there is no statistically significant increased risk of lung cancer or mesothelioma), has been discussed quite extensively (Browne, 1986; Pierce et al., 2008; Nielsen et al., 2014; Pierce et al., 2016,). Furthermore, in an analysis of an updated exposure response relationship for lung cancer and mesothelioma in chrysotile exposed cohorts, it has been reported that the no-observed adverse effect level (NOAEL) for lung cancer ranged from 1.1 to < 20 f/ml-years to 1600-3200 f/ml-years and for mesothelioma, NOAEL ranged from 100-400 f/ml-years to 800-1599 f/ml-years. The best estimates for NOAEL were reported as 89-168 f/ml-years for lung cancer while for mesothelioma NOAEL was reported as 208-415 f/ml-years (Pierce et al., 2008,

2016). Additionally, Beddington and co-workers on classification and regulation of chrysotile asbestos suggested that long term exposures at an exposure level of 0.1 f/ml is likely to be associated with relatively small increase in the risk of lung cancer (Beddington et al., 2011). These studies and others have tended to form and argue the view that the controlled use approach to chrysotile asbestos is plausible and because it has been widely applied to many other hazardous substances, it thus can successfully be applied to chrysotile asbestos. In light of recent evaluations, suggesting that exposure below 0.1 f/ml carry some considerable risk of excess cancer (ECHA, 2021), it is important that exposure controls should be strengthened with rigorous implementation of the controlled use approach in all areas of the production, use and handling of chrysotile asbestos.

The controlled use approach seems also to be motivated by research showing that there exists a threshold for chrysotile fibre below which risk of health impairment becomes insignificant (Pierce et al., 2008; Pierce et al., 2016; Goodman et al., 2020; Mezie et al., 2020; Paustenbach and Brew, 2020; Price, 2020; Paustenbach et al., 2021). On the hand, it has been argued that the controlled use approach cannot be considered to have failed in preventing ARDs., Some current scientific evidence points to the fact that development of cancer including mesothelioma do and can happen spontaneously without environmental carcinogenic factors, with the weight of evidence indicating that age, erionite exposure, genetic predisposition and therapeutic ionising radiation being some risk factors causing mesothelioma occurrence (Tomasetti and Vogelstein, 2015; Tomasetti et., 2017; Attanoos et al., 2018; Carbone et al., 2019; Paustenbach et al., 2021).

It has also been argued that chrysotile substitutes are no safer than chrysotile asbestos itself (Infante et al., 1994; Siemiatycki, 2001) and while some manmade chrysotile substitute fibres are subject to controlled use approach, chrysotile should also be subjected to same preventive measures as the substitutes. Common substitutes for asbestos include p-aramid, polyvinyl alcohol (PVA), cellulose, glass fibre, and polyacrylonitrile (PNA) (Harrison et al., 1999; Camus, 2001; Park, 2018). Furthermore, it is generally understood that key factors of fibre toxicity are dimension (diameter, length, and aspect ratio), durability (bio-persistence) and dose. Additionally, fibre type may also be a factor in determining fibre toxicity (Hesterberg et al., 1998; Harrison et al. 1999; WTO, 2000; ATSDR, 2001; Camus, 2001; Park, 2018). More significantly, bio persistence has been considered as good indicator of pathogenicity of fibres (European Commission, 1997; Hesterberg et al., 1998; Bernstein et al., 2001; Bernstein et al., 2004). Concerns about the safety of asbestos substitutes have been raised in some studies

wherein substitute materials such as p-aramid, cellulose, refractory ceramics and PVA have been cited to possibly exhibit carcinogenic effects in light of them being more bio-persistence than chrysotile asbestos (Hesterberg et al., 1998; WTO, 2000; Camus, 2001; Bernstein, 2013; Park, 2018). For instance, PVA and p-aramid fibres have been reported to be less respirable than chrysotile but more bio persistent than chrysotile, with p-aramid being shown to induce fibrosis and mesothelioma in inoculation studies (Camus, 2001). Moreover, cellulose fibre has been found to be more bio-persistent than chrysotile and refractory ceramic fibres used in complimenting p-aramid materials in brake pads but have been cited to be more carcinogenic than chrysotile (Hesterberg et al., 1994; McConnel, 1994; Camus, 2001). Nonetheless, epidemiological evidence concerning these substitutes is quite limited and the cohorts which have been studied were exposed to very low levels compared to exposures of asbestos workers in the past. Hence, exposure response differences are likely to be experienced between past cohorts and today's cohorts.

Chrysotile has been subject to models that generally overestimate risks largely because of past exposures. Ban of asbestos has been in some cases been the preferred method of choice for prevention, however, because of uncertainties with respect to the cancer-causing characteristics of chrysotile substitutes, it is imperative that the precautionary principle of ban as a preventive measure equally apply to chrysotile substitutes. In this context, a comparative risk approach that considers chrysotile asbestos on an equal level with chrysotile substitutes may possibly better inform a basis for banning chrysotile (Camus, 2001).

2.2 Lifecycle of Chrysotile Asbestos and AC products and Controlled Use Approach

In considering continued use of chrysotile asbestos and asbestos substitutes the principles of life cycle assessment may need to be taken into account. Life cycle assessment (LCA) is a tool used to assess the environmental impacts of a material, product, process or activity throughout its life cycle, from the extraction of raw materials through to processing, transport, use and disposal. LCA is commonly referred to as a "cradle-to-grave" analysis. (ISO 14040, 2006). However, in recent times, it has been extended to cover aspects to do with human health impacts of such industrial processes and activities. The motivation behind LCA has been that government policies and regulations are being informed by life cycle accountability and that businesses are not only responsible for direct production impacts but are also responsible for product, use, transport and disposal impacts, LCA have become increasingly important in evaluating risks throughout the whole cycle of cradle to grave (Urban Environment

Management (UEM) 2022). Literature and studies on LCA pertaining to asbestos and in particular chrysotile asbestos is scanty. However, a study was undertaken on comparative analysis of life cycle assessment of two types of roofing sheets, i.e. chrysotile asbestos containing sheets and polyvinyl alcohol (PVA) fibres containing sheets., taking into account a number of processes that constituted the life cycle of sheets, chief among them being;

- Extraction and processing of chrysotile asbestos
- Manufacture of cement
- Production of cellulose
- Production of PVA fibres
- Manufacture of the roofing sheets
- Transport of materials to manufacturing facility.
- The end of sheets' service time

The method used to assess the life cycle impacts examined three categories of

- Human health with focus on
 - Carcinogenic effects
 - respiratory effects
 - damage on the climate and depletion of ozone layer
 - effects by ionising radiation
- Ecosystem quality with focus on;
 - Damage caused by ecotoxic discharge
 - Damage due to acidification and eutrophication
- Associated to the removal of resources
 - Damage caused by mineral extraction
 - Damage caused by fossil fuel extraction.

The study concluded that chrysotile asbestos containing sheets were economically and ecologically more favourable than PVA containing sheets (Frazao and Fernandes, 2004). This was basically because a much high weight of PVA fibres is needed to provide the sheets with the same rigidity. One further step would be replacing the ACM pipes with full plastic pipes, e.g., PVA pipes, however, this would be beyond the substitution of asbestos only.

An evaluation of the economic, environmental and social cost implications of banning or restricting the use of chrysotile in Sri Lanka was carried with view to inform government proposed policy on replacing chrysotile with substitutes by 2024. It was argued that any attempt

to ban or restrict the most popular chrysotile roofing material that Sri Lanka currently prefers would have serious consequences in terms of the direct and indirect economic costs as well as the social and the environmental impacts that are more difficult to monetise but will have very serious consequences to the society (RIU, 2017). The report further highlights that medical records in Sri Lanka do not seem to suggest that there has been health related issues due to use of imported chrysotile and manufacture of chrysotile sheets despite use and handling of chrysotile or asbestos for more than 60 years in Sri Lanka. The reports further notes that most countries that have banned use of chrysotile were in Europe and some other industrialised nations whose gross domestic products (GDP) are about ten times higher than Sri Lanka. The direct cost to the social and economic fabric was estimated about US\$ 3.3 billion with the value of chrysotile roofing material used and chrysotile roofing inventory being the biggest cost drivers. Indirect costs were reported to be multifaceted and included impact on trade relations with other countries such as Russia where most of the chrysotile is imported. The social and economic impact of banning and restricted use of chrysotile was considered to be severe and damaging to the majority of people who will not be able to afford other alternative more expensive forms of roofing materials. The report further highlights that the environmental impact that comes with banning chrysotile will be quite significant as there will be increased production of clay tiles that will not be sustainable as it could lead to increased landslides in Sri Lanka. Additionally, any policy decision to ban chrysotile asbestos was reported to trigger the possible policies for safe removal and disposal of asbestos material of the Sri Lanka does have capacity for such huge task. The cost of removal, transportation and disposal of all current inventory was reported to cost the government an estimated **US\$ 11.25 billion**, an amount that economy may not be able to afford. Clearly, the social, economic and environmental impact that comes along with banning or restriction in the use of chrysotile as reflected in this report could be a reminder to consider seriously and strengthen the controlled use approach around which the world has learned to apply to many hazardous substances including carcinogenic materials such as silica that has been classified in the same group as chrysotile asbestos.

2.3 The ban asbestos controversy: Question of cancer risk and low levels of exposure to chrysotile asbestos fibre.

Notwithstanding the controlled use approach, it is however argued that this approach is not practical as chrysotile asbestos cannot be used or handled safely (Smith & Wright, 1996; Tossavainen, 1997; Landrigan et al., 1999; Bang et al., 2006; Lemen et al., 2004b; Lin et al.,

2007; Welch et al, 2009), and the only plausible approach to avoid risk is to ban the use of all forms of asbestos including chrysotile (LaDou et al., 2010; WHO, 2014). This prevention approach is underpinned by essentially the following aspects;

- (a) That there is no safe dose or threshold (Stayner et al., 1997; LaDou et al., 2010; IARC, 2012; van der Bij, 2013; WHO, 2014; Markowitz, 2015; Frank, 2016; Soeberg et al., 2016) and b) that since there is no safe dose or threshold then any exposure from any source (Frank, 2016; LaDou et al., 2010) or a single fibre of exposure can cause cancer and thus all asbestos types and exposures are the same with respect to asbestos disease causation.

Furthermore, proponents of ban asbestos including chrysotile indicate that safer substitutes for asbestos exists and therefore must substitute chrysotile in all its uses (LaDou et al., 2010; Park, 2018). Hence, on the basis of these argument critics of chrysotile asbestos believe that the only measure of control of exposure is to ban use of all forms of asbestos including chrysotile (LaDou et al., 2010).

Additionally, in a response to World Trade Organisation (WTO) questions with respect to harmless or less harmfulness of cellulose, PVA or aramid fibres., the EU argues that these fibres have been used for a very long time with no reported health concerns of cancer among the workers exposed whereas asbestos has been the subject of numerous scientific studies because of the large number of cases of disease found in workers over many years. Hence, such fibres qualify to substitute chrysotile asbestos as they present lower respirability than chrysotile (WTO, 2000). In another study it has been further argued that there was no justification in the continued use of chrysotile asbestos in the face of availability of less harmful asbestos substitutes citing the fact that PVA, cellulose and p-aramid were less respirable compared to chrysotile asbestos (Harrison et al., 1999). It has been reported that the diameter of PVA fibres as manufactured are well above the respirable limit of 7µm. Most PVA fibres were reported to have diameters of 10-16 µm compared to 3 µm associated with mineral fibres such as chrysotile asbestos (Harrison et al., 1999), hence the less respirability of PVA. These substitute materials have however been reported to be more biopersistent than chrysotile asbestos (Hesterberg et al., 1994; McConnel, 1994; Camus, 2001; Park, 2018).

Indeed, literature around chrysotile asbestos is extensive, its analysis and appraisal difficult. Inevitably, the argument and counter arguments regarding its use have found expression in medico-legal and political arenas generating more enormous amounts of information material

further increasing the challenges of risk evaluation (Tweedle & McCulloch, 2004). Additionally, evidence of the significant differences in potencies between fibres of chrysotile and amphiboles have led to the introduction of chrysotile hypothesis and amphiboles hypothesis. The chrysotile hypothesis postulates that the human health risks becomes acceptable at sufficiently low levels of exposure to chrysotile while the arguments advanced to support the amphiboles hypothesis have essentially been based on pathologic lung burden studies in humans and on toxicological, mechanistic and epidemiological studies wherein the mesothelioma risk observed among workers exposed to chrysotile have been argued to arise from low levels (< 1%) of tremolite amphibole fibres in commercial chrysotile (Stayner et al.,1996; Valic, 2002). These hypotheses have not been generally supported and in particular by US regulatory agencies and by the Ramazzini Society (Valic, 2002). Although it has been reported that chrysotile is less dangerous than amphiboles in its capacity to cause mesothelioma (Berry, 1999; Landrigan et al., 1999; Dement, 2001; Valic, 2002), these authors consider that lung cancer risk from chrysotile is at least as high as that from amphiboles. On the other hand, Hodgson and Darnton, (2000) concludes that there are differences in potencies in the asbestos type which potencies were observed to be in the ratio of 1:100:500 for chrysotile, amosite and crocidolite respectively. Earlier studies have suggested that at the then exposure levels below 1 f/ml the carcinogenic risk to commercial chrysotile was quite low with reports indicating that no detectable excess risk of mortality was experienced (Hughes and Waggenspack, 1979; Berry and Newhouse, 1983; Ohlson and Hogstedt, 1985; Gardner et al., 1986; Newhouse and Sullivan, 1989; Liddell et., 1997; McDonald et al., 1997; Sichletidis et al., 2008). However, Dement, (2001) maintains that control of exposure to chrysotile should not be different from the other types of asbestos namely amosite and crocidolite, hence the need to ban all forms of its use. Furthermore recent cancer risk assessment suggest that exposure levels even below the widely applied exposure limit of 0.1f/ml still carry risk of lung cancer and mesothelioma, with exposure levels of 0.1, 0.05, 0.02 and 0.01 f/ml carrying excess life time cancer (lung cancer and mesothelioma combined) of 125, 62, 25 and 12 cases per 100 000 exposed persons (ECHA, 2021).

A more radical and substantial argument in support of the chrysotile asbestos being as potent as amphiboles requiring banning all forms of asbestos including chrysotile, and a departure from the dose response of toxicology is the “any exposure theory of causation” or the any fibre theory (Behrens & Anderson, 2008). The any exposure theory contends that since asbestos is a cumulative dose response phenomenon each and every exposure to asbestos during a person’s lifetime irrespective of how small it may be, significantly contributes to the final disease such

as lung cancer, mesothelioma or asbestosis (Behrens & Anderson, 2008). This theory provided an opportunity to plaintiffs in US asbestos litigation cases to sue defendants every year for perceived health damages arising from exposure to asbestos. Such concept of dose goes far back as the 16th century where the “father of toxicology and physician Paracelsus put forward the principle that “the dose makes the poison and that the dose differentiate a poison from a remedy. The concept of dose is widely recognised in both science and legal realm and is considered as an important reasoning approach worthy consideration in assessing whether exposure cause a specific effect (Behrens & Anderson, 2008). In other words, the concept of “the dose makes the poisons” argues or suggests that low doses cannot produce same effect as high doses. The any exposure theory or any fibre theory gained some foothold in US litigation cases wherein plaintiffs were only required to show that one was exposed to asbestos material without even taking into account degree or frequency of exposure (Behrens & Anderson, 2008) Experts representing plaintiffs in litigation cases frequently opined that every exposure or single asbestos fibre a worker received from any occupational or hobby related work was a substantial factor in causing asbestos related diseases (Behrens & Anderson, 2008).

Thus, the any exposure theory or any asbestos fibre theory has the potential to expand asbestos litigation cases to any hazardous substance in any country as long as it is established that there was some form of exposure. In 1997, it was reported that the US was seized by an asbestos litigation crisis in which there was an extraordinary avalanche of claims with many employers forced into bankruptcy as a result of payment to sick individuals believed to have contracted asbestos related diseases (Bernstein, 2003). While there is clear relationship between asbestos and disease, it has been noted that the effect of exposure to asbestos on a particular individual depends on the level of exposure, type of asbestos one is exposed to and duration of exposure. This will be in keeping with the dose makes the poison principle which principle maybe important in understanding disease causation in asbestos, measures of prevention of exposure as well as on appropriate policy for asbestos control interventions.

The aspects around banning, use and handling all forms of asbestos including chrysotile have also been grounded in the linear no threshold (LNT) model of carcinogenesis that contents that every exposure to a carcinogen contributes to a cumulative linear increase in risk of developing cancer even at very low doses of exposure (Golden et al., 2019; Paustenbach et al., 2021). The LNT single hit dose responds model for carcinogenicity and mutagenicity has been in existence since 1956 when it was applied in regulatory risk assessment for radiation and later from 1977 when used for risk assessment for chemicals. However, it has been argued that assumptions

upon which the LNT was based during the early days of its formulation may no longer be valid in view of today's expanded scientific knowledge on molecular biology (Paustenbach et al., 2021). The validity of the LNT has been increasingly questioned with arguments that it has been formulated in a manner that could be scientifically invalid (Doss, 2018; Calabrese, 2019; Costantini & Borremans, 2019; Golden, 2019; Paustenbach et al., 2021). The LNT model is premised on the fact that there is no threshold below which exposure will not increase an individual's risk of developing cancer, an assertion, which has been accepted by a number regulatory agencies (Paustenbach, et al., 2021). However, it has been argued that the application of the LNT model to the real world would imply that a single exposure to any carcinogen such as asbestos has the potential to cause cancer. If such proposition were to be a valid dose-response model, then life on earth maybe be untenable (Golden et al., 2019). Yet the ban policies on all forms of asbestos including chrysotile appear to be premised essentially on the LNT model which model seem to suggest that the dose makes the poison toxicological principle is not applicable to asbestos and indeed chrysotile asbestos fibre itself and hence application of the LNT model to chrysotile asbestos effects with respect to lung cancer and mesothelioma would overestimate the true risk of acquiring these diseases (Paustenbach, et al., 2021)

Recently the EU produced a directive setting binding exposure limits to products classified as carcinogenic to humans (group 1) by IARC. Such a directive compelled member states of the EU to develop appropriate legislative frameworks covering these carcinogens which include among others silica dust, wood dust, chromium VI compounds, vinyl chloride monomer, refractory ceramics, aluminium production, boot and shoe manufacture and iron and steel foundry (EU, 2017). The setting of such binding exposure limits may suggest that among other measures, the controlled use approach may be applied to these carcinogens. Yet despite chrysotile being in the same IARC grouping of carcinogenicities, it has not been included among those carcinogens for which exposure limits have been set and has been banned alongside with other forms of amphiboles. The listing of such carcinogens with exposure limits expected to be complied with by the EU member States do not seem suggesting banning the use of these carcinogens, but perhaps effort should be directed at ensuring that at least exposure under the conditions of use for these carcinogenic substances is below the set exposure limit, below which it is expected that repeated exposure over standard working hours over possibly a working lifetime may result in minimal health risk.

Notwithstanding the provisions of convention 162 on safety in the use of asbestos, ILO in 2006 issued a resolution in support of a global ban on all forms of asbestos including chrysotile (ILO,

2010). However, this resolution appears to contradict the long-standing ILO convention 162 on safety in the use of asbestos which according to this international instrument provides a legal as well as practical guidance for comprehensive preventive measures at the national and enterprise levels in order to protect workers and prevent asbestos related diseases. Although convention 162 does prohibit use of amphiboles and use of loose friable asbestos in such applications as fireproofing, the concept of safe use of chrysotile is reflected in the convention. Additionally, ILO has also produced the code on safety in the use of asbestos to provide guidance on how asbestos in general can be used and handled safely (ILO, 1984). Furthermore, though WHO recognised a differentiated approach to regulating various forms of asbestos consistent with some provisions of ILO convention 162 (ILO, 1986), it has however called for a global ban on all forms of asbestos and their uses as a way to eliminate asbestos related diseases (WHO, 2014).

The call for a global ban on chrysotile asbestos use as a measure of control of exposure may have the potential to trigger or perhaps provide an opportunity to campaign for extending banning to all forms of mining that may present a risk of exposure to asbestos. It has been noted that asbestos fibres in the form of tremolite-actinolite have been identified in the lungs of diamond mine workers and that tremolite-actinolite and chrysotile asbestos were present in the mine tailings of these diamond mines. Furthermore, mesothelioma and pleural plaques were also diagnosed in some diamond mine workers at autopsy suggesting that diamond mine workers (Nelson, 2011), and perhaps mining of other commodities such as platinum may present a real risk of exposure to asbestos. The complex issue which then may arise in banning all activities to do with exposure to asbestos and in particular chrysotile, taken as a measure of prevention of exposure, is the need to call for a ban on all forms of mining which may present risk of exposure to asbestos. This stemming from the fact that some portion of the scientific community has considered that any source of exposure and any cumulative exposure above background cause mesothelioma and lung cancer (LaDou et al., 2010; Frank, 2016).

Consider silica dust exposure, which is widespread in many mining settings especially gold mining. Silica dust is a group 1 carcinogen same as chrysotile asbestos, yet it is subject to generally controlled use approach with respect to prevention of exposure in many workplace settings, nonetheless, there has not been a call for banning of gold mining though silica dust is as carcinogenic as chrysotile and also being a possible cocarcinogen to asbestos.

The parallel with silica dust exposure is brought into perspective to draw attention to the effect that while silica is classified in the same group of carcinogenicity with chrysotile, silica dust's approach to prevent exposure appear to be quite different from that of chrysotile. There has not been necessarily a worldwide call to banning processes that involve exposure to silica dust but rather controlled use approach. Hence within this context, it can be argued that it is plausible to apply the controlled use approach to chrysotile asbestos just like in a number of hazards. It is insightful to note that the IARC list of Group 1 carcinogens include several substances, mixtures and industrial activities that have been classified as carcinogenic, yet there has not been major call to all these except chrysotile. Thus, it may be imperative that any major policy decision such as banning chrysotile be applied uniformly across all applicable hazards. Furthermore, risk management policy decision for regulatory action around hazards should be informed by hazard characterisation coupled with comprehensive assessment that comprises of exposure data over time and actual estimation of the likely risk under actual conditions of use.

Thus, it may be argued that at present day use and current obtaining exposure limit together with effective application of the controlled use approach, risk of asbestos related disease may be adequately controlled. Furthermore, chrysotile asbestos policy may need to take into account risk assessment based on unique features of chrysotile asbestos containing material, the properties of chrysotile versus amphiboles, the present-day exposure levels together with the occupational exposure limits, the modern mining and manufacturing processes and the spirit of ILO convention 162 and its recommendation 172 together with the guidance provided by the ILO code of practice on safety in the use of asbestos. There may be merit in the use of these international instruments in preventing and reducing exposure to chrysotile asbestos to levels that may not bring material health impairment to those exposed. Banning of chrysotile use is still a plausible approach to prevention, however it may be argued that since this approach is generally premised on the fact that there is no safe level and any source of asbestos may mean material health impairment as a result of exposure, some other forms of mining other than asbestos may also need to be banned. Additionally, the any exposure or the linear no threshold model upon which banning as a measure of prevention appear to be generally premised on, may create an avalanche of possible unmerited litigation in other industrial settings as other forms of mining appear to present real risk of exposure to asbestos.

Conclusion

Asbestos exposure evokes fear, panic and anxiety across a broad range of societies around the world (Mossman et al., 1990). In the medical and scientific community, opinions on the toxicity and carcinogenicity of asbestos differ sharply. One position asserts that asbestos is extremely toxic and carcinogenic in all its forms including chrysotile, that there is no safe level of exposure, that exposure to asbestos cannot be adequately controlled in the workplace and that safer alternatives to asbestos are available and hence the only plausible measure of control is banning use of all forms of asbestos including chrysotile. On the other hand, the other position holds that there is a significant difference in risk by asbestos type or fibre type, nature of exposure, that high risks identified in epidemiological historic cohort studies are not comparable to risks under current exposure conditions. Additionally, this school of thought argues that the substitutes for asbestos may not be any safer than asbestos, pointing out that the current evidence suggests that some of the asbestos substitutes are carcinogenic. The current conditions under which asbestos substitutes are produced and used are not comparable to conditions under which chrysotile and other forms of asbestos were used in the past and thus their risk profile may differ due to the minimal hygiene conditions which existed in the past, thus drawing a possible need for a comparative approach in applying similar exposure controls, procedures and rules to chrysotile and its substitutes. On balance, banning can still be considered as a way to eliminate ARDs, but may not necessarily be the panacea for prevention of ARDs, more so that some other forms of mining such as diamond and gold mining have been associated with exposure to amphibole asbestos. Controlled use approach may perhaps still present real possibilities of prevention of exposure to levels that may present minimal risk to health impairment if effectively implemented as applied to a range of hazards with some success. In applying the controlled use approach, it is important that the entire life cycle of chrysotile asbestos need to be considered from mining, to manufacturing, repairs and demolitions and waste management. This will ensure minimal exposure throughout the entire product life cycle.

REFERENCES

Addison J & Davies LS (1990). Analysis of amphibole asbestos in chrysotile and other minerals. *The Annals of Occupational Hygiene*, 34(2), 159–175. <https://doi.org/10.1093/annhyg/34.2.159>.

Agency for Toxic Substances and Diseases Registry (ATSDR) (2001). Asbestos - Production, Import, Use and Disposal. CDC. USA. Available online at <https://www.atsdr.cdc.gov/toxprofiles/TP.asp?id=30&tid=4>. (Accessed 11/7/2018).

American Conference of Governmental Industrial Hygienist (ACGIH) (2014). Threshold limit values for chemical substances and physical agents and biological exposure indices. Cincinnati, OH: American Conference of Governmental Industrial Hygienists.

Arthur L. Frank Affidavit, State of Pennsylvania County of Philadelphia. State of Pennsylvania County of Philadelphia (2016). Available from: <https://drexel.edu/dornsife/academics/faculty/Arthur%20Frank/>. (Accessed on 15 April 2022).

Atkinson RJ (1973). Chrysotile asbestos: colloidal-silica surfaces in acidified suspensions. *J Colloid Interface Sci.* 42: 624 -628.

Badollet (1948). Research on asbestos fibre. *Can Min Metal Trans.* 51: 48-51.

Barlow CA, Grespin M & Best EA (2017). Asbestos fiber length and its relation to disease risk. *Inhalation Toxicology*, 29(12-14), 541–554. <https://doi.org/10.1080/08958378.2018.1435756>.

Bang KM, Pinheiro GA, Wood JM & Syamlal G (2006). Malignant mesothelioma mortality in the United States, 1999-2001. *International journal of Occupational and Environmental Health*, 12(1), 9–15. <https://doi.org/10.1179/oeh.2006.12.1.9>.

Beddington J, Taylor AN, Darnton A, et al. (2011). GCSA meeting on the classification and regulation of chrysotile asbestos. Government Office for Science, London, UK. Available online; https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/

<file/283166/meeting-gcsa-chrysotile-asbestos-7-march-2011.pdf>. (Accessed on 20 September 2021).

Behrens MA and Anderson WL (2008). The “Any Exposure” Theory: An Unsound basis for Asbestos Causation and Expert Testimony. Available online at http://masonlec.org/site/rte_uploads/files/Behrens_Southwestern_Any_Exposure.pdf. Accessed 14/6/2020.

Berman DW & Crump KS (2008). A meta-analysis of asbestos-related cancer risk that addresses fiber size and mineral type. *Critical Reviews in Toxicology*, 38 Suppl 1, 49–73. <https://doi.org/10.1080/10408440802273156>.

Bernstein DM, Rogers R & Smith P (2004). The bio-persistence of Brazilian chrysotile asbestos following inhalation. *Inhalation Toxicology*, 16(11-12), 745–761. <https://doi.org/10.1080/08958370490490176>.

Bernstein DM & Hoskins JA (2006). The health effects of chrysotile: current perspective based upon recent data. *Regulatory Toxicology and Pharmacology: RTP*, 45(3), 252–264. <https://doi.org/10.1016/j.yrtph.2006.04.008>.

Bernstein D, Dunnigan J, Hesterberg T, Brown R, Velasco JA, Barrera R, Hoskins J & Gibbs A (2013). Health risk of chrysotile revisited. *Critical Reviews in Toxicology*, 43(2), 154–183. <https://doi.org/10.3109/10408444.2012.756454>.

Bernstein DM, Riego Sintes JM, Ersboell BK & Kunert J (2001a). Biopersistence of synthetic mineral fibres as a predictor of chronic inhalation toxicity in rats. *Inhalation Toxicology*, 13(10), 823–849. <https://doi.org/10.1080/089583701752378133>.

Bernstein, D. M., Riego Sintes, J. M., Ersboell, B. K., & Kunert, J (2001b). Biopersistence of synthetic mineral fibres as a predictor of chronic intraperitoneal injection tumor response in rats. *Inhalation Toxicology*, 13(10), 851–875. <https://doi.org/10.1080/089583701752378142>.

Berry G (1999). Models for mesothelioma incidence following exposure to fibres in terms of timing and duration of exposure and the biopersistence of the fibres. *Inhalation Toxicology*, 11(2), 111–130. <https://doi.org/10.1080/089583799197203>.

Berry G & Newhouse ML (1983). Mortality of workers manufacturing friction materials using asbestos. *British journal of Industrial Medicine*, 40(1), 1–7.

<https://doi.org/10.1136/oem.40.1.1>.

Berry G, Gilson JC, Holmes S, Lewinsohn HC Roach SA (1979). Asbestosis: a study of dose-response relationships in an asbestos textile factory. *British Journal of Industrial Medicine*, 36(2), 98–112. <https://doi.org/10.1136/oem.36.2.98>.

Browne K (1986). A threshold for asbestos related lung cancer. *Br J Ind Med*. 43,556-558.

Available online at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1007705/pdf/brjindmed00172-0052.pdf>. (Accessed on 4 Jan 2021).

Carbone M, Adusumilli PS, Alexander HR Jr, Baas P Bardelli F, Bononi A, Bueno R, Felley-Bosco E, Galateau-Salle F, Jablons D, Mansfield AS, Minaai M, de Perrot M, Pesavento P, Rusch V, Severson DT, Taioli E, Tsao A, Woodard G, Yang H, Pass HI (2019). Mesothelioma: Scientific clues for prevention, diagnosis, and therapy. *CA: A Cancer Journal for Clinicians*, 69(5), 402–429. <https://doi.org/10.3322/caac.21572>.

Calabrese EJ (2019). The linear No-Threshold (LNT) dose response model: A comprehensive assessment of its historical and scientific foundations. *Chemico-Biological Interactions*, 301, 6–25. <https://doi.org/10.1016/j.cbi.2018.11.020>.

Camus M (2001). A ban on asbestos must be based on a comparative risk assessment. *CMAJ: Canadian Medical Association journal = journal de l'Association medicale canadienne*, 164(4), 491–494. Available online at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC80777/>. (Accessed on 10 April 2022).

Costantini D & Borremans B (2019). The linear no-threshold model is less realistic than threshold or hormesis-based models: An evolutionary perspective. *Chemico-Biological Interactions*, 301, 26–33. <https://doi.org/10.1016/j.cbi.2018.10.007>.

Cullen MR & Baloyi RS (1991). Chrysotile asbestos and health in Zimbabwe: I. Analysis of miners and millers compensated for asbestos-related diseases since independence

(1980). American Journal of Industrial Medicine, 19(2), 161–169.

<https://doi.org/10.1002/ajim.4700190204>.

Davis JM & McDonald JC (1988). Low level exposure to asbestos: is there a cancer risk? British Journal of Industrial Medicine, 45(8), 505–508.

<https://doi.org/10.1136/oem.45.8.505>.

de Klerk NH, Musk AW, Williams V, Filion PR, Whitaker D & Shilkin KB (1996). Comparison of measures of exposure to asbestos in former crocidolite workers from Wittenoom Gorge, W. Australia. American Journal of Industrial Medicine, 30(5), 579–587. [https://doi.org/10.1002/\(SICI\)1097-0274\(199611\)30:5<579::AID-AJIM5>3.0.CO;2-O](https://doi.org/10.1002/(SICI)1097-0274(199611)30:5<579::AID-AJIM5>3.0.CO;2-O).

Dement J (2001) Differences in carcinogenicity between asbestos types: EPA Asbestos Health Effects conference, May 2001.

Doss M (2018). Are We Approaching the End of the Linear No-Threshold Era? Journal of nuclear medicine: official publication, Society of Nuclear Medicine, 59(12), 1786–1793.

<https://doi.org/10.2967/jnumed.118.217182>.

Dreesen WC, Dalavalle JW, Edwards TI, et al. (1938). A study of asbestos in the asbestos textile industry. Washington DC, US Public Health Service (Public Health Bulletin No. 241).

Enarson DA., Embree V, MacLean L & Grzybowski S (1988). Respiratory health in chrysotile asbestos miners in British Columbia: a longitudinal study. British Journal of Industrial Medicine, 45(7), 459–463. <https://doi.org/10.1136/oem.45.7.459>.

European Chemicals Agency (ECHA) (2021). ECHA Scientific report for evaluation of limit values for asbestos at the workplace. Helsinki, Finland, ECHA. Available online at [4605fc92-18a2-ae48-f977-4dffdecfec11 \(europa.eu\)](https://doi.org/10.2823/342121). Accessed on July 18 2023.

European Commission (1997). O.J. L 343/19 of 13 December 1997. Commission Directive 97/69/EC of 5 December 1997 adapting to technical progress for the 23rd time Council Directive 67/ 548/EEC on the approximation of the laws regulations and administrative provisions relating to the classification, packaging and labelling of dangerous substances. Official Journal of the European Communities. Available online at

<https://eur-lex.europa.eu/legal-content/EN/TXT/PDF/?uri=CELEX:31997L0069&from=ES>.

Accessed August 7 2022.

European Union (EU) (2017). Directive (EU) 2017/2398 of the European Parliament and of the Council of 12 December 2017 amending Directive 2004/37/EC on the protection of workers from the risks related to exposure to carcinogens or mutagens at work. Available online at

<https://eur-lex.europa.eu/legalcontent/EN/TXT/PDF/?uri=CELEX:32017L2398&rid=1>.

(Accessed 19 Feb 2021).

EU Environmental Agency (2022). Term: Life Cycle Assessment.

<https://www.eea.europa.eu/help/glossary/eea-glossary/life-cycle-assessment>.

Feder IS, Tischoff I, Theile A, Schmitz I, Merget R & Tannapfel A (2017). The asbestos fibre burden in human lungs: new insights into the chrysotile debate. *The European Respiratory Journal*, 49(6), 1602534. <https://doi.org/10.1183/13993003.02534-2016>.

Frazao R and Fernandes R (2004). Comparative Analysis of the Life Cycle of AT Fibre cement and NT Fibre-cement: Final Report. International Chrysotile Association (ICA). Available online: https://chrysotileassociation.com/data/26826_EN_INETI.pdf. (Accessed August 2 2022).

Finkelstein MM & Dufresne A (1999). Inferences on the kinetics of asbestos deposition and clearance among chrysotile miners and millers. *American Journal of Industrial Medicine*, 35(4), 401–412. [https://doi.org/10.1002/\(sici\)1097-0274\(199904\)35:4<401::aid-ajim12>3.0.co;2-4](https://doi.org/10.1002/(sici)1097-0274(199904)35:4<401::aid-ajim12>3.0.co;2-4).

Gardner MJ, Winter PD, Pannett B & Powell CA (1986). Follow up study of workers manufacturing chrysotile asbestos cement products. *British Journal of Industrial medicine*, 43(11), 726–732. <https://doi.org/10.1136/oem.43.11.726>.

Gov of South Africa (2020). Asbestos abatement Regulations. Dept of Labour, South Africa.

Government of Zimbabwe (Gov of Zim) (1990). Statutory Instrument 68 of 1990: Accident Prevention and Workers Compensation Scheme. Gov of Zimbabwe.

Goodman JE, Dodge DG, Bailey LA, Prueitt RL, Peterson MK, Beck BD, Engel AM (2020). Comments on the toxic substances control act draft risk evaluation for asbestos regarding chrysotile asbestos and the occupational use of automotive brakes. Washington (DC): Comments submitted by Gradient. Available online at

https://gradientcorp.com/pdfs/news/Docket%20EPA_HQ_OPPT_2019_0501_Comments.pdf.

(Accessed on 23 April 2022).

Golden R, Bus J & Calabrese E (2019). An examination of the linear no-threshold hypothesis of cancer risk assessment: Introduction to a series of reviews documenting the lack of biological plausibility of LNT. *Chemico-biological Interactions*, 301, 2–5.

<https://doi.org/10.1016/j.cbi.2019.01.038>.

Gualtieri AF, Lusvardi G, Zoboli A, Di Giuseppe D & Lassinantti Gualtieri M (2019). Biodurability and release of metals during the dissolution of chrysotile, crocidolite and fibrous erionite. *Environmental Research*, 171, 550–557.

<https://doi.org/10.1016/j.envres.2019.01.011>.

Gylseth B, Mowé G & Wannag A (1983). Fibre type and concentration in the lungs of workers in an asbestos cement factory. *British Journal of Industrial Medicine*, 40(4), 375–379.

<https://doi.org/10.1136/oem.40.4.375>.

Harrison PT, Levy LS, Patrick G, Pigott GH & Smith LL (1999). Comparative hazards of chrysotile asbestos and its substitutes: A European perspective. *Environmental Health Perspectives*, 107(8), 607–611. <https://doi.org/10.1289/ehp.99107607>.

Hesterberg TW, Hart GA, Chevalier J, Miiller WC, Hamilton RD, Bauer J & Thevenaz, P (1998). The importance of fiber biopersistence and lung dose in determining the chronic inhalation effects of X607, RCF1, and chrysotile asbestos in rats. *Toxicology and Applied Pharmacology*, 153(1), 68–82. <https://doi.org/10.1006/taap.1998.8522>.

Hesterberg TW, Miiller WC, Mast R, McConnell EE, Bernstein DM & Anderson R (1994). Relationship between lung biopersistence and biological effects of man-made vitreous fibres after chronic inhalation in rats. *Environmental Health Perspectives*, 102 Suppl 5(Suppl 5), 133–137. <https://doi.org/10.1289/ehp.94102s5133>.

Hodgson JT & Darnton A (2000). The quantitative risks of mesothelioma and lung cancer in relation to asbestos exposure. *The Annals of Occupational Hygiene*, 44(8), 565–601.

HSE (2012). *The Control of Asbestos Regulations*, HSE, UK.

Huang JQ (1990). A study on the dose-response relationship between asbestos exposure level and asbestosis among workers in a Chinese chrysotile product factory. *Biomed Environ Sci*. 3(1):90-8. PMID: 2331328.

Infante PF, Schuman LD, Dement J & Huff J (1994). Fibrous glass and cancer. *American Journal of Industrial Medicine*, 26(4), 559–584. <https://doi.org/10.1002/ajim.4700260413>.

International Agency for Research on Cancer (IARC) (2012). *IARC Monograph on Evaluation of Carcinogenic Risks to Human, Arsenic, Metals, Fibres and Dust: A review of human carcinogens Vol. 100C* p.219 – 309. IARC/WHO.

International Chrysotile Association (ICA) (2019). *Safety in the use of Chrysotile – Requirements and Achievements*. Available online at https://chrysotileassociation.com/data/ICA_Chrysotile_Safety_Requirements_Achievements_v5F_LR.pdf. (Accessed 13 April 2022).

International Labour Organization (ILO) (1986). *Convention No. 162 Convention - Safety in the Use of Asbestos*, ILO, Geneva, Switzerland.

International Labour Organization (ILO)(1986). *R172 – Asbestos Recommendation, 1986 (No. 172)*, ILO, Geneva, Switzerland.

International Labour Organization (ILO) (2006). *Resolution concerning asbestos, 2006*. Available online at https://www.ilo.org/safework/info/standards-and-instruments/WCMS_108556/lang--en/index.htm. Accessed 10 Feb 2021.

International Labour Organization (ILO) (2006). *ILO position on safety in the use of asbestos 2006*. Available online at https://www.ilo.org/global/topics/safety-and-health-at-work/areasofwork/occupational-health/WCMS_360580/lang--en/index.htm. (Accessed on 15/3/2021).

International Labour Organisation (1984). Safety in the use of asbestos: An ILO Code of practice, ILO, Geneva.

International Standardisation Organization (ISO) (2006). ISO 14040: 2006: Environment Management – Life Cycle Assessment – Principles and Framework.

International Programme on Chemical Safety (IPCS) (1998). Chrysotile Asbestos. Environmental Health Criteria 203: Geneva, UNEP, ILO, WHO and IPCS.

Jaurand MC, Bignon J, Sebastien P & Goni J (1977). Leaching of chrysotile asbestos in human lungs. Correlation with in vitro studies using rabbit alveolar macrophages. Environmental Research, 14(2), 245–254. [https://doi.org/10.1016/0013-9351\(77\)90036-6](https://doi.org/10.1016/0013-9351(77)90036-6).

Jones RN, Diem JE, Hughes JM, Hammad YY, Glindmeyer HW & Weill H (1989). Progression of asbestos effects: a prospective longitudinal study of chest radiographs and lung function. British Journal of Industrial Medicine, 46(2), 97–105. <https://doi.org/10.1136/oem.46.2.97>.

Korchevskiy A, Rasmuson JO & Rasmuson EJ (2019). Empirical model of mesothelioma potency factors for different mineral fibres based on their chemical composition and dimensionality. Inhalation Toxicology, 31(5), 180–191. <https://doi.org/10.1080/08958378.2019.1640320>.

LaDou J, Castleman B, Frank A, Gochfeld M, Greenberg M, Huff J, Joshi TK, Landrigan PJ, Lemen R, Myers J, Soffritti M, Soskolne CL, Takahashi K, Teitelbaum D, Terracini B, & Watterson A (2010). The case for a global ban on asbestos. Environmental Health Perspectives, 118(7), 897–901. <https://doi.org/10.1289/ehp.1002285>.

Landrigan PJ, Nicholson WJ, Suzuki Y & LaDou J (1999). The hazards of chrysotile asbestos: a critical review. Industrial Health, 37(3), 271–280. <https://doi.org/10.2486/indhealth.37.271>.

Langer AM & Nolan RP (1994). Chrysotile: its occurrence and properties as variables controlling biological effects. The Annals of Occupational Hygiene, 38(4), 427–451. <https://doi.org/10.1093/annhyg/38.4.427>.

Langer AM and Pooley FD (1973). Identification of single asbestos fibres in human tissue. In: Bogovovski P, Gilson JC, Timbrell V and Wagner JC ed. Biological effects of asbestos. Lyon, IARC, p199-125 IARC Scientific publication No.8.

Lemen RA (2004). Chrysotile asbestos as a cause of mesothelioma: application of the Hill causation model. *International Journal of Occupational and Environmental Health*, 10(2), 233–239. <https://doi.org/10.1179/oeh.2004.10.2.233>.

Liddell FD, McDonald AD & McDonald JC (1997). The 1891-1920 birth cohort of Quebec chrysotile miners and millers: development from 1904 and mortality to 1992. *The Annals of Occupational Hygiene*, 41(1), 13–36. [https://doi.org/10.1016/S0003-4878\(96\)00044-0](https://doi.org/10.1016/S0003-4878(96)00044-0).

Lin RT, Takahashi K, Karjalainen A, Hoshuyama T, Wilson D, Kameda T, Chan CC, Wen CP, Furuya S, Higashi T, Chien LC & Ohtaki M (2007). Ecological association between asbestos-related diseases and historical asbestos consumption: an international analysis. *Lancet (London, England)*, 369(9564), 844–849. [https://doi.org/10.1016/S0140-6736\(07\)60412-7](https://doi.org/10.1016/S0140-6736(07)60412-7).

Markowitz S (2015). Asbestos-related lung cancer and malignant mesothelioma of the pleura: selected current issues. *Seminars in Respiratory and Critical Care Medicine*, 36(3), 334–346. <https://doi.org/10.1055/s-0035-1549449>.

McConnell EE (1994). Synthetic vitreous fibres--inhalation studies. *Regulatory Toxicology and Pharmacology: RTP*, 20(3 Pt 2), S22–S34.

McDermott M, Bevan MM, Elmes PC, Allardice JT & Bradley AC (1982). Lung function and radiographic change in chrysotile workers in Swaziland. *British Journal of Industrial Medicine*, 39(4), 338–343. <https://doi.org/10.1136/oem.39.4.338>.

McDonald JC (2001). Carcinogenicity of fibrous tremolite in workplace and general environments. EPA Asbestos Health Conference, May 2001.

McDonald AD, Case BW, Churg A, Dufresne A, Gibbs GW, Sébastien P & McDonald JC (1997). Mesothelioma in Quebec chrysotile miners and millers: epidemiology and aetiology. *The Annals of Occupational Hygiene*, 41(6), 707–719. [https://doi.org/10.1016/S0003-4878\(97\)00020-3](https://doi.org/10.1016/S0003-4878(97)00020-3).

McDonald JC & McDonald AD (1997). Chrysotile, tremolite and carcinogenicity. *The Annals of Occupational Hygiene*, 41(6), 699–705. [https://doi.org/10.1016/S0003-4878\(97\)89350-7](https://doi.org/10.1016/S0003-4878(97)89350-7).

McDonald AD & McDonald JC (1980). Malignant mesothelioma in North America. *Cancer*, 46(7), 1650–1656. [https://doi.org/10.1002/1097-0142\(19801001\)46:7<1650::aid-cnrcr2820460726>3.0.co;2-y](https://doi.org/10.1002/1097-0142(19801001)46:7<1650::aid-cnrcr2820460726>3.0.co;2-y).

McDonald JC, Liddell FDK, Dufresne, et al. (1993). The 1891 – 1920 birth cohort of Quebec chrysotile miners and millers: mortality 1976 -1988. *Br J Ind Med*, 50, 1072-81.

Mezie G, Moolgavkar S, Mowat F (2020). Comment on draft risk evaluation for asbestos for consideration by Science Advisory Committee on Chemicals (SACC). Washington (DC): Comments submitted by Exponent, Inc. Available online at <https://www.regulations.gov/document/EPA-HQ-OPPT-2019-0501-0039>. (Accessed on 23 April 2022).

Morgan A and Cralley LJ (1973). Chemical characteristics of asbestos and associated trace elements. In: Bogovski P, Gilson JC ed. *biological effects of mineral fibres*. Lyon, IARC, p113-118. IARC publication No. 8.

Mutetwa B, Moyo D & Brouwer D (2021). Trends in Airborne Chrysotile Asbestos Fibre Concentrations in Asbestos Cement Manufacturing Factories in Zimbabwe from 1996 to 2016. *International Journal of Environmental Research and Public Health*. 18 (20), 10755. <https://doi.org/10.3390/ijerph182010755>.

Mutetwa B, Moyo D & Brouwer D (2022). Job Exposure Matrix for Chrysotile Asbestos Fibre in the Asbestos Cement Manufacturing (ACM) Industry in Zimbabwe. *International Journal of Environmental Research and Public Health*, 19(5), 2680. <https://doi.org/10.3390/ijerph19052680>.

Nelson G, Murray J & Phillips JI (2011). The risk of asbestos exposure in South African diamond mine workers. *The Annals of Occupational Hygiene*, 55(6), 569–577. <https://doi.org/10.1093/annhyg/mer028>.

Nielsen LS, Bælum J, Rasmussen J, Dahl S, Olsen KE, Albin M, Hansen NC & Sherson D (2014). Occupational asbestos exposure and lung cancer--a systematic review of the literature. *Archives of Environmental & Occupational Health*, 69(4), 191–206. <https://doi.org/10.1080/19338244.2013.863752>.

Newhouse ML and Sullivan KR (1989). A mortality study of workers manufacturing friction materials: 1941 -1986. *Br J Ind Med*. 46(3), 176-179.

Ohlson CG & Hogstedt C (1985). Lung cancer among asbestos cement workers. A Swedish cohort study and a review. *British journal of Industrial Medicine*, 42(6), 397–402. <https://doi.org/10.1136/oem.42.6.397>.

Oze C and Solt K (2010). Bio-durability of chrysotile and tremolite asbestos in simulated lung and gastric fluids. *Am Mineral*, 95: 825-831. Available online at http://www.minsocam.org/msa/ammin/toc/Abstracts/2010_Abstracts/MJ10_Abstracts/Oze_p825_10.pdf. (Accessed 10 August 2021).

Park SH (2018). Types and Health Hazards of Fibrous Materials Used as Asbestos Substitutes. *Safety and Health at Work*, Volume 9(3), 360-364. Available online at <https://www.sciencedirect.com/science/article/pii/S2093791118300933#!>. (Accessed 10 April 2022).

Park SH (2018). Types and Health Hazards of Fibrous Materials Used as Asbestos Substitutes. *Safety and Health at Work*, 9(3), 360–364. <https://doi.org/10.1016/j.shaw.2018.05.001>.

Paustenbach DJ, Finley BL, Lu ET, Brorby GP & Sheehan PJ (2004). Environmental and occupational health hazards associated with the presence of asbestos in brake linings and pads (1900 to present; a "state-of-the-art" review. *Journal of Toxicology and Environmental Health. Part B, Critical reviews*, 7(1), 25–80. <https://doi.org/10.1080/10937400490231494>.

Paustenbach D, Brew D, Ligas S & Heywood J (2021). A critical review of the 2020 EPA risk assessment for chrysotile and its many shortcomings. *Critical Reviews in Toxicology*, 51(6), 509–539. <https://doi.org/10.1080/10408444.2021.1968337>.

Paustenbach D, Brew D (2020). RE: updated comments by Paustenbach on EPA draft risk evaluation for asbestos of March 30, 2020. Washington, DC: Comments Submitted By Paustenbach and Associates. Available online at <https://www.regulations.gov/document/EPA-HQ-OPPT-2019-0501-0042>. (Accessed on 23 April 2022).

Pierce JS, McKinley MA, Paustenbach DJ & Finley BL (2008). An evaluation of reported no-effect chrysotile asbestos exposures for lung cancer and mesothelioma. *Critical Reviews in Toxicology*, 38(3), 191–214. <https://doi.org/10.1080/10408440701845609>.

Pierce JS, Ruestow PS & Finley BL (2016). An updated evaluation of reported no-observed adverse effect levels for chrysotile asbestos for lung cancer and mesothelioma. *Critical Reviews in Toxicology*, 46(7), 561–586. <https://doi.org/10.3109/10408444.2016.1150960>.

Pira E, Donato F, Maida L & Discalzi G (2018). Exposure to asbestos: past, present and future. *Journal of Thoracic Disease*, 10 (Suppl 2), S237–S245. <https://doi.org/10.21037/jtd.2017.10.126>.

Pollastri S, Perchiazzi N, Lezzerini M et al. (2016). The crystal structure of mineral fibres I. Chrysotile. *Periodico di Mineralogia*. 85: 249-259. Available online at https://www.researchgate.net/publication/311425221_The_crystal_structure_of_mineral_fibres_1_Chrysotile. (Accessed 3 January 2021).

Price B (2020). Comments on: draft risk evaluation for asbestos - EPA Document # EPA-740-R1-8012, March 2020. Washington (DC): Comment submitted by Price Associates, Inc. Available online at <https://www.regulations.gov/document/EPA-HQ-OPPT-2019-0501-0062>. (Accessed 23 April 2022).

Price B & Ware, A (2009). Time trend of mesothelioma incidence in the United States and projection of future cases: an update based on SEER data for 1973 through 2005. *Critical Reviews in Toxicology*, 39(7), 576–588. <https://doi.org/10.1080/10408440903044928>.

Raffn E, Lyng E, Juel K, et al. (1989). Incidence of cancer and mortality among employees in the asbestos cement industry in Denmark. *Br J Ind Med*. 46: 90-96. Available online at <https://www.ncbi.nlm.nih.gov/pmc/articles/PMC1009733/pdf/brjindmed00130-0018.pdf>. Accessed 20 Jan 2021.

Research Intelligence Unit (RIU) (2017). Socio – Economic Impact of the Potential Prohibition of Chrysotile in Sri Lanka – Cost Evaluation. Final Report. RIU. Available online at <https://www.fcpma.lk/pdf/RIU-english.pdf>). Accessed July 21 2023.

Rubino GF, Newhouse M, Murray R, Scansetti, G, Piolatto G & Aresini G (1979). Radiologic changes after cessation of exposure among chrysotile asbestos miners in Italy. *Annals of the New York Academy of Sciences*, 330, 157–161. <https://doi.org/10.1111/j.1749-6632.1979.tb18716.x>.

Stayner L, Smith R, Bailer J, Gilbert S, Steenland K, Dement J, Brown D & Lemen R (1997). Exposure-response analysis of risk of respiratory disease associated with occupational exposure to chrysotile asbestos. *Occupational and Environmental Medicine*, 54(9), 646–652. <https://doi.org/10.1136/oem.54.9.646>.

Sichletidis L, Chloros D, Spyrtos D, Haidich AB, Fourkiotou I, Kakoura M & Patakas D (2009). Mortality from occupational exposure to relatively pure chrysotile: a 39-year study. *Respiration; International Review of Thoracic Diseases*, 78(1), 63–68. <https://doi.org/10.1159/000163443>.

Siemiatycki J. Should Canadian health care professionals support the call for a worldwide ban on asbestos? [Editorial]. *CMAJ* 2001; 164(4), 495-7. Available online at <https://www.cmaj.ca/content/cmaj/164/4/495.full.pdf>. (Accessed on 10 April 2022).

Smith AH & Wright CC (1996). Chrysotile asbestos is the main cause of pleural mesothelioma. *American Journal of Industrial Medicine*, 30(3), 252–266. [https://doi.org/10.1002/\(SICI\)1097-0274\(199609\)30:3<252::AID-AJIM2>3.0.CO;2-0](https://doi.org/10.1002/(SICI)1097-0274(199609)30:3<252::AID-AJIM2>3.0.CO;2-0).

Soeberg MJ, Leigh J & van Zandwijk N (2016). Malignant mesothelioma in Australia 2015: Current incidence and asbestos exposure trends. *Journal of toxicology and environmental*

health. Part B, Critical Reviews, 19(5-6), 173–189.
<https://doi.org/10.1080/10937404.2016.1194254>.

Suzuki Y and Yuen SR (2001). Asbestos tissue burden study on human malignant mesothelioma. *Industrial Health*. 39, 150-60. Available online at
https://www.jstage.jst.go.jp/article/indhealth1963/39/2/39_2_150/pdf/-char/en. Accessed 28 Feb 2021.

Tomasetti C, Li L & Vogelstein B (2017). Stem cell divisions, somatic mutations, cancer etiology, and cancer prevention. *Science (New York, N.Y.)*, 355(6331), 1330–1334.
<https://doi.org/10.1126/science.aaf9011>.

Tomasetti C & Vogelstein B (2015). Cancer etiology. Variation in cancer risk among tissues can be explained by the number of stem cell divisions. *Science (New York, N.Y.)*, 347(6217), 78–81. <https://doi.org/10.1126/science.1260825>.

Tweedale G & McCulloch J (2004). Chrysophiles versus chrysophobes: the white asbestos controversy, 1950s-2004. *Isis; an international review devoted to the history of science and its cultural influences*, 95(2), 239–259. <https://doi.org/10.1086/426196>.

Thomas HF, Benjamin IT, Elwood PC & Sweetnam PM (1982). Further follow-up study of workers from an asbestos cement factory. *British Journal of Industrial Medicine*, 39(3), 273–276. <https://doi.org/10.1136/oem.39.3.273>.

Tossavainen A (1997). Asbestos, asbestosis, and cancer: the Helsinki criteria for diagnosis and attribution. (1997). *Scandinavian Journal of Work, Environment & Health*, 23(4), 311–316.
[doi:10.5271/sjweh.226](https://doi.org/10.5271/sjweh.226).

Tulchinsky TH, Ginsberg GM, Iscovich J, Shihab S, Fischbein A & Richter ED (1999). Cancer in ex-asbestos cement workers in Israel, 1953-1992. *American Journal of Industrial medicine*, 35(1), 1–8. [https://doi.org/10.1002/\(sici\)1097-0274\(199901\)35:1<1::aid-ajim1>3.0.co;2-5](https://doi.org/10.1002/(sici)1097-0274(199901)35:1<1::aid-ajim1>3.0.co;2-5).

Urban Environment Management (UEM) (2022). Available online at
<https://www.gdrc.org/uem/lca/lca-define.html>

van der Bij S, Koffijberg H, Lenters V, Portengen L, Moons, KG, Heederik D & Vermeulen RC (2013). Lung cancer risk at low cumulative asbestos exposure: meta-regression of the exposure-response relationship. *Cancer Causes & Control: CCC*, 24(1), 1–12. <https://doi.org/10.1007/s10552-012-0107-7>.

Valic F (2002). The Asbestos Dilemma: 1 Assessment of Risk. *Arh Hig Rada Toksikol.* 53, 153-167. Available online at https://chrysotileassociation.com/data/valic_1_risk_assessment.pdf. Accessed 16 Dec 2020.

Viallat JR, Boutin C, Pietri JF & Fondarai J (1983). Late Progression of Radiographic Changes in Canari Chrysotile Mine and Mill Exworkers. *Archives of Environmental Health: An International Journal*, 38,1, 54-58, DOI: [10.1080/00039896.1983.10543979x](https://doi.org/10.1080/00039896.1983.10543979x).

Virta RL (2006). Worldwide Asbestos Supply and Consumption Trends from 1900 through 2003: Circular 1298. Available at: <http://pubs.usgs.gov/circ/2006/1298/c1298.pdf>. Accessed 23/4/18.

Weill H, Hughes J & Waggenpack C (1979). Influence of dose and fiber type on respiratory malignancy risk in asbestos cement manufacturing. *The American Review of Respiratory Disease*, 120(2), 345–354. <https://doi.org/10.1164/arrd.1979.120.2.345>.

Welch L, Anderson HA, Balmes J, et al. (2009) Research on Mesothelioma from Brake Exposure: Corporate Influence Remains Relevant Concern. *International Journal of Occupational and Environmental Health*, 15 (2), pp. 234-238. <http://www.ijoh.com/index.php/ijoh/article/view/1226>.

World Health Organization (WHO) (2014). Chrysotile Asbestos. Available online at; http://www.who.int/ipcs/assessment/public_health/chemicals_phc. Accessed 9/2/2020.

World Trade Organisation (2000). European Communities – Measures Affecting Asbestos and Asbestos-Containing Products: Panel Record. Available online https://www.wto.org/english/tratop_e/dispu_e/135r_a_e.pdf. Accessed August 6 2022.

Yarborough CM (2006). Chrysotile as a cause of mesothelioma: an assessment based on epidemiology. *Critical Reviews in Toxicology*, 36(2), 165–187.
<https://doi.org/10.1080/10408440500534248>.

Ye L, Qi C, Hong J, Ma X (2017). Life cycle assessment of polyvinyl chloride production and its recyclability in China. *Journal of Cleaner Production*. Volume 142 Part 4, page 2965-2972. Available online at
<https://www.sciencedirect.com/science/article/abs/pii/S0959652616318029>. (Accessed August 1, 2022).

10. APPENDICES

Supplementary material

1. Trends in Airborne Chrysotile Asbestos Fibre Concentrations in Asbestos Cement Manufacturing Factories in Zimbabwe from 1996 to 2016
2. Job Exposure Matrix for chrysotile asbestos fibre in the asbestos cement manufacturing (ACM) industry in Zimbabwe.
3. Prediction of Asbestos Related Diseases (ARDs) and chrysotile asbestos exposure concentrations in asbestos-cement (AC) manufacturing factories in Zimbabwe.

Ethical Approval

4. Ethics clearance certificate

Appendix 1. Trends in Airborne Chrysotile Asbestos Fibre Concentrations in Asbestos Cement Manufacturing Factories in Zimbabwe from 1996 to 2016.

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Content

1. Table S1: Number of chrysotile fibre personal sampling point measurements per operational area per year in the chrysotile cement manufacturing factories: 1996-2016.
2. Table S2: Number of monthly mean personal chrysotile fibre concentrations by factory, operational area and year in the chrysotile asbestos cement manufacturing factories: 1996 – 2016.

NB: The number of personal samples decreased during the latter years, especially from 2008 to 2016, largely due to limited availability of consumables required for personal sampling of chrysotile asbestos fibre in various operational areas. Personal sampling at sheeting plant mixer, fettling table, kollergang and moulded goods was done at 2 or 3 operational points per month. For the pipe section, personal sampling data was collected at 2-4 joints lathe machines and at 2-3 full length pipe lathe machines per month. For multi-cutter operational tasks, personal sampling was done generally once per month where feasible.

Table S1: Number of chrysotile fibre personal sampling point measurements per operational area per year in the chrysotile cement manufacturing factories:

	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	Totals
HARARE CHRYSOTILE CEMENT FACTORY	143	132	148	143	120	89	101	99	71	104	81	77	32	44	44	51	56	36	40	45	18	1674
Saws cutting operations	63	69	72	71	60	42	40	35	25	35	26	23	12	16	17	20	23	18	19	20	7	713
Fettling tables - scrapping and polishing	7	10	12	12	12	11	11	12	9	12	6	11	1	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	126
Kollergang	41	19	17	20	14	14	19	17	11	21	16	14	6	8	9	15	12	10	9	7	2	301
Moulded Goods	10	22	23	16	12	11	9	12	10	12	12	11	5	8	6	7	9	4	6	8	4	217
Ground Hard Waste	11	12	12	12	10	Nil	11	11	5	12	9	8	Nil	5	7	6	12	4	6	10	5	168
Laundry Room	11	Nil	12	12	12	11	11	12	11	12	12	10	8	7	5	3	Nil	Nil	Nil	Nil	Nil	149
TOTAL	143	132	148	143	120	89	101	99	71	104	81	77	32	44	44	51	56	36	40	45	18	1674
BULAWAYO CHRYSOTILE CEMENT FACTORY	54	114	179	187	117	88	50	119	108	Nil	53	80	16	14	25	22	1	12	6	4	25	1216
(a) Sheeting Plant	37	75	100	105	65	52	28	70	56	Nil	27	43	9	14	20	10	1	8	6	3	25	754
Saws cutting operations	21	38	54	59	39	33	16	42	33	Nil	17	23	6	6	5	Nil	Nil	3	3	2	16	416
Fettling tables - scrapping and polishing	5	7	10	11	7	4	3	Nil	2	Nil	1	1	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	51
Kollergang	Nil	8	11	9	8	6	3	19	20	Nil	8	13	3	5	10	8	1	4	3	Nil	5	144
Ground Hard Waste	8	16	16	17	6	4	4	5	1	Nil	Nil	2	Nil	Nil	1	Nil	Nil	Nil	Nil	1	4	85
(b) Pipe Plant (P/P)	17	39	79	82	52	36	22	49	52	Nil	26	37	7	Nil	5	12	Nil	4	Nil	1	Nil	520
P/P - Lathe machining of pipe joints	10	17	37	47	32	21	12	22	20	Nil	11	17	3	Nil	2	6	Nil	2	Nil	1	Nil	260
P/P - Lathe machining full length pipes	5	15	36	28	16	9	8	20	23	Nil	10	13	4	Nil	3	4	Nil	2	Nil	Nil	Nil	196
Multicutter	2	7	6	7	4	6	2	7	9	Nil	5	7	Nil	Nil	Nil	2	Nil	Nil	Nil	Nil	Nil	64
TOTAL																						2890

Table S2: Number of monthly mean chrysotile fibre personal concentrations by factory, operational area and year in the chryso tile asbestos cement manufacturing factories: 1996 - 2016

	1996	1997	1998	1999	2000	2001	2002	2003	2004	2005	2006	2007	2008	2009	2010	2011	2012	2013	2014	2015	2016	Totals
FACTORY AND OPERATIONAL AREA																						
HARARE CHRYSOTILE CEMENT FACTORY	60	56	72	72	70	59	65	71	54	71	62	60	28	37	35	38	42	29	32	35	15	1063
Saws cutting operations	12	12	12	12	12	11	12	12	11	12	12	10	8	9	9	11	12	11	11	10	4	
Fettling tables - scrapping and polishing	7	10	12	12	12	11	11	12	9	12	6	11	1	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	
Moulded Goods	8	12	12	12	12	11	9	12	10	12	12	11	5	8	6	7	9	4	6	8	4	
Kollergang	11	10	12	12	12	11	11	12	8	11	11	10	6	8	8	11	9	10	9	7	2	
Ground Hard Waste	11	12	12	12	10	4	11	11	5	12	9	8	Nil	5	7	6	12	4	6	10	5	
Laundry Room	11	0	12	12	12	11	11	12	11	12	12	10	8	7	5	3	Nil	Nil	Nil	Nil	Nil	
TOTAL	60	56	72	72	70	59	65	71	54	71	62	60	28	37	35	38	42	29	32	35	15	1063
BULAWAYO CHRYSOTILE CEMENT FACTORY	35	61	82	83	54	45	26	55	57	Nil	26	39	12	11	19	20	1	12	5	3	12	600
(a) Sheeting Plant	23	41	52	52	34	25	16	29	26	Nil	11	21	5	11	14	9	1	8	5	3	12	398
Saws cutting operations	8	11	11	12	8	8	4	10	12	Nil	5	7	3	3	1	Nil	Nil	3	2	2	3	416
Fettling tables - scrapping and polishing	5	7	10	11	7	4	3	Nil	2	Nil	1	1	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	Nil	51
Kollergang	Nil	8	11	9	8	5	3	10	11	Nil	4	7	2	5	8	7	1	4	3	Nil	5	111
Ground Hard Waste	7	9	11	11	6	3	4	5	1	Nil	Nil	2	Nil	Nil	1	Nil	Nil	Nil	Nil	1	4	65
(b) Pipe Plant (P/P)	12	20	30	31	20	20	10	26	31	Nil	15	18	7	Nil	5	11	Nil	4	Nil	Nil	Nil	260
P/P - La the machining of pipe joints	6	6	12	12	8	7	4	9	11	Nil	5	7	3	Nil	2	5	Nil	2	Nil	Nil	Nil	99
P/P - La the machining full length pipes	4	7	12	12	8	7	4	10	11	Nil	5	4	4	Nil	3	4	Nil	2	Nil	Nil	Nil	97
Multicutter	2	7	6	7	4	6	2	7	9	Nil	5	7	Nil	Nil	Nil	2	Nil	Nil	Nil	Nil	Nil	64
TOTAL																						1663

Appendix 2. Job Exposure Matrix for chrysotile asbestos fibre in the asbestos cement manufacturing (ACM) industry in Zimbabwe

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Content

1. Table S1: Harare Factory Post Hoc Test: Multiple comparisons showing mean differences between time periods for various job categories that reaches significance ($p < 0.05$).
2. Table S2: Bulawayo Factory Post Hoc Test: Multiple comparisons showing mean differences between time periods for various job categories that reaches significance ($p < 0.05$).

Table S1: Harare factory post hoc test showing mean differences between time periods for various jobs

Job	Time-period	Mean Difference (I-J)	Std. Error	p-value	HSD 95% Confidence Interval		
					Lower Bound	Upper Bound	
Saw cutting operator	1996-2000	2001-2008	.06543*	0.00297	0.000	0.06	0.07
		2009-2016	.11918*	0.00306	0.000	0.11	0.13
		2018-2020	.08893*	0.00402	0.000	0.08	0.10
	2001-2008	1996-2000	-.06543*	0.00297	0.000	-0.07	-0.06
		2009-2016	.05375*	0.00277	0.000	0.05	0.06
		2018-2020	.02350*	0.00380	0.000	0.01	0.03
	2009-2016	1996-2000	-.11918*	0.00306	0.000	-0.13	-0.11
		2001-2008	-.05375*	0.00277	0.000	-0.06	-0.05
		2018-2020	-.03025*	0.00387	0.000	-0.04	-0.02
	2018-2020	1996-2000	-.08893*	0.00402	0.000	-0.10	-0.08
		2001-2008	-.02350*	0.00380	0.000	-0.03	-0.01
		2009-2016	.03025*	0.00387	0.000	0.02	0.04
Moulded goods operator	1996-2000	2001-2008	-0.00415	0.00478	0.822	-0.02	0.01
		2009-2016	.05769*	0.00532	0.000	0.04	0.07
		2018-2020	0.00400	0.01300	0.990	-0.03	0.04
	2001-2008	1996-2000	0.00415	0.00478	0.822	-0.01	0.02
		2009-2016	.06184*	0.00494	0.000	0.05	0.07
		2018-2020	0.00815	0.01284	0.921	-0.03	0.04
	2009-2016	1996-2000	-.05769*	0.00532	0.000	-0.07	-0.04
		2001-2008	-.06184*	0.00494	0.000	-0.07	-0.05
		2018-2020	-.05369*	0.01305	0.000	-0.09	-0.02
	2018-2020	1996-2000	-0.00400	0.01300	0.990	-0.04	0.03
		2001-2008	-0.00815	0.01284	0.921	-0.04	0.03
		2009-2016	.05369*	0.01305	0.000	0.02	0.09
Kollergang operator	1996-2000	2001-2008	.01124*	0.00422	0.042	0.00	0.02
		2009-2016	.06414*	0.00445	0.000	0.05	0.08
		2018-2020	0.01358	0.00880	0.413	-0.01	0.04
	2001-2008	1996-2000	-.01124*	0.00422	0.042	-0.02	0.00
		2009-2016	.05290*	0.00411	0.000	0.04	0.06
		2018-2020	0.00235	0.00863	0.993	-0.02	0.02
	2009-2016	1996-2000	-.06414*	0.00445	0.000	-0.08	-0.05
		2001-2008	-.05290*	0.00411	0.000	-0.06	-0.04
		2018-2020	-.05056*	0.00874	0.000	-0.07	-0.03
	2018-2020	1996-2000	-0.01358	0.00880	0.413	-0.04	0.01
		2001-2008	-0.00235	0.00863	0.993	-0.02	0.02
		2009-2016	.05056*	0.00874	0.000	0.03	0.07
Ground hard waste operator	1996-2000	2001-2008	.02455*	0.00508	0.000	0.01	0.04
		2009-2016	.08472*	0.00510	0.000	0.07	0.10
		2018-2020	.03384*	0.01019	0.006	0.01	0.06
	2001-2008	1996-2000	-.02455*	0.00508	0.000	-0.04	-0.01
		2009-2016	.06017*	0.00513	0.000	0.05	0.07
		2018-2020	0.00929	0.01021	0.800	-0.02	0.04
	2009-2016	1996-2000	-.08472*	0.00510	0.000	-0.10	-0.07
		2001-2008	-.06017*	0.00513	0.000	-0.07	-0.05
		2018-2020	-.05089*	0.01022	0.000	-0.08	-0.02
	2018-2020	1996-2000	-.03384*	0.01019	0.006	-0.06	-0.01
		2001-2008	-0.00929	0.01021	0.800	-0.04	0.02
		2009-2016	.05089*	0.01022	0.000	0.02	0.08
Laundry room operator	1996-2000	2001-2008	0.00864	0.00461	0.243	0.00	0.02
		2009-2016	.08671*	0.00755	0.000	0.07	0.11
		2018-2020	.02761*	0.00775	0.003	0.01	0.05
	2001-2008	1996-2000	-0.00864	0.00461	0.243	-0.02	0.00
		2009-2016	.07807*	0.00712	0.000	0.06	0.10
		2018-2020	0.01897	0.00733	0.051	0.00	0.04
	2009-2016	1996-2000	-.08671*	0.00755	0.000	-0.11	-0.07
		2001-2008	-.07807*	0.00712	0.000	-0.10	-0.06
		2018-2020	-.05910*	0.00946	0.000	-0.08	-0.03
2018-2020	1996-2000	-.02761*	0.00775	0.003	-0.05	-0.01	
	2001-2008	-0.01897	0.00733	0.051	-0.04	0.00	
	2009-2016	.05910*	0.00946	0.000	0.03	0.08	

Table S2: Bulawayo factory post hoc test showing mean differences between time periods for various jobs

Job	Time-period	Mean Difference (I-J)	Std. Error	p-value	HSD 95% Confidence Interval		
					Lower Bound	Upper Bound	
Saw cutting operator	1996-2000	2001-2008	.05125*	0.00401	0.000	0.0408	0.0617
		2009-2016	.11347*	0.00588	0.000	0.0982	0.1288
		2017-2019	.11380*	0.00496	0.000	0.1009	0.1267
	2001-2008	1996-2000	-.05125*	0.00401	0.000	-0.0617	-0.0408
		2009-2016	-.06222*	0.00589	0.000	0.0469	0.0775
		2017-2019	-.06255*	0.00497	0.000	0.0496	0.0755
	2009-2016	1996-2000	-.11347*	0.00588	0.000	-0.1288	-0.0982
		2001-2008	-.06222*	0.00589	0.000	-0.0775	-0.0469
		2017-2019	0.00033	0.00657	1.000	-0.0168	0.0174
	2017-2019	1996-2000	-.11380*	0.00496	0.000	-0.1267	-0.1009
		2001-2008	-.06255*	0.00497	0.000	-0.0755	-0.0496
		2009-2016	-0.00033	0.00657	1.000	-0.0174	0.0168
Kollergang operator	1996-2000	2001-2008	.02230*	0.00548	0.000	0.0080	0.0366
		2009-2016	.06915*	0.00577	0.000	0.0541	0.0842
		2017-2019	.07944*	0.00741	0.000	0.0601	0.0987
	2001-2008	1996-2000	-.02230*	0.00548	0.000	-0.0366	-0.0080
		2009-2016	.04685*	0.00556	0.000	0.0324	0.0613
		2017-2019	.05714*	0.00725	0.000	0.0382	0.0760
	2009-2016	1996-2000	-.06915*	0.00577	0.000	-0.0842	-0.0541
		2001-2008	-.04685*	0.00556	0.000	-0.0613	-0.0324
		2017-2019	0.01029	0.00748	0.516	-0.0092	0.0298
	2017-2019	1996-2000	-.07944*	0.00741	0.000	-0.0987	-0.0601
		2001-2008	-.05714*	0.00725	0.000	-0.0760	-0.0382
		2009-2016	-0.01029	0.00748	0.516	-0.0298	0.0092
Groundhard waste operator	1996-2000	2001-2008	0.02194	0.00993	0.130	-0.0042	0.0480
		2009-2016	.06227*	0.01445	0.000	0.0243	0.1003
		2017-2019	.07144*	0.01081	0.000	0.0430	0.0999
	2001-2008	1996-2000	-0.02194	0.00993	0.130	-0.0480	0.0042
		2009-2016	0.04033	0.01604	0.066	-0.0018	0.0825
		2017-2019	.04950*	0.01286	0.001	0.0157	0.0833
	2009-2016	1996-2000	-.06227*	0.01445	0.000	-0.1003	-0.0243
		2001-2008	-0.04033	0.01604	0.066	-0.0825	0.0018
		2017-2019	0.00917	0.01660	0.946	-0.0345	0.0528
	2017-2019	1996-2000	-.07144*	0.01081	0.000	-0.0999	-0.0430
		2001-2008	-.04950*	0.01286	0.001	-0.0833	-0.0157
		2009-2016	-0.00917	0.01660	0.946	-0.0528	0.0345
Pipe joints operator	1996-2000	2001-2008	.01948*	0.00594	0.008	0.0040	0.0350
		2009-2016	.07073*	0.01031	0.000	0.0438	0.0977
		2017-2019	.08095*	0.01330	0.000	0.0462	0.1157
	2001-2008	1996-2000	-.01948*	0.00594	0.008	-0.0350	-0.0040
		2009-2016	.05126*	0.01027	0.000	0.0244	0.0781
		2017-2019	.06148*	0.01327	0.000	0.0268	0.0961
	2009-2016	1996-2000	-.07073*	0.01031	0.000	-0.0977	-0.0438
		2001-2008	-.05126*	0.01027	0.000	-0.0781	-0.0244
		2017-2019	0.01022	0.01572	0.915	-0.0308	0.0513
	2017-2019	1996-2000	-.08095*	0.01330	0.000	-0.1157	-0.0462
		2001-2008	-.06148*	0.01327	0.000	-0.0961	-0.0268
		2009-2016	-0.01022	0.01572	0.915	-0.0513	0.0308
Multicutter operator	1996-2000	2001-2008	0.00338	0.00644	0.953	-0.0136	0.0204
		2009-2016	.05615*	0.01835	0.017	0.0077	0.1046
		2017-2019	.08115*	0.01835	0.000	0.0327	0.1296
	2001-2008	1996-2000	-0.00338	0.00644	0.953	-0.0204	0.0136
		2009-2016	.05278*	0.01817	0.026	0.0048	0.1008
		2017-2019	.07778*	0.01817	0.000	0.0298	0.1258
	2009-2016	1996-2000	-.05615*	0.01835	0.017	-0.1046	-0.0077
		2001-2008	-.05278*	0.01817	0.026	-0.1008	-0.0048
		2017-2019	0.02500	0.02501	0.750	-0.0410	0.0910
	2017-2019	1996-2000	-.08115*	0.01835	0.000	-0.1296	-0.0327
		2001-2008	-.07778*	0.01817	0.000	-0.1258	-0.0298
		2009-2016	-0.02500	0.02501	0.750	-0.0910	0.0410

HSD – Tukey Honest Significance Difference: The mean difference is significant at the 0.05 level.

Appendix 3. Prediction of Asbestos Related Diseases (ARDs) and chrysotile asbestos exposure concentrations in asbestos-cement (AC) manufacturing factories in Zimbabwe.

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1. Table S1: Cumulative exposure on lung cancer risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Harare factory
2. Table S2: Cumulative exposure on lung cancer risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Bulawayo factory
3. Table S3: Cumulative exposure on mesothelioma risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Harare factory
4. Table S4: Cumulative exposure on mesothelioma risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Bulawayo factory
5. Table S5: Cumulative exposure on gastrointestinal cancer risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Harare factory
6. Table S6: Cumulative exposure on gastrointestinal cancer risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Bulawayo factory
7. Table S7: Overall summary estimates of cancer mortality cases by factory, job and duration of exposure of 1 year.

8. Table S8: Overall summary estimates of cancer mortality cases by factory, job and duration of exposure of 10 years.
9. Table S9: Overall summary estimates of cancer mortality cases by factory, job and duration of exposure of 20 years.
10. Table 10: Overall summary estimates of cancer mortality cases by factory, job and duration of exposure of 25 years.
11. Table S11: Estimates of possible asbestosis incidence after 25 years of exposure
12. Table S12: Estimated number of workers working at various jobs in the chrysotile asbestos cement manufacturing factories

Table S1: Cumulative exposure on lung cancer risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Harare factory

Job	Time period	Mean f/ml	Duration of exposure											
			1 year			10 years			20 years			25 years		
			CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid
Saw cutting operator	1996-2000	0.19	0.19	13.7	0.01	1.9	135	0.14	3.8	274	0.27	4.8	346	0.35
	2001-2008	0.13	0.13	9.4	0.009	1.3	94	0.09	2.6	187	0.19	3.3	238	0.24
	2009-2016	0.07	0.07	5.1	0.005	0.7	50	0.05	1.4	101	0.10	1.8	130	0.13
	2018-2020	0.10	0.10	7.2	0.007	1.0	72	0.07	2.0	144	0.14	2.5	180	0.18
Fettling table operator	1996-2000	0.12	0.12	8.6	0.009	1.2	86	0.09	2.4	173	0.17	3.0	216	0.22
	2001-2008	0.12	0.12	8.6	0.009	1.2	86	0.09	2.4	173	0.17	3.0	216	0.22
	2009-2016	-	-	-	-	-	-	-	-	-	-	-	-	-
	2018-2020	0.11	0.11	7.9	0.008	1.1	79	0.08	2.2	158	0.16	2.8	202	0.20
Moulded goods operator	1996-2000	0.11	0.11	7.9	0.008	1.1	79	0.08	2.2	158	0.16	2.8	202	0.20
	2001-2008	0.11	0.11	7.9	0.008	1.1	79	0.08	2.2	158	0.16	2.8	202	0.20
	2009-2016	0.05	0.05	3.6	0.004	0.5	36	0.04	1.0	72	0.07	1.3	94	0.09
	2018-2020	0.11	0.11	7.9	0.008	1.1	79	0.08	2.2	158	0.16	2.8	202	0.20
Kollergang operator	1996-2000	0.13	0.13	9.4	0.01	1.3	94	0.09	2.6	187	0.19	3.3	238	0.24
	2001-2008	0.12	0.12	8.6	0.009	1.2	86	0.09	2.4	173	0.17	3.0	216	0.22
	2009-2016	0.07	0.07	5.0	0.005	0.7	50	0.05	1.4	101	0.10	1.8	130	0.13
	2018-2020	0.12	0.12	8.6	0.007	1.2	86	0.09	2.4	173	0.17	3.0	216	0.22
Ground hard waste operator	1996-2000	0.16	0.16	11.5	0.01	1.6	115	0.12	3.2	230	0.23	3.3	238	0.24
	2001-2008	0.13	0.13	9.4	0.009	1.3	94	0.09	2.6	187	0.19	3.3	238	0.24
	2009-2016	0.07	0.07	5.0	0.005	0.7	50	0.05	1.4	101	0.07	1.8	130	0.13
	2018-2020	0.12	0.12	8.6	0.009	1.2	86	0.09	2.4	173	0.16	3.0	216	0.22
Laundry room operator	1996-2000	0.13	0.13	9.4	0.009	1.3	94	0.09	2.6	187	0.19	3.3	238	0.24
	2001-2008	0.13	0.13	9.4	0.009	1.3	94	0.09	2.6	187	0.19	3.3	238	0.24
	2009-2016	0.05	0.05	3.6	0.004	0.5	36	0.04	1.0	72	0.07	1.3	94	0.09
	2018-2020	0.11	0.11	7.9	0.008	1.1	79	0.08	2.2	158	0.15	2.8	202	0.20
Overall factory		0.11	0.11	7.9	0.008	1.1	79	0.08	2.2	158	0.16	2.8	202	0.20

% Incid – Percentage incidence; CE – Cumulative. Cases/10⁵ – cancer mortality/morbidity cases per 100 000 workers exposed.

Table S2: Cumulative exposure on **lung cancer** risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Bulawayo factory

		Duration of exposure												
		1 year				10 years			20 years			25 years		
Job	Time period	Mean f/ml	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid
Saw cutting operator	1996-2000	0.17	0.17	12.2	0.01	1.7	122	0.12	3.4	245	0.25	4.3	310	0.30
	2001-2008	0.12	0.12	8.6	0.01	1.2	86	0.09	2.4	173	0.17	3.0	216	0.20
	2009-2016	0.06	0.06	4.3	0.005	0.6	36	0.04	1.2	86	0.09	1.5	108	0.11
	2018-2020	0.05	0.05	3.6	0.007	0.5	36	0.04	1.0	72	0.07	1.3	94	0.09
Fettling table operator	1996-2000	0.17	0.17	12.2	0.01	1.7	122	0.12	3.4	245	0.25	4.3	310	0.30
	2001-2008	0.12	0.12	8.6	0.009	1.2	86	0.09	2.4	173	0.17	3.0	216	0.22
	2009-2016	-	-	-	-	-	-	-	-	-	-	-	-	-
	2018-2020	-	-	-	-	-	-	-	-	-	-	-	-	-
Moulded goods operator	1996-2000	-	-	-	-	-	-	-	-	-	-	-	-	-
	2001-2008	-	-	-	-	-	-	-	-	-	-	-	-	-
	2009-2016	-	-	-	-	-	-	-	-	-	-	-	-	-
	2018-2020	-	-	-	-	-	-	-	-	-	-	-	-	-
Kollergang operator	1996-2000	0.14	0.14	10.1	0.01	1.4	101	0.10	2.8	202	0.20	3.5	252	0.25
	2001-2008	0.12	0.12	8.6	0.009	1.2	86	0.09	2.4	173	0.17	3.0	216	0.22
	2009-2016	0.07	0.07	5.0	0.005	0.7	50	0.05	1.4	101	0.10	1.8	130	0.13
	2018-2020	0.06	0.06	4.3	0.004	0.6	36	0.04	1.2	86	0.09	1.5	108	0.11
Ground hard waste operator	1996-2000	0.13	0.13	9.4	0.009	1.3	94	0.09	2.6	187	0.19	3.3	238	0.24
	2001-2008	0.11	0.11	7.9	0.008	1.1	79	0.08	2.2	158	0.16	2.8	202	0.20
	2009-2016	0.07	0.07	5.0	0.005	0.7	50	0.05	1.4	101	0.10	1.8	130	0.13
	2018-2020	0.06	0.06	8.6	0.009	0.6	36	0.04	2.4	173	0.17	1.5	108	0.11
Laundry room operator	1996-2000	-	-	-	-	-	-	-	-	-	-	-	-	-
	2001-2008	-	-	-	-	-	-	-	-	-	-	-	-	-
	2009-2016	-	-	-	-	-	-	-	-	-	-	-	-	-
	2018-2020	-	-	-	-	-	-	-	-	-	-	-	-	-
Table S2 continued.....														
		Duration of exposure												
		1 year				10 years			20 years			25 years		
Job	Time period	Mean f/ml	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid
Pipe joints operators	1996-2000	0.13	0.13	9.4	0.01	1.3	94	0.09	2.6	187	0.19	3.3	238	0.24
	2001-2008	0.11	0.11	7.9	0.01	1.1	79	0.08	2.2	158	0.16	2.8	202	0.20
	2009-2016	0.05	0.05	3.6	0.005	0.5	36	0.04	1.0	72	0.07	1.3	94	0.09
	2018-2020	0.05	0.05	3.6	0.005	0.5	36	0.04	1.0	72	0.07	1.3	94	0.09

Fulllength pipe operator	1996-2000	0.13	0.13	9.4	0.01	1.3	94	0.09	2.6	187	0.19	3.3	238	0.24
	2001-2008	0.11	0.11	7.9	0.01	1.1	79	0.08	2.2	158	0.16	2.8	202	0.20
	2009-2016	0.07	0.07	5.0	0.01	0.7	50	0.05	1.4	101	0.10	1.8	130	0.13
	2018-2020	-	-	-	-	-	-	-	-	-	-	-	-	-
Multicutter operator	1996-2000	0.13	0.13	9.4	0.01	1.3	94	0.09	2.6	187	0.19	3.3	238	0.24
	2001-2008	0.12	0.12	8.6	0.01	1.2	86	0.09	2.4	173	0.17	3.0	216	0.22
	2009-2016	0.07	0.07	5.0	0.01	0.7	50	0.05	1.4	101	0.10	1.8	130	0.13
	2018-2020	0.04	0.04	2.9	0.003	0.4	29	0.03	0.8	58	0.06	1.0	72	0.07
Overall factory		0.12	0.12	8.6	0.009	1.2	86	0.09	2.2	158	0.16	3.0	216	0.22

% Incid – Percentage incidence; CE – Cumulative. Cases/10⁵ – cancer mortality/morbidity cases per 100 000 workers exposed.

Table S3: Cumulative exposure on **mesothelioma** cancer risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Harare factory

Job	Time period	Mean f/ml	Duration of exposure											
			1 year			10 years			20 years			25 years		
			CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid
Saw cutting operator	1996-2000	0.19	0.19	8.0	0.008	1.9	70	0.07	3.8	138	0.14	4.8	174	0.17
	2001-2008	0.13	0.13	5.8	0.006	1.3	48	0.05	2.6	95	0.10	3.3	120	0.12
	2009-2016	0.07	0.07	3.7	0.004	0.7	26	0.03	1.4	52	0.05	1.8	66	0.06
	2018-2020	0.10	0.10	4.8	0.005	1.0	37	0.04	2.0	73	0.07	2.5	91	0.09
Fettling table operator	1996-2000	0.12	0.12	5.5	0.006	1.2	44	0.04	2.4	88	0.09	3.0	109	0.11
	2001-2008	0.12	0.12	5.5	0.006	1.2	44	0.04	2.4	88	0.09	3.0	109	0.11
	2009-2016	-	-	-	-	-	-	-	-	-	-	-	-	-
	2018-2020	0.11	0.11	5.1	0.005	1.1	41	0.04	2.2	81	0.08	2.8	102	0.11
Moulded goods operator	1996-2000	0.11	0.11	5.1	0.005	1.1	41	0.04	2.2	81	0.08	2.8	102	0.11
	2001-2008	0.11	0.11	5.1	0.005	1.1	41	0.04	2.2	81	0.08	2.8	102	0.11
	2009-2016	0.05	0.05	3.0	0.003	0.5	19	0.02	1.0	37	0.04	1.3	48	0.09
	2018-2020	0.11	0.11	5.1	0.005	1.1	41	0.04	2.2	81	0.08	2.8	102	0.11
Kollergang operator	1996-2000	0.13	0.13	5.8	0.006	1.3	48	0.05	2.6	95	0.10	3.3	120	0.12
	2001-2008	0.12	0.12	5.5	0.006	1.2	44	0.04	2.4	88	0.09	3.0	109	0.11
	2009-2016	0.07	0.07	3.7	0.004	0.7	26	0.03	1.4	52	0.05	1.8	66	0.06
	2018-2020	0.12	0.12	5.5	0.004	1.2	44	0.04	2.4	88	0.09	3.0	109	0.11
Ground hard waste operator	1996-2000	0.16	0.16	6.9	0.007	1.6	59	0.06	3.2	117	0.12	3.3	120	0.12
	2001-2008	0.13	0.13	5.8	0.006	1.3	48	0.05	2.6	95	0.10	3.3	120	0.12
	2009-2016	0.07	0.07	3.7	0.004	0.7	26	0.03	1.4	52	0.05	1.8	66	0.07
	2018-2020	0.12	0.12	5.5	0.006	1.2	44	0.04	2.4	88	0.09	3.0	109	0.11
Laundry room operator	1996-2000	0.13	0.13	5.8	0.006	1.3	48	0.05	2.6	95	0.10	3.3	120	0.11
	2001-2008	0.13	0.13	5.8	0.006	1.3	48	0.05	2.6	95	0.10	3.3	120	0.11
	2009-2016	0.05	0.05	3.0	0.003	0.5	19	0.02	1.0	37	0.04	1.3	48	0.05
	2018-2020	0.11	0.11	5.1	0.005	1.1	41	0.04	2.2	81	0.08	2.8	102	0.11
Overall factory		0.11	0.11	5.1	0.005	1.1	41	0.04	2.2	81	0.08	2.8	102	0.10

% Incid – Percentage incidence; CE – Cumulative. Cases/10⁵ – cancer mortality/morbidity cases per 100 000 workers exposed

Table S4: Cumulative exposure on Mesothelioma risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Bulawayo factory

		Duration of exposure												
		1 year				10 years			20 years			25 years		
Job	Time period	Mean f/ml	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid
Saw cutting operator	1996-2000	0.17	0.17	7.2	0.007	1.7	63	0.06	3.4	124	0.12	4.3	156	0.16
	2001-2008	0.12	0.12	5.4	0.005	1.2	44	0.04	2.4	88	0.09	3.0	109	0.11
	2009-2016	0.06	0.06	3.3	0.003	0.6	23	0.02	1.2	44	0.04	1.5	55	0.06
	2018-2020	0.05	0.05	3.0	0.003	0.5	19	0.02	1.0	37	0.04	1.3	48	0.05
Fettling table operator	1996-2000	0.17	0.17	7.2	0.007	1.7	63	0.06	3.4	124	0.12	4.3	156	0.16
	2001-2008	0.12	0.12	5.4	0.005	1.2	44	0.04	2.4	88	0.09	3.0	109	0.11
	2009-2016	-	-	-	-	-	-	-	-	-	-	-	-	-
	2018-2020	-	-	-	-	-	-	-	-	-	-	-	-	-
Moulded goods operator	1996-2000	-	-	-	-	-	-	-	-	-	-	-	-	-
	2001-2008	-	-	-	-	-	-	-	-	-	-	-	-	-
	2009-2016	-	-	-	-	-	-	-	-	-	-	-	-	-
	2018-2020	-	-	-	-	-	-	-	-	-	-	-	-	-
Kollergang operator	1996-2000	0.14	0.14	6.2	0.006	1.4	52	0.05	2.8	102	0.10	3.5	127	0.13
	2001-2008	0.12	0.12	5.4	0.005	1.2	44	0.04	2.4	88	0.09	3.0	109	0.11
	2009-2016	0.07	0.07	3.7	0.004	0.7	26	0.03	1.4	52	0.05	1.8	66	0.07
	2018-2020	0.06	0.06	3.3	0.003	0.6	23	0.02	1.2	44	0.04	1.5	55	0.06
Ground hard waste operator	1996-2000	0.13	0.13	5.8	0.006	1.3	48	0.05	2.6	95	0.10	3.3	120	0.12
	2001-2008	0.11	0.11	5.1	0.005	1.1	41	0.04	2.2	81	0.09	2.8	102	0.10
	2009-2016	0.07	0.07	3.7	0.004	0.7	26	0.03	1.4	52	0.05	1.8	66	0.07
	2018-2020	0.06	0.06	3.3	0.003	0.6	23	0.02	2.4	88	0.09	1.5	55	0.06
Laundry room operator	1996-2000	-	-	-	-	-	-	-	-	-	-	-	-	-
	2001-2008	-	-	-	-	-	-	-	-	-	-	-	-	-
	2009-2016	-	-	-	-	-	-	-	-	-	-	-	-	-
	2018-2020	-	-	-	-	-	-	-	-	-	-	-	-	-
Table S4 continued.....														
		Duration of exposure												
		1 year				10 years			20 years			25 years		
Job	Time period	Mean f/ml	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid
Pipe joints operators	1996-2000	0.13	0.13	5.8	0.006	1.3	48	0.05	2.6	95	0.10	3.3	120	0.12
	2001-2008	0.11	0.11	5.1	0.005	1.1	41	0.04	2.2	81	0.08	2.8	102	0.11
	2009-2016	0.05	0.05	3.0	0.003	0.5	19	0.02	1.0	37	0.04	1.3	48	0.05
	2018-2020	0.05	0.05	3.0	0.003	0.5	19	0.02	1.0	37	0.04	1.3	48	0.05

Fulllength pipe operator	1996-2000	0.13	0.13	5.8	0.006	1.3	48	0.05	2.6	95	0.10	3.3	120	0.12
	2001-2008	0.11	0.11	5.1	0.005	1.1	41	0.04	2.2	81	0.08	2.8	102	0.10
	2009-2016	0.07	0.07	3.7	0.004	0.7	26	0.03	1.4	52	0.05	1.8	66	0.07
	2018-2020	-	-	-	-	-	-	-	-	-	-	-	-	-
Multicutter operator	1996-2000	0.13	0.13	5.8	0.006	1.3	48	0.05	2.6	95	0.10	3.3	120	0.12
	2001-2008	0.12	0.12	5.5	0.006	1.2	44	0.04	2.4	88	0.09	3.0	109	0.11
	2009-2016	0.07	0.07	3.7	0.004	0.7	26	0.03	1.4	52	0.05	1.8	66	0.07
	2018-2020	0.04	0.04	2.6	0.003	0.4	16	0.02	0.8	30	0.03	1.0	37	0.04
Overall factory		0.12	0.12	5.5	0.006	1.2	44	0.04	2.2	81	0.08	3.0	109	0.11

% Incid – Percentage incidence; CE – Cumulative. Cases/10⁵ – cancer mortality/morbidity cases per 100 000 workers exposed

Table S5: Cumulative exposure on gastrointestinal cancer risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Harare factory

Job	Time period	Mean f/ml	Duration of exposure											
			1 year			10 years			20 years			25 years		
			CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid
Saw cutting operator	1996-2000	0.19	0.19	1.3	0.001	1.9	13.7	0.01	3.8	27.4	0.03	4.8	34.6	0.03
	2001-2008	0.13	0.13	0.9	0.001	1.3	9.3	0.009	2.6	18.7	0.02	3.3	23.8	0.02
	2009-2016	0.07	0.07	0.5	0.0005	0.7	5.0	0.005	1.4	10.1	0.01	1.8	12.9	0.01
	2018-2020	0.10	0.10	0.7	0.001	1.0	7.2	0.007	2.0	14.4	0.01	2.5	18.0	0.02
Fettling table operator	1996-2000	0.12	0.12	0.8	0.001	1.2	8.6	0.009	2.4	17.2	0.02	3.0	21.6	0.02
	2001-2008	0.12	0.12	0.8	0.001	1.2	8.6	0.009	2.4	17.2	0.02	3.0	21.6	0.02
	2009-2016	-	-	-	-	-	-	-	-	-	-	-	-	-
	2018-2020	0.11	0.11	0.8	0.001	1.1	7.9	0.008	2.2	15.8	0.02	2.8	20.2	0.02
Moulded goods operator	1996-2000	0.11	0.11	0.8	0.001	1.1	7.9	0.008	2.2	15.8	0.02	2.8	20.2	0.02
	2001-2008	0.11	0.11	0.8	0.001	1.1	7.9	0.008	2.2	15.8	0.02	2.8	20.2	0.02
	2009-2016	0.05	0.05	0.3	0.0003	0.5	3.6	0.004	1.0	7.2	0.01	1.3	9.4	0.01
	2018-2020	0.11	0.11	0.8	0.001	1.1	7.9	0.008	2.2	15.8	0.02	2.8	20.2	0.02
Kollergang operator	1996-2000	0.13	0.13	0.9	0.001	1.3	9.3	0.009	2.6	18.7	0.02	3.3	23.8	0.02
	2001-2008	0.12	0.12	0.8	0.001	1.2	8.6	0.009	2.4	17.2	0.02	3.0	21.6	0.02
	2009-2016	0.07	0.07	0.5	0.0005	0.7	5.0	0.005	1.4	10.1	0.01	1.8	12.9	0.01
	2018-2020	0.12	0.12	0.8	0.001	1.2	8.6	0.009	2.4	17.2	0.02	3.0	21.6	0.02
Ground hard waste operator	1996-2000	0.16	0.16	1.1	0.001	1.6	11.5	0.01	3.2	23.0	0.02	3.3	23.8	0.02
	2001-2008	0.13	0.13	0.9	0.001	1.3	9.3	0.009	2.6	18.7	0.02	3.3	23.8	0.02
	2009-2016	0.07	0.07	0.5	0.0005	0.7	5.0	0.005	1.4	10.1	0.01	1.8	12.9	0.01
	2018-2020	0.12	0.12	0.8	0.001	1.2	8.6	0.009	2.4	17.2	0.01	3.0	21.6	0.02
Laundry room operator	1996-2000	0.13	0.13	0.9	0.001	1.3	9.3	0.009	2.6	18.7	0.02	3.3	23.8	0.02
	2001-2008	0.13	0.13	0.9	0.001	1.3	9.3	0.009	2.6	18.7	0.02	3.3	23.8	0.02
	2009-2016	0.05	0.05	0.3	0.0003	0.5	3.6	0.004	1.0	7.2	0.01	1.3	9.3	0.01
	2018-2020	0.11	0.11	0.8	0.001	1.1	7.9	0.008	2.2	15.8	0.02	2.8	20.2	0.02
Overall factory		0.11	0.11	0.8	0.001	1.1	7.9	0.008	2.2	15.8	0.02	2.8	20.2	0.02

% Incid – Percentage incidence; CE – Cumulative. Cases/10⁵ – cancer mortality/morbidity cases per 100 000 workers exposed

Table S6: Cumulative exposure on gastrointestinal cancer risk by job, time period, exposure duration of 1, 10, 20 and 25 years: Bulawayo factory

		Duration of exposure												
		1 year				10 years			20 years			25 years		
Job	Time period	Mean f/ml	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid
Saw cutting operator	1996-2000	0.17	0.17	1.2	0.001	1.7	12.2	0.01	3.4	24.5	0.02	4.3	31.0	0.03
	2001-2008	0.12	0.12	0.8	0.001	1.2	8.6	0.009	2.4	17.3	0.02	3.0	21.6	0.02
	2009-2016	0.06	0.06	0.4	0.0004	0.6	4.3	0.004	1.2	8.6	0.009	1.5	10.8	0.01
	2018-2020	0.05	0.05	0.4	0.0004	0.5	3.6	0.004	1.0	7.2	0.007	1.3	9.3	0.01
Fettling table operator	1996-2000	0.17	0.17	1.2	0.001	1.7	12.2	0.01	3.4	24.5	0.02	4.3	31.0	0.03
	2001-2008	0.12	0.12	0.8	0.001	1.2	8.6	0.009	2.4	17.3	0.02	3.0	21.6	0.02
	2009-2016	-	-	-	-	-	-	-	-	-	-	-	-	-
	2018-2020	-	-	-	-	-	-	-	-	-	-	-	-	-
Moulded goods operator	1996-2000	-	-	-	-	-	-	-	-	-	-	-	-	-
	2001-2008	-	-	-	-	-	-	-	-	-	-	-	-	-
	2009-2016	-	-	-	-	-	-	-	-	-	-	-	-	-
	2018-2020	-	-	-	-	-	-	-	-	-	-	-	-	-
Kollergang operator	1996-2000	0.14	0.14	1.0	0.001	1.4	10.1	0.01	2.8	20.1	0.02	3.5	25.2	0.03
	2001-2008	0.12	0.12	0.8	0.001	1.2	8.6	0.009	2.4	17.3	0.02	3.0	21.6	0.02
	2009-2016	0.07	0.07	0.5	0.0005	0.7	5.0	0.005	1.4	10.1	0.01	1.8	12.9	0.01
	2018-2020	0.06	0.06	0.4	0.0004	0.6	4.3	0.004	1.2	8.6	0.009	1.5	10.8	0.01
Ground hard waste operator	1996-2000	0.13	0.13	0.9	0.001	1.3	9.3	0.009	2.6	18.7	0.02	3.3	23.8	0.02
	2001-2008	0.11	0.11	0.8	0.001	1.1	7.9	0.008	2.2	15.8	0.02	2.8	20.1	0.02
	2009-2016	0.07	0.07	0.5	0.0005	0.7	5.0	0.005	1.4	10.1	0.01	1.8	12.9	0.01
	2018-2020	0.06	0.06	0.4	0.0004	0.6	4.3	0.004	2.4	17.3	0.02	1.5	10.8	0.01
Laundry room operator	1996-2000	-	-	-	-	-	-	-	-	-	-	-	-	-
	2001-2008	-	-	-	-	-	-	-	-	-	-	-	-	-
	2009-2016	-	-	-	-	-	-	-	-	-	-	-	-	-
	2018-2020	-	-	-	-	-	-	-	-	-	-	-	-	-
Table S6 continued.....														
		Duration of exposure												
		1 year				10 years			20 years			25 years		
Job	Time period	Mean f/ml	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid	CE f/ml-years	Cases per/10 ⁵	% Incid
Pipe joints operators	1996-2000	0.13	0.13	0.9	0.001	1.3	9.3	0.009	2.6	18.7	0.02	3.3	23.8	0.02
	2001-2008	0.11	0.11	0.8	0.001	1.1	7.9	0.008	2.2	15.8	0.02	2.8	20.1	0.02
	2009-2016	0.05	0.05	0.3	0.0003	0.5	3.6	0.004	1.0	7.2	0.01	1.3	9.3	0.01
	2018-2020	0.05	0.05	0.3	0.0003	0.5	3.6	0.004	1.0	7.2	0.01	1.3	9.3	0.01

Fulllength pipe operator	1996-2000	0.13	0.13	0.9	0.001	1.3	9.3	0.009	2.6	18.7	0.02	3.3	23.8	0.02
	2001-2008	0.11	0.11	0.8	0.001	1.1	7.9	0.008	2.2	15.8	0.02	2.8	20.1	0.02
	2009-2016	0.07	0.07	0.5	0.0005	0.7	5.0	0.005	1.4	10.1	0.01	1.8	12.9	0.01
	2018-2020	-	-	-	-	-	-	-	-	-	-	-	-	-
Multicutter operator	1996-2000	0.13	0.13	0.9	0.001	1.3	9.3	0.009	2.6	18.7	0.02	3.3	23.8	0.02
	2001-2008	0.12	0.12	0.9	0.001	1.2	8.6	0.009	2.4	17.3	0.02	3.0	21.6	0.02
	2009-2016	0.07	0.07	0.5	0.0005	0.7	5.0	0.005	1.4	10.1	0.01	1.8	12.9	0.01
	2018-2020	0.04	0.04	0.3	0.0003	0.4	2.9	0.003	0.8	5.7	0.006	1.0	7.2	0.01
Overall factory		0.12	0.12	0.9	0.001	1.2	8.6	0.009	2.2	15.8	0.02	3.0	21.6	0.02

% Incid – Percentage incidence; CE – Cumulative. Cases/10⁵ – cancer mortality/morbidity cases per 100 000 workers

Table S7: Overall summary estimates of cancer mortality cases by factory, job and duration of exposure of 1 year.

Job	Harare factory							Bulawayo Factory						
	N	Mean ±SD	CE	Cancer cases per 100 000 exposed				N	Mean ±SD	CE	Cancer cases per 100 000 exposed			
				Lung	Meso	Gast	Tot				Lung	Meso	Gast	Tot
Saw cutting operator	254	0.12±0.05	0.12	8.6	5.5	0.84	14.9	137	0.12±0.05	0.12	8.6	5.5	0.84	14.9
Fetting table operator	130	0.12±0.03	0.12	8.6	5.5	0.84	14.9	51	0.16±0.06	0.16	11.5	6.9	1.13	19.5
Moulded goods operator	197	0.10±0.04	0.10	7.2	4.8	0.70	12.7	-	-	-	-	-	-	-
Kollegang operator	212	0.10±0.04	0.10	7.2	4.8	0.70	12.7	126	0.10±0.04	0.10	7.2	4.8	0.70	12.7
Ground hard waste operator	176	0.12±0.04	0.12	8.6	5.5	0.84	14.9	76	0.11±0.04	0.11	7.9	5.1	0.77	13.8
Laundry operator	163	0.12±0.03	0.12	8.6	5.5	0.84	14.9	-	-	-	-	-	-	-
Pipe joints operator	-	-	-	-	-	-	-	103	0.11±0.03	0.11	7.9	5.1	0.77	13.8
Full length pipe operator	-	-	-	-	-	-	-	97	0.12±0.04	0.12	8.6	5.5	0.84	14.9
Multicutter operator	-	-	-	-	-	-	-	66	0.12±0.03	0.12	8.6	5.5	0.84	14.9
Overall factory	1132	0.11±0.04	0.11	7.9	5.1	0.77	13.8	656	0.12±0.04	0.12	8.6	5.5	0.84	14.9

N—number of monthly-averaged personal chrysotile fibre concentrations, 1996—2020; SD—standard deviation; CE – Cumulative exposure in f/ml-years; Lung – lung cancer; Meso – Mesothelioma; Gast – Gastrointestinal cancer; Tot – Total estimated cancers at respective cumulative exposure

Table S8: Overall summary estimates of cancer mortality cases by factory, job and duration of exposure of 10 years.

Job	Harare Factory							Bulawayo Factory						
	N	Mean ±SD	CE	Cancer cases per 100 000 exposed				N	Mean ±SD	CE	Cancer cases per 100 000 exposed			
				Lung	Mes	Gast	Tot				Lung	Meso	Gast	Tot
Saw cutting operator	254	0.12±0.05	1.2	86.4	44	8.6	139	137	0.12±0.05	1.2	86.4	44	8.6	139
Fettling table operator	130	0.12±0.03	1.2	86.4	44	8.6	139	51	0.16±0.06	1.6	115	44	8.6	168
Moulded goods operator	197	0.10±0.04	1.0	72.0	37	7.2	116	-	-	-	-	-	-	-
Kollegang operator	212	0.10±0.04	1.0	72.0	37	7.2	116	126	0.10±0.04	1.0	72	37	7.2	116
Ground hard waste operator	176	0.12±0.04	1.2	86.4	44	8.6	139	76	0.11±0.04	1.1	79	41	7.9	128
Laundry operator	163	0.12±0.03	1.2	86.4	44	8.6	139	-	-	-	-	-	-	-
Pipe joints operator	-	-	-	-	-	-	-	103	0.11±0.03	1.1	79	41	7.9	128
Full length pipe operator	-	-	-	-	-	-	-	97	0.12±0.04	1.2	86.4	44	8.6	139
Multicutte r operator	-	-	-	-	-	-	-	66	0.12±0.03	1.2	86.4	44	8.6	139
Overall factory	1132	0.11±0.04	1.1	79.2	40	7.9	127	656	0.12±0.04	1.2	86.4	44	8.6	139

N—number of monthly-averaged personal chrysotile fibre concentrations, 1996—2020; SD—standard deviation; CE – Cumulative exposure in f/ml-years; Lung – lung cancer; Meso – Mesothelioma; Gast – Gastrointestinal cancer; Tot – Total estimated cancers at respective cumulative exposure

Table S9: Overall summary estimates of cancer mortality cases by factory, job and duration of exposure of 20 years

Job	Harare Factory							Bulawayo Factory						
	N	Mean ±SD	CE	Cancer cases per 100 000 exposed				N	Mean ±SD	CE	Cancer cases per 100 000 exposed			
				Lung	Mes	Gast	Tot				Lung	Meso	Gast	Tot
Saw cutting operator	254	0.12±0.05	2.4	173	88	17	278	137	0.12±0.05	2.4	173	88	17	278
Fettling table operator	130	0.12±0.03	2.4	173	88	17	278	51	0.16±0.06	3.2	230	117	22	369
Moulded goods operator	197	0.10±0.04	2.0	144	73	14	231	-	-	-	-	-	-	-
Kollegang operator	212	0.10±0.04	2.0	144	73	14	231	126	0.10±0.04	2.0	144	73	14	231
Ground hard waste operator	176	0.12±0.04	2.4	173	88	17	278	76	0.11±0.04	2.2	158	81	16	255
Laundry operator	163	0.12±0.03	2.4	173	88	17	278	-	-	-	-	-	-	-
Pipe joints operator	-	-	-	-	-	-	-	103	0.11±0.03	2.2	158	81	16	255
Full length pipe operator	-	-	-	-	-	-	-	97	0.12±0.04	2.4	173	88	17	278
Multicutter operator	-	-	-	-	-	-	-	66	0.12±0.03	2.4	173	88	17	278
Overall factory	1132	0.11±0.04	2.2	158	81	16	255	656	0.12±0.04	2.4	173	88	17	278

N—number of monthly-averaged personal chrysotile fibre concentrations, 1996—2020; SD—standard deviation; CE – Cumulative exposure in f/ml-years; Lung – lung cancer; Meso – Mesothelioma; Gast – Gastrointestinal cancer; Tot – Total estimated cancers at respective cumulative exposure

Table S10: Overall summary estimates of cancer mortality cases by factory, job and duration of exposure of 25 years

Job	Harare Factory							Bulawayo Factory						
	N	Mean ±SD	CE	Cancer cases per 100 000 exposed				N	Mean ±SD	CE	Cancer cases per 100 000 exposed			
				Lung	Meso	Gast	Tot				Lung	Meso	Gast	Tot
Saw cutting operator	254	0.12±0.05	3.0	216	109	22	347	137	0.12±0.05	3.0	216	109	22	347
Fettling table operator	130	0.12±0.03	3.0	216	109	22	347	51	0.16±0.06	4.0	288	146	29	463
Moulded goods operator	197	0.10±0.04	2.5	180	91	18	289	-	-	-	-	-	-	-
Kollegang operator	212	0.10±0.04	2.5	180	91	18	289	126	0.10±0.04	2.5	180	91	18	289
Ground hard waste operator	176	0.12±0.04	3.0	216	109	22	347	76	0.11±0.04	2.8	203	102	20	325
Laundry operator	163	0.12±0.03	3.0	216	109	22	347	-	-	-	-	-	-	-
Pipe joints operator	-	-	-	-	-	-	-	103	0.11±0.03	2.8	203	102	20	325
Full length pipe operator	-	-	-	-	-	-	-	97	0.12±0.04	3.0	216	109	22	347
Multicutter operator	-	-	-	-	-	-	-	66	0.12±0.03	3.0	216	109	22	347
Overall factory	1132	0.11±0.04	2.8	203	81	20	304	656	0.12±0.04	3.0	216	109	22	347

N—number of monthly-averaged personal chrysotile fibre concentrations, 1996—2020; SD—standard deviation; CE – Cumulative exposure in f/ml-years; Lung – lung cancer; Meso – Mesothelioma; Gast – Gastrointestinal cancer; Tot – Total estimated cancers at respective cumulative exposure.

Table S11: Estimates of asbestosis incidence after 25 years of exposure

Job	Harare factory					Bulawayo factory				
	N	Mean ±SD	CE	% Incidence	Cases per 100 000	N	Mean ±SD	CE	% Incidence	Cases per 100 000
Saw cutting operator	254	0.12±0.05	3.0	0.16	160	137	0.12±0.05	3.0	0.16	160
Fettling table operator	130	0.12±0.03	3.0	0.16	160	51	0.16±0.06	4.0	0.22	220
Moulded goods operator	197	0.10±0.04	2.5	0.14	140	-	-	-	-	-
Kollergang operator	212	0.10±0.04	2.5	0.14	140	126	0.10±0.04	2.5	0.14	140
Ground hard waste operator	176	0.12±0.04	3.0	0.16	160	76	0.11±0.04	2.8	0.15	150
Laundry room operator	163	0.12±0.03	3.0	0.16	160	-	-	-	-	-
Pipe joints operator	-	-	-	-	-	103	0.11±0.04	2.8	0.15	150
Full length pipe operator						97	0.12±0.04	3.0	0.16	160
Multicutter operator						66	0.12±0.04	3.0	0.16	160
Overall factory	1132	0.11±0.04	2.8	0.15	150	656	0.12±0.03	3.0	0.16	160

N—number of monthly-averaged personal chrysotile fibre concentrations, 1996—2020; SD—standard deviation;
CE – Cumulative exposure in f/ml-years.

Table S12: Estimated number of workers working at various jobs in the chrysotile asbestos cement manufacturing factories

Job	Time period	Harare factory		Bulawayo	
		No. of workers by time period	Maximum possible workers per job	No. of workers by time period	Maximum workers per job
Saw cutting operator	1996-2000	54	54	54	54
	2001-2008	45		54	
	2009-2016	27		54	
	2018-2020	3		54	
Fettling table operator	1996-2000	15	15	15	15
	2001-2008	15		15	
	2009-2016	15		15	
	2018-2020	-		15	
Moulded Goods operator	1996-2000	18	18	-	-
	2001-2008	18		-	
	2009-2016	18		-	
	2018-2020	-		-	
Kollergang operator	1996-2000	12	12	12	12
	2001-2008	12		12	
	2009-2016	12		12	
	2018-2020	-		12	
Ground hard waste operator	1996-2000	6	6	6	6
	2001-2008	6		6	
	2009-2016	6		6	
	2018-2020	-		6	
Laundry room operator	1996-2000	2	2	-	-
	2001-2008	2		-	
	2009-2016	2		-	
	2018-2020	-		-	
Pipe joints operators	1996-2000	-	-	27	27
	2001-2008	-		27	
	2009-2016	-		27	
	2018-2020	-		27	
Fulllength pipe operator	1996-2000	-	-	27	27
	2001-2008	-		27	
	2009-2016	-		27	
	2018-2020	-		27	
Multi-cutter operator	1996-2000	-	-	12	12
	2001-2008	-		12	
	2009-2016	-		12	
	2018-2020	-		12	
Total Average			107		153

Appendix 3: Ethics Approval



R14/49 Mr B Mutetwa

HUMAN RESEARCH ETHICS COMMITTEE (MEDICAL) CLEARANCE CERTIFICATE NO. M181157

NAME: Mr B Mutetwa
(Principal Investigator)
DEPARTMENT: School of Public Health
Division of Occupational Health
Medical School
University

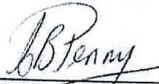
PROJECT TITLE: Occupational exposure to chrysotile asbestos fibre in the chrysotile asbestos cement manufacturing industry in Zimbabwe

DATE CONSIDERED: 30/11/2018

DECISION: Approved unconditionally

CONDITIONS:

SUPERVISOR: Professor D Brouwer


APPROVED BY: 
Dr CB Penny, Chairperson, HREC (Medical)

DATE OF APPROVAL: 16/05/2019

This clearance certificate is valid for 5 years from date of approval. Extension may be applied for.

DECLARATION OF INVESTIGATORS

To be completed in duplicate and ONE COPY returned to the Research Office Secretary on the 3rd Floor, Phillip Tobias Building, Parktown, University of the Witwatersrand, Johannesburg.
I/we fully understand the conditions under which I am/we are authorized to carry out the above-mentioned research and I/we undertake to ensure compliance with these conditions. Should any departure be contemplated, from the research protocol as approved, I/we undertake to submit details to the Committee. I agree to submit a yearly progress report. When a funder requires annual re-certification, the application date will be one year after the date when the study was initially reviewed. In this case, the study was initially reviewed in November and will therefore reports and re-certification will be due early in the month of November each year. Unreported changes to the application may invalidate the clearance given by the HREC (Medical).


Principal Investigator Signature

17/5/2019
Date

PLEASE QUOTE THE CLEARANCE CERTIFICATE NUMBER IN ALL ENQUIRIES