EVALUATION OF OCCUPATIONAL EXPOSURE TO WOOD DUST AND NOISE AMONG SAWMILL WORKERS IN THE GERT SIBANDE DISTRICT MUNICIPALITY

by

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Dissertation submitted in fulfilment of the requirements for the degree of

MASTER OF HEALTH SCIENCES IN ENVIRONMENTAL HEALTH

in the

Department of Life Sciences

Faculty of Health and Environmental Sciences

at the

Central University of Technology, Free State

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BLOEMFONTEIN, SOUTH AFRICA

NOVEMBER 2022

Declaration

I, Rathipe Moeletsi, with student number: _____, do at this moment declare that, except the references made to work done by other authors, which have been duly acknowledged, this work was done by me under supervision. I declare further that I have not previously submitted this dissertation, partially or in full, at any other tertiary institution in respect of a qualification.

10/11/2022

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Acknowledgements

- Many thanks to the Almighty God, who has given me the strength, knowledge and wisdom to complete this dissertation.
- I would like to send my sincere thanks and appreciation to my main supervisor Dr. F.S. Raphela from the Central University of Technology, Free State, for his advice and inspiration during my study in the Faculty of Health Science.
- I would like to express my deepest gratitude to Mr D. Seleme, the Occupational Hygienist from Omohau Solutions for monitoring instruments and assistance provided during noise and wood dust monitoring at the sawmills.
- I would like to express my sincere thanks and appreciation to all persons who have contributed positively to the development of this dissertation.
- The Chief Directorate: Occupational Health and Hygiene from the National Department of Employment and Labour is highly acknowledged for the noise and air monitoring training for hazardous chemical agents and the necessary information provided.
- I would like to thank the management and the sawmill workers who participated in this study for their assistance, cooperation and understanding. You have helped me through the process, and I will always be grateful for the help you have provided in my life.

Dedications

I dedicate this dissertation to the sawmill workers working at the wet and dry mills located within the Gert Sibande District Municipality in Mpumalanga Province.

† In loving memories of my late grandfather Motsamai, father Tsila and mother Elizabeth Rathipe †

Summary

Wood dust is a human carcinogen based on an increased risk of nasal and sinonasal cancer. Occupational exposure to hazardous noise above the 85 decibels, A-weighted (85 dB[A]), eight-hours time-weighted average (8-hour TWA) can cause noise induced hearing loss (NIHL). The aim of the study was to measure and determine the time-weighted average (TWA) occupational exposure level (OEL) to wood dust and noise and compare the results with the OEL for wood dust and the noise rating limit for noise and assess hearing loss and respiratory symptoms and use of personal protective equipment (PPE) among sawmill workers within the Gert Sibande District Municipality. A comparative cross-sectional study comprising of 137 exposed and 20 unexposed workers randomly selected, using simple random sampling was undertaken between January and March 2021. Self-reported hearing loss and respiratory symptoms were evaluated using a self-administered questionnaire. Personal and area wood dust exposure levels were measured using calibrated Giliair-3 personal air sampling pumps, while personal and area noise surveys were conducted using calibrated SV104IS noise dosimeters (SVANTEK, Poland) and a type 1 sound level meter (Soundpro SE/DL, U.S.A.).

Data was analysed using the Microsoft Excel 2019 analysis Tool Pak to obtain a summary of descriptive statistics. The geometric mean, standard deviation, minimum and maximum values were calculated. Mann-Whitney U test was used to test the significance of the differences between values in sawmill A and B while *t*-test was used to compare the mean time-weighted averages of occupational exposure levels to noise from sawmills A and B. Discrete data from questionnaire results were presented as percentages and tables. The *t*-test was used to compare continuous variables, while the chi-square test was used to test categorical responses. Fisher's test was employed when the anticipated number was below 5. A significance level of 0.05 was used. The prevalence of symtoms such as chest pains or shortness of breath was higher among the unexposed group (50%) than the exposed group (44%). Furthermore, the participants in the exposed group suffered from tinnitus (ringing in the ears) (50%) and ear infections (21.43%). Moreover, the unexposed group reported suffering from tinnitus (33%) and ear infections (66.67%). The exposed group (86.86%) reported always wearing the personal protective equipment (PPE) than the unexposed group (75%) who wear it sometimes.

Moreover, the exposed group (48.48%) did not wear PPE consistently due to not being available while all participants from the unexposed group reported other reasons for not wearing it. The geometric mean(GSD) for personal respirable wood dust exposure level at sawmill A was 0.9(4.8) mg/m^3 while at sawmill B was 0.57(0.75) mg/m^3. The geometric mean(GSD) for personal respirable wood dust exposure level at sawmill A was 0.9(4.8) mg/m^3 while at sawmill B was 0.57(0.75) mg/m^3. The geometric mean(GSD) for personal total inhalable wood dust exposure level at sawmill A was 0.37(0.94) mg/m^3 while at sawmill B was 1.19(16.91) mg/m^3. Moreover, the geometric mean(GSD) for area respirable wood dust exposure at sawmill A was 0.13(0.09) mg/m^3 while at sawmill B was 0.8(0.6) mg/m^3. Likewise, the geometric mean(GSD) for area total inhalable wood dust exposure at sawmill A was 0.13(0.16) mg/m^3 while at sawmill B was 0.54(0.55) mg/m^3. The geometric mean(GSD) for area noise exposure level at sawmill A was 90.05(8.02) dB(A) while at sawmill B was 90.14(7.94) dB(A). The geometric mean(GSD) for personal noise exposure level at sawmill A was 92.26(4.35) dB(A) while at sawmill B was 92.24(2.65) dB(A).

Occupational exposure to wood dust and noise was associated with respiratory symptoms such as chest pains or shortness of breath and hearing loss conditions or symptoms such as tinnitus (ringing in the ears) and ear infections (H_a accepted and H_0 rejected). The majority of the results for wood dust samples were below the South African OEL, while majority of the results for noise exposure levels were above the noise rating limit (H_a accepted and H_0 rejected). A non-significant difference on the levels of exposure to noise was observed when comparing the results of sawmill A to sawmill B (H_0 accepted and H_a rejected). Similarly, a non-significant difference on the levels of exposure to wood dust was observed when comparing the results of sawmill A to sawmill B (H_0 accepted and H_a rejected). The findings suggest that sawmill owners should take steps to lower the levels of exposure to noise and wood dust to help protect workers' health.Implementation of engineering and administrative controls supplemented by appropriate use of the fit-tested hearing protective devices (HPDs) with the higher noise reduce reduction (NRR) or single number rating (SNR) and the respiratory protection devices (RPDs) with the higher assigned protection factor (APF) is recommended based on the exposure levels recorded.

Tobacco or cigarette smoking should be discouraged because when associated with exposure to wood dust and noise may increase risk of respiratory symptoms and development of high frequency hearing loss.

Keywords: Occupational exposure, wood dust, cancer, noise level, noise-induced hearing loss

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Abbreviations

µm	micrometer or micron (10^{-6} meters)
ACGIH	American Conference of Governmental Industrial Hygienists
APF	Assigned protection factor of RPD, P1=4, P2=10, P3=20
COPD	Chronic Obstructive Pulmonary Disease
CVD	Cardiovascular disease
dB(A)	Decibel(s), A-weighted average
FFP	Filtering face piece, number indicates filter type
h/hr	hour(s)
HPDs/RPDs	Hearing protective devices or respiratory protective devices
ISO	International Organisation for Standardisation
kHz	kilohertz, the unit of sound frequency
$L_{Req,\ 8hr}$	8-hour equivalent continuous rating level
$L_{Req,\ T}$	Equivalent continuous rating level for a given period of time
mg/m^3	milligrams (10^{-3} gm) per cubic metre
mmHg	millimetres of mercury
NIHL	Noise Induced Hearing Loss
NIOSH	National Institute for Occupational Safety and Health
NRR/SNR	Noise reduction rating or Single number rating
OEL	Occupational Exposure Limit
OSHA	Occupational Safety and Health Administration
PEL/REL	Permissible exposure limit or Recommended exposure limit
PPE	Personal protective equipment
SANAS	South African National Accreditation System
SANS	South African National Standards, SANS 10083:2013
SLM	Sound Level Meter

Definition of terms

A-weighted decibel [dB(A)] is the measurement of sound pressure levels as perceived by humans, calculated by the use of a spectral sensitivity factor (A-filter) that weighs sound pressure levels by frequency to match the sensitivity of the human ear**.**

Action level is the concentration specified on 29 CFR part 1910 of OSHA for that particular substance, computed as an average over 8-hours' time which require certain actions such as exposure monitoring and medical surveillance. Always lower than the permissible exposure limit (PEL) and in reality, the action level frequently corresponds to 50% or 1/2 of the permissible exposure level.

Assigned Protection Factor (APF) is a respirator protection level that the respirator is expected to provide to a worker when an effective respiratory program is implemented at the workplace as defined on EN 529: 2005.

Attenuation of the HPD is the HPD defined on SANS 1451-1, SANS 1451-2 and SANS 1451-3 with proven capability of reducing the sound exposure to which the wearer is exposed to. HPDs efficiency levels are determined using OSHA, NIOSH and HSE prescribed methods for attenuation to 85 dB(A) and the residual noise at the ears must be between 70 to 84dB as per SANS 50458:2008.

Carcinogen refers to any chemical substance or mixture that causes cancer or raises its occurrence and is categorised by the Globally Harmonised System (GHS) as a known or suspected human carcinogen falling either in category 1 or 2.

Criteria level (L_c) is the steady noise level allowed over a whole 8-hour work shift, most places use 85 dB(A) as the acceptable level.

Decibels (**dB**) refer to the used units for measuring sound pressure levels on a logarithmic scale.

Derate refers to the use of a fraction of a hearing protector's noise reduction rating (NRR) to calculate the noise exposure of a worker wearing that hearing protector.

Dermatitis is an inflammatory condition on the skin characterised by skin irritation

Dose is the level of an environmental contamination multiplied by the duration (length of time) of exposure to the contaminant while dose-response is the relationship between the dose and the health effects. On the curve, the dose (X-axis) is plotted against the response (Y-axis).

Equivalent aerodynamic diameter (**AED**) is the diameter of a sphere of unit density (one gram per cubic centimetre), which exhibits the same aerodynamic properties as the particle of any shape or density being measured.

Equivalent continuous rating level ($L_{Req,T}$) refers to the equivalent continuous A-weighted sound pressure level ($L_{Aeq,T}$) for a given period of time plus specified corrections for the impulsiveness of the sound and is computed as follows:

Equation 1: $L_{Req,\,Ts} = 10 \log \frac{1}{n} \left(\sum_{i=1}^{n} 10^{LReq,Ti/10} \right)$, dB(A)

Where, $L_{Req,Ts}$ is the average equivalent continuous rating level,
$L_{Aeq,Ti}$ is the individual equivalent continuous rating level measured by a SLM and
n is the number of measurement position (three or more).

Eight(8)-hour equivalent continuous rating level ($L_{Req,8h}$) refers to the rating level normalised to an 8-hour workday and computed as follows:

Equation 2: $L_{Req,8h} = L_{Req,\,Ts} + 10 \log \frac{Ts}{8}$, dB(A),

Where $L_{Req,8h}$ is an equivalent continuous 8-hour rating level,
$L_{Req,\,Ts}$ is an equivalent continuous rating level determined over the duration of the work shift while Ts is the total working shift in hours and
8 is the reference work shift in hours.

Eight (8)-hour time weighted average (8-hour TWA) is the average airborne concentration of a specific substance allowed over an 8-hour workday and a 5-day workweek**.**

Equivalent sound level is the equivalent sound level over a given time period calculated when sound levels vary over time, which happens frequently with occupational noise. The A-weighted sound level is represented by $L_{Aeq,\,T}$, which is the A-weighted sound level averaged over time (*T*). In occupational research and regulations, a typical exposure time, T is 8 hours. The parameter is denoted by the notation $L_{Aeq,8h}$.

Filter cassette is the protective casing that holds the filter media**.**

Exposure action values are noise exposure levels defined by the control of noise at work regulation of 2005 of the United Kingdom and the European Union Directive 2003/10/EC for physical agents (noise). The employer is required to take a particular action to monitor the exposure levels if the value is exceeded. There is a lower exposure action values for the daily or weekly personal noise exposure of 80 dB(A) and the upper exposure action values of 85 dB(A) where the use of HPD is mandatory if noise exposure cannot be reduced by engineering or other technical measures.

Genotoxicity is the ability of a substance to cause harm to a cell's genetic material. Such compounds are classified as genotoxic or mutagenic substances.

Hearing protective devices (HPDs) is the technical term for earplugs or earmuffs that can be worn to lower the level of sound entering the ear. It is worn in places where the noise exposure level is at 85 dB(A) and above regardless of exposure time.

Hypersensitivity pneumonitis is a lung disorder in which the lungs get inflamed as a result of an allergic reaction arising from exposure to dusts of animal and vegetable origin or the inflammation of the terminal bronchioles and alveoli.

Inhalable fraction refers to the fraction of airborne material that passes through the mouth and nose during breathing and is thus available for deposition anywhere along the respiratory tract.

***L*Aeq, 8hr (*L*Req, 8hr)** is an average sound level, also known as A-weighted equivalent-continuous sound level or rating level, standardized to an 8-hour period using a 3 dB exchange rate. It is similar to $L_{EX,8h}$ which according to the ISO 1999 standards is defined as the value for A-weighted sound pressure level of a continuous, steady sound within specified time interval, T_m and has the same mean-square sound pressure as a sound under consideration whose levels varies with time.

Leukaemia is a group of blood diseases whereby excessive amount of aberrant white blood cells are formed in the bone marrow.

***L*EX, T** is a logarithm that considers both the exposure and the actual time worked as determined by a noise dosimeter.

Lignin is an essential component of plants' secondary cell walls which is primarily found in wood.

Local exhaust ventilation (**LEV**) is an engineering technique for extracting contaminated air from the source or close by to reduce workers' exposure. It is among the most effective techniques available, compared to general or diluted ventilation.

Lung cancer refers to the type of cancer that starts in the lungs and is characterised by expansion of aberrant lung cells. It may be caused by a number of substances including smoking.

Noise dosimeter is basically the SLM designed to measure a worker's noise exposure integrated over a period of time. Personal noise exposure level is the amount of noise to which an individual employee is exposed and is measured using a noise dosimeter. Noise surveys involve measuring noise levels at selected locations throughout an entire plant or sections to identify noisy areas using SLM.

Noise induced hearing impairment occurs primarily at higher frequencies between 3000 and 6000 Hz, with the highest impact around 4000 Hz.

Noise induced hearing loss (NIHL) is a permanent sensorineural hearing loss that develops gradually, initially affecting higher hearing frequencies. It is mostly related to the cochlear hair cell damages and often occurs in patients with a history of long-term exposure to harmful noise level.

Noise rating limit for hearing conservation programme (HCP) means the value of an 8-hour rating level ($L_{Req,\ 8h}$), at or above 85 dB(A) in which hearing impairment is likely to result.

Noise zone means a noise zone as contemplated in regulation 9(a) of the Noise Induced Hearing Loss Regulation of 2003 promulgated under the Occupational Health and Safety Act,1993 (Act No. 85 of 1993) as amended. The noise zone must be demarcated according to SANS 1186-1 and hearing conservation measures specified on clause 5 of SANS 10083:2013 should be implemented.

Noise reduction rating (**NRR**) is the HPD noise reduction rating, as supplied by the manufacturer.

Occupational asthma refers to the type of asthma characterised by allergic constriction of the bronchi and bronchioles that is accompanied by bronchial inflammation due to exposure to irritants at work.

Occupational Exposure Limit (**OEL**) is defined by the Occupational Exposure Standards (OES), as an average concentration of an airborne substance over a reference period at which, there is no evidence that exposure to that concentration by inhalation on a daily basis will likely cause harm to employees as defined in EH40/2005.

Ototoxicity is an ear poisoning brought on by medications (drugs) or substances that interfere with hearing or auditory capabilities. The substances that lead to ototoxicity are known as ototoxicants.

Percentage loss of hearing (**PLH**) is the measure of hearing loss determined in accordance with annexure E of SANS 10083:2013 standard.

Percentage loss of hearing shift (**PLH shift**) is a shift in PLH from the baseline that exceeds 10% during diagnosis for compensation for NIHL (see SANS 10083:2013 diagnostic audiology.

Permanent threshold shift is the shift or difference between the normal hearing level and the level at which hearing recovers after prolonged exposure to noise.

Personal monitoring is a process used to establish the concentration of an airborne chemical within the employee's breathing zone. Breathing zone is the space within 20 to 30 cm of the person's nose and mouth while fixed place or static monitoring is used to measure the amount of chemicals at a particular fixed site at the workplace in order to gather data on the possible sources that may be contributing to the exposure, among other things as described in HSG 173:2006 standard.

Personal protective equipment (**PPE**) for this study refer mainly on RPDs and HPDs which are specialised equipment worn by workers to protect them against exposure to noise and wood dust.

Respirable fraction refers to the fraction of airborne material that reaches the gaseous exchange region of the lung.

Respirator or respiratory zone means a respiratory zone as contemplated in regulation 8(a) of the Hazardous Chemical Substance Regulation of 1995 promulgated under the OHS Act, 1993 (Act No. 85 of 1993). The respiratory zone must be demarcated and identified by notice, sign or pictograms as prescribed on EN 132:1999.

Respiratory protective equipment (**RPE**) is a particular type of PPE worn to protect the individual wearer against inhalation of hazardous substances in the workplace as defined on HSG 53:2013. For the purpose of choosing RPD for wood dust, wood dust results are divided by OEL and compare the results to APF. The RPD with the highest APF is then chosen, fitted, and worn as directed.

SANS 1008:2013 means the code of practice for the measurement and assessment of occupational noise for hearing conservation purpose.

Sawdust refers to fine wood dust particles produced from sawing of wood.

Sawmills are the establishments that perform wood processing, sawn wood, preservation, sawmilling, drying of sawn wood and wood working.

Sensitiser refers to respiratory sensitisers and cutaneous sensitisers, that after repeated exposure to hazardous chemical substances, causes a sizable majority of those exposed to experience an allergic reaction in normal tissue.

Sound level meter (**SLM**) is used to take readings at ear height where workers are standing to record area readings. It determines locations of high and low noise exposure and track amount of time spent during a shift in noisy environments while noise dosimeter measure sound and time spent at noise levels to get individualised exposure.

Sound levels is the sensitivity of the human ear to sounds of various frequencies. A spectral sensitivity factor is used to weigh the sound pressure level at various frequencies (A-filter) in order to take into account the perceived loudness of a sound. These sound pressure values are A-weighted and reported as decibels [dB(A)].

Standard threshold shift (**STS**) is a measure of substantial hearing loss defined as a 10 dB increase in hearing threshold averaged over 2, 3 and 4 kHz in the same ear as compared to the individual's baseline as per OSHA. It also refers to a 15 dB or more shift in either ear at 0.5, 1, 2, 3, 4 or 6 kHz with the same shift at same test frequency in the same ear on an instant retest as per NIOSH definition with no age corrections allowed**.**

Sound pressure level (***L*p**) is a measure of the air vibrations that make up sound. The loudness of a sound is measured on a logarithmic scale using decibels (dB) to represent the range of sound pressure levels that the human ear can perceive [20 micropascals (µPa) to 200 Pascal (Pa)].

Threshold limit value – ceiling (TLV-C) is the maximum concentration of a hazardous chemical substance that should not be exceeded during any part of the working shift or exposure.

Threshold limit value – ceiling (TLV-C) is the exposure limit that should not be exceeded even instantaneously. The accepted limit for the peak sound pressure level is 140 dB for an impulsive sound as per OSHA occupational noise standard 29 CFR 1910.95. This limit is also used in United States of America(USA) viz. ACGIH, the United Kingdom(UK), European Union (EU) and ISO 1999:1990 although France use the 135 dB (C-weighted).

Threshold limit value – **time-weighted average** (**TLV-TWA**) is the time-weighted average concentration of a hazardous material in the air that is deemed to be safe for workers to repeatedly be exposed to, day after day, throughout the duration of their working lives. This concentration is averaged over 8-hour workday and 40-hour workweek.

Threshold of hearing (Threshold shift) refers to the sound level that can be heard by a normal ear. It is characterised by a change in hearing thresholds of an average 10 dB or more at 2, 3 and 4 kHz in either ear (worse hearing).

Tinnitus refers to the ringing/buzzing/clanging/clicking/whistling/humming sound in the ears, which is uncomfortable and can cause sleep disruption.

Time-weighted average (TWA) refers to the average rate for occupational noise exposure among workers on a daily basis (normalised to an 8-hour workday), taking into consideration the average noise levels and the amount of time spent in each location.

Wood dust is a broad phrased term covering a range of airborne dusts. Hardwoods and softwoods are the two categories into which all types of timber fall. Hardwoods such as beech, ash, oak, mahogany, and teak are made from deciduous trees that can grow in both temperate and tropical climates while conifers like scots pine, yew, and cedar make up the majority of the softwoods species also called gymnosperm.

Chapter 1: Introduction

1.1. Background

Wood has been used by humans ever since ancient times and will continue to be the most crucial element as the demand for wood and wood products grows (Forest Products Laboratory, 1999; Ramage, Burridgeb, Busse-Wicher, Fereday, Reynolds, Shah, Wu, Yu, Fleming, Densley-Tingley, Allwood, Dupree, Linden and Scherman, 2017; Ross, 2010). Wood is an important renewable natural resource that is widely used in buildings and manufacturing furniture (Alwis, 1998). Over 1 000 species of trees are widely used in mining, pulp and paper mills, and cabinet manufacturing factories (Chamber and Nunes, 2016). South Africa began using treated and untreated timber in, 1652, mainly for construction projects, mining applications, roof trusses, and manufacturing pallets, boxes, ceilings, doors, and floor tiles (Sawmilling South Africa, 2014; Showers, 2010).

Between 1910 and 1919, the exotic species of black wattle (*Acacia mearnsii*), pine, and *Eucalyptus* trees were introduced into the South African forests mainly for growth and cultivation (Sawmilling South Africa, 2014; Tourism Marketer, 2013a). Between 1970 and 1980, the state issued a public notice, stopping the expansion of state sawmilling, resulting in the privatization of sawmills (Sawmilling South Africa, 2014; Tourism marketer, 2013b). In the early 2000s, there were approximately 330 private sawmills that were active in the South African markets, processing pine, *Eucalyptus* trees, and other exotic tree species (Caveney, 2019; Heyl, Maltitz, Evans and Segoale, 2000; Knysna museums, 2021; South Africa forest online, 2017; Sawmilling South Africa, 2014). Pine trees were the dominant species widely used in South Africa mainly because 73% of structural pines were exported for use in making furniture.

In general, sawmill industries are viewed as not an attractive investment, even though they were estimated to contribute about R46 billion to the country's gross domestic product (GDP), while forestry and other manufacturing sectors were estimated to contribute around R35.4 billion (Sawmilling South Africa, 2014). Timber processing factories differed from each other by their dimensions because large-scale sawmills have a log intake of 200 000 per cubic meter (m^3) per annum while small-scale sawmills have a fewer log intake of 5 000 m^3 per annum (Heyl *et al*., 2000). Timber processing

factories or sawmilling is an establishment where fresh logs or dry timbers are sawed into timber boards (Adhikari and Ozarska, 2018; Edmond, Oluniyi and Bamidele, 2014). Timber processing factories usually generate wood dust particles that contain 92(100) % wood dust and 8(0) % formaldehyde (Universal Forestry Products, 2010). Wood dust can be a health hazard if compliance with occupational health and safety is neglected (Awosan, Ibrahim, Yunusa, Isah, Ango and Michael, 2018; Buliga, Rongo and Mamuya, 2017; Health and Safety Authority, 2010; Zobel and Sprague, 1998; Hermaline, 2017). Wood dust is mainly generated when machines cut or shape wood. Fine wood dust particles are emitted during the process and become suspended in the air and pose a health risk (Sussell, Periakaruppan and Burr, 2005).

When these fine dust particles are inhaled they can deposit in the nose or throat, upper bronchial region, or lungs depending on the strength of airflow, shape, density, chemical and toxicological properties, aerodynamic diameter, and size of the wood dust particles (Alwis, 1989; Mazzoli and Favoni, 2012). Contrary to that, the 85 dB(A), 8-hour National Institute for Occupational Safety and Health (NIOSH) recommended exposure limit (REL) and 90 dB(A), 8-hour Occupational Safety and Health Administration (OSHA) permissible exposure limit (PEL) may not protect workers against noise induced-hearing loss (NIHL) due to exposure to unwanted sound (NIOSH, 1998). Mining, construction, manufacturing, transportation and agricultural industries are characterized as the noisiest industries, contributing to the development of NIHL (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004). The NIOSH predicted a 40-year lifetime risk of NIHL among workers exposed to 90 dB(A) OSHA PEL over an 8-hour day or 40-hour week with 1 in 4 workers (25%) with NIHL while the 85 dB(A) NIOSH PEL have 1 in 12 workers (8%) with NIHL when exposed over an 8-hour day or 40-hour week (Anthony, 2016; NIOSH, 1998). NIHL was estimated to cost between 0.2 to 2 % of the country's GDP (Lie, Skogstad, Johannessen, Tynes, Mehlum, Nordby, Engdahl and Tambs, 2016; Nandi and Dhatrak, 2008).

1.2. Statement of the problem

A large number of epidemiological studies have confirmed wood dust as a human carcinogen (Demers, Stellman, Colin and Boffetta, 1998; Demers, Teschke and Kennedy, 1997; International Agency for Research on Cancer (IARC), 1995; Kauppinen, Vincent, Liukkonen, Grzebyk, Kauppinen, Welling, Arezes, Black, Bochmann, Campelo, Costa, Elsigan, Goerens, Kikemenis, Kromhout, Miguel, Mirabelli, Mceneany, Pesch, Plato, Schlu¨nssen, Schulze, Sonntag, Verougstraete, de Vicente, Wolf, Zimmermann, Husgafvel-Pursiaine and Savolainen, 2006; Schlünssen and Schaumburg, 1998; Stellman, Demers, Colin and Boffetta, 1998). Numerous studies have found that exposure to hardwood dust at levels greater than 5mg/m^3 increases the risk of developing nasal and sinonasal cancer (Barcenas, Delclos, Zein, Tortolero-Luna, Whitehead and Spitz, 2005; Straumfors, Olsen, Daae, Afanou, McLean, Corbin, Mannetje, Ulvestad, Bakke, Johnsen, Douwes and Eduard, 2018; Kauppinen *et al.*, 2006; Youldena, Cramba, Petersb, Porcedduc, Møllerd, Fritschib and Baadea, 2013).

Despite this, more than one million workers worldwide are exposed to wood dust particles in concentrations greater than 5mg/m^3 (Kauppinen *et al.*, 2006; Schlünssen and Schaumburg, 1998). Wood dust exposure between 0.5 and 1 mg/m^3 has not been linked with sinonasal cancer, despite the fact that exposure to wood dust below 0.5 mg/m^3 can cause asthma, bronchial hyperresponsiveness and pulmonary function impairment, while exposure above 0.5 mg/m^3 can cause pulmonary effects (Kauppinen *et al*., 2006; Scientific Committee on Occupational Exposure Limits (SCOEL), 2003). Cancer is the leading cause of death in Europe, accounting for 53% of all deaths, and nearly 3 500 people die each year in the United Kingdom due to carcinogenic exposure (European Commission, 2018).

Several international studies have confirmed that, workers in the sawmills are exposed to harmful sound (Ramage-Morin and Gosselin, 2008). Ratnasingam, Natthondan, Loras and McNully (2010) conducted a noise survey in South East Asia and found the highest noise levels of 130 decibels A-weighted (dB(A)) average, at the rough milling section of the furniture manufacturing factory. Antonio, Antonio, Evangelista and Doddato (2013) measured noise levels in an Italian sawmill and reported noise levels

ranging from 85 to 110dB(A). Vermal, Demers, Finkelstein, Shaw, Kurtz, Verma and Welto (2007) reported noise levels ranging from 55 to 117dB(A) at an Ontario sawmill and veneer/plywood plants, which were greater than the Canadian limit of 85dB(A). The sound levels produced by sawmill machines can range between 80 to 120 dB(A) while idle saws can produce noise levels of up to 95dB(A) (Choudhari, Dhote and Patil, 2011; Ugbebor and Yorkor, 2015).

According to Concha-Barrientos, Campbell-Lendrum and Steenland (2004), nearly 30 million workers in the United States of America (USA) and five million workers in Germany were reported to be exposed to hazardous noise at work. According to the World Health Organization (WHO), NIHL affects approximately 250 million people in developing countries. Likewise, the British University of Columbia reported 27 000 workers suffering from heart disease as a result of exposure to excessive noise (Ugbebor and Yorkor, 2015). The global loss of NIHL on production, early retirement, and unemployment of hearing loss patients costs the countries approximately 105 billion United States (US) dollars per year and this does not include the costs of other negative health effects such as stress and fatigue (Adhikari and Sahu, 2016). According to Antonio *et al.* (2013), NIHL has been estimated to costs Canada about 18 billion dollars per year. The major health hazards at the sawmill are loud sounds generated by the noisy equipment and airborne wood dust particles produced during the sawing process (Buliga, Rongo and Mamuya, 2017). Osuchukwu, Osuchukwu, Eko and Otaren (2015) conducted an air monitoring for respirable dust in Calabar and found a respirable wood dust level of 31.75 mg/m^3, which was greater than the 5 mg/m^3 OSHA PEL and 1 mg/m^3 NIOSH REL.

On the contrary, noise exposure can cause occupational stress which is the leading cause of coronary heart disease (CHD) (Kivimäki, Virtanen, Elovainio, Kouvonen, Väänänen and Vahtera, 2006). Several researchers have found an association between occupational stress and worker morbidity and mortality (Carrère, Evans, Palsane and Rivas, 1991). Stress, stroke and abnormal behavior are caused by hearing loss (Otoghile, Onakoya and Otoghile, 2018). Also, extreme noise is linked to hypertension and CHD (Talbott, Helmkamp, Mathews, Kuller, Cottington and Redmond, 1985; van Kempen, Kruize, Boshuizen, Ameling, Staatsen and de Hollander, 2002). Workers who leave a healthy lifestyle are less likely to develop cardiovascular disease (CVD), while those smoking cigarettes/tobacco are more likely

to develop hearing loss (Fransen, Topsakal, Hendrickx, Van Laer, Huyghe, van Eyken, Lemkens, Hannula, Jensen, Demeester, Tropitzsch, Bonaconsa, Mazzoli, Espeso, Verbruggen, Huyghe, Kunst, Manninen, Diaz-Lacava, Steffens, Wienker, Pyykkö, Cremers, Kremer, Dhooge, Stephens, Orzan, Pfister, Bille, Parving, Sorri, van de Heyning and van Camp, 2008)

Sawmill operators are constantly exposed to harmful sound and the attenuation level of the hearing protection devices (HPDs) may offer insufficient protection (under protection) as the residual noise at the ears may be above 85dB, with risks of additional noise exposures or may offer too much protection (overprotection) as the residual noise at the ears may be below 70dB, resulting in communication problems among co-workers due to earplugs blocking sounds that they need to hear such as voices and warning signals (South African National Standard (SANS), 2008; Uppin, Ogunfowokan, Mbada, Olatubi and Ogungbemi, 2014). Further, engineering controls implemented at the sawmills may be ineffective, and compliance with HPDs usage may be less than 100% (Koehncke, 1999). There is a lack of accurate epidemiological data on the prevalence, risk factors and costs of NIHL, as well as hyperacusis, lung, and bronchial diseases which account for 14% and 75% of reportable diseases, respectively (Rus, Daud, Musa and Naing, 2008; Straumfors *et al*., 2018). Also, because of limited statistics globally, the European Union community database on the prevalence of hearing loss can be used as a control mechanism to reduce NIHL as a burden of occupational disease worldwide (Antonio *et al*., 2013).

The assessment of noise levels at the sawmill will provide information on the harmful effects of exposure to loud sound for the measurement of hearing acuity or audiometric evaluation of workers to determine the presence of NIHL. Evaluating the concentration of wood dust levels will assist in determining an association between work-related exposure and health outcome in an epidemiological study where measurement of the arithmetic mean may be applicable. There is a need to determine the accurate noise dose delivered to workers' ears while wearing HPDs because 27% of workers may be overexposed to loud sound despite wearing HPDs (Koehncke, 1999). The study aims to provide empirical evidence on the extent of worker exposure to loud sound and its health effects on the hearing capability to help policymakers to propose measures that will help protect workers' health (Gyamfi, Amankwaa, Sekyere and Boateng, 2016).

1.3. Aim of the study

The aim of the study was to measure and determine the time-weighted average (TWA) occupational exposure levels (OEL) to wood dust and noise and assess hearing loss and respiratory symptoms among sawmill workers within the Gert Sibande District Municipality.

1.3.1. Objectives of the study

The objectives of the study were to:

- Measure and determine TWA exposure levels of wood dust in the sawmill factories.
- Measure and determine TWA exposure levels of noise in the sawmill factories.
- Compare the findings to the South African OEL for wood dust and noise rating limit for noise.
- Assess the prevalence of hearing loss and respiratory symptoms and use of personal protective equipment (PPE) among sawmill workers.
- Recommend appropriate measures to be implemented to reduce the risk.

1.3.2. Research question

What are the levels of exposure to noise and wood dust and associated hearing loss and respiratory symptoms and use of PPE among sawmill workers within the Gert Sibande District Municipality?

1.3.3. Hypotheses

H_0 There is no statistical significant difference in the levels of exposure to noise between sawmill A and sawmill B.

H_a There is a statistical significant difference in the levels of exposure to noise between sawmill A and sawmill B.

H_0 There is no statistical significant difference in the levels of exposure to wood dust between sawmill A and sawmill B.

H_a There is a statistical significant difference in the levels of exposure to wood dust between sawmill A and sawmill B.

H_0 The exposure levels of wood dust and noise in the sawmills do not exceed the South African OEL for wood dust and noise-rating limit for noise.

H_a The exposure levels of wood dust and noise in the sawmills exceed the South African OEL for wood dust and noise-rating limit for noise.

H_0 Exposure to wood dust and noise is not associated with hearing loss and respiratory symptoms among sawmill workers.

H_a Exposure to wood dust and noise is associated with hearing loss and respiratory symptoms among sawmill workers.

1.4. Ethical clearance and approval

The ethical approval (Clearance No.: UFS-HSD2019/2236/3006) was obtained from the Health Science Research Ethics Committee of the Free State University (see Appendix A). Permission to conduct the study was granted by the managers in charge of the sawmills and the participants gave consent to take part in the study. Participation in the study was voluntary and participants were allowed to withdraw at any time

1.5 Justification

The purpose of the study was to determine the levels of occupational exposure to noise and wood dust and make recommendations on how to reduce carcinogens, NIHL, or mutagenic exposure among sawmill workers to help protect their health. The major challenge in preventing cancer is the absence of information about where and how many employees are exposed to carcinogens (Peters, Ge, Hall, Davies and Demers, 2015; European Commission, 2018). The reasons for conducting air monitoring and noise surveys at the workplace are to monitor workers' exposure to physical agents (noise) and chemical agents (wood dust) for compliance comparisons with the noise-rating limit and the occupational exposure limit (OEL) (Davies and Henderson, 2009a; Driscoll, 1995; Driscoll, Milk and Burgess, 2009; Health and Safety Executive (HSE), 2006; HSE, 2020). In addition, it is to ascertain if exposure levels meet the local or international standards and to establish a relationship between exposure and health outcomes (diseases) in an occupational epidemiological study where measurement of the central tendency or arithmetic mean may be applicable (Kothari, 2004; Ramachandran, 2008; Rappaport, Kromhout and Symanski, 1993). The exposure levels of workers doing the same task may differ over time among workers, locations, and shifts, and the sampling strategy must be able to quantify this variation and produce accurate exposure assessments (Kromhout, Symansk and Rappaport, 1993; Ramachandran, 2008). A literature review of recent occupational hygiene journals or books must be consulted, and appropriate sampling strategies must be carefully selected to address the research problem.

A good hearing loss prevention and/or respiratory protection program can help sawmill factories and the workers who work there (Franks, Stephenson and Merry, 1996). Workers may experience reduced weariness, and their general health may improve. Eventually, the factories can benefit from a reduction in medical costs and claims. This will improve workers' confidence and efficiency. However, the presence of a hearing loss prevention or respiratory protection program is not a guarantee against protection from occupational hearing loss or cancer. Management must create policies and programs to address the integration of the hearing loss prevention program or respiratory program into the company's policy. In addition, an employee with the ultimate task of implementing management systems must be appointed in writing (Franks, Stephenson and Merry, 1996).

The government should consider revising its policies in consultation with other social partners in order to help employers purchase machinery that satisfies local and international emission limits (HSE, 2017; Wang, Zhou, Li, Kong, Wang, Guo, Zhang, He, Guo and Chen, 2018). The consultation will help in developing effective guidelines for reducing hearing loss and cancer as a burden of occupational disease worldwide. A register on hearing loss or mortality rate caused by nasal or sinonasal cancer published on journals, newsletters, and websites will provide information to other professionals, such as epidemiologists or health care professionals (Davies and Henderson, 2009a; Davies and Henderson, 2009b; Driscoll, Milk and Burgess, 2009; Hirst, 2010; International Organization for Standardization (ISO), 1999). Employers and employees at the sawmills will benefit as knowledge gained or shared through the process will guide them to develop programs that will help them protect against exposure to wood dust and noise.

About 5 million people die each year due to tobacco use. It is estimated that by 2030, this number will increase to 10 million, with 70% of deaths occurring each year in developing countries (Blackadar, 2016; Chang and Adami, 2006; Simpson, Niven, Pickering, Fletcher, Oldham and Francis, 1999). An employee who quits smoking at work can save up to 12 months of costs per year (Ekpu and Brown, 2015). Implementing smoke-free workplaces may encourage many new smokers to reduce their cigarette intake. Due to a drop in the number of occurrences, workplace interventions are likely to improve the economy by fostering a healthier and more productive workforce (Ekpu and Brown, 2015). It is important to adopt policies and disseminate information about the health risks of smoking, consuming alcohol, taking medicinal drugs, and being exposed to wood dust and noise at high or low frequencies (Chang and Adami, 2006; Blackadar, 2016; Simpson *et al*., 1999).

Schulte and Howard (n.d.) illustrated that there were concerns that technology would reduce or eliminate jobs by replacing humans with robots. However, Abdychev, Alonso, Alper, Desruelle, Kothari, Liu, Perinet, Rehman, Schimmelpfennig and Sharma (2018) argued that ageing and ill workers will need more money to pay for pensions and healthcare services. If noise and wood dust exposure are not well managed, most retired and ill-working people will not be able to afford consultation with a private otorhinolaryngologist, speech therapist, or occupational doctor or nurse.

Consequently, the government will have to carry the financial burden and render services to the poor. The government must invest in infrastructure and health to provide the foundation of the country and influence the opportunities from the fourth industrial revolution. The policies of today for the jobs of the future will create a flexible educational system, smart urbanization, and trade integrations that will strengthen the social safety net and connect us (Abdychev *et al*., 2018; Department of Environmental Affairs and Development Planning, n.d.).

1.6 Structure of the dissertation

The background, rationale, problem statement, aim and objectives of this study, hypotheses, significance, and scope of this study are outlined in Chapter 1. Chapter 2 outlines a literature review on wood dust, noise, and related health effects. Results on exposure to wood dust among sawmill workers at the timber processing factories are presented in Chapter 3, while the results of exposure to noise are provided in Chapter 4. Chapter 5 presents findings on hearing loss and respiratory symptoms linked with exposure to wood dust and noise at timber processing factories within the Gert Sibande District Municipality. Conclusion and recommendations for chapters 3, 4, and 5 are presented in Chapter 6. The chapters are presented in article format. Chapter 3 has been published in the *Annals of Agriculture and Environmental Medicine*, while Chapter 4 is in the Journal of *Applied Artificial Intelligence*, and Chapter 5 has been submitted for publication in the *BMC Public Health Journal*.

1.7 Scope of the study

A comparative cross-sectional study comprising of 137 exposed and 20 unexposed workers aged between 18 and 65 years was undertaken from January to March 2021. The study was carried out at the sawmill factories located within the Gert Sibande District Municipality in Mpumalanga Province. The municipality covers an area of about 31 841 square kilometre.The factories were selected based on their size, location, number of workers, and wood type being processed. Participants in this study comprise chipper operators, unscramble operators, log operators, welders, stopper operators, trim saw operators, bell drivers, profile cutters, log frame operators, general workers, staffer operators, door cutters, housekeepers, and grader operators. The administrative staff (office workers) and maintenance personnel constituted the unexposed group because of their occupations, which did not expose them to wood dust and noise under normal circumstances. Twenty-two participants took part in the personal noise monitoring, while 34 participated in the wood dust monitoring (16 for respirable and 18 for total inhalable wood dust) as per the NIOSH sampling strategy.

Area noise surveys were carried out at the finger joint, knotty pine, and profile doors, the saw extractor machine next to the door house, the saw shop, workshop, dry mill, boiler house, green chain, the wet mill, and the chipper machine next to the dry mill. The results were plotted on the floor diagram to show the locations of the noise areas that were at or above the noise rating limit. Area respirable wood dust monitoring was done at the finger joint, green chain, dry mill adjacent to the profile cutter machine 1, dry mill next to the opticut machine 1, dry mill next to the profile cutter machine 2, and wet mill. Total inhalable dust monitoring was done at the knotty pine and profile house, door house, dry mill, wet mill, saw shop, door house next to the profile cutter 1, green chain next to the chipper machine 2, knotty pine and profile house next to the profile cutter 2, door house next to the staffer machine 1, and knotty house next to the profile cutter machine 2.

1.8 References

Abdychev, A., Alonso, C., Alper, E., Desruelle, D., Kothari, S., Liu, Y., Perinet, M., Rehman, S., Schimmelpfennig, A. and Sharma, P. (2018) *The future of work in Sub-Saharan Africa*. Washington: International Monetary Fund.

Adhikari, A. and Sahu, S. (2016) prevalence of noise induced hearing loss (NIHL) among sawmill workers in West Bengal, India. *International Journal of Current Research and Academic Review* **4**(9): 12-20.

Alwis, K.U. (1998) *Occupational exposure to wood dust*. *(Ph.D. Thesis)*. Australia: University of Sydney.

Anthony, R.T. (2016) *Noise monitoring*. Lowa: Lowa-Illinois Safety Council.

Antonio, C.D., Antonio, P.D., Evangelista, C. and Doddato, V. (2013) The assessment of the sawmill noise. *Journal of Agricultural Engineering* **155**:768-772.

Awosan, K.J., Ibrahim, M.T.O., Yunusa, E.U., Isah, B.A., Ango, U.M. and Michael, A. (2018) Knowledge of workplace hazards, safety practices and prevalence of workplace-related health problems among sawmill workers in Sokoto, Nigeria. *International Journal of Contemporary Medical Research* **5**(10): J5 – J12.

Barcenas, C.H., Delclos, G.L., EL-Zein, R., Tortolero-Luna, G., Whitehead, L.W. and Spitz, M.R. (2005) Wood dust exposure and the association with lung cancer risk. *American Journal of Industrial Medicine* **47**(4): 349-357.

Blackadar, C.B. (2016) Historical review of the causes of cancer. *World Journal of Clinical Oncology* **7**(1): 54–86. DOI:10.5306/wjco.v7.i1.54.

Buliga, V., Rongo, L.M.B. and Mamuya, S.H.D. (2017) Dust exposure and respiratory health symptoms among workers in small scale sunflower oil industry in Singida Tanzania. *International Journal of Research Granthaayah* **5**(12): 452–463.

Carrère, S., Evans, G.W., Palsane, M. and Rivas, M. (1991) Job strain and occupational stress among urban public transit operators. *Journal of Occupational Psychology* **64**(4): 305-316.

Caveney, P. (2019) *The Thesen homes of Knysna.* Available from: http://www.theheritageportal.co.za/article/thesen-homes-knysna.(Accessed on 9 April 2021).

Chamber, P. and Nunes, E. (2016) Work-related asthma among workers in the wood processing industry: A review. *Current Allergy and Clinical Immunology Journal* **29**(2):110-117.

Chang, E.T. and Adami, H. (2006) The enigmatic epidemiology of nasopharyngeal carcinoma. *Cancer Epidemiology, Biomarkers and Prevention* **15**(10):1765-1777. DOI: 10.1158/1055-9965.

Choudhari, V.P., Dhote, D.S. and Patil, C.R. (2011) Assessment and control of sawmill noise.*International Conference of Chemical and Environmental Sciences* **1**(1):299-303.

Concha-Barrientos, M., Campbell-Lendrum, D. and Steenland, K. (2004) *Occupational noise: Assessing the burden of disease from work-related hearing impairment at national and local levels*. Geneva: WHO.

Davies, B. and Henderson, J. (2009a) *W501 student manual measurements of hazardous chemical substances*. Australia: University of Wollongong.

Davies, B. and Henderson, J. (2009b) *W505 student manual control of hazardous substances. Australia*: University of Wollongong.

Demers, P.A., Teschke, K. and Kennedy, S.M. (1997) What to do about softwood? A review of respiratory effects and recommendations regarding exposure limits. *American Journal of Industrial Medicine* **31**: 385–398.

Demers, P.A., Stellman, S.D., Colin, D. and Boffetta, P. (1998) Non-malignant respiratory disease mortality among woodworkers participating in the American cancer society cancer prevention study-ii (CPS-II). *American Journal of Industrial Medicine* **34**: 238–243.

Department of Environmental Affairs and Development Planning (DEADP). (n.d.) *Managing air quality at the sawmills*. Pretoria: Government Printer.

Driscoll, D. (1995) *Occupational Safety and Health Administration (OSHA) technical manual (OSHATM). Section III: Chapter 5 - Noise*. Available from: https://www.osha.gov/otm/section-3-health-hazards/chapter-5.(Accessed on 8 Jan 2021).

Driscoll, D.P.P.E, Milk, K. and Burgess, M. (2009) *W503 student manual noise: measurements and its effects*. UK: British Occupational Hygiene Society (BOHS).

Edmond, O.A., Oluniyi, A.A. and Bamidele, O.O. (2014) Re-engineering technical vocational education and training (TVET) towards safety practice skill needs of sawmill workers against workplace hazards in Nigeria. *Journal of Education and Practice* **5**(7)**:** 142-149.

Ekpu, V.W. and Brown, K. (2015) The Economic Impact of Smoking and of Reducing Smoking Prevalence: Review of Evidence. *Tobacco Use Insights* **8**(1):1–35. DOI:10.4137/TUI.S15628.

European Commission. (2018) *Draft European parliament legislative resolution the proposal for a directive of the European parliament and of the council amending Directive 2004/37/EC on the protection of workers from the risks related to exposure to carcinogens or mutagens at work (COM (2018)0171 – C8-0130/2018 – 2018/0081(COD)).*Available from: http://www.europarl.europa.eu/doceo/document/A-8-2018-0382_EN.html. (Accessed on 11 February 2020).

Forest Products Laboratory. (1999) *Wood handbook: wood as an engineering material. General Technical Report FPL, GTR-113.* Madison, Wisconsin, USA: Department of Agriculture and Forest Services.

Franks, J.R., Stephenson, M.R. and Merry, C.J. (1996) *Preventing occupational hearing loss: A practical guide*. Cincinnati, Ohio: NIOSH.

Fransen, E., Topsakal, V., Hendrickx, J., Van Laer, L., Huyghe, J.R., Van Eyken, E., Lemkens, N., Hannula, S., Mäki-Torkko, E., Jensen, M., Demeester, K., Tropitzsch, A., Bonaconsa, A., Mazzoli, M., Espeso, A., Verbruggen, K., Huyghe, J., Huygen, P.L.M., Kunst, S., Manninen, M., Diaz-Lacava, A., Steffens, M., Wienker, T.F., Pyykkö, I., Cremers, C.W.R.J., Kremer, H., Dhooge, I., Stephens, D., Orzan, E., Pfister, M., Bille, M., Parving, A., Sorri, M., Van de Heyning, P. and Van Camp, G. (2008) Occupational noise, smoking, and a high body mass index are risk factors for age-related hearing impairment and moderate alcohol consumption is protective: A European population-based multicenter study. *Journal of the Association for Research in Otolaryngology* **9**(3): 264-263.DOI:10.1007/s10162-008-0123-1.

Gyamfi, C.K.R., Amankwaa, I., Sekyere, F.O. and Boateng, D. (2016) Noise exposure and hearing capabilities of quarry workers in Ghana: A cross-sectional study. *Journal of Environmental and Public Health* **2016**(1)*:*705- 4276. DOI:10.1155/2016/7054276.

Health and Safety Executive (HSE). (2006) *Monitoring strategies for toxic substances, HSG 173* .2nd ed. London: HSE.

Health and Safety Executive (HSE). (2020) *EH40/2005 workplace exposure limit*. London: TSO.

Health and Safety Executive (HSE). (2017) *Controlling airborne contaminants at work: A guide to local exhaust ventilation (LEV), HSG 258.* 3th ed. London: TSO.

Hermaline, J. (2017) A study to assess the effectiveness of video assisted teaching programme (VATP) on knowledge regarding occupational hazards and safety measures among workers of selected sawmill at Thiruvarur. *International Journal of Recent Research and Applied Studies* **4**(22): 86-89.

Heyl, L., Maltitz, G.V., Evans, J. and Segoale, R. (2000) *Issues and opportunities for small-scale sawmilling in South Africa: An Eastern Cape case study. Instruments for sustainable private sector forestry, South Africa series.* London and Pretoria: International Institute for Environment and Development and CSIR- Environmental.

Hirst, A. (2010) *W201 Basic principles in occupational hygiene*. UK: OHTA

International Agency for Research on Cancer (IARC). (1995) *Wood dust and formaldehyde, IARC monographs on the evaluation of carcinogenic risks to humans.* Lyon, France: WHO.

International Organization for Standardization (ISO). (1999) *Acoustics: Determination of occupational noise exposure and estimation of noise-induced hearing impairment*. Geneva: ISO.

Kauppinen, T., Vincent, R., Liukkonen, T., Grzebyk, M., Kauppinen, A, Welling, I., Arezes, P., Black, N., Bochmann, F., Campelo, F., Costa, M., Elsigan, G., Goerens, R., Kikemenis, A., Kromhout, H., Miguel, S, Mirabelli, D., Mceneany, R., Pesch, B., Plato, N., Schlu¨nssen, V., Schulze, J., Sonntag, R., Verougstraete, V., de Vicente, M.A., Wolf, J., Zimmermann, M., Husgafvel-Pursiaine, K. and Savolainen, K. (2006) Occupational exposure to inhalable wood dust in the Member States of the European Union. *Annals of Occupational Hygiene* **50**(6): 549–561.

Kivimäki, M., Virtanen, M., Elovainio, M., Kouvonen, A., Väänänen, A. and Vahtera, J. (2006) Work stress in the aetiology of coronary heart disease: A meta-analysis. *Scandinavian Journal of Work, Environment and Health* **32**(6): 431-442.

Knysna museums. (2021) *The timber merchants: Knysna museums.* Available from: https://www.knysnamuseums.co.za/pages/the-timber-merchants/. (Accessed on 7 February 2021).

Koehncke, N. (1999) *An investigation of noise in Alberta sawmills. (Master Thesis)*. Canada: University of Alberta, Edmonton.

Kothari, C. R. (2004) *Research methodology: Methods and techniques*. 2nd ed. New Delhi: New Age International limited.

Kromhout, H., Symanski, E. and Rappaport, S. M. (1993) A comprehensive evaluation of within and between-worker components of occupational exposure to chemical agents. *Annal of Occupational Hygiene* **37**(3): 153-270.

Lie**,** A., Skogstad, M., Johannessen, H.A., Tynes, T., Mehlum, I.S., Nordby, K., Engdahl, B. and Tambs, K. (2016) Occupational noise exposure and hearing: A systematic review. *International Archives of Occupational and Environmental Health* **89**(3):351-372. DOI:10.1007/s00420-015-1083-5.

Mazzoli, A. and Favoni, O. (2012) Particle size, size distribution and morphological evaluation of airborne dust particles of diverse woods by Scanning Electron Microscopy and image processing program. *Power Technology* **225**: 65-71.

Nandi, S.S. and Dhatrak, S.V. (2008) Occupational noise-induced hearing loss in India. *Indian Journal of Occupational and Environmental Medicine* **12**(2): 53-56.

NIOSH. (1998) *Criteria for a recommended standard for occupational noise exposure revised criteria.* Cincinnati, Ohio: NIOSH.

Osuchukwu, N.C., Osuchukwu, E.C. and Eko, J.E. (2015) Occupational exposure to wood dust in Calabar Municipality, Cross River State, Nigeria. *International Journal of Science and Research (IJSR)* **4**(1):1414-1420.

Otoghile, B., Onakoya, P.A. and Otoghile, C.C. (2018) Auditory effects of noise and its prevalence among sawmill workers. *International Journal of Medicine and Medical Sciences* **10**(2):27-30.

Peters, C.E., Ge, C.B., Hall, A.L., Davies, H.W. and Demers, P.A. (2015). CAREX Canada: An enhanced model for assessing occupational carcinogen exposure. *Occupational and Environmental Medicine* **72**: 64–71.

Ramachandran, G. (2008) Toward Better Exposure Assessment Strategies—The New NIOSH Initiative. *Annals of Occupational Hygiene* **52**(5):297–301.

Rappaport, S.M., Kromhout, H. and Symanski, E. (1993) Variation of exposure between workers in homogeneous exposure groups. *American Industrial Hygiene Association Journal* **54**(11):654-662. DOI: 10.1080/15298669391355198.

Ratnasingam, J., Natthondan, V., Loras, F. and McNulty, T. (2010) Dust, noise and chemical solvents exposure of workers in the wooden furniture Industry in South East Asia. *Journal of Applied Sciences* **10**: 1413-1420.

Ramage, M.H., Burridgeb, H., Busse-Wicher, M., Fereday, G., Reynolds, T., Shah, D.U., Wu, G., Yu, L., Fleming, P., Densley-Tingley, D., Allwood, J., Dupree, P., Linden, P.F. and Scherman, O. (2017) The wood from the trees: The use of timber in construction. *Renewable and Sustainable Energy Reviews* **68**(1): 333-359.

Ramage-Morin, P.L. and Gosselin, M. (2008) Canadians vulnerable to workplace noise. *Health Reports* **29**(8): 9-17.

Ross, R.J. (ed). (2010) *Wood handbook: wood as an engineering material. General technical report FPL-GTR-190.* Madison, U.S.A.: Dept. of Agriculture, Forest Service, Forest Products Laboratory.

Rus, R.M., Daud, Z., Musa, K.I. and Naing, L. (2008) Knowledge, attitude and practice of sawmill workers towards noise-induced hearing loss in Kota Bharu, Kelantan. *Malaysian Journal of Medical Sciences* **15**(4): 28-34.

South African National Standard (SANS 50458). (2008) *Hearing protectors – Recommendations for selection, use, care and maintenance – Guidance document*. Pretoria: South African Bureau of Standards.

South Africa forest online. (2017) *Celebrating 125 years in the timber business*. Available from: http://saforestryonline.co.za/articles/celebrating-125-years-in-the-timber-business/. (Accessed on 07 February 2021).

Sawmilling South Africa (SSA). (2014) *Sawmilling in South Africa.*Pretoria:Government Printer.

Schlünssen, V. and Schaumburg, I. (1998) Asthma, bronchitis and chronic obstructive pulmonary disease in occupational exposure to wood. *Ugeskr Laeger* **160**(5):609-615.

Schulte, P. and Howard, J. (n.d.) *Future of work*. Washionton DC: NIOSH.

Scientific Committee on Occupational Exposure Limits (SCOEL). (2003) *Recommendation from the Scientific Committee on Occupational Exposure Limits: Risk assessment for wood dust.* (SCOEL/SUM/102). [S.l.: S.n.].

Simpson, J.C.G., Niven, R.M., Pickering, C.A.C., Oldham, L.A., Fletcher, A.M. and Francis, H. C. (1999) Comparative personal exposures to organic dusts and endotoxin. *Annals of Occupational Hygiene* **43**(2): 107–115.

Showers, K.B. (2010) Prehistory of Southern African forestry: from vegetable garden to tree plantation. *Environment and History* **16**(3):295-322.

Stellman, S.D., Demers, P.A., Colin, D. and Boffetta, P. (1998) Cancer mortality and wood dust exposure among participants in the American Cancer Society Cancer Prevention Study-II (CPS-II). *American Journal of Industrial Medicine* **34**(3):299-237.

Straumfors, A., Olsen, R., Daae, H.L., Afanou, A., McLean, D., Corbin, M., Mannetje, A.T, Ulvestad, B., Bakke, B., Johnsen, H.L., Douwes, J. and Eduard, W. (2018) Exposure to wood dust, microbial components and terpenes in the Norwegian Sawmill industry. *Annals of work Exposures and Health* **62**(6): 674-688.

Sussell, A., Periakaruppan, P. and Burr, G. (2005) *NIOSH health evaluation: Ikens hardwood floor services.* Madison, Wisconsin: NIOSH.

Talbott, E., Helmkamp, J., Mathews, K., Kuller, L., Cottington, E. and Redmond, G. (1985) Occupational noise exposure, NIHL, and the epidemiology of high blood pressure. *American Journal of Epidemiology* **121**(4):501–514.

Tourism Marketer. (2013a) *Sabie, Mpumalanga, South Africa: A short biography of Hans Merensky, Renowned prospector, successful farmer and philathropist.* Available from: https://sabie.co.za/blog/?p=2014. (Accessed on 07 March 2021).

Tourism marketer. (2013b) *Sabie: History of South African forest investments*. Available from: https://sabie.co.za/blog/?p=2042. (Accessed on 07 February 2021).

Ugbebor, J.N. and Yorkor, B. (2015) Assessment and evaluation of noise pollution levels in selected sawmill factories in Port Harcourt, Nigeria. *International Journal on Emerging Technologies* **6**(2): 01-08.

Universal Forestry Products (UFP). (2010) *Material safety data sheet for wood dust.* Available from: http://blogs.nwic.edu/rocketteam/files/2011/09/wooddust-165.pdf. (Accessed on 13 March 2019).

Uppin, F.A., Ogunfowokan, A.A, Mbada, C., Olatubi, M.I. and Ogungbemi, A.V. (2014) Occupational Hazards awareness and safety practices among Nigerian sawmill workers. *Journal of Medical Science and Public Health* **3**(10):1244-1248.

Van Kempen, E.E.M.M., Kruize, H., Boshuizen, H.C., Ameling, C.B., Staatsen, B.A.M. and de Hollander, A.E.M. (2002) The association between noise exposure and blood pressure and ischemic heart disease: A meta-analysis. *Environmental Health Perspectives* **110**(3):307–317.

Vermal, D.K., Demers, C., Finkelstein, M., Shaw, D., Kurtz, L., Verma, P. and Welto, T. (2007) Occupational exposure to chemicals, biological and physical agents in Ontario sawmill and veneer/plywood plants. *Research Gate*. Available from: https://www.researchgate.net/publication/267821918_Occupational_Exposure_to_Chemical_Biological_and_Physical_Agents_in_Ontario_Sawmill_and_Veneer_Plywood_Plants. (Accessed on 23 April 2019).

Wang, D., Zhou, M., Li, W., Kong, W., Wang, Z., Guo, Y., Zhang, X., He, M., Guo, H. and Chen, W. (2018) Occupational noise exposure and hypertension: The Dongfeng-Tongji Cohort Study. *Journal of the American Society of Hypertension* **12**(2):71-79. DOI: 10.1016/j.jash.2017.11.001.

Youldena, D.R., Cramba, S.M, Petersb, S., Porcedduc, S.V., Møllerd, H., Fritschib, L. and Baadea, P.D. (2013) International comparisons of the incidence and mortality of sinonasal cancer. *Cancer Epidemiology* **1**(1):1-26.

Zobel, B.J. and Sprague, J.R. (1998) *Juvenile wood in forest trees.* USA: Springer-verlag berling Heiderburg.

Chapter 2: Literature Review

Abstract

Forests are a valuable natural resource. About 0.3% of trees are harvested each year for domestic and global use. Softwood saw logs processed at the sawmills are estimated to be 4.1 million per cubic meter per annum on an average, while hardwood saw logs consumed per year were estimated to be 260 000 cubic meter. Sawmills are workplaces where fresh or dry timber is sawed into timber boards. Sawmill operations are classified as one of the most hazardous processes because fine dust particles are produced during the process. These fine dust particles are generated at an average speed of 10m/s. Likewise, these particles are larger than 10µm in aerodynamic diameter and can easily stuck in the nasal passages, while those smaller than 10µm can permeate deeper into the gas exchange region of the lung. It is claimed that the concentration of tannin and other related compounds in wood contributes to the adverse health effects.

Similarly, sawmill workers are constantly exposed to noise levels above 85 dB(A), which may cause NIHL. The NIOSH recommended an exposure limit of 85 dB(A) for an 8-hour period, while OSHA set the permissible exposure limit of 90 dB(A), which may not be effective in protecting workers against auditory health effects such as tinnitus, temporary or permanent threshold shift, as well as non-auditory effects such as irritation, cardiovascular disease, psychological stress, and hypertension. HPDs with attenuation protection ranging between 70 and 84 dB(A) can help protect against NIHL when used properly in conjunction with other engineering and administrative controls. It is critical to implement policies on smoking restrictions and educate workers about the health risks of exposure to noise, wood dust, smoking cigarettes, and using ototoxic drugs. Tobacco use, heredity, socioeconomic status, and high blood pressure have been linked to an increased risk of cardiovascular diseases.

Keywords: Wood dust, cancer, occupational noise, hearing loss

2.1. Introduction

Forests are a valuable natural resource (Vermal, Demers, Finkelstein, Shaw, Kurtz, Verma and Welto, 2007). The Canadian forests comprised 66% softwood, 22% mixed wood, and 12% hardwood, while the Ontario forests comprised 58% softwood, 26% mixed wood, and 16% hardwood (Vermal *et al*., 2007). The species of trees within the woodland are categorized either as hardwood and softwood. Softwood or coniferous trees, such as pine, fir, spruce, cedar, and others, are classified as gymnosperms and produce seeds without coverings. Conversely, deciduous trees or hardwoods, viz. oak, beech, and walnut, are categorized botanically as angiosperms due to the fact they produce seeds with coverings (International Agency for Research on Cancer (IARC), 2012b; IARC, 2012a; SCOEL, 2003). About 0.3% of indigenous trees are harvested each year for the domestic or global market (Vermal *et al*., 2007).

Moving fresh logs from the woodlands to the mill requires a variety of equipment (Agbana, Joshua, Daikwo and Metiboba, 2016; Odibo, Nwaogazie, Achalu and Ugbebor, 2018). At the log yard, logs are positioned on the deck by a bell logger, cut into lengths with a cut-off saw, debarked to remove the bark, and then allowed to run through chains to be sawn by a circular saw. After that, boards are chosen, and scraps are permitted to proceed to the chipper. All of the slabs are subsequently broken up into chips by the chipper, which are then blown into vans. Following the edger, boards move over the trimmer to be cut to the pre-selected lengths on both ends before falling onto a set of chains to be stacked (Alwis, 1998; Adhikari and Ozarska, 2018). The average speed of the fine, dry dust particles produced by this method is 10 m/s (Proto, Zimbalatti and Negri, 2010). Dust particles with a diameter of less than 5 micrometers (µm), are released in tiny amounts when thin wood is processed (Proto, Zimbalatti and Negri, 2010). The factories that make cabinets and furniture as well as sawmills were found to emit the highest fraction of fine wood dust particles (National Cancer Institute, 2018).

Wood dust is made up of dust particles with an aerodynamic diameter greater than 10µm, which can become trapped in the nasal cavity (Alwis, Mandryk and Hocking, 1999a; Davies, Teschke and Demers, 1999a; Löfstedt, Hagström, Bryngelsson, Holmström and Rask-Andersen, 2017; IARC, 1995a). The average diameter of wood is normally between 10 and 30µm (Proto, Zimbalatti and Negri, 2010). Wood dust is

produced when machines cuts or shapes wood (Adeoye, Adeomi, Abodunrin, Olugbenga-Bello and Abdulsalam, 2015; FAO, 2010; Hagström, Axelsson, Arvidsson, Bryngelsson, Lundholm and Eriksson, 2008a; Harper and Muller, 200b; Löfstedt *et al*., 2017; National cancer institute, 2018; National Toxicology Program (NTP), 2016). The physical and chemical properties of wood dust depend on the species of trees, working conditions, growing environments and other factors (Health council of the Netherlands, 2000; IARC, 2012b; WorkSafe New Zealand, 2019). Wood is composed of polymeric compounds such as extracts, hemicellulose, cellulose and lignin (Occupational health clinics for Ontario worker's, 2016; SCOEL, 2003).

2.2. Literature search strategy and selection criteria

A literature search on PubMed, Science Direct, Scopus, Google Scholar and reference from related articles in English from the 1st of January 1970 to the 1st of February 2021, using the 1st search terms: [‘’Occupational noise”, “cardiovascular diseases”, “hypertension”, “blood pressure”, “myocardial infarction”, “coronary heart diseases”], 2nd search terms: [“Occupational noise”, “health effects”, “hearing loss”], 3rd search terms [“Occupational noise”, “hearing impairment], 4th search terms: [“wood dust”, “exposure”, “health effects”] and 5th search terms: [“wood dust”, “cancer”] were consulted. The IARC monographs, Word Health Organisation (WHO), US Department of Labour, OSHA, ACGIH, NIOSH CDC, Department of Energy (DOE), EU standards and British journal of work exposure and Health, HSE books and International Journals of medicine *et cetera* were also consulted.

The author searched and consulted PRISMA on published systematic reviews. The author focused on research papers published in the past. However, research papers that denoted influential research or deemed crucial to understand recent findings were used. Reports from recent conferences that were pertinent to the study were included. The first searches were limited to materials with full abstract to rule suitability for full text retrieval. Papers were excluded if they were not relevant to the intended nature of our review. In addition, references to key sources were included to strengthen and provide up-to-date and easy-to-access point-of-reference to assist interested parties in seeking further information on subject matter or more importantly in improving workers’ health.

2.3 Absorption, distribution, metabolism and excretion of hazardous substances

The amount of wood dust, that settles in the nasal cavity depends on the individual's breathing rate (Brown, Gordon, Price and Asgharian, 2013; WorkSafe New Zealand, 2019). Inhaled dust deposits in the nose and throat, upper bronchial region or lungs, depending on airflow strength, shape, density, chemical and toxicological properties, and aerodynamic particle size (Sussell, Periakaruppan and Burr 2005; WorkSafe New Zealand, 2019). According to the Canadian Centre for Occupational Health and Safety (CCOHS) (2022) and WorkSafe New Zealand (2019), dust particles with a diameter of 0.5 to 5µm can deposit in the lower respiratory tract, and coarse particles with a diameter of 2.5 to 10µm can deposit in the nasal area by impaction. According to Alwis, Mandryk and Hocking (1999), dust particles with diameter greater than 10µm micrometers are trapped in the nasal cavity. Diffusion can cause the accumulation of tiny dust particles with a diameter below 0.01µm in the nasopharynx (WorkSafe New Zealand, 2019).

Wood dust particles deposited in the nasal cavity can be rapidly removed by sniffing, sneezing, or a mucociliary escalator, whereas particles deposited in the nasal region of the pharynx may be retained longer due to the absence of cilia in some areas of the nasal part of the pharynx (CCOHS, 2022; WorkSafe New Zealand, 2019). Other factors that can affect the deposition and absorption of wood dust in the nasal passages include smoking and lung disease (Lie, Skogstad, Johannessen, Tynes, Mehlum, Nordby, Engdahl and Tambs, 2016). Exposure to wood dust can impair ciliary clearance and cause mucosal stasis. Impaired mucociliary clearance can result in constant interaction with the airway epithelium. Impaired mucociliary clearance may allow entry of particulate antigens into lymphoid tissues associated with the nasopharynx, increasing atopy (CCOHS, 2022; WorkSafe New Zealand, 2019).

2.4. Health effects associated with wood dust exposure

Wood dust exposure can cause respiratory symptoms like wheezing, mucus secretion, mucosal irritation, shortness of breath, dyspnea, toxic alveolitis or pulmonary mycotoxins, and bronchial asthma (Tobin, Ediagbonya, Okojie and Asogun, 2016). Tobin *et al*. (2016) established that breathing in wood dust increases the risk of developing laryngeal and pharyngeal cancer, chronic obstructive pulmonary disease, and idiopathic pulmonary fibrosis. Chest tightness, skin irritability, nasal congestion, and hypersecretion are health problems linked to exposure to wood dust (Yamanaka, 2000). In addition, exposure to organic dusts may also increase the risk of organic dust toxic syndrome (ODTS), chronic bronchitis, occupational asthma, hypersensitivity pneumonitis, and mucosal irritation (Alwis, Mandryk and Hocking, 1999). It is thought that concentrations of tannins and other similar chemicals in wood are the main cause of these health effects (Sussell, Periakaruppan and Burr, 2005).

2.4.1. Wood dust exposure and associated risks of cancer

Men's chance of developing adenocarcinoma may be increased by exposure to wood, and that risk rises with exposure period (Hayes, Gerin, Raatgever and de Bruyn, 1986; NTP, 2016). According to Klintenberg, Olofsson, Hellquist and Sökjer (1984), adenocarcinoma is a rare form of nasal and sinus cancer that typically affects the ethmoid sinus. Nasal and sinus adenocarcinomas are primarily brought on by wood dust exposure (NTP, 2000; WHO, 1995a; WHO, 1995b; WHO, 1997). According to Luce, Gérin, Leclerc, Morcet, Brugère and Goldberg (1993) and Nylande and Dement (1993), exposure to formaldehyde and wood dust may increase the risk of nasal adenocarcinoma. Sinonasal adenocarcinoma has been linked to exposure to hardwood dust in previous research, but it was unclear whether exposure also affected the upper respiratory system (Demers, Kogevinas, Boffetta, Leclerc, Luce, Gérin, Battista, Belli, Bolm-Audorf, Brinton, Colin, Comba, Hardell, Hayes, Magnani, Merler, Morcet, Preston-Martin, Matos, Rodella, Vaughan, Zheng and Vainio, 1995a; Hernberg, Westerholm, Schultz-Larsen, Degerth, Kuosma, Englund, Engzell, Hansen and Mutanen, 1983; Vaughan and Davis, 1991).

Workers are more likely to develop paranasal sinuses and nasal cavities if they are exposed to formaldehyde, wood dust, varnishes, and adhesives (Olsen, Jensen, Hink, Faurbo, Breum and Jensen, 1984). Squamous cell carcinoma is the most typical kind of cancer (National cancer institute, 2019). In Alberta, squamous cell carcinoma and adenocarcinoma are the two most common causes of sinus cancer (Yamanaka, 2000). According to Luce, Leclerc, Bégin, Demers, Gérin, Orlowski, Kogevinas, Belli, Bugel, Bolm-Audorff, Brinton, Comba, Hardell, Hayes, Magnani, Merler, Preston-Martin, Vaughan, Zheng and Boffetta (2002), squamous cell carcinoma is more likely to develop in people who have been exposed to asbestos fibers. Moreover, exposure to sawdust and formaldehyde may raise the incidence of nasal cancer and nasopharyngeal cancer, specifically squamous cell carcinoma (Hildesheim, Dosemeci, Chan, Chen, Cheng, Hsu, Chen, Mittl, Sun, Levine, Chen, Brinton and Yang, 2001; Vaughan, Stewart, Teschke, Lynch, Swanson, Lyon and Berwick, 2000; Blair, Saracci, Stewart, Hayes and Shy, 1990). Squamous cell carcinoma is more common in women than in males. This has been observed in workers who process fresh wood for more than 30 years. The chances of adenocarcinoma, however, were observed to be higher in males than in women and to rise with prolonged exposure (Demers *et al*., 1995a).

Leclerc, Cortes, Gerin, Luce and Brugere (1994) found that 59 men had squamous cell carcinoma and 82 had adenocarcinoma in a study of woodworkers in France. Moreover, two of the 82 men who had adenocarcinoma had been exposed to hardwood and various types of wood dust. In addition to the nasal cavity, which is the target organ of the nasal airways, wood dust has been linked to an increased risk of cancer in other tissues (NTP, 2016). Despite the lack of evidence, squamous cell carcinoma of the nasal cavity (sinonasal cancers) includes laryngeal, nasopharyngeal, and Hodgkin's disease (NTP, 2016). According to Hansen and Larsen (1995) and Luce *et al*. (2002), formaldehyde exposure in the workplace increases the risk of sinonasal cancer. Although sinus cancers are a rare form of tumor, 440 cases are reported per year in the United Kingdom, making it one of the listed occupational diseases by the Industrial Injury Disability Benefits Scheme (Department of Work and Pensions, 2017).

In the early 2000s, the prevalence of nasal and sinonasal cancer rose by 0.8 cases per 100,000 people per year in Canada and 0.5 cases per 100,000 people per year in Alberta (Yamanaka, 2000). Sinonasal epithelial cancer is primarily caused by exposure to wood dust at levels below 1 and above 5 mg/m^3 (Worksafe New Zealand, 2019). Exposure to wood dust is a major cause of sinus tumors, and exposure to hardwood dust increases the risk of cytotoxic effects leading to inflammation (Wultsch, Nersesyan, Kundi, Wagner, Ferk Jakse and Knasmueller, 2015). Wood dust exposures between 0.5 and 1 mg/m^3 have not been associated with the development of sinonasal cancer, but exposures below 0.5 mg/m^3 have been linked to increased risks of bronchial asthma, bronchial hyperreactivity, and pulmonary dysfunction, while exposures above 0.5 mg/m^3 have been linked to pulmonary side effects (Kauppinen, Vincent, Lükkonen, Gusevik, Kauppinen, Welling, Arezes, Black, Bochmann, Campello, Costa, Ercigan, Gehrens, Kikemenis, Cromhout, Miguel, Mirabelli, McKennie, Pesch, Plato, Schrunsen, Schulze, Sonntag, Verogstrathe, Devicente, Wolf, Zimmermann, Husgafvel-Pursiaine and Savolainen, 2006; SCOEL, 2003).

According to Taskinen, Kyyrönen, Sallmén, Virtanen, Liukkonen, Huida, Lindbohm and Anttila (1999), exposure to formaldehyde increases the chance of spontaneous abortions and reduces fertility in women. Exposure to formaldehyde has been linked to rat nasal cancers, according to an experimental study (Hernberg *et al*.,1983). Exposure to formaldehyde, wood dust, and tobacco smoking increases the risk of developing nasopharyngeal cancer (Yu and Yuan, 2002). Furthermore, smoking cigarettes is the leading cause of respiratory health problems (Buliga, Rongo and Mamuya, 2017). Nasopharyngeal cancer, sinonasal cancer, and nasal adenocarcinoma risk increases following exposure to both hardwood and softwood dust (Saejiw, Chaiear and Sadhra, 2009). Nasal and sinonasal cancer are linked to exposure to hardwood dust (Kauppinen *et al*., 2006; Hernberg *et al*., 1983). Despite the lack of evidence from case control or cohort literature studies to support this claim (WorkSafe New Zealand, 2019), inhaling dust can increase the risk of nasal, paranasal, and nasopharyngeal cancers among exposed workers.

Exposure to wood dust increases the risk of developing multiple myeloma, sinonasal cancer, nasopharyngeal cancer, haematopoietic and lymphatic malignancies (Demers and Boffetta, 1998; Demers, Boffetta, Kogevinas, Blair, Miller, Robinson, Roscoe, Winter, Colin, Matos and Vainio, 1995b; Cherrie, Hutchings, Ng, Mistry, Corden, Lamb,

Jime'nez, Shafrir, Sobey, Tongeren and Rushton, 2017). The risk of pharyngeal and laryngeal cancers may also be increased by welding exposure (Bush, Portnoy, Saxon, Terr and Wood, 2006). Laryngeal cancer appeared to be mostly caused by cigarette smoking and alcohol drinking (Muscat and Wynder, 1992; Dietz, Ramroth, Urban, Ahrens and Becher, 2004; Wynder, Covey, Mabuchi and Mushinski, 1976). Exposure to asbestos is also connected to laryngeal cancer (Bush *et al*., 2006; Gustavsson, Jakobsson, Johansson, Lewin, Norell and Rutkvist, 1998).

The negative health effects of wood dust depend completely on the type of wood being processed (Department of Environmental Affairs and Development Planning, n.d.). Also, it depends on the physiochemical properties of the plant species, environmental factors, the length of exposures, the composition of the dust, and the distribution of the particle size (WorkSafe New Zealand, 2019). The chance of an inflammatory reaction in the nasal cavity and the impaired mucociliary clearance may escalate with prolonged exposure to wood dust (NTP, 2016). Paranasal sinus and nasal cavity cancer may be diagnosed by tests prepared to examine the sinuses and nasal cavity (National cancer institute, 2019). Surgery is frequently used to treat sinonasal cancer (Youldena, Cramba, Petersb, Porcedduc, Møllerd, Fritschib and Baadea, 2013).

2.4.2. Cancer and non-cancer health effects associated with wood dust exposure in the respiratory system

Exposure to wood dust may impair the respiratory system and increase the risk of cancer (Alwis, 1998). On the lower respiratory tract, issues can include EAA, idiopathic pulmonary fibrosis, idiopathic pulmonary cough, chronic bronchitis, and occupational asthma, while on the upper respiratory tract, issues can include impaired mucociliary clearance, increased nasal mucosal metaplasia, a rise in the frequency of nasal symptoms, ocular irritation, altered olfactory function, pharyngeal problems, and chronic bronchitis (WorkSafe New Zealand, 2019). Green or dry wood dust is the primary cause of obstructive and restrictive pulmonary effects, while exposure to green pine dust is associated with an increased risk of atopy.

2.4.3. Paranasal sinus and nasal cavity cancer

According to the Occupational Health Clinics for Ontario Workers (OHCOW) (2016) exposure to hardwood and softwood dust may increase the risk of sinonasal adenocarcinoma and non-malignant respiratory effects. Exposure to wood dust is linked to lung function abnormalities, anaphylaxis, paranasal sinus and nasal cavity cancers (Proto, Zimbalatti and Negri, 2010). The most common cause of nasal cavity and paranasal sinus cancer is wood dust (Kauppinen *et al*., 2006; Siemiatycki, Richardson, Straif, Latreille, Lakhani, Campbell, Rousseau and Boffetta, 2004; National Cancer Institute, 2008). According to Kauppinen *et al*. (2006), wood dust can cause cancer in humans. Similarly, paranasal sinus and nasal cavity cancers are caused by malignant cells that develop in the tissues of the nasal cavity and paranasal sinuses (National Cancer Institute, 2019).

Exposure to wood dust and smoking tobacco reduce the lung's capacity for clearing dust and increase the risk of respiratory symptoms (Cancer Council Australia, 2019). In comparison to a 40-year-old male who does not smoke but is exposed to wood dust, the former has a higher risk of developing paranasal sinus and nasal cavity cancer (National cancer Institute, 2019). Nasal cavity and paranasal sinus cancer symptoms include sinus issues, nosebleeds, sinus pressure, headaches, runny nose, unhealed sore within the nose, lumps on the face, numbness, swelling, eyes issue, pains in the upper teeth, and pressure or discomfort in the ear (Nasal cancer institute, 2019).

2.4.4. Nasal cancer

Exposure to hardwood dust such as oak and beech wood causes nasal cancer (CCOHS, 2017; Yamanaka, 2000). According to a case-referent study conducted on sawmill workers, breathing in wood dust that is mixed with both hardwood and softwood increases the risk of nasal cancer, specifically squamous cell cancer (Yamanaka, 2000). Despite the absence of case-referent studies to measure this association, softwood is also associated with an increased risk of squamous cell cancer (Yamanaka, 2000). Nasal cancer might not be primarily caused by softwood dust, such as those from pine and spruce (Hernberg *et al*., 1983). Exposure to formaldehyde is linked to nasal cancer (Hayes *et al*., 1986). According to Hernberg *et al.* (1983), hardwood dust is associated with nasal adenocarcinoma, a rare malignancy

that affects around 1 in 1,000 persons annually. Adenocarcinoma is a malignancy of the glandular or secretory tissues of the nasal canals. Dr. R.G. Macbeth first identified it among woodworkers in the little town of High Wycombe in 1963. It is the most significant cell type of nasal cancer (Yamanaka, 2000). Other histological varieties of nasal cancer include squamous cells, sarcoma, anaplastic and transitional cell carcinomas (Harbo, Grau, Bundgaard, Overgaard, Elbrønd, Søgaard and Overgaard, 1997).

Nasal cancer is the leading cause of nasal obstruction, epistaxis (nose bleeds), foul odor, and facial pain (Yamanaka, 2000). Additionally, it may be diagnosed by using specialized radiographic tools such as computerized tomography and magnetic resonance imaging scans, which provide an image of the extent of the cancer association. The diagnosis is confirmed using biopsies from cancer sites, and pathological specimens of squamous cell carcinoma, adenocarcinoma, and other cancers can be determined (National Cancer Institute, 2019; Yamanaka, 2000). Treatment for nasal cancer depends on the type, location, and extent of lymph nodes or glands affected after diagnosis. Surgery, radiation dosimetry, and radiotherapies like intensity-modulated radiation therapy (IMRT) and proton therapy are the most common treatments, even though the prognosis of the diseases varies subject to the extent of cancer involvement as it is in a progressive phase at the time of first detection (National Cancer Institute, 2019; Harbo *et al*., 1997). In addition, a combination of cytotoxic chemotherapeutics and targeted biological agents is included in the management of sinonasal cancer patients. Cancer growth is slow and asymptomatic, even after diagnosis and treatment, the survival rate is 30% in five years (Yamanaka, 2000). Office endoscopy and high-resolution imaging are used to diagnose it.

2.5. Carcinogenic effects of wood dust exposure

Wood dust has been classified by the IARC as a human carcinogen based on epidemiological data (Kauppinen *et al.*, 2006; IARC, 1995a; Universal Forestry Product, 2010). Wood dust is a well-known human carcinogen. (NTP, 2016). It was identified by the ACGIH as a confirmed human carcinogen (Universal Forestry Product, 2010; Spee, van de Rijdt-Van Hoof, van Hoof, Noy and Kromhout, 2007). It was also identified by the NIOSH as a potential occupational carcinogen (NTP, 2016). Species of both hardwood and softwood, including *Thuja plicata*, are regarded as sensitisers (WorkSafe New Zealand, 2019). Hardwood dust may be classified as a sensitiser and a confirmed or suspected human carcinogen depending on the type of timber being treated. Hard wood species including birch, teak, mahogany, and walnut have been classified by the ACGIH as suspected human carcinogens (Universal Forestry Product, 2010). Because there is an insufficient information to differentiate between wood dust species based on their potential to cause cancer, the IARC, DECOS, NTP, and SCOEL have come to a conclusion that all species of wood must be considered carcinogenic (WorkSafe New Zealand, 2019).

2.5.1. Carcinogenic effects of wood dust exposure according to the International Agency for Research on Cancer (IARC)

There are abundant facts that wood dust increases the risk of developing nasal, paranasal, and nasopharyngeal cancer (IARC,1995a; Kauppinen *et al*., 2006). Wood dust is linked with an increased risk of sinonasal cancer (IARC, 1995a; WorkSafe New Zealand, 2019). Wood dust is the leading cause of nasal cavity, paranasal sinus, and nasopharynx cancer, mainly squamous cell carcinomas (IARC, 1995a). Tobacco smoking may escalate the risk of sinonasal and nasopharyngeal cancer (IARC, 1995a). Notwithstanding the lack of evidence on cellular assays, tissue injuries, impaired ciliary clearance, and direct and indirect genotoxicity are the primary mechanisms of carcinogenesis of wood dust (IARC, 1995a).

According to Cancer Council Australia (2019), the consequences of wood dust and formaldehyde exposure might be short-term or long-term. Short-term health outcomes include respiratory irritations, congestion, and eye and skin irritations, whereas long-term effects comprise asthma, lung fibrosis, and nasal, sinus, and nasopharyngeal cancers. Formaldehyde is a cancer-causing agent in humans and the leading cause of leukemia, nausea, dermatitis, and nasopharyngeal cancer (Cancer Council Australia, 2019; Tang, Bai, Duong, Smith, Li and Zhang, 2009). Additives and wood preservatives have been linked to sinonasal cancer despite the lack of supporting evidence. The IARC established that there are some inadequate facts on the causal relationship between wood dust exposure and cancer of the nasopharynx, oropharynx, hypopharynx, lungs, etc. (IARC, 1995b).

2.5.2. Carcinogenic effect of wood dust exposure according to the Scientific Committee on Occupational Exposure Limits (SCOEL)

Inhaling wood dust at concentrations greater than 1 mg/m^3 increases the risk of upper respiratory symptoms (WorkSafe New Zealand, 2019). These symptoms include nosebleeds, rhinosinusitis, long-lasting colds, nasal congestion, nasal mucus stasis, cytological modifications of mucus, and histological modifications of the nasal epithelium. Moreover, the non-carcinogenic effects of wood dust exposure on the lower respiratory tract include asthma, cough, chronic obstructive pulmonary disease, alteration of respiratory function parameters, cryptogenic fibrosing alveolitis, and EAA. Wood dust exposure is linked with an increased risk of sinonasal cancer, notwithstanding the rarity of this disease. Individuals exposed to wood dust are at greater risk of respiratory function impairment and pulmonary symptoms, even though a sawmill worker who processed *Pinus radiata* developed atopy and lung function abnormalities due to excessive dust exposure (WorkSafe New Zealand, 2019). According to SCOEL (2003), wood dust above 0.5 mg/m^3 causes lung problems, but exposure below 0.5 mg/m^3 elevates bronchial asthma, for example in *Thuja plicata*. The levels between 0.5 and 1 mg/m^3 are lower than the levels at which sinonasal cancer was discovered (SCOEL, 2003).

2.5.3. Carcinogenic effects of wood dust exposure according to the Dutch Expert Committee on Occupational Standards (DECOS)

Studies conducted on animals and animal cells exposed to beech, oak, and organic solvent extract detected deoxyribonucleic acid (DNA) damage, micronucleus formation, and chromatid abruptions or chromosomal aberration (NTP, 2016). Additionally, higher rates of DNA single strand breaks, DNA healing, and small nucleus creation were detected in the peripheral blood leukocytes of employees exposed to wood dust. Hardwood extracts and condensates are genotoxic *in vitro* and may cause gene mutations and chromosomal aberrations; correspondingly, softwood extracts and condensates are also genotoxic *in vitro* and can cause gene mutations, chromosomal aberrations, and chromatid exchange (WorkSafe New Zealand, 2019).

2.5.4. Carcinogenic effects of wood dust exposure according to the American Conference for Governmental Industrial Hygienists (ACGIH)

The ACGIH has classified wood dust as a confirmed human carcinogen. Inhalation of wood dust increases the risk of developing asthma, EAA, wheezing, catarrh, chronic obstructive pulmonary disease, flu-like symptoms, an itchy throat, and eye irritation, while exposure to endotoxin and glucan increases the risk of developing lung function impairment and work-related symptoms (WorkSafe New Zealand, 2019; Douwes, McLean, Slater and Pearce, 2001; Douwes, Thorne, Pearce and Heederik, 2003). Moreover, cabinet makers have a greater incidence of sneezing, persistent cough, breathlessness, nasal discharge, or obstruction. Exposure to Rimu dust (Dacrydium cupressinum) is the major cause of upper respiratory effects and eye symptoms. Additionally, wood dust exposure is the leading cause of irritation and allergic contact dermatitis related to Type I and Type IV hypersensitivity (WorkSafe New Zealand, 2019). Similarly, respiratory health effects and mucosal symptoms such as rhinitis and asthma are caused by exposure to pine, beech, walnut, maple, and fir, while conjunctivitis is induced by western red cedar (*Thuja plicata*). Mandryk, Alwis, and Hocking (1999) discovered that sawmill workers had a significantly higher incidence of consistent wheezing, catarrh, and chronic obstructive pulmonary disease than controls.

2.6. Previous research studies on wood dust exposure and risk of cancer

Kawachi, Pearce and Fraser (1989) conducted research among 19904 male patients above the age of 20 who were registered for cancer in New Zealand from 1980 to 1984 to ascertain the relation between sawdust and cancer risk. Sawmill workers had the greatest risks of liver and lung cancers, with odds ratios of 1.76 and 3.55, while carpenters had the highest risk of lip and lung cancers, with odds ratios of 2.28 and 1.28, respectively. Furthermore, foresters and loggers reported the highest prevalence of nasopharyngeal carcinoma with an odds ratio of 6.02. Demers *et al*. (1995a) conducted a case-control study among 680 males and 2349 controls, as well as 250 females and 787 controls, to assess the relationship between sawdust and sinonasal cancer. Their findings revealed that sawdust exposure escalates the risk of adenocarcinomas in men and women, with the risk being higher in men and increasing with length of exposure. The prevalence of squamous cell carcinoma seemed to be higher in women who worked in workplaces with higher exposure to wood dust. An increased chance of squamous cell carcinoma was seen in males exposed to excessive fresh wood dust over 30 years. Inhalation of sawdust increased the risk of sinonasal adenocarcinoma, although the evidence for squamous cell carcinomas was inconclusive (Demers *et al*., 1995a).

Kauppinen, Partanen, Nurminen, Nickels, Hernberg, Hakulinen, Pukkala and Savonen (1986) undertook a cohort study among 3805 men who were employed in the particle board, plywood, sawmill, and formaldehyde glue industries for more than a year between 1944 and 1981 to determine if exposure to terpenes and other wood products increases the risk of cancer. Exposure to terpenes and other coniferous wood-related products over five years was linked with an increased risk of respiratory cancer. Siew, Kauppinen, Kyyrönen, Heikkilä and Pukkala (2012) conducted a cohort study among men employed in a Finnish wood processing factory from 1970 to 1994 to determine if sawdust exposure increased the risk of nasal cancer. It was concluded that wood dust increased the risk of nasal cancer. To determine if wood dust exposure increases the risks of oral and pharyngeal cancer, Smailyte (2012) performed a study in Lithuania among 1080 men and 438 women employed in the wood processing factories for more than a year between 1947 and 1996. The study discovered a significant correlation between softwood dust and the prevalence of oral and pharyngeal cancer.

Eriksson, Stjernberg, Levin, Hammarstrom and Ledin (1996) undertook a study among 48 sawmill workers to determine if exposure to terpenes increased the risk of lung function and respiratory symptoms. Employees unprotected from sawdust longer than 1825 days were at higher risk of developing methacholine reactivity and increased risk of eye irritations, while those exposed for less than 5 years were at greater risk of developing bronchial reactivity. Siew, Martinsen, Kjaerheim, Sparén, Tryggvadottir, Weiderpass and Pukkala (2017) carried out a case-control study in Finland, Sweden, Norway and Iceland comprising 393 cases of nasal adenocarcinoma, 1747 cases of nasopharyngeal cancer, and 2446 other nasal cancers diagnosed among males to establish whether wood dust exposure increases the risk of nasal adenocarcinomas. Continuous inhalation of softwood or mixed wood was related to an increased risk of nasal cancer. Ratnasingam, Ioras, Tadin, Wai and Ramasamy (2014) conducted a study among 2324 employees to find out if sawdust and wood coating increase the risk of developing chronic respiratory disease. Exposure to wood dust was linked with respiratory symptoms or eye irritation (red eyes) rather than chronic respiratory diseases, while exposure to wood coating was linked with chronic respiratory diseases.

Cherrie *et al*. (2017) conducted a study on 25 preselected mixtures of hazardous substances to determine if implementing five (5), three (3), and one (1) mg/m^3 of OEL that is enforced by some countries will reduce the incidence of cancer in current or future cases. The implementation of 5 mg/m^3 OEL was predicted to increase the number of cancer cases by 14,000 from 2010 to 2069, resulting in 200 fatalities over 59 years of exposure. The implementation of 3 mg/m^3 OEL was predicted to prevent 500 cancer cases, which could result in 6,300 deaths over 59 years of exposure. Likewise, implementing the 1 mg/m^3 OEL was estimated to reduce the number of cancer cases by 3,900. Approximately 8% of employees are currently unprotected from inhalable wood dust beyond 1 mg/m^3 OEL, while 1% are exposed to inhalable wood dust beyond the 5 mg/m^3 limit set by the European Union.

Vaughan *et al*. (2000) performed a case-referent study between 1987 and 1993 among 196 newly diagnosed nasopharyngeal cancer patients and 244 controls to investigate whether exposure to sawdust and formalin escalates the chance of nasopharyngeal carcinoma (NPC). Besides sawdust, occupational exposure to formaldehyde was linked with an increased risk of NPC. The relationship between formaldehyde and an increased risk of NPC was limited to squamous cell carcinomas and was stronger among cigarette smokers than non-smokers. Exposure to formaldehyde over 18 years was linked with an increased risk of NPC. The Department of Work and Pensions (2017) conducted a study to determine if being unprotected from sawdust escalates the risk of sinonasal cancer. Relevant reports from IARC (2012a), Young *et al*. (2012), and a peer-reviewed article on the occupational cause of sinonasal cancer by Alonso-Sardón *et al*. (2015) and an exploratory study were identified. Wood dust exposure was linked to sinonasal tumors or cancer of the nasal passage and associated airway sinusitis or nasal carcinoma.

Hertzman, Teschke, Ostry, Hershler, Dimich-Ward, Kelly, Spinelli, Gallagher, McBride and Marion (1997) conducted a historic cohort study among employees exposed to chlorophenate for more than a year at a sawmill in British Columbia to determine if exposure to chlorophenates increases the prevalence of Hodgkin disease. An increase in the prevalence of Hodgkin disease was linked to chlorophenates. Five studies found that there was no link between chlorophenates and liposarcoma in the woodworking population. Other studies indicated that non-Hodgkin's lymphoma was associated with metal-working occupations and exposure to solvents rather than benzene and formaldehyde. The study found that there were about 37 deaths caused by non-Hodgkin's lymphoma in a 40-year mortality follow-up and 65 cases of cancer prevalence over a 20-year follow-up. Youldena *et al*. (2013) carried out a study by outsourcing data from various countries and other accessible sources to determine if being unprotected from sawdust increases the chance of sinonasal cancer. It was reported that smoking cigarettes or tobacco was the main risk factor for sinonasal cancer, mainly squamous cell carcinoma. The mean 12-month incidence of sinonasal cancer was 5 to 10 cases per million in men and 2 to 5 cases per million in women. The European Union Council set 5 mg/m^3 OEL for inhalable wood dust in 1999 and 1 to 1.5 mg/m^3 in 2005 to reduce the incidence of cancer.

In 1997, the USA set TLVs of 5 and 1 mg/m^3 for softwood dust and hardwood dust, respectively. In 2005, these limits were reduced to 1 mg/m^3 for all sawdust. The same OEL is widely used in other parts of the world. The incidence rate of cancer among males exposed to inhalable dust above 1 mg/m^3 was 1.7% in Denmark and Ireland, 1.3% in the Netherlands, 1.2% in Italy and Finland, and 0.8% in Sweden. Denmark and Ireland had the highest incidence of sinonasal cancer, while Sweden reported the lowest incidence. These standards are implemented in Europe and other parts of the USA. The criteria have been introduced in less developed countries to prevent sinonasal cancer from becoming a burden (Youldena *et al*., 2013). The Health Council Committee of the Netherlands (HCCN) (2000) reviewed studies to ascertain whether sawdust was a genotoxic cancer-causing agent. The committee reported that hardwood dust was a cancer-causing agent with weak genotoxicity assets and cytotoxicity ability *in vivo*, as well as softwood dust. Hardwood dust can induce sinonasal adenocarcinoma in humans. A meta-analysis revealed that unprotected exposure to hardwood and softwood dust can increase the risks of nasopharyngeal and epidermoid carcinomas of the nose, even though there was uncertainty because of mixed exposure (HCCN, 2000). DECOS considers softwood dust as a suspected human carcinogen even though experimental studies performed on animals to detect carcinogenicity through inhalation did not show any evidence of carcinogenic properties (HCCN, 2000).

The Minister of Social Affairs in the Netherlands accepted linear extrapolation recommendations in similar elusive situations even though their application would result in a 5.8 mg/m^3 inhalable dust reference lifetime risk for nasal cancer, representing 1 in 250 adenocarcinomas, and 0.06 mg/m^3 for 1 in 25 000, both for occupational exposure over the course of 40 years. There is data showing that extracts and condensates of hardwood cause gene mutation and chromosomal aberration in vitro and are considered genotoxic *in vitro*. The genotoxic *in vitro* effects of deoxyribonucleic acid (DNA) and the genotoxic *in vivo* effects of softwood extracts have not been investigated extensively (HCCN, 2000). DNA single strands broke in rats' hepatocytes in vitro as well as the micronuclei in rodents' tissues *in vivo* (SCOEL, 2003). The link between hardwood dust and varieties of cancer in the sinonasal cavity and other places in the body is subject to argument, as well as the relationship between softwood and cancer, which remains obscure (HCCN, 2000).

Mandryk, Alwis and Hocking (2000) conducted a study among 100 sawmill workers, 34 from dry mills, 53 from green mills, and 34 from a control group, to determine if exposure to sawdust increases the incidence of lung functions. Sawdust exposure was linked with respiratory ailments and pulmonary function among sawmill employees. Employees at the dry mill had a higher prevalence of stuffy nose, cough, sternutation, sinus problems, influenza-like illness, eye and throat irritation, and chronic bronchitis compared to green mill workers. Sardón, Chamorro, García, Sena, Rodero, Herrera, Marcos and Canelo (2015) carried out a systemic review of 114 studies. A meta-analysis study revealed that sawdust was linked to an increased risk of nasal adenocarcinoma even though there was a great degree of heterogeneity. Jagtap and Deshmukh (2018) carried out a cross-sectional study among 180 sawmill employees and 180 controls to establish if there was a relationship between wood dust and other diseases. The study found that morbidity among sawmill workers was higher in respiratory cutters (84.21%), supervisors (87.50%), others (77.22%), and musculoskeletal cutters (77.19%).

Kauppinen *et al*. (1986) performed a study on 32 million workers in 15 nation states of the European Union (EU) exposed to the IARC agents covered by carcinogen exposure (CAREX) between 1990 and 1993 to determine the number of employees exposed as well as the sources. The familiar sources of carcinogens were solar radiation (9.1 million unprotected workers, smoking tobacco (7.5 million), crystalline silica (3.2 million), diesel exhaust (3.1 million), radon (2.7 million), wood dust (2.6 million), lead and inorganic lead compounds (1.5 million) and benzene (1.4 million). Austria (800 000), Belgium (700 000), Denmark (700 000), Finland (500 000), France (4.9 million), Germany (8.2 million), Great Britain (5.0 million), Greece (900 000), Ireland (300 000), Italy (4.2 million), Luxembourg (50 000), the Netherlands (1.1 million), Portugal (1.0 million), Spain (3.1 million), and Sweden (800 000) were announced to be the top countries with the highest number of unprotected workers. The overall number of employees exposed to chemical agents was 42 million, which is an average of 1.3 employees per exposed group.

Peters, Ge, Hall, Davies and Demers (2015) carried out a study utilizing CARcinogen Exposure (CAREX) Canada, which yields approximations of the incidence of occupational exposure to 44 cancer-causing agents without categorization by industry, occupation, region, or gender to determine the total number of employees unprotected from deadly chemicals. Approximately 338, 000 Canadian employees were exposed to carcinogens. CAREX Canada estimated that 1.9 million night workers, 1.5 million solar ultraviolet radiation workers, and 781 000 diesel engine exhaust workers were unprotected from 44 well-known, likely, and alleged deadly chemicals. From 1996 to 2001, Vallières, Pintos, Parent, and Siemiatycki (2015) conducted a study comprising 533 communities and 1349 control cases in Study 1 and 894 communities and 736 control cases in Study 2 to determine the link between inhaling sawdust and the prevalence of lung cancer. Escalation of lung cancer was seen in studies 1 and 2 due to excessive wood dust exposure.

't Mannetje, Kogevinas, Luce, Demers, Bégin, Bolm-Audorff, Comba, Gérin, Hardell, Hayes, Leclerc, Magnani, Merler, DipStat and Boffetta (1999) conducted a study in which they pooled data for prevalent cases (104 females and 451 men) and control cases (241 females and 1,464 men) to ascertain whether wood and leather dust were linked with sinonasal cancer. Unprotected from wood and leather dust were linked with adenocarcinomas rather than squamous cell carcinoma. Thirty-nine percent (39%) of all sinonasal cancers occurred in males compared to 11% in females. Hubbard, Lewis, Richards, Johnston and Britton (1996) carried out a case-control study among 218 patients and 569 controls who had cryptogenic fibrosing alveolitis (CFA) to evaluate if wood dust or metal exposure is the leading cause of this disorder. Metal or wood dust exposure was associated with cryptogenic fibrosing alveolitis.

2.7. Types of monitoring for wood dust

There are three types of monitoring utilized for wood dust: personal monitoring, breathing zone sampling and area monitoring, or general air sampling (Davies and Henderson, 2009a; Health and Safety Executive (HSE), 2000; HSE 2006; Harmse, 2000; Leidel, Busch and Lynch, 1997). During personal sampling, the sampling device is attached to the employee and worn continuously during working hours to measure the employee's personal exposure to hazardous substances. The breathing zone method entails another person measuring air in the employee's breathing zone while holding the sampling device. For general air sampling, the sampler is placed at a fixed location inside the working area (Leidel, Busch and Lynch, 1997). Fixed-place monitoring, on the other hand, measures the amount of hazardous substances at or near the source of emission (HSE, 2006). If the person performing the task does not move from a specific position, static monitoring at breathing or head height may be useful (Jankewicz, Lee, Pisaniello and Tkaczuk, 2008). The results of area monitoring may not accurately reflect the employees' exposure to potentially hazardous substances. Consequently, the results may not be compared with the occupational exposure limit (HSE, 2000; HSE, 2020; HSE, 2015).

Fixed-place or static monitoring is used to check the effectiveness of controls, identify emission sources, or determine the background contaminant concentration of a hazardous chemical substance (HSE, 2006). Samples taken for determining employee exposure are taken only by the personal or breathing zone method (Leidel, Busch and Lynch, 1997). The Australian Occupational Exposure Limit mandates personal monitoring for wood dust and formaldehyde (Jankewicz *et al*., 2008). Most workplace exposure limits (WELs) require monitoring of personal exposures to ensure compliance with WELs. The objective of personal monitoring is to determine the concentration of airborne substances inside the worker's breathing zone. The breathing zone is the distance between 20 and 30 cm from the employee's nose and mouth (HSE, 2000; HSE, 2006). The monitor is clipped on the collar within the employee's breathing zone, which is around 300 mm in radius from the midpoint between the mouth and nose. Since the monitor travels with the worker everywhere during his or her work shift, it can accurately document the daily exposure (Jankewicz *et al*., 2008).

There are two types of monitoring strategies used for monitoring personal exposure to wood dust, namely, representative and worst-case sampling (HSE, 2006). Representative sampling considers possible workplace influences, whereas worst-case sampling can be taken where a high-risk employee can be clearly identified (HSE, 2006). Employees assumed to be highly exposed to substances at or above the OEL can be selected for monitoring. However, for each work activity with a unique method, the maximum risk employee must be selected in each work operation. If the maximum-risk employee cannot be selected in the work operation, then employees who perform similar tasks are grouped for a random sampling of a group with high risk (HSE, 2006). Leidel, Busch and Lynch (1977) found that five samples would be enough to ensure, with a confidence level of 90%, that one sampling period would occur during 33% of the time on most occasions with maximum exposure level. If there are less than ten employees in the SEG, only five should be included in the monitoring exercise for representative measures. Conversely, if there are more than ten employees in the SEG, the Occupational Exposure Sampling Strategies Manual (OESSM) or BS EN 689 must be used (HSE, 2006). After determining the number of employees to sample, a random number table may be used to select employees and measure their exposure (Leidel, Busch and Lynch, 1997).

The duration of the monitoring must be sufficient for the results to be representative of the exposure period including peak exposures so that the TWA exposure may be calculated (HSE, 2006). The airflows and activities may differ, and positioning the sampler on the right lapel, left lapel, or helmet may cause an over or underestimation of the normal concentration of the inhaled substance(s) (HSE, 2006). Before placing sampler during monitoring exercise, consideration should be given to the processes, tasks and work practices so that the results can be interpreted correctly (HSE, 2006). Once the personal exposure levels within an area have been measured and compared with the OEL values, each area must be categorised in classification bands to determine various homogeneous groups within the area.

2.7.1 Sampling equipment used for monitoring wood dust

The operation of a sawmill generates wood dust particles of different sizes, compositions and concentrations (Alwis, 1998). The size distribution of sawdust comprises of dust particles with diameter larger than 10 µm aerodynamic diameter that could stuck in the nasal cavity making inhalable mass sampling the best suitable method for monitoring wood dust (Alwis, 1989). Inhalable particulate matter (IPM) sampling is the most preferred monitoring technique to estimate risk of nasal tumor (Alwis, 1998; Brown *et al.*, 2013; Comite' Europe'e' de Normalisation (CEN),1993). IPM sampling is used for personal monitoring because size distribution and concentration of wood dust differs by position owing to presence of local sources such as distance from source, employee mobility, ventilation air movement patterns and individual habits (Alwis, 1998; Harmse, 2000; Leidel, Busch and Lynch, 1977). Most studies have employed total dust samplers which are not accurate for monitoring inhalable wood dust (Alwis, 1998). Alwis (1998) recommended the use of respirable dust sampling when exposure is asthma from sawmills processing western red cedar.

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The 37 mm plastic close-face cassette (CFC) used by NIOSH lacks the required sampling efficiency to collect larger dust particles to match the ISO inhalable fraction and underestimates the inhalable fraction above 20 to 25µm (Kauffer, Wrobel, Görner, Rott, Grzebyk, Simon and Witschger, 2010). In a laboratory context, the 37 mm open-face cassette (OFC) used in Sweden, Spain and Finland collects a higher concentration of total dust and undersampled particles. The OFC and CFC samplers do not comply with the CEN/ACGIH/ISO inhalable curve (ISO, 1995; Kenny *et al*., 1997). The Institute of Occupational Medicine (IOM) sampling displays a good performance orientation at wind speed of 0.5 to 1m/s (Kenny, Aitken, Baldwin, Beaumont and Maynard, 1999; Lee, Harper, Slaven, Lee, Rando and Maples, 2011). Nevertheless, the sampler is susceptible to major bias at constant directional airflow and is prone to projectile entry in some environments (Jime'nez, van Tongeren and Cherrie, 2011). The huge inlet entry of the IOM sampler increases its potential to aspire particles above 100 µm that are present within the worker's breathing zone (0.3 meters, or 11.8 inches' radius of the worker's nose and mouth), although this is not covered by the inhalable fractions. The inhalation efficiency of the human nose and mouth decreases as particle size increases (Lee *et al*., 2011). The sampling efficiency of IOM and conical inhalable samplers decreases with increasing wind speed. The IOM and CIS samplers meet inhalable criteria at 1m/s average wind speed (Lee *et al*., 2011).

The seven-hole sampler undersamples and shows poor precision at wind speeds of 0.5 and 1 m/s in a laboratory setting (Jime'nez, van Tongeren and Cherrie, 2011). The ACCU-CAP™ can prevent the loss of the interior wall of the cassettes when included in the CFC (Puskar, Fergon, Harkins, Hecker, 1992). The CFC must be located on the wearer body with the opening facing at an angle of 45° (Buchan, Soderholm and Tillery, 1986; Lee *et al*., 2011). The CFC must be positioned in a direction facing outward from the body (Kauffer *et al*., 2010). The button sampler underestimates particles above 25µm due to the spherical glass test causing particles to be trapped on the porous screen (Getschman, 2013). The inlet screen minimizes the collection of particles above 100µm. The sampler is relatively insensitive to wind speed, direction and has shown minimal internal wall loss in laboratory studies (Aizenberg, Grinshpun, Willeke, Smith and Baron, 2000; Lie, Skogstad, Johannessen, Tynes, Mehlum, Nordby, Engdahl and Tambs, 2000). Samplers do not provide matching results as discussed below.

2.7.2 Conversion factor of total dust results to inhalable dust

There are no well-established procedures existing to change total dust results to inhalable dust (SCOEL, 2003). The commonly used correction factor is 2.5 (ACGIH, 2005). The correction factor was developed from CFC and IOM samplers set next to each other in various aerosols of different sizes (Lee *et al*., 2011). The ratio in the woodworking industry was 3.5 due to the effect of the positioning of the close-face cassette sampling (Lee *et al*., 2011). The use of a 2.5 performance ratio for all dust must be done with caution due to the wide variety of dust samplers and types of dust (Getschman, 2013). The variance depends on mass concentration and particle size, even though the absolute value of the OEL for inhalable dust might be set at or double the absolute value of what would have been the equivalent limit value for total dust (ACGIH, 2012). A conversion factor of 2 and 3 obtained by an open-face filter holder for total dust can be compared to the limit value (SCOEL, 2003). The Danish organic dust results can be multiplied by 1.18 to convert to total dust and multiplied by 1.59 to convert to inhalable dust (Kauppinen *et al*., 2006; SCOEL, 2003). In addition, French results can be multiplied by 1.59 to convert to Danish total dust, while Finnish total dust results can be multiplied by 2 to convert to inhalable dust. The Dutch measurement methods must be compared to the German and British methods since they directly measure the concentration of inhalable dust (Kauppinen *et al*., 2006).

2.7.3 Limitations of the current NIOSH strategy

The OESSM outlines a strategy for assessing compliance for a single worker (a maximum risk employee) on a single day using one or two measurements for compliance comparison with OEL. This strategy does not require knowledge of the exposure variability or the statistical calculations needed to estimate it, only the sampling and analytical variability linked to each measurement are accounted for. Furthermore, the strategy cannot detect poorly controlled exposures, even though a 50% power calculation to detect an unacceptable exposure profile for a 25% exceedance fraction can be undertaken. However, the NIOSH strategy is efficient in requesting a few measurements to be undertaken, but it cannot identify work-case scenarios that are non-compliant, even though the compliance status depends on the number of measurements taken. That having been said, the strategy encourages an increase in the number of measurements (Ramachandran, 2008).

The NIOSH sampling strategy has limits, as it cannot be used when a substance does not have OELs or dermal risks and its data cannot be used for risk management or epidemiological studies (Ramachandran, 2008). Moreover, when sampling SEG or maximum exposure risk workers, the strategy is likely to overestimate exposures and underestimate variability, resulting in a lack of epidemiological data representation. A study conducted by Hall, Teschke, Davies, Demers and Marion (2002) at a sawmill in British Columbia ascertained that compliance samples did not overestimate the mean exposure levels at the job level. A comprehensive exposure assessment strategy that assesses the health risks of all substances among workers every day instead of selecting the maximum-risk employee on a single day for a particular substance with OEL can be useful for baseline sampling and decide to initiate or discontinue specific exposure control as well as for epidemiological studies (Ramachandran, 2008).

Ramachandran (2008) proposed an observational approach for assigning workers to a group with similar exposure to hazardous substances, tasks, or work environments and obtaining measurement data from such similarly exposed groups (SEGs). Rappaport, Kromhout and Symanski (1993) proposed a classification scheme in which the entire work population selected randomly and divided into similar exposed groups, or sampling similar job designations and divide them into groups, followed by conducting multiple samples for each sampled worker in the SEG and using mixed models for statistical estimating of the between- and within-worker constituents of variance. However, this scheme can be impractical due to resource constraints because medium-sized processing buildings with 100 activities and 15 to 20 substances per activity will require more than 1500 SEG chemical task mixtures with multiple samples from several workers in each SEG, and this can be impossible to achieve (Rappaport, Lyles and Kupper, 1995). The observational approach proposed by AIHA requires the exposure profile for SEG to be classified as very low exposure (zero), highly controlled exposure (1), well-controlled exposure (two), controlled exposure (three), and poorly controlled exposure (four). Moreover, these classifications are based on a 95% exposure distribution for SEG less than 1% of OEL at 1 to 10, 10 to 50, 50 to 100, or above 100% (Hewett, Logan, Mulhausen, Ramachandran and Banerjee, 2006). The approach is used to classify workers even though potential misclassification exists, although the risks for some SEGs are negligible with significance for others.

Hewett (2006, as cited in Ramachandran, 2008) contends that serious SEGs can be monitored when assessing within and between worker variability, while SEGs with exposure profiles above the OEL will require control measures to reduce the exposures and samples are not required, the same is true for SEGs with exposures below 10 % of the OEL. However, when all the SEGs exposure profiles are above 10% of the OEL, about 6 to 10 samples per SEG are required to be taken to detect exposure profiles above the OEL, with a 25% exceedance fraction of above 90% for 6 samples and increasing to above 99% for 10 samples (AIHA, 2006; Navy and Marine Corps Public Health Centre, 2021; Ramachandran, 2008). In SEGs with exposure profiles between 50 and 100% of OEL, multiple samples per worker in the SEG may be required to characterize the components of variance, and this can be achieved by using the accurate expert judgment of an occupational hygienist that is documented to classify workers in the SEGs and obtain sufficient samples if needed (Ramachandran, 2008). Hygienists must not underestimate exposures that will lead to inadequate protection for some workers (Hagström, Lundholm, Eriksson and Liljelind, 2008b).

2.7.4. Guidelines for wood dust exposure

Exposure to wood dust can cause asthma, lung function impairment, upper respiratory problems and eye, skin and throat irritations (Hirst, 2009; Hirst, 2010; Occupational Health Clinic of Ontario Worker (OHCOW), 2016). Wood dust is classified as hard and softwood (Alwis, 1998; IARC, 1995b; WorkSafe New Zealand, 2019). Most studies and nations, are using OEL for wood dust, total dust, hardwood and softwood of 5mg/m^3 measured as either total or inhalable dust while others use 2 mg/m^3 apart from Belgium which use 3 mg/m^3 OEL for hardwood and softwood dust (Alwis, 1998; Scheeper, Kromhout and Boleij, 1995; Straumfors, Olsen, Daae, Afanou, McLean, Corbin, Mannetje, Ulvestad, Bakke, Johnsen, Douwes and Eduard, 2018). The ACGIH have set OEL for soft wood at 5mg/m^3 (Diwe, Duru, Iwu, Merenu, Uwakwe, Oluoha, Ogunniyan, Madubueze and Ohale, 2016). Thailand and OSHA PEL have 5mg/m^3 OELs for reparable wood dust (Occupational Health Clinic of Ontario Worker, 2016; Siripanish, 2013). South Africa, Britain, Germany, Irelan, Italy, Spain, Turkey, UK and European Union (EU) have 5mg/m^3 OEL for hardwood dust and South Africa anticipate to revise this limit to 2 mg/m^3 with 5 mg/m^3 OEL for softwood dust (Chamber and Nunes, 2016; Simpson, Niven, Pickering, Fletcher, Oldham and Francis,1999).

When OEL for a specific chemical is not available, exposure to total inhalable dust must be kept below 10 mg/m^3 OEL and 5 mg/m^3 OEL for respirable dust (South Africa, Hazardous Chemical Substance Regulation, 1995). Contrary to that, Health and Safety Executive (SHE) (2020) specified that, if softwood dust might be mixed with hardwood, the OEL applicable for hardwood dust must applied to all wood dust present in the mixture. However, hardwood dust is classified as human carcinogen while softwood is a sensitiser and a suspected human carcinogen (American Conference of Governmental Industrial Hygienists (ACGIH), 2012; Davies and Henderson, 2009a, 2009b; Spee *et al*., 2007). If workers are exposed consecutively to more than one agent, the statistic on EN 689 of 1995 needs to be taken into considerations when determining limit of detection.

The occupational exposure limits (OEL) may not be effective in reducing workers' exposure to chemicals. The industry views the ACGIH TLV as an achievable level rather than a health-based level (Occupational Health Clinics for Ontario Workers (OHCOW), 2012; OHCOW, 2016). Wood dust exposure levels below 1 to 1.5 mg/m^3 are unlikely to cause significant symptoms (Kauppinen *et al*., 2006; SCOEL, 2003). The OEL needs to be lowered for its effectiveness in reducing exposure. In addition, sampling must be undertaken in workplaces where exposures are at or above the OEL. Ayalew, Gebre and Wael (2015) indicated that 71% of measurements taken at the Ethiopian sawmills exceeded the 5 mg/m^3 OEL set by the European Union. However, in the absence of legislative obligations, employers may decide not to measure at all for fear of being deemed non-compliant (Occupational Health Clinics for Ontario Workers, 2012). Based on the measurements results obtained, approved RPD with higher assigned protection factor (APF) to be used as a supplement to the engineering and administrative control in respect of shorter duration of exposure.

2.8. Occupational exposure to noise

Noise is an unwanted sound, and continuous exposure to noise is the main source of sleeping disturbance, hearing impairment, high blood pressure, stress, and other physical, physiological, and psychological effects (Koehncke, 1999; Seidman and Standring, 2010; Stansfeld and Matheson, 2003; Ugbebor and Yorkor, 2015). Health outcomes develop from unprotected exposure to noise levels between 80 and 84 dB(A) (Cantley, Galusha, Cullen, Dixon-Ernst, Rabinowitz and Neitzel, 2015). In the sawmill, the running of high machinery engines and air cylinder exhausts generates considerable noise levels (Koehncke, 1999). Dost (1974a) conducted a noise survey at a California lumber mill that processed softwood and discovered that the noise levels for the chipper tender, tail sawyer, and planer tender feeder were 104, 106, and 107 dB(A), respectively. Myles *et al*. (1971, as cited in Koehncke, 1999) did a similar research study on logging machinery in Eastern Canada and found that the average noise levels produced by the chain saws and skidders were 104 dB(A) and 106 dB(A). Noise levels generated by idling saws can vary from 80 to 96 dB(A) and 100 to 114 dB(A) while under load. These levels are greater than 108 dB(A) and 105 dB(A) produced by the wood planer and tail sawyer machines, and nearly 80% of saw blades idle during operational time (Koehncke, 1999).

Koehncke (1999) conducted personal monitoring for noise in Alberta, Canada, and recorded 95 dB(A) noise levels. About 27% of sawmill employees were unprotected from noise levels beyond 85 dB(A) NIOSH REL (Kling, Demers, Alamgir and Davies, 2012). Nearly 250 million employees worldwide are unprotected from noise levels above 90.2 dB(A), and 28% of these workers have NIHL (Robinson, Whittaker, Acharya, Singh and Smith, 2015). Exposure to noise levels above 130 dB(A) is the leading cause of 34.7% of permanent threshold shift (PTS) (Ratnasingam, Natthondan, Ioras and McNulty, 2010). About 33.4 million employees in the USA were unprotected from excessive noise, and 10 million of them have NIHL (Cantley *et al*., 2015; Ahmed, Dennis, Badran, Ismail, Ballal, Ashoor and Jerwood, 2001; Nelson, Nelson, Concha-Barrientos and Fingerhut, 2005).

2.9. Health effects associated with exposure to noise

Prolonged exposure to loud noise is slightly associated with high blood pressure, or hypertension. Moreover, long-term unprotected exposure to noise levels between 65 and 70 dB(A) is linked with cardiovascular effects (Jarup, Babisch, Houthuijs, Pershagen, Katsouyanni, Cadum, Dudley, Savigny, Seiffert, Swart, Breugelmans, Bluhm, Selander, Haralabidis, Dimakopoulou, Sourtzi, Velonakis and Vigna-Taglianti, 2008). However, elevated blood pressure is linked with road traffic noise exposure (Babisch, 2002; Skogstad, Johannessen, Tynes, Mehlum, Nordby and Lie, 2016; van Kempen and Babisch, 2012). The Scientific Literature for Cardiovascular Diseases pointed out that exposure to extreme noise is linked with an increased risk of high blood pressure and CVDs (Skogstad *et al*., 2016). Exposure to extreme noise is also linked to hypertension, irregular heart rhythms, sleep disorders, hearing defects, irritability, stress, misperception, and a decline in production (European agency for safety and health at work, 2005; Olayinka, 2013).

Health effects of exposure to noise include acoustic trauma, tinnitus, temporary threshold shift, destruction of speech communication, and permanent threshold shift (Otoghile, Onakoya and Otoghile, 2018), as well as 87% of headaches and 72% of hearing impairments (Aremu, Aremu and Olukanni, 2015). The incidences of ringing in the ears are higher among older people with hypertension, exposure to loud sounds, a history of smoking, and hearing impairments (Shargorodsky, Curhan and Farwell, 2010). Unprotected exposure to loud noise is linked with an increased risk of sensorineural hearing loss (SNHL), cardiovascular diseases, and harmful effects like ischemic heart diseases and acute myocardial infarction (Davies, Teschke, Kennedy, Hodgson, Hertzman and Demers, 2005). Occupational exposure to loud sounds is also linked to an increased risk of coronary heart disease and hypertension (Liu, Young, Yu, Bao and Chang, 2016a). Heart and circulatory disease is the main cause of death among men aged between 12 and 14 years (Virkkunen, Kauppinen and Tenkanen, 2005). Moreover, occupational noise is the main cause of metabolic syndromes such as hypertension, a high Body Mass Index (BMI), and dyslipidemia. NIHL and ringing in the ears are the most common auditory health results of occupational noise exposure (Otoghile, Onakoya and Otoghile, 2018).

Exposure to excessive noise is associated with an increased risk of extra-auditory health effects such as stress, decline in performance, hypertension, sleep disorders, and heart diseases (Cantley *et al*., 2015). The health effects related to noise exposure include a decline in performance functions, annoyance, and psychiatric symptoms (see figure 2.1) (Liu *et al*., 2016a). However, exposure to noise levels above 85dB(A) is associated with an increased risk of auditory health effects such as NIHL, tinnitus, acoustic trauma, hearing sensitivity, and PTS, as well as non-auditory health effects such as psychological, social, physical, and economic effects (Basner, Babisch, Davis, Brink, Clark, Janssen and Stansfeld, 2014; Cantley *et a*l., 2015; Owoyemi, Falemara and Owoyemi, 2016; Concha-Barrientos, Campbell-Lendrum and Steenland, 2004; European agency for safety and health at work, 2005; Münzel *et al*., 2014; Timmins and Granger, 2010).

In addition, exposure to noise levels above the 80 dB(A) action level is the leading cause of injuries among men, while unprotected exposure to noise levels above the 85 dB(A) NIOSH recommended exposure limit (REL) and the 90 dB(A) OSHA permissible exposure level leads to NIHL (Clark and Bohne 1999; Kling *et al.*, 2012; Stansfeld and Matheson, 2003). Helzner, Cauley, Pratt, Wisniewski, Zmuda, Talbott, de Rekeneire, Harris, Rubin, Simonsick, Tylavsky and Newman (2005) opined that reduced cochlear blood flow or the ototoxic effects of nicotine on the cochlear hair cells cause NIHL in cigarette smokers. The conceptual model of noise and its associated health effects on the standard of living is detailed in figure 2.1 below

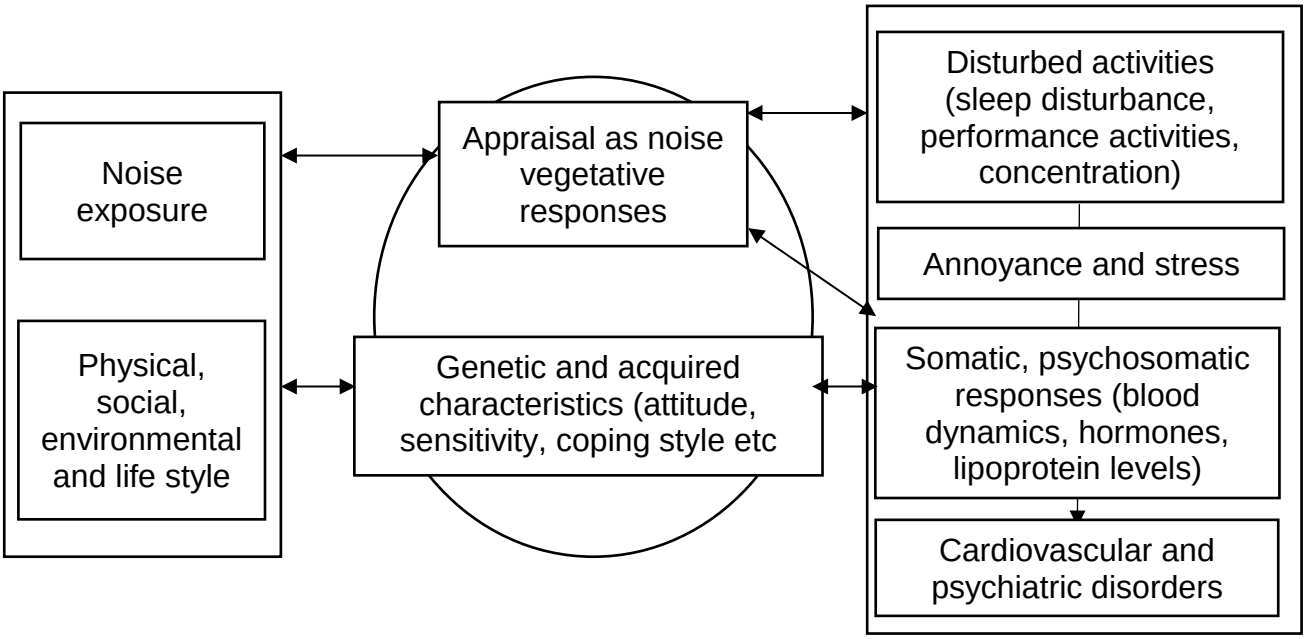


Figure 2.1.: The conceptual model of noise and its health effects based on cognitive stimulus response model (adapted in full from Davies and van Kamp, 2012; Passchier-Vermeer and Passchier, 2000; van Kempen, Kruize, Boshuizen, Ameling, Staatsen and de Hollander, 2002)

2.9.1. Annoyance

The health outcomes of unprotected exposure to noise and their impact on the auditory system are well recognized (Münzel *et al*., 2014). Annoyance is the most commonly reported community health effect initiated by exposure to transport noise (Clark and Stansfeld, 2007; World Health Organization (WHO), 2011). Acoustic elements like sources of sound, levels, and length of experience and non-acoustical factors like beliefs and noise sensitivity about whether noise could be reduced by those accountable influence annoyance responses (Clark and Stansfeld, 2007). Noise interferes with job performance, causes annoyance, and alters social behavior (Stansfeld and Matheson, 2003). It is the leading cause of sleep disturbances and an impairment of cognitive performance (Passchier-Vermeer and Passchier, 2000). Furthermore, epidemiological studies have proven that noise pollution is linked with an increased risk of arterial high blood pressure, stroke, and heart attack (Münzel *et al*., 2014). Noise may affect health by causing an annoyance response, which causes a stress response and subsequent illness in both children and adults (Clark and Stansfeld, 2007).

Noise affects the quality of life in a larger population (Clark and Stansfeld, 2007; Dalton, Cruickshanks, Klein, Klein, Wiley and Nondahl, 2003). The duration and quality of sleep are influenced by the environment, education, counseling, and measures of public health (Halperin, 2014). Sleep deprivation increases sympathetic nervous system activity. Epidemiological studies have discovered that sleep deprivation increases the risk of hypertension, diabetes mellitus, and coronary heart disease (Mosendane, Mosendane and Raal, 2008; Nagai, Hoshide and Kario, 2010). Adequate sleep may prevent cardiovascular diseases (Nagai, Hoshide and Kario, 2010).

2.9.2. Cardiovascular diseases (CVD)

Exposure to noise levels above 85 dB(A) increases the risk of cardiovascular disease or mortality (Gopinath, Thiagalingam, Teber and Mitchell, 2001; Virtanen and Notkola, 2002). Cardiovascular disease is a major burden on the economy, society, and health care services (Backé, Seidler, Latza, Rossnagel and Schumann, 2012). It accounts for 45% of on-duty deaths and is a major cause of diseases among firefighters (Soteriades, Smith, Tsismenakis, Baur and Kales, 2011). It is the primary cause of death and the main contributor to the burden of occupational diseases worldwide (Navas-Acien, Guallar, Silbergeld and Rothenberg, 2007; Backé *et al*., 2012; Thayer, Yamamoto and Brosschot, 2010). Occupational noise exposure is the most significant contributor to cardiovascular diseases (van Kempen *et al*., 2002). Heart and circulatory disease are the leading causes of premature deaths and disabilities, as well as the leading contributors to rising health-care costs (de Backer, Ambrosioni, Borch-Johnsen, Brotons, Cifkova, Dallongeville, Ebrahim, Faergeman, Graham, Mancia, Cats, Orth-Gomér, Perk, Pyörälä, Rodicio, Sans, Sansoy, Sechtem, Silber, Thomsen, Wood, Albus, Bages, Burell, Conroy, Deter, Hermann-Lingen, Humphries, Fitzgerald, Oldenburg, Schneiderman, Uutela, Williams, Yarnell, Priori, Garcia, Blanc, Budaj, Cowie, Dean, Deckers, Burgos, Lekakis, Lindahl, Mazzotta, McGregor, Morais, Oto, Smiseth, Trappe, Budaj, Agardh, Bassand, Deckers, Godycki-Cwirko, Heagerty, Heine, Home, Priori, Puska, Rayner, Rosengren, Sammut, Shepherd, Siegrist, Simoons, Tendera and Zanchetti, 2003).

The fundamental pathology of cardiovascular disease is atherosclerosis (de Backer *et al.*, 2003). In 1990, the European Atherosclerosis Society (EAS), European Society of Cardiology (ESC), and European Society for Hypertension (ESH) agreed to collaborate on guidelines for preventing coronary heart disease in clinical practice with the aim of averting ischaemic heart disease and heart and circulatory disease (de Backer *et al.*, 2003). Exposure to noise contributes to increased risks of arterial hypertension, cardiovascular risk factors, and the prevalence of cardiovascular diseases (Münzel *et al*., 2014). Unprotected from chronic traffic noise was linked with an increase in the prevalence of heart and circulatory disease (Babisch, Beule, Schust, Kersten and Ising, 2005; Davies, Vlaanderen, Henderson and Brauer, 2009c). There is a link between cardiovascular disease risk factors like total cholesterol and hearing loss in elderly people, with the risks being higher for women at lower frequencies (Gates, Cobb, D'Agostino and Wolf, 1993). Additionally, lower frequency presbycusis is linked to microvascular disease that leads to atrophy of the stria vascularis. The risk of cardiovascular disease is caused by lifestyle and physiological factors (de Backer *et al*., 2003). Several interrelated behavioral and physiological mechanisms may increase the risk of cardiovascular diseases. Moreover, the most probable behavioral changes are weight gain and cigarette smoking, while the most plausible physiological mechanisms are lipid change, metabolic syndrome, type II diabetes, atherosclerosis, autonomic nervous system activation, and inflammation (Puttonen, Härmä and Hublin, 2010).

An experimental and observational study has established that unprotected exposure to excessive sound results in irritation, sleep disruption, daytime sleepiness, and an increase in the incidence of hypertension, cardiovascular disease (CVD), and cognitive impairment of performance in both learners and staff (Basner *et al*., 2014; Münzel *et al.*, 2014; Passchier-Vermeer and Passchier, 2000). An elevated blood pressure (basal and fixed systolic or diastolic blood pressure) at any given age or gender is a potential co-founder of all forms of cardiovascular diseases (Samuelsson, Wilhelmsen, Andersson, Pennert and Berglund, 1987; Kannel, 1974). However, hypertension is the most common and a potential contributor to cardiovascular mortality (Jarup *et al*., 2008; Kannel, 1974; Kannel, 1993). Smoking cigarettes, heredity, socio-economic status (SES), hyperlipidemia, and hypertension escalate the risk of CVD (Eriksson, 2019).

There is a significant correlation between SES and cardiovascular risk factors such as smoking, hypotension, hypertension, and high-density lipoprotein cholesterol (Winkleby, Jatulis, Frank and Fortmann, 1992). Moreover, education is the key risk factor for SES, while advancing the educational level is the socio-economic predictor of good health (Winkleby *et al*., 1992). Cardiovascular diseases are related to parasympathetic activities and heart rate variability (HRV) (Thayer, Yamamoto and Brosschot, 2010). Circadian rhythms, disturbed socio-temporal patterns, social support, stress behaviors, and biochemical changes are the main contributing factors to cardiovascular diseases (Bøggild and Knutsson, 1999). People with insulin resistance syndrome are at the highest risk of CVD (de Backer *et al*., 2003). Hypertension, obesity, family history, and work stress are the most important changeable and non-changeable risk factors for cardiovascular disease (Koslowski, 1998). Noise exposure is the primary cause of the stress response, which increases the secretion of catecholamines and cortisol (Babisch, 2002; Otoghile, Ediale, Ariyibi, Otoru, Kuni and Maan, 2019). The estimated cost of stress-related disease is 100 billion euros, and job stress affects production (Koslowski, 1998).

Several cross-sectional studies have associated effort-rewards imbalance and job strain at work, which are characterized by chronic work stress, with coronary heart disease (Aboa-Eboulé, Brisson, Maunsell, Mâsse, Bourbonnais, Vézina, Milot, Théroux, Dagenais, 2007; Kivimäki, Virtanen, Elovainio, Kouvonen, Väänänen and Vahtera, 2006; Peter and Siegrist, 2000). Shift work may increase the risks of cardiovascular diseases by 40%, while psychosocial factors, behavioral and physiological mechanisms, passive smoking, monotonous or sedentary work, and other stressful jobs may add to an increased risk of cardiovascular diseases (Bøggild and Knutsson, 1999; Puttonen, Härmä and Hublin, 2010). Various chemicals, such as solvents and organophosphates, may escalate the risk of cardiovascular diseases (Bøggild and Knutsson, 1999). High concentrations of bad cholesterol and triglycerides in combination with lower concentrations of good cholesterol were linked with increased risks of stroke, coronary heart disease, and atherosclerosis (Psaty, Heckbert, Atkins, Siscovick, Koepsell, Wahl, Longstreth Jr., Weiss, Wagner, Prentice and Furberg, 1993). CVD can be prevented by eating healthy, exercising, and avoiding tobacco and alcohol intake. Figure 2.2 depicts occupational noise exposure and related diseases.

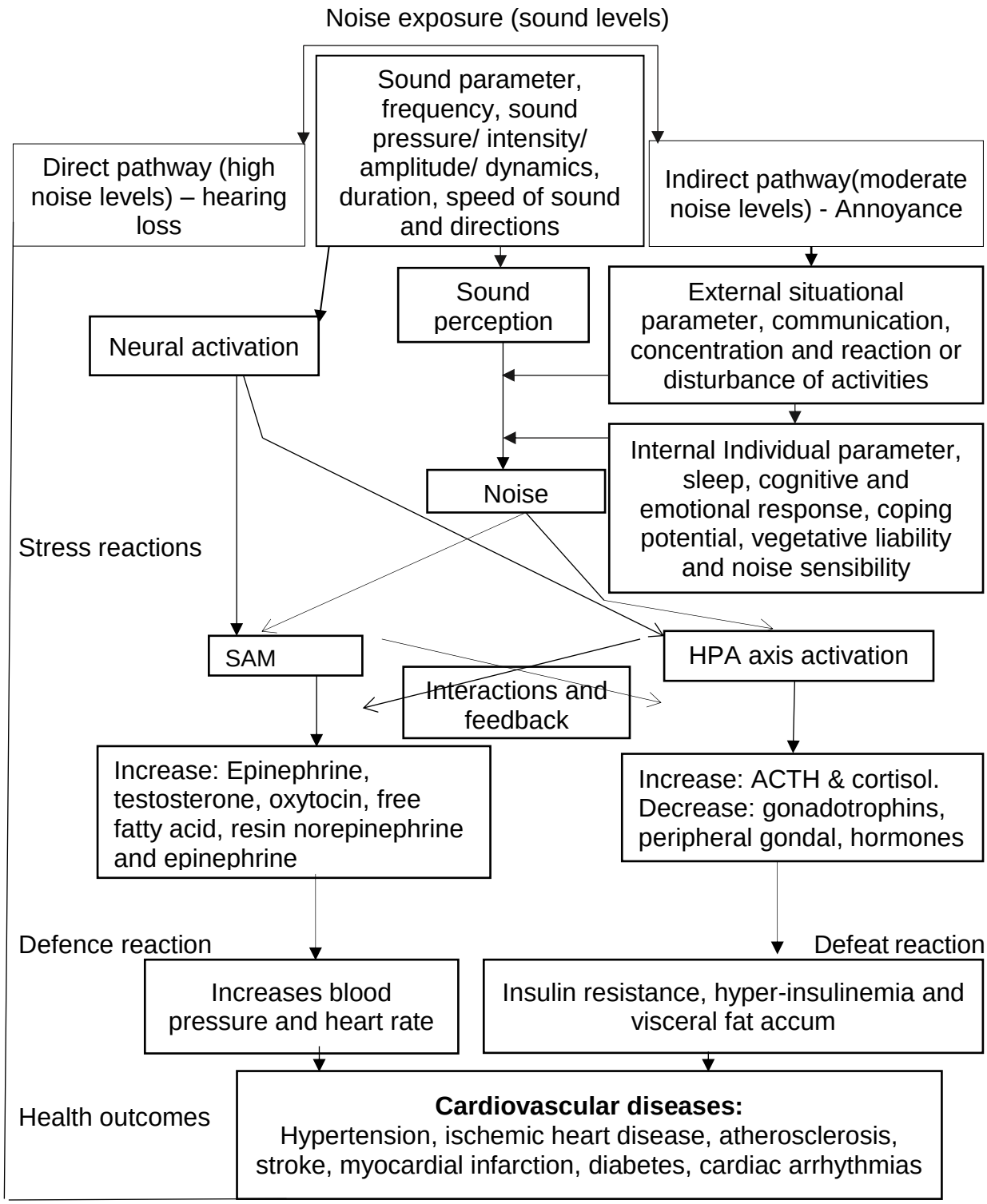


Figure 2.2.: Noise and cardiovascular disease risk model (adapted and modified from Babisch, 1998; Davies, 2002; Münzel *et al.*, 2014).

Abbreviations:

HPA – Hypothalamic pituitary adrenal

SAM – Sympathetic adrenomedullary system

ACTH – Adrenocortico-tropin hormone

2.9.3. Stroke (Brain attack)

In Sweden, around 25,800 people had a stroke or an ischemic stroke in 2017, with a prevalence of 360 per 100,000 people, and 26% of these people died within 28 days (Eriksson, 2019). About 75% of stroke victims were aged 70 or older, even though stroke can affect people of all ages (Eriksson, 2019). Stroke is common among men aged 85 and older because the mortality rate is much higher (Eriksson, 2019). Stroke and coronary heart disease are inversely linked to socio-economic status amongst women and men (Eriksson, 2019). A meta-analysis and literature showed that the risk of coronary artery disease in females with low socio-economic status may be greater (Eriksson, 2019).

Shift work is linked with vascular events such as 1.6% ischaemic stroke, 7% myocardial infarction, and 7.3% coronary events, which have some implications for public policy and industrial medicine (Vyas, Garg, Iansavichus, Costella, Donner, Laugsand, Janszky, Mrkobrada, Parraga and Hackam, 2012). Epidemiological research has shown that environmental noise increases the risk of brain attack, pulmonary hypertension, and myocardial infarction (Münzel *et al*., 2014). Exposure to aircraft noise is related to an increased risk of brain attack, coronary artery disease, heart disease, and circulatory disease (Hansell, Blangiardo, Fortunato, Floud, de Hoogh, Fecht, Ghosh, Laszlo, Beevers, Gulliver, Best, Richardson and Elliott, 2013). Despite the paucity of evidence, lead exposure is linked with an increase in the incidence of stroke, coronary artery disease, and peripheral arterial diseases (Navas-Acien *et al*., 2007). Furthermore, mortality from myocardial infarction and stroke develops rapidly before medication can be administered, and numerous therapeutic interventions are irrelevant because the mass prevalence of CVD was linked with style of living and modifiable physiological factors, and adjustments on these factors showed a decline in the incidence of death and disease mainly in persons with recognized type 2 diabetes (T2D) or unrecognized diabetic cardiac impairment (UDCI) CVD (de Backer *et al*., 2003; Schernthaner *et al*., 2018).

Stamler, Stamler, Neaton, Wentworth, Daviglus, Garside, Dyer, Liu and Greenland (1999) found that the long-term mortality rate from stroke, cardiovascular disease, and coronary heart diseases was significantly lower on individuals who did not smoke or have diabetes or myocardial infarction, and their life expectancy was much greater or longer. Extreme noise exposure is the leading cause of elevated cholesterol and triglyceride levels in male smokers (Mersereau, n.d.). About 27% of male and 11% of female deaths are attributable to smoking (Ekpu and Brown, 2015). Smoking causes 80% of lung cancer, bronchitis, and emphysema deaths and 17% of heart disease deaths (Ekpu and Brown, 2015). Moreover, more than a quarter of all cancer deaths are attributable to smoking, while 600,000 deaths per year are caused by second-hand smoking. Women and small children are more vulnerable. Unprotected from noise levels above 85 dB(A) and smoking over 30 packets of cancer sticks every day may increase the risks of hearing loss by 1.64 (the odds ratio is 1.56) for those who smoke between 10 and 20 packets of cigarettes per day (Mersereau, n.d.).

Tobacco smoking is estimated to cost the country over US$ 500 billion in GDP (Ekpu and Brown, 2015). Smoking is responsible for 15% of the typical health care expenditures in high-income nations. Ekpu and Brown (2015) opine that reducing the prevalence of smoking would result in significant cost savings to health care facilities, governments, and society at large. High carbohydrate consumption is also linked with a high risk of mortality, while total fat and certain types of fat lower the risk. However, total fat and different types of fat are not related to cardiovascular deaths, disease, or myocardial infarction. Nevertheless, saturated fat consumption was inversely linked with stroke (Dehghan, Mente, Zhang, Swaminathan, Li, Mohan, Iqbal, Kumar, Wentzel-Viljoen, Rosengren, Amma, Avezum, Chifamba, Diaz, Khatib, Lear, Lopez-Jaramillo, Liu, Gupta, Mohammadifard, Gao, Oguz, Ramli, Seron, Sun, Szuba, Tsolekile, Wielgosz, Yusuf, Yusufali, Teo, Rangarajan, Dagenais, Bangdiwala, Islam, Anand and Yusuf, 2017). As a result, global dietary guidelines should be adopted to reduce the risks.

2.9.4. Ischemic Heart Disease (IHD)

Despite a lack of epidemiological data, exposure to occupational noise above the 85 dB(A) ACGIH Threshold Limit Values (TLVs) increases the risk of IHD or mortality (Suadicani, Hein and Gyntelberg, 2012; van Kempen *et al*., 2002). Likewise, there are insufficient epidemiological data on a causal relationship between shift work and IHD (Frost, Kolstad and Bonde, 2009). Babisch, Ising and Gallacher (2003) conducted a prospective cohort study among 3950 middle-aged men to determine if annoyance and disturbance caused by traffic sounds increase the prevalence of ischemic heart disease. They found that traffic noise was linked with an escalating risk of IHD (Babisch, Ising and Gallacher, 2003; Babisch, Ising, Gallacher, Sweetnam and Elwood, 1999). In addition, Smith, Ben-Shlomo, Beswick, Yarnell, Lightman and Elwood (2005) discovered that a high circulation of cortisol to testosterone escalates the risk of IHD by mediating insulin resistance syndrome. Siegrist, Peter, Junge, Cremer and Seidel (1990) developed a model of work-related socio-emotional distress to identify the incongruity between higher workload and lower control over occupational status, namely, job insecurity, poor promotional prospects, and status inconsistency-induced distress conditions. They concluded that a redefined model of work-related socio-emotional distress could best explain the high prevalence of IHD among men (Siegrist *et al*., 1990).

2.9.5. Hypertension (HPT)

The World Health Organization (WHO) described hypertension as pressure in the artery when the heart beats, which is SBP beyond 140 mmHg, as well as pressure in the artery when the heart rests between heart beats, which is DBP beyond 90 mmHg (Shrestha and Shiqi, 2017). High blood pressure is the main cause of heart and circulatory disease. Occupational exposure to noise levels above 85 dB(A) increases the risk of hypertension (Chang, Hwang, Liu, Chen, Wang, Bao and Lai, 2013; Eriksson, 2019; Stokholm, Bonde, Christensen, Hansen and Kolstad, 2013; Sbihi, Davies and Demers, 2008; Skogstad *et al*., 2016; Talbott, Findlay, Kuller, Lenkner, Matthews, Day and Ishii, 1990; Virkkunen, Härmä, Kauppinen and Tenkanen, 2007). There is sufficient evidence that exposure to excessive noise increases the risk of hypertension, however, evidence on the prevalence of CVD is limited (Davies *et al*., 2005).

Long-term reductions of six millimeters in DBP are associated with a 30 to 40% reduction in strokes and 20 to 25% reduction in coronary heart diseases (Shrestha and Shiqi, 2017). Nevertheless, high blood pressure is related to increased risks of coronary heart disease and stroke (National Centre for Chronic Disease Prevention and Health Promotion, 2020). Chronic exposure to environmental noise affects cardiovascular systems, escalating the risks of hypertension, heart attacks, coronary artery disease, and angina pectoris (Owoyemi, Falemara and Owoyemi, 2016). Occupational exposure to excessive noise increases the risk of hypertension, SBP and DBP levels depending on the duration of exposure (Davies, 2002). Noise influences SBP, DBP, and heart rate, resulting in hypertension (Otoghile, Onakoya and Otoghile, 2019). However, hypertension is prevalent in the elderly, and heredity, advancing age, sedentary life, lifestyle, and diet may escalate its likelihood. The repetitive stimulation of the sympathetic nervous system and neuroendocrine system due to physiological stress reactions to noise and temporary increases in blood pressure leading to hypertension (Davies *et al*., 2005). Furthermore, the continuous oversecretion of cortisol caused by noise exposure may result in visceral fat accumulation and insulin resistance. Similarly, a rise in SBP and DBP may heighten the risk of hypertension (Davies *et al*., 2005).

According to Navas-Acien *et al.* (2007), there is adequate data associating lead exposure with the prevalence of hypertension, while residential noise exposure is linked with an increase in the incidence of high blood pressure (Bluhm, Berglind, Nordling and Rosenlund, 2007; Barregard, Bonde and Öhrström, 2009). However, transport noise has been shown to have a mild impact. Literature revisions have revealed that transport noise has a moderate influence on hypertension, cardiovascular diseases, catecholamine secretion, and psychological symptoms (Clark and Stansfeld, 2007). Industrial and community-based studies have associated aircraft and road traffic noise exposure with psychological symptoms, while occupational exposure to sound is linked to an increase in catecholamine secretion (Stansfeld and Matheson, 2003). Additionally, environmental and occupational exposure to noise is linked with hypertension, although community-based studies indicate a minimal connection between noise exposure and cardiovascular disease (Stansfeld and Matheson, 2003). Correspondingly, epidemiologic studies provide substantial evidence linking environmental noise with an increased risk of stroke, arterial hypertension, and myocardial infarction (Münzel *et al*., 2014).

An experimental and observational analysis have shown that nighttime sound is the leading cause of sleep disturbances, a rise in blood pressure, heart rate, elevated stress hormone levels, oxidative stress, functional atherosclerosis, and pulmonary arterial hypertension (Münzel *et al*., 2014). Unprotected from transport noise and sounds increases the risk of high blood pressure and coronary artery disease (Clark and Stansfeld, 2007). According to Kaplan and Keil (1993), there is an opposite relationship among SES indicators and hypertension, smoking, good and bad cholesterol, BMI, alcoholism, and diabetes mellitus. A stressful psychosocial work environment, for example, shift work, is a major cause of hypertension and atherogenic lipids (Peter, Alfredsson, Knutsson, Siegrist and Westerholm, 1999). Exposure to noise when driving a bus may increase health risks due to hypertension rates (Ragland, Winkleby, Schwalbe, Holman, Morse, Syme and Fisher, 1987). Endothelial dysfunction for setting hypercholesterolemia, diabetes mellitus, chronic smoking, and hypertension are reliant on the creation of reactive oxygen species and a decrease in the vascular bioavailability of nitric oxide (Schulz, Jansen, Wenzel, Daiber and Münzel, 2008). Chobanian, Bakris, Black, Cushman, Green, Izzo Jr., Jones, Materson, Oparil, Wright Jr. and Roccella (2003) reported that thiazide-type diuretics can be used to treat patients with uncomplicated hypertension either alone or in combination with other drugs such as angiotensin-receptor blockers, beta-adrenergic blocking agents, dihydropyridines and nondihydropyridines, and angiotensin-converting enzyme inhibitors.

Additionally, patients with hypertension may need two antihypertensive medications or more to keep their BP below 140/90 mmHg or below 130/80 mmHg in case of patients with diabetes mellitus or chronic renal disease. However, if blood pressure is above 20/10 mmHg, the doctor should consider prescribing the first therapy with two agents, of which one must be a thiazide-type diuretic, which is the most effective therapy for controlling hypertension if patients are motivated, which may develop if patients have positive experiences and trust in the clinician, while, empathy may build trust and serve as a potential motivator (Chobanian *et al*., 2003). Systolic BP is caused by a decline in hardening of the trunk artery, which normally arises during conversely, higher diastolic blood pressure is linked to exercise, diet, and lifestyle (Otoghile, Onakoya and Otoghile, 2019; Weber, Schiffrin, White, Mann, Lindholm, Knerson, Flck, Carter, Materson, Ram, Cohen, Cadet, Jean-Charles, Taler, Kountz, Townsend, Chalmers, Ramirez, Bakris, Wang, Schutte, Bisognano, Touyz, Sica and Harrap, 2013).

2.9.6. Blood Pressure (BP)

Exposure to noise levels above 85 dB(A) NIOSH REL increases the risk of hypertension (Lee, Kang, Yaang, Choy and Lee, 2009). Noise is the leading cause of high blood lipids and noise-induced vascular damage, which results in a rise in blood pressure and pulse (Otoghile, Onakoya and Otoghile, 2019). Hypertension increases the prevalence of cardiovascular morbidity and death (Kales, Tsismenakis, Zhang and Soteriades, 2009). Other than hypertension, hypotension above 140 mmHg is a risk factor for heart and circulatory disease in persons aged 50 and above. However, the risk of cardiovascular diseases rises at 115/75 mmHg and doubles with a 20/10 mmHg increase. An individual who is 55 years old may be normotensive with a 90% lifetime risk of developing hypertension and may be considered prehypertensive if SBP is 120 to 139 mmHg or DBP is 80 to 89 mmHg (Chobanian *et al*., 2003). Health promotion is necessary to improve lifestyle adjustments and prevent cardiovascular diseases (Chobanian *et al*., 2003). Children unprotected from chronic aircraft noise might have difficulties with long-term memory and reading comprehension, which might increase the risk of developing high blood pressure (Stansfeld and Matheson, 2003).

Notwithstanding inadequate credible data, there is a strong link relating to work stressors, shift work, noise and high blood pressure (Kristensen, 1989). Evidence from cohort studies suggests that railway noise increases the risk of high blood pressure, while traffic noise increases the risk of blood pressure in diabetic patients (Dratva, Phuleria, Foraster, Gaspoz, Keidel, Künzli, Liu, Pons, Zemp, Gerbase and Schindler, 2012). Skogstad *et al*. (2016) hypothesized that the link between occupational sound exposure and the prevalence of hypertension and cardiovascular diseases is stronger than the link between unprotected exposure to occupational sound and heart and circulatory disease mortality. Lalande, Hétu, and Lambert (1986) discovered that noise exposure between 85 and 95 dB(A) increases the incidence of NIHL at 4000 Hz in pregnant women when exposed to noise of a lower frequency, like 0.5 kHz or less. Prolonged unprotected exposure to occupational noise can escalate the incidence of hypertension and fatigue in pregnant women (European Agency for Safety and Health at Work, 2005). Unprotected exposure to sound during pregnancy can cause adverse outcomes for the fetus. Correspondingly, Timmins and Granger (2010) assert that exposure to extreme noise during pregnancy might impair the fetus hearing.

The use of HPD by pregnant workers cannot protect the fetus, and the intrauterine measurement has shown that the fetus may not be protected against loud sound. One study discovered an extreme intrauterine noise attenuation of 10 dB at 4 kHz, while another study found a noise attenuation of 20 dB at 4 kHz, and the noise inside the uterus was greater than 2 to 5 dB at 250 Hz (European agency for safety and health at work, 2005). Contrary to that, psychosocial stress can increase the risk of SBP among men with independent adjustments of age, education, smoking habits, alcohol intake, BMI, leisure time, and physical activity, with no significant differences in job strain categories found among women and DBP in both genders (Cesana, Sega, Ferrario, Chiodini, Corrao and Mancia, 2003).

A decision to start treatment by patients rely on a total cardiovascular risk evaluation given the increase in the existence of risk facts, target organ damage, etc. Additionally, the choice of antihypertensive medication for clients with recognized heart and circulatory disease depends on the underlying CVD (Chobanian *et al*., 2003). Patients with a hypertensive crisis of systolic blood pressure above 180 mmHg or diastolic blood pressure above 120 mmHg must start treatment immediately (Chobanian *et al*., 2003). SBP above 140 mmHg and diastolic pressure above 90 mmHg increase the prevalence of CVD. To decrease SBP/DBP below 140/90 mmHg, antihypertensive medication should be used to benefit those with a high cardiovascular risk profile and diabetes mellitus (Chobanian *et al*., 2003). In many interventional studies, BP was controlled by using medicines such as water pills, beta receptor antagonists, angiotensin-converting enzyme inhibitors, calcium antagonists, and ARBs, while the polytherapy treatment was essential in the normal patient-contact experience. In clients with numerous morbidities requiring treatment, the use of multiple medications can be problematic, and a wellness program may be vital to solving the issue. BP reduction should be obtained gradually in all patients. The aim of treatment is to keep BP below 140/90 mmHg. However, in clients with diabetes mellitus and those at great risk of fatal heart and circulatory disease, BP must be managed below 130/80 mmHg (Chobanian *et al*., 2003).

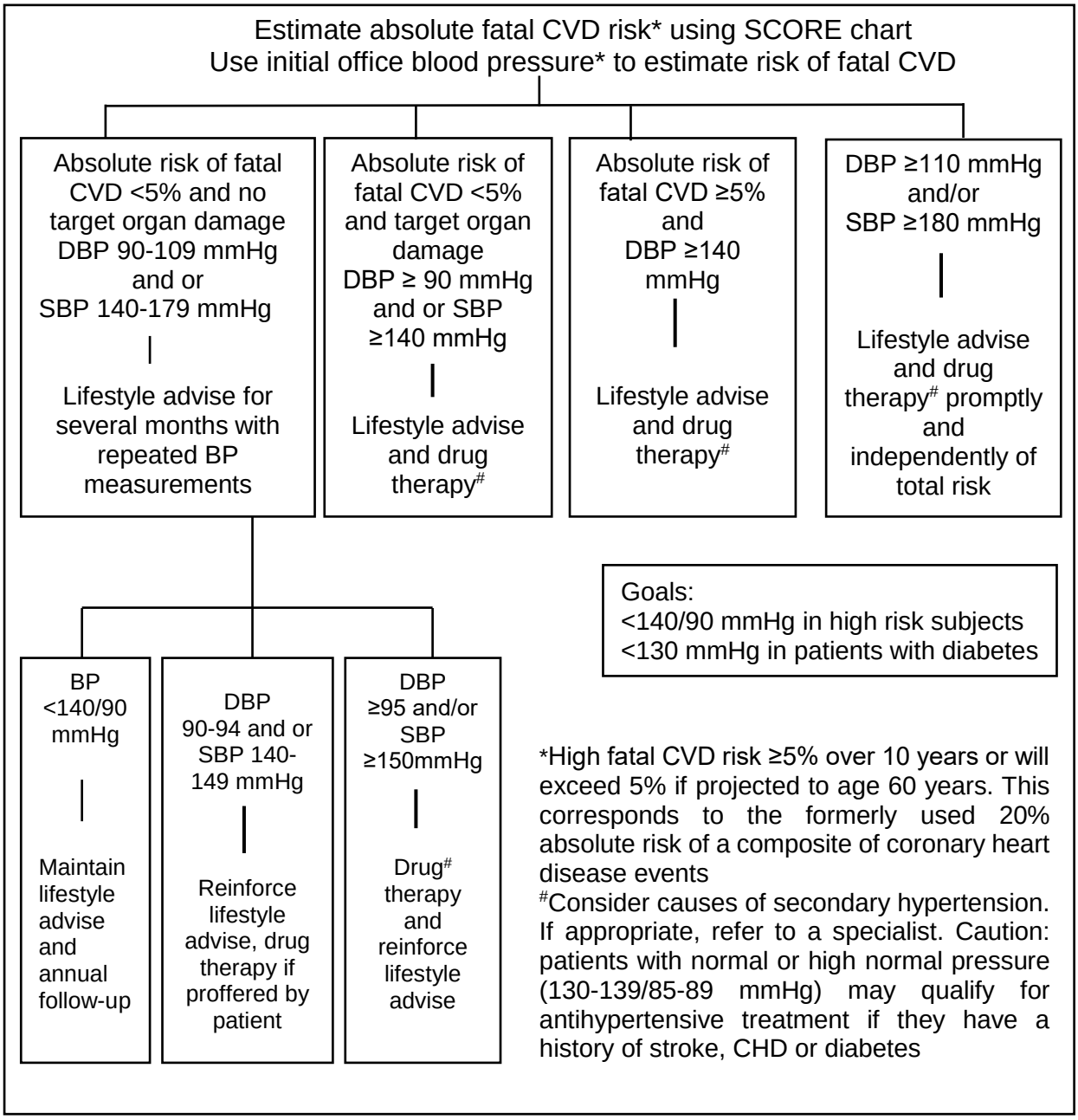


Figure 2.3: Guidelines for managing high blood pressure (adapted in full from Chobanian *et al*., 2003).

2.9.7. Coronary Heart Disease (CHD)

Continuous exposure to noise levels above 85 dB(A) may escalate the risk for CHD (Eriksson, 2019; Virkkunen, Kauppinen and Tenkanen, 2005; Virkkunen *et al*., 2006). CHD results from the hardening of the coronary arteries, or it is a clinical disorder caused by damaged coronary arteries and is known as coronary artery disease or atherosclerosis (Glass, 1980). Myocardial infarction is the most common form of clinical CHD (Glass, 1980). The escalation of coronary heart disease mortality between 1930 and 1950 was linked to coronary atherosclerosis, while a decline in coronary atherosclerosis in the early 20th century was associated with a decline in smoking and cholesterol levels (Dalen, Alpert, Goldberg and Weinstein, 2014). CHD is the main source of mortality in industrialized countries and a major cause of disease burden in developing countries (Gaziano, Bitton, Anand, Abrahams-Gessel and Murphy, 2010; Eriksson, 2019). Hypertension, cigarette smoking, and HBM increase the incidence of CHD because a person may smoke heavily and accept bad eating habits to deal with irritations produced by sound (Virkkunen, Kauppinen and Tenkanen, 2005).

CHD escalate the prevalence of CVD death and morbidity by 5 to 7 times (Wilt, Bloomfield, MacDonald, Nelson, Rutks, Ho, Larsen, McCall, Pineros and Sales, 2004). CHD is the leading cause of mortality in the USA, which accounts for over 425 000 losses each year (Sattelmair, Pertman, Ding, Kohl, Haskell and Lee, 2011). In 2001, coronary heart disease caused over 7.3 million deaths globally (Gaziano *et al*., 2010). Due to socioeconomic changes, an increase in life expectancy, and the acquisition of lifestyle-related risk factors, the CHD burden in low- to middle-income nations has increased rapidly (Gaziano *et al*., 2010). Chronic industrial noise exposure is linked with coronary heart disease in young male current smokers (Gan, Davies and Demers, 2011). Work stress is the main cause of heart disease through psychoneuroendocrine mechanisms. However, work stress increases the risk of CHD by 50% among workers (Kivimäki *et al*., 2006). Transport noise exposure may escalate the risks of coronary heart disease in bus drivers (Rosengren, Anderson and Wilhelmsen, 1991; Wang and Lin, 2001; Ragland, Winkleby, Schwalbe, Holman, Morse, Syme and Fisher, 1987). Bus driving increases the prevalence of CHD (Rosengren, Anderson and Wilhelmsen, 1991). Shift work also increases the occurrence of CHD mortality due to brain attacks. Shift work can affect the health of an individual, depending on smoking habits and age. Shift work may reduce sleep, leading to sleepiness (Eriksson, 2019).

Socio-economic status (SES) is a cardiovascular disease risk factor and is associated with a decline in coronary mortality (Winkleby *et al*., 1992). Shift work affects pregnancy results and is linked to peptic ulcers and CHD (Knutsson, 2003). Chronic work stress is also linked with an increased risk of CHD (Peter and Siegrist, 2000). Chronic job strain after the first myocardial infarction is also linked with an increased risk of CHD (Aboa-E´boule´ *et al*., 2007).

A healthy lifestyle decreases the risk of job strain from coronary artery diseases (Kivimäki, Nyberg, Fransson, Heikkilä, Alfredsson, Casini, Clays, De Bacquer, Dragano, Ferrie, Goldberg, Hamer, Jokela, Karasek, Kittel, Knutsson, Koskenvuo, Nordin, Oksanen, Pentti, Rugulies, Salo, Siegrist, Suominen, Theorell, Vahtera, Virtanen, Westerholm, Westerlund, Zins, Steptoe, Singh-Manoux and Batty, 2003). Depression is associated with the prevalence of mortality in clients with CHD after acute myocardial infarction (AMI), even though bradycardia range mediates the outcome of clinical depression or major depressive disorder on casualty survival after AMI (Carney, Blumenthal, Freedland, Stein, Howells, Berkman, Watkins, Czajkowski, Hayano, Domitrovich and Jaffe, 2005). Treatment can improve depression and bradycardia ranges and patient survival (Carney *et al*., 2005). In CHD patients, depressive symptoms or behavioral factors, namely, physical inactivity, may predict adverse cardiovascular outcomes (Whooley, de Jonge, Vittinghoff, Otte, Moos, Carney, Ali, Dowray, Na, Feldman, Schiller and Browner, 2008). Depression and anxiety are significant personality characteristics of the social environment and risk factors for CHD following myocardial infarction. Increased risk of CHD was associated with social isolation, hostility, interpersonal conflict, and job stress (Smith and Ruiz, 2002). Psychosocial control may help in CHD disease and death (Smith and Ruiz, 2002).

Physical activity is associated with a 20% to 30% reduction in the risk of CHD. However, the dose-response relationship is essential. Individuals who engaged in 150 minutes or a week of moderate-intensity leisure time, which corresponds to the minimum amount recommended by United States federal guidelines, had a 14% lesser chance of CHD compared to those who reported no free time physical exercise, while, individuals engaged in five hours or seven days of lifestyle activities of moderate intensity had a 20% lesser chance of CHD. A person who is physically active at levels lower than the minimum recommended amount has a considerably reduced risk of

CHD (Sattelmair *et al*., 2011). Administration of amlodipine to clients who have coronary heart disease or ischemic heart disease and normal BP may reduce cardiovascular events, but smaller treatment effects were observed with enalapril, and intravascular ultrasound showed that amlodipine slows atherosclerosis progression (Nissen, Tuzcu, Libby, Thompson, Ghali, Garza, Berman, Shi, Buebendorf and Topol, 2004). A randomized controlled trial demonstrated that lowering cholesterol levels and bad cholesterol reduces cardiovascular events and mortality. Statin therapy may decrease disease and death in adults who have CHD with LDL-C levels as low as 100 mg/dL (2.59 mmol/L) before treatment (Wilt *et al*., 2004). The identification and characterization of modifiable risk factors for CHD are vital for public health and clinical medicine (Sattelmair *et al*., 2011). The development of atherosclerosis, thrombosis and hypertension is associated with genetic heredity, physio-chemical determinants and psychosocial stress (Peter and Siegrist, 1999).

2.9.8. Myocardial Infarction (MI)

Shift work is a risk factor for MI and atherosclerosis (Haupt, Alte, Dörr, Robinson, Felix, John and Völzke, 2008). The number of fatalities attributable to acute MI is reduced with effective treatment (Dalen *et al*., 2014). From the mid-1960s to the present, primary and secondary preventions have reduced the prevalence of acute MI and sudden cardiac fatalities (Dalen *et al*., 2014). Unprotected from chronic sound is linked with the prevalence of acute MI deaths (Davies *et al*., 2005). Prolonged unprotected exposure to traffic sounds may escalate the risks of MI (Selander, Nilsson, Bluhm, Rosenlund, Lindqvist, Nise and Pershagen, 2009). Latest clinical trials have revealed that some form of therapeutic prevention of coronary events, revascularizations, embolic or thrombotic stroke, and peripheral arterial disease can be undertaken due to the interrelated aetiologies of MI, ischaemic stroke, and peripheral arterial diseases (de Backer *et al*., 2003). Even though Alfredsson, Hammar and Hogstedt's (1993) study on 9446 male bus drivers in Sweden found that bus drivers had an increased risk of MI. Previous studies have established that urban bus drivers are at high risk of suffering from coronary heart disease. Job strain, irregular working hours, sedentary jobs, automobile exhaust fumes, and noise in the work environment of bus drivers might be contributing factors to the escalating risks (Alfredsson, Hammar and Hogstedt, 1993).

In 2017, the National Board of Health and Welfare (NBHW) reported approximately 25,300 cases of acute MI with a prevalence ratio of 340 per 100,000 inhabitants (Eriksson, 2019). MI was more prevalent in men than women and was linked with educational levels. MI prevalence was greater among individuals with low education than among those with tertiary education, while increasing with age. Unprotected from industrial noise over 20 years may increase the risk of MI (Eriksson, 2019). Industrial noise might stimulate the vegetative nervous system and hormone systems, leading to an escalation of hypertension, affecting the pulse, and causing the discharge of cortisol. Psaty, Heckbert, Koepsell, Siscovick, Raghunathan, Weiss, Rosendaal, Lemaitre, Smith and Wahl (1995) found that large dosages of short acting calcium channel blockers administered to hypertensive patients were linked to a 60% increase in MI risk. A high dosage of short-acting calcium channel blockers is related to the prevalence of MI (Psaty *et al*., 1995). Chronic exposure to noise is also related to the prevalence of MI (Willich, Wegscheider, Stallmann and Keil, 2006). In postmenopausal women, long-term oestrogen replacement treatment was related to a low frequency of MI (Heckbert, Weiss, Koepsell, Lemaitre, Smith, Siscovick, Lin and Psaty, 1997). A cross-sectional study was conducted among 5419 adult Finnish men to determine if sleeping for nine (9) or six (6) hours increases the risk of MI. Those who slept more than 9 hours had an increased risk of MI, whereas those who slept less than 6 hours per night had an escalated incidence of symptomatic coronary heart disease or ischemic heart disease (Partinen, Putkonen, Kaprio, Koskenvuo and Hilakivi, 1982).

Willich *et al*. (2006) conducted a case-control study among 4115 patients who were admitted to 32 major hospitals in Berlin between 1998 and 2001 with confirmed cases of acute MI to evaluate if environmental noise, industrial noise, and annoyance increase the risk of MI. There was a slight increase in MI risks among women, which was attributed to annoyance from exposure to environmental noise rather than industrial noise. Environmental noise was related to an increase in the risk of MI among men and women, while industrial noise was associated with an increased prevalence of MI in males. Unprotected exposure to chronic sound is linked to the prevalence of MI or adverse pathophysiological effects. The risk was closely linked with noise rather than annoyance, which was the main cause of cardiovascular disease (Willich *et al*., 2006). An increase in plasma homocysteine level increases the risks of developing congestive heart failure (CHF) in adults without a history of MI (Vasan, Beiser, D'Agostino, Levy, Selhub, Jacques, Rosenberg and Wilson, 2003).

An observational study has suggested that unopposed oestrogens may reduce the incidence of coronary artery disease in postmenopausal females, while retrospective studies or case-referent studies establish that the reduced incidence of MI linked with the use of oestrogen alone was related to the results of other cohort, cross-sectional, and case-control studies (Psaty, Heckbert, Atkins, Lemaitre, Koepsell, Wahl, Siscovick and Wagner, 1993). The three main types of hearing loss are discussed below.

2.9.9. Noise Induced Hearing Loss (NIHL)

NIHL is the most compensable occupational disease worldwide (ACGIH, 2005a; ACGIH, 2012; ACGIH, 1995; Nandi and Dhatrak, 2008). Hearing loss accounts for 4.2 million disability-adjusted life years (Cantley *et al*., 2015). Around 10 million workers in the USA suffer from NIHL, and almost 288 million dollars were spent on hearing aids. Nevertheless, it is difficult to evaluate the impact of NIHL on the economy (Cantley *et al*., 2015; Neitzel, Andersson and Andersson, 2016). NIHL occurs when the cochlea sensory hair cells are harmed by severe acoustic force in the inner ear (Sliwinska-Kowalska and Davis, 2012; Koehncke, 1999; Nandi and Dhatrak, 2008). The anatomy, physiological structure, and functions of the human ear have been detailed elsewhere (Alberti, 2006; Driscoll, Mikl and Burgess, 2009). Infections, tumors, ageing, hazardous noise exposure, and certain chemicals and pharmaceuticals (ototoxins) may cause hearing loss (Timmins and Granger, 2010). Excessive noise exposure can cause a temporary threshold shift that results in a decline in hearing sensitivity that recovers within a few minutes to hours and a permanent threshold shift that leads to irreversible sensorineural hearing loss (SNHL) from repeated exposure (NIOSH, 1998).

Prolonged exposure to industrial noise can cause hearing loss, mainly SNHL, conductive hearing loss (CHL) and mixed hearing loss (MHL) (Alshuaib, Al-Kandari and Hasan, 2015). SNHL is an illness caused by inner ear and auditory nerve damage (Koehncke, 1999). Conductive hearing loss (CHL) is a disorder that obstructs the transmission of sound from traveling through the auricle or pinna and tympanic cavity (Koehncke, 1999). CHL is usually traceable to outer and middle ear diseases (NIOSH, 1998). SNHL is the most common category of hearing loss, which is caused by inner ear injury and is bilateral and symmetrical, affecting higher frequencies such as 3, 4, and 6 kHz and spreading towards low frequencies such as 0.5, 1, and 2 kHz (Nandi and Dhatrack, 2008).

Noise induced hearing impairment occurs mainly at higher frequencies of 3 and 6 kHz with the highest impact at 4 kHz (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004; Le, Straatman, Lea and Westerberg, 2017; Nandi and Dhatrak, 2008; Rabinowitz, 2000). The ear is most sensitive to 3, 4 and 6 kHz frequencies due to harmonic amplification of ear canal and unconditional sensitivity. The effect of noise on individuals depends on the physical character of the sound, duration of exposure, amplitude, and frequency (NIOSH, 1998). The range of sound and duration of exposure are critical in the development of NIHL (Alberti, 2006). Age-related hearing loss (presbycusis) occurs when the degeneration of hair cells causes a slow loss of hearing in old age. Older people are susceptible to presbycusis, which results in a progressive loss of hearing in later years (Alberti, 2006; Engdahl and Tambs, 2010; Nandi and Dhatrak, 2008). Once the cochlear and vestibular sensory cells deteriorate, they will not improve, resulting in irreversible SNHL. Additionally, head injuries may cause SNHL, which can disturb the cochlea. In contrast, MHL is a combination (mixture) of SNHL and CHL. Chronic otitis media, acoustic trauma, and Meniere's disease are examples of mixed hearing loss (Driscoll, Mikl and Burgess, 2009). Sawmill employees who are exposed to high noise levels must undergo regular blood pressure monitoring at the clinic. In addition, the proper use of HPDs (ear muffs and plugs) will protect employees from exposure to the harmful effects of loud sound (Otoghile *et al*., 2019).

Exposure to industrial noise is the major source of deafness or hearing loss (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004). The threshold values for binaural hearing are classified as normal hearing below 10 dB, mild hearing between 10 and 25 dB, and impaired hearing above 25 dB (Cantley *et al*., 2015). Additionally, high frequencies of 3000, 4000, and 6000 Hz reflect the range that is most susceptible to NIHL (McBride and Williams, 2001). Disabling hearing impairments among adults occur at a permanent hearing threshold level (PHTL) of 40 or 41 dB, although most individuals can only differentiate words spoken in a raised voice at one meter (Timmins and Granger, 2010). According to Tu, Dogaru and Friedman (2017), the severity of hearing loss can be classified using the American Speech-Language-Hearing Association Threshold-Based Classification System of degree of hearing loss as 0 to 15 dB for normal hearing loss (HL), 16 to 24 dB for slight HL, 25 to 40 dB for mild HL, 41 to 55 dB for moderate HL, 56 to 70 dB for moderately severe HL, 71 to 90dB for severe HL and beyond 91dB for profound HL.

Noise induced hearing loss (NIHL) is an irreversible occupational disease (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004). NIHL interferes with a worker's ability to hear instructions or warning signs (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004). Other effects of NIHL include self-isolation, anxiety, irritability, costs for hearing aids, and workers' compensation (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004). Male employees have a higher prevalence of NIHL advancement than female employees, as well as workers aged 25 years and older than those aged less than 25 years (Thepaksorn, Koizumi, Harada, Siriwong and Neitzel, 2018). Persons with 237412 days are mostly troubled by disabling hearing impairment (Sam *et al*., 2017; WHO, 2012). Employees with more than five years of experience and ex-smokers are at higher risk of developing NIHL than those with less than five years of experience who are current smokers or non-smokers. Employees who never use HPDs are more susceptible to NIHL than those who do (Thepaksorn *et al*., 2017). A 55-year-old person who is exposed to noise for 35 years will develop hearing impairment with a threshold of hearing greater than or equal to 25 dB at 1, 2, and 3 kHz, with 3% of exposure to noise at 85 dB(A), 10% at 90 dB(A), and 49% at 100 dB(A) resulting in substantial impairment (Malchaire, 2000).

Exposure to 80 dB(A) steady state noise over 10 years can cause a slight hearing loss, while 85 dB(A) can result in a 10 dB hearing threshold at most sensitive frequencies (NIOSH, 1998). Hearing thresholds of 25 dB at 0.5, 1, 2, and 3 kHz frequencies lead to NIHL exceeding 2 dB after 40 years (ACGIH, 2005a). Exposure to continuous noise levels above 85 or 90 dB(A) can cause NIHL (Lalande, Hétu and Lambert, 1896; Stansfeld and Matheson, 2003; Verbeek, Kateman, Morata, Dreschler and Mischke, 2012). The WHO placed a limit of 75 dB for industrial noise exposure for 8 hours' (Owoyemi, Falemara and Owoyemi, 2016). Exposure to noise levels below 90 dB(A) can cause ear fatigue and the development of temporary hearing loss or tinnitus (Alberti, 2006). As a result of severe metabolic stress, the hair cells may become fatigued and hearing may become less acute. TTS can develop at hearing threshold levels of 78, 85, and 140 dB and change from short-term to long-term (Alberti, 2006). Unprotected exposure to industrial sound levels beyond 115 and 155 dB(A) for 8 hours after 5 months' pregnancy can cause hearing loss in the foetus (ACGIH, 2012; Timmins and Granger, 2010). The use of HPDs by the pregnant worker will protect her but not the fetus (Mersereau, n.d.).

2.9.9.1. Diagnosis of NIHL

When hearing loss is suspected in employees who have been exposed to excessive noise, a full occupation exposure history, clinical examination, and audiometry evaluation must be done (Driscoll, Mikl and Burgess, 2009). If the assessment reveals the existence of NIHL, a recommendation letter for a complete audiological assessment must be authorized (Rabinowitz, 2000). Employees who will be registered in hearing conservation programs, or hearing loss prevention program (HLPP), are required to receive a baseline audiogram within 30 days of employment (NIOSH, 1998). Audiometry is used to determine the auditory threshold of employees to pure tones between 0.25 and 8 kHz at sound levels between 10 and 110 dB. Prior to the test, employees must not be exposed to noise for 16 hours before the test to eliminate the effects of temporary threshold shift (TTS) (Nandi and Dhatrak, 2008). Employees must not be exposed to noise levels above 85 dB(A) for at least 12 hours before a baseline audiogram test, and HPD must not be worn in lieu of the required waiting periods (NIOSH, 1998). Audiometric testing comprises bone conduction, air conduction, or pure-tone audiometry, where the hearing threshold is tested independently in both ears at 0.5 to 6 kHz, while the 8 kHz frequency is tested to obtain useful information about the aetiology of NIHL (NIOSH, 1998). Air conditioning is assessed by placing earphones on the worker's ears, while for bone conduction, a vibrator is placed on the worker's skull behind the ears (Nandi and Dhatrak, 2008).

The results of a hearing test are presented on a chart, whereas comparing air and bone conduction permits categorizing of conductive, sensorineural, and NIHL on the audiogram, as seen as a dip notch in an audiogram at a maximum frequency of 4 kHz (Nandi and Dhatrak, 2008). In addition, the variance concerning the ears must not go beyond 15 dB at low frequencies of 500, 1000, and 2000 Hz and 30 dB at high frequencies of 3000, 4000, and 6000 Hz. The hearing abilities of the males on the left ear are a little bit poorer compared with the right by almost 4 dB at 4 kHz (Eriksson, 2019). When an audiogram detects a change in the HTL in either ear at or above 5 dB at 0.5, 1, 2, 3, 4 or 6 kHz, an optional retest must be conducted instantaneously to determine whether significant threshold shift (STS) or persistent threshold shift (PTS) has occurred (NIOSH, 1998). If the initial and subsequent thresholds for the left or right ear at 1 kHz disagree within 5 dB, stop testing, refit the earphones, and re-instruct the listener. Then test at 1 kHz again, followed by 0.5 kHz, and finally 1 kHz (Martin, 1997;

Martin, 1983). If thresholds at 1 kHz agree by 5 dB, the test is concluded. If not, the listener is to be referred for further consultation (Martin,1986; Martin, 1998). An ear canal electrocochleography, high-frequency audiometry, and word recognition tasks can be used to identify signs of noise damage in the hair cells and neurons (Liberman, Epstein, Cleveland, Wang and Maison, 2016). A study conducted on exposed employees in Germany and unexposed office employees in Canada established that hearing impairments were beyond 105 decibels hearing level (dB HL) at 2, 3, and 4 kHz, which was equivalent to above 35 dB HL with a prevalence ratio of one (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004).

The study conducted on animals using mid-cochlear synaptopathy revealed that the noise damage in animals showed an increase in high-frequency thresholds and basilar hair cells due to lesser exposure to noise. The noise damage in humans showed an increase in the high-frequency threshold linked to mid-cochlear synaptopathy (Liberman *et al*., 2016). Noise damage in humans accelerates aging and has been linked to further damage of sensory cells and cochlear neurons. The damage to cochlear synaptic connections caused by noise can happen within hours after exposure. The therapeutic window in which the sensory cells and sensory neurons can be reconnected might be lengthy. Neurotrophin-based therapy can be used or local delivery of neurotrophic factors can be made directly to the round window membrane of the inner ear. Cochlear synaptopathy measurements are required in clinical trials of regenerative therapies to identify candidates and track treatment efficacy (Liberman *et al*., 2016).

2.9.9.2. Treatment of NIHL

There is no surgical or medical treatment for noise induced hearing loss (NIHL). For quite some time, aminoglycoside antibiotics have been used to produce the animal model of hearing loss. Platinum-based chemotherapeutic agents are effective in the treatment of malignant diseases and can cause permanent hearing loss as they damage the hair cells of the inner ear, resulting in functional deficits. To protect the inner ear against ototoxicity, antioxidant medicines are administered to provide upstream protection and prevent the activation of cell-death sequences (Rybak and Craig, 2005). Medicine or medical treatment may be ineffective in treating NIHL (Dhere, Pawar, Patil and Pawar, 2009). Lynch and Kil (2005) argued that safe and effective medications such as compounds and otoprotectants will be authorized for the treatment of NIHL even though they will have an impact on medical cost, disability compensation, and several quality of life concerns. Due to damaged hair cells that cannot regenerate, sensorineural hearing loss becomes irreversible. However, its progression can be treated by using preventative, prosthetic, and regenerative measures (Hong, Kerr, Poling, Dhar, 2013). Protein kinase inhibitors that block apoptosis via stress-activated protein kinase provide effective protection for acoustic trauma and ototoxicity. Cochlear implants may partly replace the function and sensory innervation of the lost auditory sensory cell. There is evidence that mammalian vestibular hair cells can be replaced slowly, but there is no evidence that the organ of corti can be replaced (Matsui, Parkr, Ryals and Cotache, 2005).

According to the World Health Organization (2015), NIHL treatment methods focus on people in severely noisy environments, such as the military. To prevent further damage, there are administration approaches that include placing the exposed individual in a quiet room or administering hyperbaric oxygen therapy immediately after exposure to loud sound (WHO, 2015). Post-exposure antioxidant and pharmacological therapy have shown to be effective in preventing further sensory cell damage. Antioxidants may alleviate cochlear damage caused by noise and ototoxicity (Fausti, Wilmington, Helt, Helt and Konrad-Martin, 2005). Currently, no pharmacological agents exist for the prevention or reversal of NIHL (WHO, 2015). Nevertheless, there have been recent advancements in this area, and it is claimed that preventive medications are expected to be available in the time that is to come. Other scientific areas of study include gene and stem-cell therapies (WHO, 2015).

Treatment options for hearing loss are mostly based on medical devices, including hearing aids and cochlear implants (Le, Straatman, Lea and Westerberg, 2017). There are currently no pharmacological therapeutics in use. Gene therapy has been successfully used to treat hearing loss in animal model systems. Moreover, approaches that are based on stem cells and gene therapy and may have the potential to restore or maintain auditory function are beginning to emerge. At present, the only management options for the treatment of NIHL include the use of hearing aids and counseling workers with mild to moderate levels of NIHL (WHO, 2015). During audiometric testing for ototoxic medication, all employees working in noisy areas and pregnant workers must be counselled, and pregnant workers must be relocated to less noisy areas if noise levels exceeds 115 dB(A) peaks. The cardiovascular health should be part of a hearing conservation program (Sriwattanatamma and Breysse, 2000).

2.10. Previous studies on occupational noise exposure and related risks

Gan, Davis and Demers (2011) conducted a cross-sectional study between 1999 and 2004 in the United States of America among 6307 employees to determine if exposure to chronic noise is linked with the incidence of coronary heart disease (CHD). Unprotected from chronic noise was linked with an increased risk of CHD among young male smokers. Two to three incidences of angina pectoris, ischaemic heart disease or coronary artery disease, heart attack, and isolated diastolic blood pressure were associated with exposure to chronic noise. Sbihi, Davies and Demers (2008) conducted a cohort study in British Columbia among 10 872 sawmill workers exposed to noise above 85 dB(A), 90 dB(A), and 95 dB(A) to assess if noise exposure increases the risk of hypertension. Unprotected from excessive noise levels beyond 85 dB(A) was linked with an increased risk of hypertension. Davies, Teschke, Kennedy, Hodgson, Hertzman and Demers (2005) conducted cohort research among 27,464 blue-collar workers who worked for a year at British Columbia lumber mills to determine if exposure to chronic noise increased the risk of acute myocardial infarction. Unprotected from excessive noise was related to an increase in the prevalence of fatal heart attacks.

Eriksson, Andersson, Schiöler, Söderberg, Sjöström, Rosengren and Torén (2018) conducted a study on 5753 men randomly selected among 10 000 men in Gothenburg to assess whether exposure to industrial noise increases the incidence of ischaemic heart disease and brain attack. Unprotected from industrial noise was linked with an increased prevalence of CHD than stroke. Dzhambov and Dimitrova (2016) conducted a study in Spain, Russia, and Bulgaria to determine if noise exposure levels beyond 75 and 80 dB(A) for 20 years escalate the risk of ischemic heart disease or coronary heart disease. The study pointed out that workers exposed to noise levels above 75 and 80 dB(A) had a higher incidence of developing ischemic heart disease. Gupta, Malhotra, Tripathi and Dev (2017) investigated whether exposure to noise levels above 90 dB(A) increased the risk of hypertension among 120 male textile mill workers between the ages of 35 and 55, as well as 30 workers in the areas of weaving, spinning, packaging, and administration. Unprotected from industrial noise levels above 90 dB(A) was linked to an increased risk of hypertension. In addition, prolonged exposure may increase the incidence of mild to moderate blood pressure changes, depending on the intensity of the sound, which might cause cardiovascular disorders, strokes, and myocardial infarction.

Chang, Hwang, Liu, Chen, Wang, Bao and Lai (2013) carried out a longitudinal study among 578 Chinese men to establish the link between noise and the 10-year incidence of hypertension (HPT). Prolonged exposure to industrial noise levels beyond 85 dB(A) was linked to a high risk of hypertension in men. In addition, being unprotected from chronic noise was also linked to an increase in the incidence of cardiovascular disease, ischemic heart disease, acute myocardial infarctions, coronary heart diseases, and stroke. Davies, Teschke, Kennedy, Hodgson and Demers (2009b) carried out a quantitative retrospective cohort study among 27,499 sawmill employees in order to evaluate whether noise exposure increases the incidence of fatal heart attacks. Unprotected from excessive noise was associated with an increase in the occurrence of heart and circulatory disease. Moreover, Wang, Zhou, Li, Kong, Wang, Guo, Zhang, He, Guo and Chen (2018) carried out a transverse or prevalence study among 10,636 employees to determine if exposure to unprotected noise escalates the incidence of hearing loss and high blood pressure. Unprotected from occupational noise was linked with the prevalence of hypertension.

Liu, Young, Yu, Bao and Chang (2016) carried out a cohort study among 1002 employees in China to evaluate if noise exposure increases the risk of hypertension. Unprotected exposure to industrial noise was linked to an increase in the incidence of high blood pressure at a frequency of 4 kHz. In addition, participants unprotected from noise levels beyond 80 dB(A) over an 8-year period were likely to develop high blood pressure. Similarly, Shrestha and Shiqi (2017) carried out a transverse study or prevalence study among 274 workers in an iron and steel manufacturing factory in Guangzhou to determine if occupational exposure to noise at 85, 85 to 90 and above 90 dB(A) were linked with hypertension. Unprotected from industrial noise was linked with the occurrence of high blood pressure, which was highly influenced by body mass index and obesity. Age, work duration, and sleep time also presented an exact relationship with the occurrence of high blood pressure. Davies (2002) conducted a pre-existing retrospective cohort study among 27499 lumber workers to determine if industrial noise exposure increases the risk of cardiovascular mortality. Unprotected from occupational noise beyond 85 dB(A), NIOSH REL was linked with an increased risk of acute myocardial infarction.

However, Akbari, Safari, Kazemi, Nourmoradi, Mahaki and Dehghan (2017) conducted a study among 120 textile male workers exposed to 95 dB(A) noise levels to determine if ear plugs, earmuffs, or ear plugs alone can decrease the level of blood pressure among workers. The findings indicated that the use of earmuffs decreased systolic and diastolic blood pressure, while the use of ear plugs only lowered systolic blood pressure. The use of earmuffs was recommended to control blood pressure changes among textile workers. Picard, Girard, Simard, Larocque, Leroux and Fernan (2008) carried out a study among 52,982 male workers whose hearing was examined at least once between 1983 and 1996 and who were regularly exposed to 80 dB(A) noise levels to determine if noise levels above 90 dB can cause accidents. Industrial noise levels above 90 dB(A) and NIHL were associated with the cause of 12.2% of accidents.

Otoghile, Onakoya and Otoghile (2018) carried out a prospective community-based study among 410 males and 10 female sawmill workers in Ile-Ife to identify the health effects linked to noise exposure. NIHL and tinnitus were linked to noise exposure among sawmill workers. Liu, Xu, Ding, Zhang, Pan, Liu, Ding, Zhao, Wang, Han, Yang and Zhu (2016b) conducted a study among 738 employees at a Chinese coal mine in Datun Xuzhou to assess if industrial noise increases the risk of hypertension.

Unprotected from industrial noise was linked to an increase in the incidence of high blood pressure and NIHL among Chinese coal miners. Moreover, industrial noise exposure increases the incidence of high blood pressure, which might escalate hypertension. Eriksson (2019) conducted a study comprising of 10,000 men born between 1915 and 1925 as well as 7,133 men and 4,496 women to determine if noise or shift work increases the risks of coronary disease and acute myocardial infarction. The researchers discovered that noise exposure and shift work increase the risk of cerebrovascular disease. However, exposure to noise above 85 dB(A) increases the risks of cerebrovascular disease (Fujino, Iso and Tamakoshi, 2007). Men exposed to noise levels at or above 75 dB(A) had a higher risk of coronary heart disease than women at a paper mill exposed to 90 dB(A) over 65 years, with a higher risk of acute myocardial infarction deaths (Eriksson *et al*., 2021).

Morata, Dunn, Kretschmer, Lemasters and Keith (1993) carried out an experimental study among 50 exposed and 51 unexposed workers to noise and toluene, or 39 organic solvent mixtures. They found that solvent exposure had toxic effects on the auditory system. The adjusted relative risk estimates for noise and toluene were four times higher for the noise group, elevenfold higher for sound and methylbenzene, and fivefold higher for solvent mixtures. However, Daniell, Swan, McDaniel, Camp, Cohen and Stebbins (2006) conducted a study on 76 companies and undertook 983 personal noise dosimetry measurements and interviewed 1557 employees at the Washington State workers' compensation registry after identifying 10 companies in each of the eight industries selected to determine if companies had noise control measures in place. It was found that most companies give limited or no attention to noise controls and rely mainly on HPDs to prevent hearing loss, yet 38% of workers did not regularly utilize HPDs. In addition, the poor use of HPD was attributable to insufficient company efforts. The proportion of a worker's exposure to noise was one point fivefold to threefold greater when using the 3-dB NIOSH doubling rule.

Likewise, Fernandez, Jeffers, Lall, Liberman and Kujawa (2015) carried out a cochlear aging study to compare noise exposure that causes permanent synaptic damage without hair cell loss with noise exposure that does not cause synaptopathy or hair cell loss. Adult mice were exposed to 8 to 16 kHz, 100(91) dB SPL for 2 hours and evaluated between 1 hour and 20 months after exposure. The study revealed that interactions between noise and aging may require an acute synaptopathy, but a single

synaptopathic exposure might accelerate cochlear aging. Between 1972 and 1974, Pyykkö, Starck, Färkkilä, Hoikkala, Korhonen and Nurminen (1981) carried out longitudinal research on hearing loss among a group of lumberjack's employees to determine if exposure to chainsaw sound may increase the risk of NIHL. They found that lumberjacks with vibration-induced white finger (VWF) had NIHL above 10 dB compared to those without VWF when classified according to age. NIHL increased with duration of exposure to chainsaw sound. Advanced NIHL was caused by vibrations present in both disorders through a common mechanism that causes vasoconstriction in the digital blood vessels and cochlear as a result of sympathetic nervous system activity. Liberman, Epstein, Cleveland, Wang and Maison (2016) indicated that synapses in the middle of hair cells and cochlear nerve terminals degenerate, leading to difficulties in understanding speeches in noisy settings, which might lead to tinnitus or hyperacusis, while the Summating Potential/Action Potential (SP/AP) ratio may be useful in diagnosing unseen hearing loss.

Fransen, Topsakal, Hendrickx, van Laer, Huyghe, van Eyken, Lemkens, Hannula, Mäki-Torkko, Jensen, Demeester, Tropitzsch, Bonaconsa, Mazzoli, Espeso, Verbruggen, Huyghe, Huygen, Kunst, Manninen, Diaz-Lacava, Steffens, Wienker, Pyykkö, Cremers, Kremer, Dhooge, Stephens, Orzan, Pfister, Bille, Parving, Sorri, van de Heyning and van Camp (2008) collected nine subsamples from a total of 4,083 subjects between the age of 53 and 67 years utilising nine audiological centres across Europe. A multicenter study was organized to identify risk factors contributing to presbycusis impairment in order to determine the relationship involving unprotected exposure to chronic sound and the prevalence of NIHL. The study found a significant correlation between unprotected exposure to chronic sound and NIHL at frequencies above 1 kHz. In addition, smoking was associated with an increased risk of NIHL at 3, 4 and 6 kHz with dose-dependent effects (Mizoue, Miyamoto and Shimizu, 2003). Effects of smoking were higher when taking into account cardiovascular events. Moreover, tall persons had improved hearing on average, with noticeable results below the 2 kHz frequency. A greater BMI was related to NIHL at all frequencies. Furthermore, adequate liquor drinking is inversely linked to hearing loss. The study proposed a healthy lifestyle to protect against presbycusis (Fransen *et al*., 2008).

Leigh (2011) utilized primary and secondary data sources from literature to determine the maximum compensation claims between injuries and diseases. The data revealed that occupational injuries accounted for 77% of all compensation claims, while diseases accounted for 23%. Occupational injuries and diseases costs were as sizable as the cost of cancer. Less than 25% of these costs were covered by compensation, while members of society bear the burden. Sprinzl and Riechelmann (2010) carried out a PubMed literature search using the keywords "presbycusis/geriatric and hearing aids/cochlear implants/electric acoustic stimulation/middle ear implants" and "elderly and cochlear implants" to determine devices for treating presbycusis. Hearing aids (His) and cochlear implants (Cis) were the most commonly used devices for treating mild-to-severe presbycusis. Hearing aids were offered for the treatment of mild (26 to 40 dB) or moderate (41 to 55 dB) hearing loss, provided patients were appropriately fitted, eager, inspired, and used them. Depending on the type and severity of the hearing loss and the specific requirements of the patient, electric-acoustic stimulation and active middle ear implants may be viable treatments for presbycusis. Cis treatment for severe-profound presbycusis has been associated with positive quality of life (QoL) and speech perception. In several studies, QoL results exceeded the expectations of elderly patients.

Hoar, Blair, Holmes, Boysen, Robel, Hoover and Fraumeni Jr. (1986) consulted and reviewed literature on the adverse health effects of occupational exposure risk factors on fertility and related reproductive outcomes to determine an association between occupational exposure and female infertility. NIHL was associated with herbicide use, mainly phenoxyacetic acids. Pesticide exposure can cause miscarriages and ovarian toxicity, leading to infertility. Literature supported the hypothesis that women employed in occupationally exposed jobs have a higher risk of infertility despite a lack of conclusive data because most literature on female fertility focuses on menstrual cycle changes and pregnancy complications rather than occupational exposure and female infertility (Figa`-Talamanca, 2006). Sheiner, Sheiner, Hammel, Potashnik and Carel (2003), argue that infertility is largely influenced by psychological distress. Nurminen (1995) hypothesizes that occupational exposure to noise above 85 dB(A) over 8 hours and rotating schedules may have negative effects on birth weight and length of gestation or reproduction.

Moreover, shift work has been associated with early fetal loss. Despite little evidence, noise exposure and shift work are linked with menstrual disruption and anovulatory infertility. Venners, Wang, Chen, Wang, Chen, Guang, Huang, Ryan, O'Connor, Lasley, Overstreet, Wilcox and Xu (2004) assessed the health effects of paternal smoking on pregnancy loss in a prospective cohort study of 526 newlyweds, non-smoking female textile workers in China between 1996 and 1998. Despite inconsistent findings from prior research, paternal smoking was associated with spontaneous abortions. The study found a relationship between substantial paternal smoking and premature pregnancy loss due to maternal or paternal exposure. Pouryaghoub, Mehrdad and Mohammadi (2007) conducted cross-sectional research among 206 male smokers and non-smokers exposed to loud sounds above 85 dB(A) at a large food producing factory to study an association between smoking and NIHL. Smoking was linked with an increased risk of NIHL. It was proposed that more studies and follow-ups be conducted on smoking and noise exposure levels beyond 85 dB(A) to understand the underlying processes. Smokers who work in noisy environments must attend periodic awareness training on quitting smoking. Starck, Toppila and Pyykkö (1999) examined the health outcome of smoking cigarettes on hearing loss among 199 forest and 171 shipyard workers. The health effects of aging on hearing loss were corrected by Robinson's model for an audiologically screened population. It was reported that smoking, high blood pressure, and Raynaud's phenomenon, taken together, were linked with an increased risk of hearing loss. Smoking without any risk factors was not linked with sensorineural hearing loss (Starck, Pyykkö and Pekkarinen, 1999).

Karasek, Baker, Marxer, Ahlbom and Theorell (1981) carried out a case-control study to examine cardiovascular cerebrovascular deaths (CHD-CVD) among Swedish males and the association between particular job characteristics and consequent heart and circulatory disease. Hectic and psychologically demanding jobs were related to an increase in the prevalence of ischaemic heart disease or coronary artery disease symptoms and premature CHD-CVD death. The lower decision latitude (LDL), expressed as low intellectual discretion (LID) and low personal schedule freedom (LPSF), was linked with an increase in the prevalence of coronary artery disease. LID predict the development of coronary heart disease symptoms, while LPSF among workers with the minimum statutory education increases the risks of CHD-CVD death.

The association develops after controlling for confounders such as age, education, smoking, and overweight. Talbott, Helmkamp, Mathews, Kuller, Cottington and Redmond (1985) assessed hearing loss and blood pressure among 197 randomly selected males from the noisiest plant and 169 control subjects from a factory. The researchers discovered that exposure to noise can increase the risks of hearing loss and high blood pressure among men. Lie *et al*. (2016) reviewed 698 articles in full text and selected 187 for investigating the linkage between NIHL and age. Seven to twenty-one percent of hearing loss among workers was caused by occupational noise exposure. It was hard to differentiate between NIHL and presbycusis because the majority of hearing losses are age-related and men lose more hearing than women. Hearing may be affected by heredity, socio-economic status, and ethnicity, along with cigarette smoking, vibration, hypertension, diabetes mellitus, and hazardous substances. Hearing loss is associated with advancing age. Cruickshanks, Klein, Klein, Wiley, Nondahl and Tweed (1998) administered an audiometric test to 3753 participants between the ages of 48 and 92 years to find a correlation between sound and hearing loss. It was found that exposure to environmental noise contributed to the development of presbycusis.

Agrawal, Platz and Niparko (2008) performed an audiometric test on 5742 Americans between the ages of 20 and 69 to investigate the incidence of hearing loss. NIHL was reported to be prevalent among elderly people. The prevalence differs across racial or ethnic groups, and earlier longitudinal or follow-up studies have established an association between hearing loss and other identifiable risk factors. The findings suggested that prevention of hearing loss by adjusting risk factors and screening should begin in early adulthood. Chang, Jain, Wang and Chan (2003) administered a 24-hour ambulatory blood pressure test and a 16-hour noise exposure assessment on 20 automobile employees to determine an association between sound and the risk of systolic BP. Unprotected from occupational noise was linked with SBP. There was a substantial consistency of 16 plus or minus 6 mmHg in sleep time SBP between the two exposed groups, as well as a minimum rise of 1 mmHg in SBP for each 1 dB(A) increase in noise experienced at a 60-minute lag time during a normal workshift.

Gates *et al.* (1993) investigated an association between presbycusis, cardiovascular disease, and the hearing status of elderly patients in a cohort study of 1662 elderly men and women that were compared to a 30-year prevalence of cardiovascular disease. Hypertension and systolic blood pressure were associated with hearing thresholds in both men and women, whereas blood glucose levels were linked to a low pure-tone average in women. High-density lipoprotein levels were inversely linked to low-frequency hearing thresholds only in women. There was a smaller but statistically significant association between cardiovascular disease and hearing status among the elderly, which was greater among women than men at lower frequencies. Low frequency presbycusis was linked to microvascular disease, resulting in atrophy of the stria vascularis. van Kempen *et al.* (2002) investigated an association between community and industrial noise exposure and the prevalence of coronary artery disease and high blood pressure by conducting a systematic review. It was found that exposure to occupational and community noise was linked with the prevalence of heart and circulatory diseases. Likewise, being unprotected from traffic noise was associated with the prevalence of heart attacks and total coronary heart disease or coronary artery disease. The causal relationship relating to noise and coronary artery disease was lacking due to its limited exposure character, the adjustment of potential confounders, and the incidence of publication bias.

van Kempen and Babisch (2012) carried out a meta-analysis on 27 published observational studies in English, German, and Dutch between 1970 and 2010 to determine the link relating to exposure to transportation sounds and high blood pressure. Transportation noise exposure was linked to an escalated incidence of cardiovascular illness. Babisch *et al*. (2005) performed a hospital-based case–referent study comprising 1881 patients aged 20 to 69 years with confirmed cases of myocardial infarction and 2234 controls to identify a correlation between traffic sound exposure and the prevalence of heart and circulatory disease. Unprotected to chronic noise was linked with cardiovascular diseases. Bluhm *et al*. (2007) conducted population-based research on 667 subjects aged 19 to 80 years to find a link between residential transportation noise and the incidence of hypertension. Unprotected exposure to transportation sounds was linked with hypertension. Kleemola *et al*. (2000) performed a cohort study comprising of 20 179 Finnish males and females between the ages of 30 and 59 to determine if coffee drinking increased the risk of coronary heart disease. Coffee drinking was not related to an escalated prevalence of coronary

disease or death, despite a modest rise in coronary heart mortality in males. All factors contributing to excessive coffee consumption were largely accounted for by the effects of smoking and a higher serum cholesterol level. The prevalence of smoking and the mean serum cholesterol level increase as coffee consumption increases. Navas-Acien *et al*. (2007) conducted an observational study regarding lead and cardiovascular end points to establish if lead exposure escalates the incidence of hypertension, heart and circulatory disease, and pulse rate. Lead exposure was linked with an increased risk of hypertension but not cardiovascular disease or heart rate. Rosenlund *et al*. (2006) performed case-referent studies among Stockholm residents aged 45 to 70 years to evaluate if prolonged exposure to air pollution increased the risk of myocardial infarction. Prolonged exposure to outdoor and indoor air pollution was linked with cardiovascular and circulatory disease mortality.

Aboa-Éboulé *et al*. (2007) undertook a prospective longitudinal study comprising 972 males and females between the ages of 35 and 59 who resumed work after an initial heart attack to investigate if noise exposure increases the risk of coronary heart disease (CHD). Prolonged job strain after the first myocardial infarction was linked to an increase in the prevalence of persistent coronary heart disease. Kurth, Gaziano, Cook, Logroscino, Diener and Buring (2006) carried out a prospective cohort study involving 27,840 women aged 45 years and older to determine if active migraine with aura increases the risk of cardiovascular disease and angina pectoris. Active migraine with aura was linked with a higher incidence of major cardiovascular diseases, myocardial infarctions, ischemic strokes, death from ischemic cardiovascular diseases, coronary revascularization, and angina. There was no association between active migraine without aura and an increased risk of any cardiovascular event. Peter and Siegrist (2000) performed a cross-sectional study among 11,636 Dutch-employed men and women to assess the relationship between the effects of job demand control (JDC) and effort reward imbalance (ERI) models on employee well-being. ERI and job strain had an adverse effect on the well-being of men and women, as well as young and old individuals. Employee well-being was negatively impacted by high psychological and physical efforts and low rewards. Torre, Cruickshanks, Klein, Klein and Nondahl (2005) examined the association between cardiovascular disease variables and distortions of product otoacoustic emissions (DPOAE) among 1501 participants having complete cardiovascular and DPOAE from 1998 to 2000. Cardiovascular disease variables were associated with cochlear impairment.

The study provided data on a possible sex-specific link between CVD and DPOAEs in older adults. Kivimäki *et al*. (2006) conducted research among 83 014 participants to determine the link between work stress and relative risks of coronary heart disease. The findings revealed a 50% CHD rate among workers with work stress.

Tomei, Fioravanti, Cerratti, Sancini, Tomao, Rosati, Vacca, Palitti, Di Famiani, Giubilati, De Sio and Tomei (2010) carried out a meta-analysis on 18,658 occupationally exposed subjects to determine a link between chronic noise and increased risks of cardiovascular disease. There was a significant increase in dystolic blood pressure and diastolic blood pressure in highly exposed subjects compared with middle- to low-exposed subjects. However, the exposed had a higher heart rate than the unexposed. Moreover, incidence of high blood pressure and electrocardiogram aberration was greater among the exposed than the control. Ferraro, Taylor, Eisner, Gambaro, Rimm, Mukamal and Curhan (2013) conducted a cohort study among 45 748 men and 196 357 women without a history of ischemic heart disease and a cohort study among 242 105 health professionals in the USA to determine if exposure to noise increases the risk of CHD; 19 678 participants reported a history of kidney stones, while 16 838 incidents of CHD in men developed 24 years and 18 years after follow-up in women. A history of kidney stones was linked to an increased prevalence of ischaemic heart disease or coronary artery disease among females than males.

Schulte *et al*. (2007) carried out a conceptual model for an interrelationship between work, obesity, and industrial health and safety to highlight ethical, legal, and social issues linked to the whole consideration of obesity's role in industrial health and safety. Obesity remained the highest risk factor for work-related asthma and CVD. It can adjust workers' responses to work-related stress, their immune responses to chemical exposure, and their risk of disease due to job-related neurotoxins. The risk of obesity escalates with duration of exposure due to poor controls in the working environment. Liu and Tanaka (2002) carried out a case-control study comprising 260 men aged 40 and 79 years who were admitted to hospitals with acute myocardial infarction between 1996 and 1998 and 445 men without acute myocardial infarction to determine if overtime work increases the risk of acute myocardial infarction. Overtime work and insufficient sleep were linked with increased risks of acute myocardial infarction.

2.11. Noise monitoring techniques

Noise evaluation techniques including the procedures to be followed have been documented in detail elsewhere (Driscoll, Mikl and Burgess, 2009; Goelzer, Hansen and Sehrndt, 2001). There are two techniques used to evaluate noise, viz: zonal (area) and personal noise surveys (Malchaire and Piette, 1997). The zonal method involves locating integrating SLM at a particular position near the noise source. It is used to measure noise above 80dB(A) to identify types of HPD to be used and list noise sources for appropriate control (Driscoll, Mikl and Burgess, 2009). The microphone is positioned 1.500 meter above floor level for standing person or 0.800 meter above seat for seated person and 1 meter away from any reflecting surfaces. A windshield is used to protect the microphone against wind to avoid sampling errors. This technique offers the most precise area noise exposure results with limited or no hindrance to frequency response of microphone. However, findings do not accurately reflect workers' exposure. The zonal method is the most efficient method for noise surveys (Rockwell, 1983). The duration per sample point must be elongated to be representative of the whole tasks or each task. If the source is constant, a period of 30 seconds to one minute may be sufficient. A map should be drawn and each contour line must demonstrate the noise levels around one source drawn manually (Neitzel, Andersson and Andersson, 2016).

A dosimeter is used to measure worker's exposure to noise of varying sound or workers who do not work in a fixed workstation (Anthony, 2016; Driscoll, Mikl and Burgess, 2009). The dosimeter is worn on the worker body for part or whole day to measure sound to integrate to a period of time (ISO, 2009; ISO, 1999). The microphone is positioned at mid-top of operator's shoulder or in operator's hearing zone following manufacturer specifications (Driscoll, Mikl and Burgess, 2009). The time for each measurement duration must be long enough to be representative of sound of each or all task(s) performed. A number of studies have revealed that this technique minimises errors in sampling and is regarded as the standard method (Erlandsson, Hakansson, Iversson and Nilsson, 1979; Shackleton and Piney, 1984). There are two methods used for selecting samples to monitor, namely, worst-case sampling and random sampling. Worst-case sampling is used when not all individuals can be sampled, selecting the maximum risk or worst-case employees (Malchaire and Piette, 1997).

This involves identifying an individual through careful observation during a walk-through survey. This includes proximity to the noise source, time spent near the process and movement patterns within the workplace. Differences in worker habits may also have an influence on the levels of exposure even when performing similar task. To identify similar exposure groups with same job functions and exposure to similar sound sources require observation and interviewing employees (Malchaire and Piette, 1997). At least three individuals from a group of four or more employees can be nominated for evaluation. Three dosimeter samples are proposed to establish peak or worst-case representation of noise exposure that any employee within a comparable exposure group may generally experience. This strategy is usually the most suitable to ensure regulatory compliance (Malchaire and Piette, 1997).

2.11.1. Random sampling of homogeneous exposure group (HEG)

The OESSM or BS EN 689 can be used to determine the total dosimeters necessary to detect maximum 20% of noise exposed individuals within 95% level of certainty (Leidel, Busch and Lynch, 1977). The total samples per employee is determined to maximise likelihood of encountering loudest circumstances (Malchaire and Piette, 1997). Five workers can be sampled to achieve a confidence level of 90% and one sampling period occurs at 0.33 on most occasions with the maximum sound level (Leidel, Busch and Lynch, 1977; Malchaire and Piette, 1997). The OESSM technical manual Appendix A gives the number of employees to sample in order to be 95% or 90% sure that one of the workers from the 20% of the homogeneous exposure group would be most exposed. If the maximum risk employees cannot be selected for worst-case monitoring, then employees will be grouped according to the similarity of exposures and a sample from each group must be selected randomly for monitoring. Random selection of monitoring periods during observational intervals is recommended for obtaining representative samples. At least 10 number of measurement points must be taken and confirmed that no systematic, special cause, unpredictable, and signal variation among the measured sound levels. The monitoring will be adequate to foresee exposures within a 5% confidence level (Malchaire and Piette, 1997).

2.11.2. Statistical Monitoring

When it is difficult to determine representative sampling, statistical evaluation may be employed. It is a preferred method for employees whose job activities or categorizations include highly variable work schedule, exposed to extremely variable sound sources or random movement through the workday (Malchaire and Piette, 1997). The purpose of statistical sample is to obtain adequate noise exposure information to make cognisant judgements concerning risk assessment. It is suggested that the selection must comprise of workers with daily noise exposure beyond 85 dB(A) on 0.05 of their shift, approximately 13 days in 365 days. When using statistical monitoring and it is vital to keep randomness in the approach when choosing workers to be sampled (Malchaire and Piette, 1997).

2.11.3. Guidelines for occupational noise exposure

Occupational noise exposure does not cause death but result in serious morbidity such as deafness (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004; Nelson *et al*., 2005). Occupational noise is responsible for 16% of deafness with a higher proportion in males (22%) than females (11%) because of variations in occupational classifications, economic sectors of employment and working life (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004). Almost 89% of the total burden of hearing loss are in the age between 15 to 59 years with 11% remaining in ages beyond 60 years (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004).The South African Department of Labour [Noise Induced Hearing Loss Regulations, 2003 promulgated under the Occupational health and Safety Act, 1993 (Act 85 of 1993)] order that, all employees who are exposed to noise levels at or above the 85 dB(A) 8-hour time-weighted avarage (TWA) to undergo medical surveillance (Noise Induced Hearing Loss Regulations, 2003; NIOSH,1998). According to Circular Instruction Number 171, NIHL is compensable at or above 10% percentage loss of hearing. This is usually calculated from the baseline audiogram accordance to annexure E of SANS 10083 and takes into account the hearing loss at frequencies of 0.5, 1, 2, 3 and 4 KHz (SANS, 2013; South African Department of Labour, Circular Instruction No. 171, 2001).

2.12. Conclusion

The OELs may not be effective in preventing carcinogens, asthmagens or mutagenic exposure (Occupational health clinic for Ontario workers, 2012). The OELs have some limitations in preventing occupational diseases. Compliance with OELs neither 85dB(A) noise rating limit will not protect workers against exposure to sensitisers or hazardous noise (Themann and Masterson, 2019). Workers develop cancer and NIHL despite compliance with the limits due to non-adherence to RPD and HPD. Kauppinen, Toikkanen, Pedersen, Young, Ahrens, Boffetta, Hansen, Kromhout, Maqueda Blasco, Mirabelli, de la Orden-Rivera, Pannett, Plato, Savela, Vincent and Kogevinas (2000) reported that about 22 million employees in Europe are unprotected from carcinogens. Exposure to wood dust between 0.1 to 2.7 mg/m^3 can increase the risk of respiratory symptoms or pulmonary functions while exposure above 0.25 mg/m^3 can cause dust to accumulate in the lungs (Occupational health clinic for Ontario workers, 2012). Some substances can cause hearing loss alone or either in conjunction with noise exposure. These substances are known as ototoxins or neurotoxic and can affect the cochlea and the auditory neurological pathways (NIOSH, 1989). The Occupational Exposure limits (OELs) have not been adjusted to reflect the risks of hearing loss and OEL must be used with caution when using OEL for substances with ototoxic potential. In addition, the risks are likely to be higher if there is exposure to multiple ototoxicants and combination of exposure to noise and ototoxicants can have an additive or probably synergistic effect on the risk of hearing loss. Hearing loss prevention programs should consider including ototoxins exposure on the management program (NIOSH 1989). NIOSH estimated 40 years work life of developing hearing loss to employees over 8 hours' day or 40 hours' week with 1 in 12 workers (8%) developing NIHL from exposure to 85dB(A) NIOSH REL and 1 in 4 workers (25%) developing NIHL from exposure to 90dB(A) OSHA PEL (Anthony, 2016; NIOSH, 1998). Research are needed to be undertaken to determine the possible roles of chemicals in the development of NIHL or cancer in the female reproductive system (NIOSH, 1998; Driscoll, Milk and Burgess, 2009; Weiderpass and Labrèche, 2012). The main aims are to inform health policies to establish standards to reduce inequalities in survival rates (Coleman, Forman, Bryant, Butler, Rachet, Maringe, Nur, Tracey, Coory, Hatcher, McGahan, Turner, Marrett, Gjerstorff, Johannesen, Adolfsson, Lambe, Lawrence, Meechan, Morris, Middleton, Steward and Richards, 2011). Lifestyle factors contribute to development of hearing loss, cancer as well as infertility.

2.13. References

Aboa-Eboulé, C., Brisson, C., Maunsell, E., Mâsse, B., Bourbonnais, R., Vézina, M., Milot, A., Théroux, P. and Dagenais, G. R. (2007) Job strain and risk of acute recurrent coronary heart disease events. *Journal of the American Medical Association* **298**(14):1652-1660. DOI:10.1001/jama.298.14.1652.

Adeoye, O.A., Adeomi, A.A., Abodunrin, O.L., Olugbenga-Bello, A.I. and Abdulsalam, S.T. (2015) Awareness of Occupational hazards and health problems among sawmill workers in Osun state, Nigeria. *International Journal of Research* **2**(1):1-14.

Adhikari, S. and Ozarska, B. (2018) Minimizing environmental impacts of timber products through the production process from sawmill to final products. *Environmental Systems Research* **7**(1):1-6.

Agbana, B.E., Joshua, A.O., Daikwo, M.A. and Metiboba, L.O. (2016) Knowledge of occupational hazards among sawmill workers in Kwara State, Nigeria. *Niger Postgraduate Medical Journal* **23**: 25-32.

Agrawal, Y., Platz, E.A. and Niparko, J.K. (2008) Prevalence of hearing loss and differences by demographic characteristics among US adults: data from the National Health and Nutrition Examination Survey, 1999-2004. *Archives of Internal Medicine* **168**(14):1522-1530. DOI:10.1001/archinte.168.14.1522.

American Industrial Hygiene Association (AIHA). (2006) *A strategy for assessing and managing occupational exposures*. 3rd ed. Fairfax, VA: AIHA Press

Ahmed, H.O., Dennis, J.H., Badran, O., Ismail, M., Ballal, S.G., Ashoor, A. and Jerwood, D. (2001) Occupational noise exposure and hearing loss of workers in two plants in eastern Saudi Arabia. *Annals of Occupational Hygiene* **45**(5):371-380.

Aizenberg, V., Grinshpun, S.A., Willeke, K., Smith, J. and Baron, P. A. (2000) Performance characteristics of the button personal inhalable aerosol sampler. *American Industrial Hygiene Association Journal* **61**:398–404.

Alshuaib, W. B., Al-Kandari, J. M. and Hasan, S. M. (2015) *Classification of hearing loss. In (ed.), Update on Hearing Loss*. London: IntechOpen.

Akbari, J. Safari, S., Kaze, M., Noumoradi, H., Mahaki, B. and Dehghan, H. (2018) The impact of hearing protection devices (HPDs) on blood pressure in workers Exposed to Noise: A cross-sectional study in a textile industry. *Iranian Journal of Health, Safety and Environment* **5**(3): 1066-1071.

Alberti, P.W. (2006) *The anatomy and physiology of the ear and hearing*. Available from:https://asram.in/asram/study%20materials/files/14-04-2020/Ear%20Mohanty%20Sir.pdf. (Accessed on 15 February 2021).

Alfredsson, L., Hammar, N. and Hogstedt, C. (1993) Incidence of myocardial infarction and mortality from specific causes among bus drivers in Sweden. *International Journal of Epidemiology* **22**(1):57-61. DOI:10.1093/ije/22.1.57.

Alwis, K.U. (1998) *Occupational exposure to wood dust*. (*P.h.D Thesis*). New South Wales, Australia: University of Sydney.

Alwis, K.U., Mandryk, J. and Hocking, A.D. (1999) Exposure to biohazards in wood dust: Bacteria, fungi, endotoxins, and (1→3)-βeta-D-glucans. *Applied Occupational and Environmental Hygiene* **14**(9): 598–608. DOI:10.1080/104732299302404.

American Conference of Governmental Industrial Hygienists (ACGIH). (1995) *Threshold limit values for chemical substances and physical Agents and the biological Exposure Indices.* Cincinnati, Ohio: ACGIH.

American Conference of Governmental Industrial Hygienists (ACGIH). (2012) *Threshold limit values (TLVs) for chemical substances and physical agents and the biological exposure indices (BEIs).* Cincinnati, Ohio: ACGIH.

American Conference of Governmental Industrial Hygienists (ACGIH). (2005a) *TLVs and BEIs based on the documentations of the TLV for chemical substances and physical agents and biological exposure indices.* Cincinnati, Ohio: ACGIH

American Conference of Governmental Industrial Hygienists (ACGIH). (2005) *Documentation of the threshold limit values and biological exposure indices – wood dusts.* 7th ed. Cincinnati, Ohio: ACGIH.

Anthony, R.T. (2016) *Noise monitoring*. Available from: https://hwc.public-health.uiowa.edu/wp-content/uploads/2016/05/3D-Renee-Anthony-Conducting-Noise-Exposure-Assessments.pdf.(Accessed on 22 December 2020).

Aremu, A.O., Aremu, A.S. and Olukanni, D.O. (2015) Assessment of noise pollution from sawmill activities in Ilorin, Nigeria. *Nigerian Journal of Technology* **34**(1):72-79.

Babisch, W. (1998) Epidemiological studies of the cardiovascular effects of noise – a critical appraisal. *Noise and Health* **1**:24-39.

Babisch, W. (2002) The noise/stress concept, risk assessment and research needs. *Noise Health* **4**:1–11.

Babisch, W., Beule, B., Schust, M., Kersten, N. and Ising, H. (2005) Traffic Noise and Risk of Myocardial Infarction. *Epidemiology* **16**(1):33–40.

Babisch, W., Ising, H., Gallacher, J.E.J., Sweetnam, P.M. and Elwood, P.C. (1999) Traffic noise and cardiovascular risk: The caerphilly and speedwell studies, 3rd phase-10-year follow up. *Archives of Environmental & Occupational Health* **54**:210–216.

Backé, E., Seidler, A., Latza, U., Rossnagel, K. and Schumann, B. (2012) The role of psychosocial stress at work for the development of cardiovascular diseases:A systematic review. *International Archives of Occupational and Environmental Health* **85**:67–79.

Barregard, L., Bonde, E. and Öhrström, E. (2009) Risk of hypertension from exposure to road traffic noise in a population-based sample. *Occupational and Environmental Medicine* **66**(6):410-415. DOI:10.1136/oem.2008.042804.

Basner, M., Babisch, W., Davis, A., Brink, M., Clark, C., Janssen, S. and Stansfeld, S. (2014) Auditory and non-auditory effects of noise on health. *Lancet* **383**(9925):1325–1332. DOI:10.1016/S0140-6736(13)61613-X.

Blair, A., Saracci, R., Stewart, P.A., Hayes, R.B. and Shy, C. (1990) Epidemiologic evidence on the relationship between formaldehyde exposure and cancer. *Scandinavian Journal of Work Environmental and Health* **16**(6):381-393. DOI:10.5271/sjweh.1767.

Bluhm, G. L.,Berglind, N., Nordling, E. and Rosenlund, M. (2007) Road traffic noise and hypertension.*Journal of Occupational and Environmental Medicine* **64**(2):122–126.

Bøggild, H. and Knutsson, A. (1999) Shift work, risk factors and cardiovascular disease. *Scandinavian Journal of Work, Environment and Health* **25**(2):85-99.

Brown, J. S., Gordon, T., Price, O. and Asgharian, B. (2013) Thoracic and respirable particle definitions for human health risk assessment. *Part Fibre Toxicol* **10**:1-12.

Buchan, R.M., Soderholm, S.C. and Tillery, M.I. (1986) Aerosol sampling efficiency of 37-mm filter cassettes. *American Industrial Hygiene Association Journal* **47**:825–831. DOI:10.1080/15298668691390728.

Buliga, V., Rongo, L.M.B. and Mamuya, S.H.D. (2017) Dust exposure and respiratory health symptoms among workers in small scale sunflower oil industry in Singida Tanzania. *International Journal of Research – Granthaayah* **5**(12): 452-463.

Bush, R.K., Portnoy, J.M., Saxon, A., Terr, A.I. and Wood, R.A. (2006) The medical effects of mold exposure. *Journal of Allergy and Clinical Immunology* **117**(2):326-333. DOI: 10.1016/j.jaci.2005.12.001.

Canadian Centre for Occupational Health and Safety (CCOHS). (2017) *Wood dust - health effects: OSH answers fact sheets.* Available from: https://www.ccohs.ca/oshanswers/chemicals/wood_dust.html.(Accessed on 20 December 2019).

Cancer Council Australia. (2019) *Wood product.* Available from: https://www.cancer.org.au/cancer-information/causes-and-prevention/workplace-cancer/wood-products.(Accessed on 22 February 2020).

Cantley, L.F., Galusha, D., Cullen, M.R., Dixon-Ernst, C., Rabinowitz, P.M. and Neitzel, R.L. (2015) Association between ambient noise exposure, hearing acuity, and risk of acute occupational injury. *Scandinavian Journal of Work, Environment and Health* **41**(1):75-83. DOI:10.5271/sjweh.3450.

Carney, R.M., Blumenthal, J.A., Freedland, K.E., Stein, P.K., Howells, W.B., Berkman, L.F., Watkins, L.L., Czajkowski, S.M., Hayano, J., Domitrovich, P.P. and Jaffe, A.S. (2005) Low heart rate variability and the effect of depression on post-myocardial infarction mortality.*Archives of Internal Medicine* **165**(13):1486-1491. DOI:10.1001/archinte.165.13.1486.

Canadian Centre for Occupational Health and Safety (CCOHS). (2022) *OSH answers fact sheets:How do particulates enter the respiratory system*?. Available from: https://www.ccohs.ca/oshanswers/chemicals/how_do.html. (Accessed on 22 February 2022).

Cesana, G., Sega, R., Ferrario, M., Chiodini, P., Corrao, G. and Mancia, G. (2003) Job strain and blood pressure in employed men and women: a pooled analysis of four Northern Italian population samples. *Psychosomatic Medicine* **65**(4):558-63. DOI: 10.1097/01.psy.0000041473.03828.67.

Chang, T.Y., Hwang, B.F., Liu, C.S., Chen, R.Y., Wang, V.S., Bao, B.Y. and Lai, J.S. (2013) Occupational noise exposure and incident hypertension in men: a prospective cohort study. *American Journal of Epidemiology* **177**(1):818–825.

Chang, T.Y., Jain, R.M., Wang, C.S. and Chan, C.C. (2003) Effects of occupational noise exposure on blood pressure. *Journal of Occupational and Environmental Medicine* **45**(12):1289-1296. DOI: 10.1097/01.jom.0000100003.59731.3d.

Chamber, P. and Nunes, E. (2016) Work-related asthma among workers in the wood processing industry: A review. *Journal of Current Allergy and Clinical Immunology* **29**(2):110-117.

Cherrie, J.W., Hutchings, S., Ng, M.G., Mistry, R., Corden, C., Lamb, J., Jime´nez, A.S., Shafrir, A., Sobey, M., Tongeren, M.V. and Rushton, L. (2017) Prioritising action on occupational carcinogens in Europe: A socioeconomic and health impact assessment. *British Journal of Cancer* **117**:274–281.

Chobanian, A.V., Bakris, G.L., Black, H.R., Cushman, W.C., Green, L.A., Izzo, J.L. Jr, Jones, D.W., Materson, B.J., Oparil, S., Wright, J.T. Jr and Roccella, E.J. (2003) Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. *Hypertension* **42**(6):1206-1252.

Clark, C. and Stansfeld, S. A. (2007) The Effect of Transportation Noise on Health and Cognitive Development: A Review of Recent Evidence. *International Journal of Comparative Psychology* **20**(2):145-158.

Clark, W.W. and Bohne, B.A. (1999) Effects of Noise on Hearing. *Journal of the American Medical Associations (JAMA)* **281**(17):1658–1659.

Coleman, M.P., Forman, D., Bryant, H., Butler, J., Rachet, B., Maringe, C., Nur, U., Tracey, E., Coory, M., Hatcher, J., McGahan, C. E., Turner, D., Marrett, L., Gjerstorff, M.L., Johannesen, T.B., Adolfsson, J., Lambe, M., Lawrence, G., Meechan, D., Morris, E.J., Middleton, R., Steward, J. and Richards, M.A. (2011) Cancer survival in Australia, Canada, Denmark, Norway, Sweden, and the UK, 1995–2007 (The International Cancer Benchmarking Partnership): An analysis of population-based cancer registry data. *Lancet* **377**:127–138. DOI:10.1016/S0140-6736(10)62231-3.

Comite' Europe'e' de Normalisation (CEN). (1993) *EN 481:1993. Workplace atmosphere-size fraction definition for measurement of airborne particulates in the workplace*. Brussels:CEN.

Concha-Barrientos, M., Campbell-Lendrum, D. and Steenland, K. (2004) *Occupational noise: Assessing the burden of disease from work-related hearing impairment at national and local levels*. Geneva: WHO.

Cruickshanks, K.J., Klein, R., Klein, B.E., Wiley, T.L., Nondahl, D.M. and Tweed, T.S. (1998) Cigarette smoking and hearing loss: the epidemiology of hearing loss study. *Journal of the American Medical Association* **279**(21):1715-1719. DOI:10.1001/jama.279.21.1715.

Dalen, J.E., Alpert, J.S., Goldberg, R.J. and Weinstein, R.S. (2014) The epidemic of the 20(th) century: coronary heart disease. *The American Journal of Medicine* **127**(9):807-812. DOI: 10.1016/j.amjmed.2014.04.015.

Dalton, D.S., Cruickshanks, K. J., Klein, B. E. K., Klein, R., Wiley, T. L. and Nondahl, D. M. (2003) The impact of hearing loss on quality of life in older adults. *The Gerontologist* **43**(5):661–668.

Daniell, W.E., Swan, S.S., McDaniel, M.M., Camp, J.E., Cohen, M.A. and Stebbins, J.G. (2006) Noise exposure and hearing loss prevention programmes after 20 years of regulations in the United States. *Occupational and Environmental Medicine* **63**(5):343-351.DOI:10.1136/oem.2005.024588.

Davies, B. and Henderson, J. (2009a) *W501 student manual measurements of hazardous chemical substances*. Australia: University of Wollongong.

Davies, B. and Henderson, J. (2009b) *W505 student manual control of hazardous substances*. Australia: University of Wollongong

Davies, H. and Kamp, I.V. (2012) Noise and cardiovascular disease: a review of the literature 2008-2011. *Noise Health* **14**(61):287-291. DOI:10.4103/1463-1741.104895.

Davies, H.W. (2002) *Exposure to occupational noise and risk of cardiovascular disease: A retrospective cohort study. (Ph.D. Thesis)*. Columbia: University of British Columbia.

Davies, H.W., Teschke, K. and Demers, P.A. (1999a) A field comparison of inhalable and thoracic size selective sampling techniques. *Annals of Occupational Hygiene* **43**: 381–392.

Davies, H.W., Teschke, K., Kennedy, S.M., Hodgson, M.R. and Demers, A.P. (2009b) A retrospective assessment of occupational noise exposures for a longitudinal epidemiological study. *Occupational and Environmental Medicine* **66**(6):388-394.

Davies, H.W., Teschke, K., Kennedy, S.M., Hodgson, M.R., Hertzman, C. and Demers, P.A. (2005) Occupational Exposure to Noise and Mortality from Acute Myocardial Infarction. *Epidemiology* **16**(1):25-32.

Davies, H.W., Vlaanderen, J.J., Henderson, S.B. and Brauer, M. (2009c) Correlation between co-exposures to noise and air pollution from traffic sources. *Occupational and Environmental Medicine* **66**(5):347-350.

de Backer, G., Ambrosioni, E., Borch-Johnsen, K., Brotons, C., Cifkova, R., Dallongeville, J., Ebrahim, S., Faergeman, O., Graham, I., Mancia, G., Cats, V.M., Orth-Gomér, K., Perk, J., Pyörälä, K., Rodicio, J.L., Sans, S., Sansoy, V., Sechtem, U., Silber, S., Thomsen, T., Wood, D., Albus, C., Bages, N., Burell, G., Conroy, R, Deter, H.C., Hermann-Lingen, C., Humphries, S., Fitzgerald, A., Oldenburg, B., Schneiderman, N., Uutela, A., Williams, R., Yarnell, J., Priori, S.G., Garcia, M.A.A., Blanc, J., Budaj, A., Cowie, M., Dean, V., Deckers, J., Burgos, E.F., Lekakis, J, Lindahl, B., Mazzotta, G., McGregor, K., Morais, J., Oto, A., Smiseth, O., Trappe, H., Budaj, A., Agardh, C., Bassand, J., Deckers, J., Godycki-Cwirko, M., Heagerty, A., Heine, R., Home, P., Priori, S., Puska, P., Rayner, M., Rosengren, A., Sammut, M., Shepherd, J., Siegrist, J., Simoons, M., Tendera, M. and Zanchetti, A. (2003) European guidelines on cardiovascular disease prevention in clinical practice: Third Joint Task Force of European and other Societies on Cardiovascular Disease Prevention in Clinical Practice (constituted by representatives of eight societies and by invited experts). *European Heart Journal* **24**(17):1601–1610.

Dehghan, M. , Mente, A., Zhang, X., Swaminathan, S., Li, W., Mohan, V., Iqbal, R., Kumar, R., Wentzel-Viljoen, E., Rosengren, A., Amma, L.I., Avezum, A., Chifamba, J., Diaz, R., Khatib, R., Lear, S., Lopez-Jaramillo, P., Liu, X., Gupta, R., Mohammadifard, N., Gao, N., Oguz, A., Ramli, A.S., Seron, P., Sun, Y., Szuba, A., Tsolekile, L., Wielgosz, A., Yusuf, R., Yusufali, A.H., Teo, K., Rangarajan, S., Dagenais, G., Bangdiwala, S. I., Islam, S., Anand, S.S. and Yusuf, S. (2017) Associations of fats and carbohydrate intake with cardiovascular disease and mortality in 18 countries from five continents (PURE): A prospective cohort study. *Lancet* **390**(10107):2050-2062. DOI:10.1016/S0140-6736(17)32252-3.

Demers, P.A. and Boffetta, P. (1998) *Cancer risk from occupational exposure to wood dust: A pooled analysis of epidemiological studies*. *IARC Technical Report No. 30*. Lyon: IARC.

Demers, P.A., Kogevinas, M., Boffetta, P., Leclerc, A., Luce, D., Gérin, M., Battista, G., Belli, S., Bolm-Audorf, U., Brinton, L.A., Colin, D., Comba, P., Hardell, L., Hayes, R.B., Magnani, C., Merler, E., Morcet, J.F., Preston-Martin, S., Matos, E., Rodella, S., Vaughan, T.L., Zheng, W. and Vainio, H. (1995a) Wood dust and sino-nasal cancer: Pooled reanalysis of twelve case-control studies. *American Journal of Industrial Medicine* **28**(2):151-166.

Demers, P.A., Boffetta, P., Kogevinas, M., Blair, A., Miller, B.A., Robinson, C.F., Roscoe, R.J., Winter, P.D., Colin, D., Matos, E. and Vainio, H. (1995b) Pooled reanalysis of cancer mortality among five cohorts of workers in wood-related industries. *Scandinavian Journal of Work, Environment and Health* **21**(3):179-190.

Dost, W. A. (1974a) Noise levels in soft wood lumber mills. *Forest Products Journal* **24**:25–27.

Dost, W. A. (1974b) Sawmill noise at the operating level. *Forest Products Journal* **24**:13–17.

Department of Environmental Affairs and Development Planning (DEADP). (n.d.) *Managing air quality at the sawmills.* Available from: https://www.westerncape.gov.za/assets/departments/environmental-affairs-development-planning/managing-air-quality-at-sawmills.pdf.(Accessed on 26 October 2019).

Diwe, K.C., Duru, C.B., Iwu, A.C., Merenu, I.A., Uwakwe, K.A., Oluoha, U.R., Ogunniyan, T.B., Madubueze, U.C. and Ohale, I. (2016) Occupational hazards, safety and hygienic practices among timber workers in a South Eastern State, Nigeria. *Occupational Diseases and Environmental Medicine* **4**(1): 63-71.

National Institute for Occupational Safety and Health (NIOSH). (1998) *Criteria for a recommended standard for Occupational noise exposure revised criteria.* Cincinnati, Ohio: NIOSH.

Department of work and pension. (2017) *Nasal carcinoma and occupational exposure to wood dust: Report by the Industrial Injuries Advisory Council in accordance with Section 171 of the Social Security Administration Act 1992 reviewing nasal carcinoma and occupational exposure to wood dust*. UK: APS Group.

Dhere, A.M., Pawar, C.B., Patil, D.A. and Pawar, J.A. (2009) Noise induced hearing loss (NIHL) in saw mill and printing press workers in Akluj Town of Solapur district. *Journal of Environmental Science and Engineering* **51**(3):187-190.

Dietz, A., Ramroth, H., Urban, T., Ahrens, W. and Becher, H. (2004) Exposure to cement dust, related occupational groups and laryngeal cancer risk: results of a population based case-control study. *International Journal of Cancer* **108**(6):907-911.

Douwes, J., McLean, D., Slater, T. and Pearce, N. (2001) Asthma and other respiratory symptoms in New Zealand pine processing sawmill workers. *American Journal of Industrial Medicine* **8**:608–615.

Douwes, J., Thorne, P., Pearce, N. and Heederik, D. (2003) Bioaerosol health effects and exposure assessment: progress and prospects. *The Annals of Occupational Hygiene* **47**(3): 187–200.

Dratva, J., Phuleria, H.C., Foraster, M., Gaspoz, J.M., Keidel, D., Künzli, N., Liu, L.J., Pons, M., Zemp, E., Gerbase, M.W. and Schindler, C. (2012) Transportation noise and blood pressure in a population-based sample of adults. *Environmental Health Perspectives* **120**(1):50-505. DOI:10.1289/ehp.1103448.

Driscoll, D.P., Mikl, K. and Burgess, M. (2009) *W503 student manual noise: measurement and its effects.* Australia: University of Wollongong.

Dzhambov, A.M. and Dimitrova, D.D. (2016) Occupational noise and ischemic heart disease: A systematic review. *Noise Health* **18**(83): 167–177.

Edmond, O.A., Oluniyi, A.A. and Bamidele, O.O. (2014) Re-Engineering Technical Vocational Education and Training (TVET) Towards safety practice skill needs of sawmill workers against workplace hazards in Nigeria. *Journal of Education and Practice* **5**(7):142-149.

Ekpu, V.W. and Brown, K. (2015) The economic impact of smoking and of reducing smoking prevalence: Review of evidence. *Tobacco Use Insights* **8**(1):1–35. DOI:10.4137/TUI.S15628.

Engdahl, B and Tambs, K. (2010) Occupation and the risk of hearing impairment – results from the Nord-Trøndelag study on hearing loss. *Scandinavian Journal of Work, Environment and Health* **36**(3):250–257.

Eriksson, H. (2019) *Work-related cardiovascular disease*. *(Ph.D. Thesis*). University of Gothenburg, Sahlgrenska Academy.

Eriksson, H.P., Andersson, E., Schiöler, L., Söderberg, M, Sjöström, M., Rosengren, A. and Torén, K. (2018) Longitudinal study of occupational noise exposure and joint effects with job strain and risk for coronary heart disease and stroke in Swedish men. *British Medical Journal (BMJ*) **8**(4):e019160. DOI:10.1136/bmjopen-2017-019160.

Eriksson, H.P., Söderberg, M., Neitzel, R.L., Torén, K. and Andersson, E. (2021) Cardiovascular mortality in a Swedish cohort of female industrial workers exposed to noise and shift work. *Int Arch Occup Environ Health* **94**(2):285-293.

Erlandsson, B., Hakansson, H., Iversson, A. and Nilsson, P. (1979) Comparison between stationary and personal noise dose measuring systems.*Ada Otolaryngologica Supplement* **360**:105-108.

European Agency for Safety and Health at Work. (2005) *Risk observatory: Noise in Figures*. Luxembourg: European Agency for Safety and Health at Work. Available from: https://osha.europa.eu/en/publications/report-noise-figures.(Accessed on 14 February 2021).

FAO. (2010) *Global forest resources assessment 2000*. Rome, Italy.

Fausti, S.A., Wilmington, D.J., Helt, M.A.P.V., Helt, W.J. and Konrad-Martin, D. (2005) Hearing health and care: The need for improved hearing loss prevention and hearing conservation practices. *Journal of Rehabilitation Research and Development* **4**(2):45–62. DOI:10.1682/jrrd.2005.02.0039.

Fernandez, K.A., Jeffers, P.W., Lall, K., Liberman, M.C. and Kujawa, S.G. (2015) Aging after noise exposure: acceleration of cochlear synaptopathy in "recovered" ears. *Journal of Neuroscience* **35**(19):7509-7520.DOI:10.1523/JNEUROSCI.5138-14.2015.

Ferraro, P.M., Taylor, E.N., Eisner, B.H., Gambaro, G., Rimm, E.B., Mukamal, K.J. and Curhan, G.C. (2013) History of kidney stones and the risk of coronary heart disease. *Journal of the American Medical Association* **310**(4):408-415.

Figa`-Talamanca, I. (2006) Occupational risk factors and reproductive health of women. *Occupational Medicine* **56**:521–531. DOI:10.1093/occmed/kql114.

Fransen, E., Topsakal, V., Hendrickx, J., Van Laer, L., Huyghe, J.R., Van Eyken, E., Lemkens, N., Hannula, S., Mäki-Torkko, E., Jensen, M., Demeester, K., Tropitzsch, A., Bonaconsa, A., Mazzoli, M., Espeso, A., Verbruggen, K., Huyghe, J., Huygen, P.L.M., Kunst, S., Manninen, M., Diaz-Lacava, A., Steffens, M., Wienker, T.F., Pyykkö, I., Cremers, C.W.R.J., Kremer, H., Dhooge, I., Stephens, D., Orzan, E., Pfister, M., Bille, M., Parving, A., Sorri, M., Van de Heyning, P. and Van Camp, G. (2008) Occupational noise, smoking, and a high body mass index are risk factors for age-related hearing impairment and moderate alcohol consumption is protective: A European population-

based multicenter study. *Journal of the Association for Reseach in Otolaryngology* **9**(3): 264-263.DOI:10.1007/s10162-008-0123-1.

Frost, P., Kolstad, H.A. and Bonde, J.P. (2009) Shift work and the risk of ischemic heart disease - a systematic review of the epidemiologic evidence. *Scandinavian Journal of Work, Environment and Health* **35**(3):163-179. DOI:10.5271/sjweh.1319.

Fujino, Y., Iso, H. and Tamakoshi, A. (2007) JACC study group: A prospective cohort study of perceived noise exposure at work and cerebrovascular diseases among male workers in Japan. *Journal of Occupational Health* **49**:382–388.

Gan, W.Q., Davies, H.W. and Demers, P.A. (2011) Exposure to occupational noise and cardiovascular disease in the United States: The National Health and Nutrition Examination Survey 1999–2004. *Journal of Occupational and Environmental Medicine* **68**:183-190.

Gates, G.A., Cobb, J.L., D'Agostino, R.B. and Wolf, P.A. (1993) The relation of hearing in the elderly to the presence of cardiovascular disease and cardiovascular risk factors. *Archives of Otolaryngology - Head and Neck Surgery* **119**(2):156-161.

Gaziano, T.A., Bitton, A., Anand, S., Abrahams-Gessel, S. and Murphy, A. (2010) Growing epidemic of coronary heart disease in low- and middle-income countries. *Current Problems in Cardiology* **35**(2):72-115. DOI: 10.1016/j.cpcardiol.2009.10.002.

Getschman, B.J. (2013) *Evaluation of the validity of the inhalable and "total" dust concentration ratio. (Master Thesis).* Lowa: University of Lowa.

Glass, D.C. (1980) *Stress, Behaviour Patterns, and Coronary Disease*. In: Blankstein, K.R., Pliner, P. and Polivy J. (Eds.).*Assessment and Modification of Emotional Behavior. Advances in the Study of Communication and Affect.* Boston, MA: Springer.

Goelzer, B., Hansen, C.H. and Sehrndt, G. (2001) *Occupational exposure to noise: evaluation, prevention and control*.Available from: https://edocs.tib.eu/files/e01fn02/346867487.pdf. Accessed on 4 April 2021).

Gopinath, B., Thiagalingam, A., Teber, E. and Mitchell, P. (2001) Exposure to workplace noise and the risk of cardiovascular disease events and mortality among older adults. *Preventive Medicine* **53**:390–394.

Gupta, S., Malhotra, V., Tripathi, Y. and Dev, P. (2017) Blood pressure variations in textile mill middle-aged male workers exposed to noise. *The Free Library*. Available from: https://www.thefreelibrary.com/Blood pressure variations in textile mill middle-aged male workers...-a0550390516. (Accessed on 5 January 2021).

Gustavsson, P., Jakobsson, R., Johansson, H., Lewin, F., Norell, S. and Rutkvist, L. (1998) Occupational exposures and squamous cell carcinoma of the oral cavity, pharynx, larynx, and oesophagus: A case-control study in Sweden. *Occupational and Environmental Medicine* **55**: 393–400.

Hagström, K. Lundholm, C., Eriksson, K. and Liljelind, I. (2008a) Variability and determinants of wood dust and resin acid exposure during wood pellet production: measurement strategies and bias in assessing exposure-response relationships. *Annals of Occupational Hygiene* **52**:685–694.

Hagström, K., Axelsson, S., Arvidsson, H., Bryngelsson, I.L., Lundholm, C. and Eriksson, K. (2008b) Exposure to wood dust, resin acids, and volatile organic compounds during production of wood pellets. *Journal of Occupational and Environmental Hygiene* **5**(5):296-304. DOI:10.1080/15459620801957225.

Hall, A.H., Teschke, K., Davies, H., Demers, P. and Marion, S. (2002) Exposure levels, determinants of softwood dust exposures in BC lumber mills, 1981-1997. *American Industrial Hygiene Association (AIHA) Journal* **63**:709-714.

Halperin, D. (2014) Environmental noise and sleep disturbances: A threat to health? *Sleep Science* **7**(4):209-212. DOI: 10.1016/j.slsci.2014.11.003.

Hansell, A.L., Blangiardo, M., Fortunato, L., Floud, S., de Hoogh, K., Fecht, D., Ghosh, R.E., Laszlo, H.E., Pearson, C., Beale, L., Beevers, S., Gulliver, J., Best, N., Richardson, S. and Elliott, P. (2013) Aircraft noise and cardiovascular disease near Heathrow airport in London: small area study. *British Medical Journal* **347**: 1136-5432. DOI:https://doi.org/10.1136/bmj.f5432.

Hansen, J. and Ølsen, J.H. (1995) Formaldehyde and cancer morbidity among male employees in Denmark. *Cancer Causes Control* **6**:354–360.

Harmse, L. (2000) Implementing an air monitoring programme. *Occupational Health South Africa* **6**(2):14-17.

Harbo, G., Grau, C., Bundgaard, T., Overgaard, M., Elbrønd, O., Søgaard, H., and Overgaard, J. (1997) Cancer of the nasal cavity and paranasal sinuses. A clinico-pathological study of 277 patients. *Acta Oncologica* **36**(1):45-50.

Harper, M. and Muller B.S. (2002) An evaluation of total and inhalable samplers for the collection of wood dust in three wood products industries. *Journal of Environmental Monitoring* **4**:648–656.

Haupt, C.M., Alte, D., Dörr, M., Robinson, D.M., Felix, S.B., John, U. and Völzke, H. (2008) The relation of exposure to shift work with atherosclerosis and myocardial infarction in a general population. *Atherosclerosis* **201**(1):205-211.

Hayes, R.B., Gerin, M., Raatgever, J.W. and de Bruyn, A. (1986) Wood-related occupations, wood dust exposure, and sinonasal cancer. *American Journal of Epidemiology* **124**(4):569–577.

Health and Safety Executive (HSE). (2006) *Monitoring strategies for toxic substances, HSG 173* .2nd ed. London: HSE.

Health and Safety Executive (HSE). (2020) *EH40/2005 workplace exposure limit*. London: TSO.

Health and Safety Executive. (2015) *MDHS 14/4: The general methods for sampling and gravimetric analysis of respirable, thoracic and inhalable aerosols.* UK: HSE.

Health Council of the Netherlands (HCN). (2000) *Hardwood and softwood dust: Evaluation of the carcinogenicity and genotoxicity.* The Hague: HCN.

Heckbert, S.R., Weiss, N.S., Koepsell, T.D., Lemaitre, R.N., Smith, N.L., Siscovick, D.S., Lin, D. and Psaty, B.M. (1997) Duration of estrogen replacement therapy in relation to the risk of incident myocardial infarction in postmenopausal women. *Archives of Internal Medicine* **157**(12):1330–1336.

Helzner, E.P., Cauley, J.A., Pratt, S.R., Wisniewski, S.R., Zmuda, J.M., Talbott, E.O., de Rekeneire, N., Harris, T.B., Rubin, S.M., Simonsick, E.M., Tylavsky, F.A. and Newman, A.B. (2005) Race and Sex Differences in Age-Related Hearing Loss: The Health, Aging and Body Composition Study. *Journal of the American Geriatrics Society* **53**(12):2119-2127.

Hernberg, S., Westerholm, P., Schultz-Larsen, K., Degerth, R., Kuosma, E., Englund, A., Engzell, U., Hansen, H.S. and Mutanen, P. (1983) Nasal and sinonasal cancer. Connection with occupational exposures in Denmark, Finland and Sweden. Scandinavian *Journal of Work, Environment and Health* **9**(4):315-326.

Hertzman, C., Teschke, K., Ostry, A., Hershler, R., Dimich-Ward, H., Kelly, S., Spinelli, J.J., Gallagher, R.P., McBride, M. and Marion, S.A. (1997) Mortality and cancer incidence among sawmill workers exposed to chlorophenate Wood Preservative. *American Journal of Public Health* **87**(1):71-79.

Hewett, P., Logan, P., Mulhausen, J., Ramachandran, G. and Banerjee, S. (2006) Rating exposure control using bayesian decision analysis. *journal of occupational and environmental Hygiene* **3**: 568–581.DOI:10.1080/15459620600914641.

Hildesheim, A., Dosemeci, M., Chan, C.C., Chen, C.J., Cheng, Y.J., Hsu, M.M., Chen, I.H., Mittl, B.F., Sun, B., Levine, P.H., Chen, J.Y., Brinton, L.A. and Yang, C.S. (2001) Occupational exposure to wood, formaldehyde, and solvents and risk of nasopharyngeal carcinoma. *Cancer Epidemiology, Biomarkers & Prevention Journal* **10**(11):1145-1153.

Hirst, A. (2010) *W201 Student manual basic principles in occupational hygiene.* UK: OHTA.

Hirst, A. (2009) *W507 Student manual health effects of hazardous substances.* UK: OHTA.

Hoar, S.K., Blair, A., Holmes, F.F., Boysen, C.D., Robel, R.J., Hoover, R. and Fraumeni, J.F., Jr. (1986) Agricultural herbicide use and risk of lymphoma and soft-tissue sarcoma. *Journal of the American Medical Association* **256**:1141–1147.

Hong, O.S., Kerr, M.J., Poling, G.L. and Dhar, S. (2013) Understanding and preventing noise-induced hearing loss. *Disease-a-Month* **59**(4):110-118.

Hubbard, R., Lewis, S., Richards, K., Johnston, I. and Britton, J. (1996) Occupational exposure to metal or wood dust and aetiology of cryptogenic fibrosing alveolitis. *Lancet* **347**(8997):284-289. DOI:10.1016/s0140-6736(96)90465-1.

International Agency for Research on Cancer (IARC). (1995a) *Wood dust. In wood dust and formaldehyde. IARC Monographs on the evaluation of carcinogenic risk of chemicals to humans, vol. 62.* Lyon, France: IARC.

International Agency for Research on Cancer (IARC). (1995b) *Wood dust and formaldehyde*. *IARC Monograph of evaluation of carcinogenic risks to humans* **62**: 1-405.

International Agency for Research on Cancer (IARC). (2012a) Working group on the evaluation of carcinogenic risks to humans: Chemical agents and related occupations. IARC Monogr. *Evaluating Carcinogenic Risks to Humunan* **100**(PtF):9-562.

International Agency for Research on Cancer (IARC). (2012b) *Chemical Agent and Related Occupations IARC monographs evaluation carcinogenic risks to humans.* 100F: PMID:183356-40. Lyon, France: IARC.

International Organization for Standardization (ISO). (1999) *Acoustics: Determination of occupational noise exposure and estimation of noise-induced hearing impairment.* Geneva: ISO.

International Organization for Standardization (ISO). (2009) *Acoustics: Determination of occupational noise exposure - Engineering method, ISO 9612:2009*. Geneva: ISO.

International Organization for Standardization (ISO). (1995) Air *quality: Particle size fraction definitions for health-related sampling, ISO 7708:1995*. Geneva:ISO.

Jagtap, A.A. and Deshmukh, J. (2018) Comparative study of morbidities in sawmills workers from central India: a cross sectional study. *International Journal of Community Medicine and Public Health* **5**(7):2846-2852.

Jankewicz, G., Lee, S.G., Pisaniello, D. and Tkaczuk, M. (2008) Benchmarking *of exposures to wood dust and formaldehyde In selected industrles In Australia.* Australian: ASCC, Canberra, A.C.T.

Jarup, L., Babisch, W., Houthuijs, D., Pershagen, G., Katsouyanni, K., Cadum, E., Dudley, M.L., Savigny, P., Seiffert, I., Swart, W., Breugelmans, O., Bluhm, G., Selander, J., Haralabidis, A., Dimakopoulou, K., Sourtzi, P., Velonakis, M. and Vigna-Taglianti, F. (2008) Hypertension and exposure to noise near airports: hyena study. *Environmental Health Perspectives* **116**(3):329–333.

Jime'nez, A.S., van Tongeren, M. and Cherrie, J.W. (2011) A *review of monitoring methods for inhalable hardwood dust: SHE can Report 937/1A.1-18.* [s.l.:s.n.].

Kales, S.N., Tsismenakis, A.J., Zhang, C. and Soteriades, E.S. (2009) Blood pressure in firefighters, police officers, and other emergency responders. *American Journal of Hypertension* **22**(1):11-20. DOI:10.1038/ajh.2008.296.

Kannel, W.B. (1974) Role of blood pressure in cardiovascular morbidity and mortality. *Progress in Cardiovascular Diseases* **17**(1): 5-24.

Kannel, W.B. (1993) Hypertension as a risk factor for cardiac events-epidemiologic results of long-term studies. *Journal of Cardiovascular Pharmacology* **21**(2):27-37. DOI: 10.1097/00005344-199321002-00006.

Kaplan, G.A. and Keil, J.E. (1993) Socioeconomic factors and cardiovascular disease: a review of the literature. *Circulation* **88**(4):1973-1998.

Karasek, R., Baker, D., Marxer, F., Ahlbom, A. and Theorell, T. (1981) Job decision latitude, job demands, and cardiovascular disease: A prospective study of Swedish men. *American Journal of Public Health* **71**(7):694–705.

Kauffer, E., Wrobel, R., Görner, P., Rott, C., Grzebyk, M., Simon, X. and Witschger, O. (2010) Site comparison of selected aerosol samplers in the wood industry. *Annals of Occupational Hygiene* **54**(2):188–203.

Kauppinen, T., Toikkanen, J., Pedersen, D., Young, R., Ahrens, W., Boffetta, P., Hansen, J., Kromhout, H., Maqueda Blasco, J., Mirabelli, D., de la Orden-Rivera, V., Pannett, B., Plato, N., Savela, A., Vincent, R. and Kogevinas, M. (2000) Occupational exposure to carcinogens in the European Union (EU). *Occupational and Environmental Medicine* **57**(1):10–18. DOI:10.1136/oem.57.1.10.

Kauppinen, T., Vincent, R., Liukkonen, T., Grzebyk, M., Kauppinen, A., Welling, I., Arezes, P., Black, N., Bochmann, F., Campelo, F., Costa, M., Elsigan, G., Goerens, R., Kikemenis, A., Kromhout, H., Miguel, S, Mirabelli, D., Mceneany, R., Pesch, B., Plato, N., Schlu¨nssen, V., Schulze, J., Sonntag, R., Verougstraete, V., de Vicente, M.A., Wolf, J., Zimmermann, M., Husgafvel-Pursiaine, K. and Savolainen, K. (2006) Occupational exposure to inhalable wood dust in the Member States of the European Union. *Annals of Occupational Hygiene* **50**(6):549–561.

Kauppinen, T.P., Partanen, T.J. Nurminen, M.M., Nickels, J.I., Hernberg, S.G., Hakulinen, T.R. Pukkala, E.I. and Savonen, E.T. (1986) Respiratory cancers and chemical exposures in the wood industry: a nested case-control study. *Occupational and Environmental Medicine* **43**:84-90.

Kawachi, I., Pearce, N. and Fraser, J. (1989) New Zealand Cancer Registry-Based Study of Cancer in Wood Workers. *Cancer* **64**(12): 2609 – 2613.

Kenny, L.C. Aitken, R., Chalmers, C., Fabries, J.F., Gonzalez-Fernadez, E., Kromhout, H., Liden, G., Mark, D., Riediger, G. and Prodi, V. (1997) A Collaborative European study of personal inhalable aerosol sampler performance. *Annals of Occupational Hygiene* **41**:135-153.

Kenny, L.C., Aitken, R.J., Baldwin, P.E.J., Beaumont, G.C. and Maynard, A.D. (1999) The sampling of personal inhalable aerosol sampler in very low air movement. *Journal of Aerosol Science* **30**:627: 638.

Kivimäki, M., Nyberg, S.T., Fransson, E.I., Heikkilä, K., Alfredsson, L., Casini, A., Clays, E., De Bacquer, D., Dragano, N., Ferrie, J.E., Goldberg, M., Hamer, M., Jokela, M., Karasek, R., Kittel, F., Knutsson, A., Koskenvuo, M., Nordin, M., Oksanen, T., Pentti, J., Rugulies, R., Salo, P., Siegrist, J., Suominen, S.B., Theorell, T., Vahtera, J., Virtanen, M., Westerholm, P.J., Westerlund, H., Zins, M., Steptoe, A., Singh-Manoux, A. and Batty, G.D. (2013) IPD-Work Consortium. Associations of job strain and lifestyle risk factors with risk of coronary artery disease: a meta-analysis of individual participant data. *Canadian Medical Association Journal (CMAJ)* **185**(9):763-769.

Kivimäki, M., Virtanen, M., Elovainio, M., Kouvonen, A., Väänänen, A. and Vahtera, J. (2006) Work stress in the aetiology of coronary heart disease-a meta-analysis. *Scandinavian Journal of Work, Environment and Health* **32**(6):431-442.

Kleemola, P., Jousilahti, P., Pietinen, P., Vartiainen, E. and Tuomilehto, J. (2000) Coffee consumption and the risk of coronary heart disease and death. *Archives of Internal Medicine* **160**(22):3393-3400. DOI:10.1001/archinte.160.22.3393.

Kling, R.N., Demers, P.A., Alamgir, H. and Davies, H.W. (2012) Noise exposure and serious injury to active sawmill workers in British Columbia.*Occupational Environmental Medicine* **69**:211-216. DOI:10.1136/oem.2010.058107.

Klintenberg, C., Olofsson, J., Hellquist, H. and Sökjer, H. (1984) Adenocarcinoma of the ethmoid sinuses. A review of 28 cases with special reference to wood dust exposure. *Cancer* **54**(3):482-488.

Knutsson, A. (2003) Health disorders of shift workers. *Occupational Medicine* **53**(2): 103–108.

Koehncke, N. (1999) *An investigation of noise in Alberta sawmills*. (*Master Thesis*). Canada: University of Alberta, Edmonton.

Koslowski, M. (1998) *Modelling the stress-strain relationship in work settings*. London: Routledge.

Kristensen, T.S. (1989) Cardiovascular diseases and the work environment. A critical review of the epidemiologic literature on chemical factors. *Scandinavian Journal of Work, Environment and Health* **15**(4):245-264. DOI:10.5271/sjweh.1854.

Kurmis, A.P. and Apps, S.A. (2007) Occupationally-acquired noise-induced hearing loss: A senseless workplace hazard. *International Journal of Occupational Medicine and Environmental Health* **20**(2):127–136.

Kurth, T., Gaziano, J.M., Cook, N.R., Logroscino, G., Diener, H.C. and Buring, J.E. (2006) Migraine and risk of cardiovascular disease in women. *Journal of the American Medical Association* **296**(3):283-291. DOI:10.1001/jama.296.3.283.

Lalande, N.M., Hétu, R. and Lambert, J. (1986) Is occupational noise exposure during pregnancy a risk factor of damage to the auditory system of the fetus? *American Journal of Industrial Medicine* **10**(4):427-435.

Le, T.N., Straatman, L.V., Lea, J. and Westerberg, B. (2017) Current insights in noise-induced hearing loss: a literature review of the underlying mechanism, pathophysiology, asymmetry, and management options. *Journal of otolaryngology - Head and Neck Surgery* **46**(1):1-41. DOI:10.1186/s40463-017-0219-x.

Leclerc, A., Cortes, M.M., Gerin, M., Luce, D. and Brugere, J. (1994) Sinonasal cancer and wood dust exposure. *American Journal of Epidemiology* **140**(1):340-349.

Lee, J.H., Kang, W., Yaang, S.R., Choy, N. and Lee, C.R. (2009) Cohort study for the effect of chronic noise exposure on blood pressure among male workers in Busan, Korea. *American Journal of Industrial Medicine* **52**: 509–517.

Lee, T., Harper, M., Slaven, J.E., Lee, K., Rando, R.J. and Maples, E.H. (2011) Wood dust sampling: field evaluation of personal samplers when large particles are present. *Annals of Occupational Hygiene* **55**(2):180-191. DOI:10.1093/annhyg/meq075.

Leidel, N.A., Busch, K.A. and Lynch, J.R. (1977) *Occupational exposure sampling strategy manual.* Cincinnati, U.S.A: NIOH.

Leigh, J.P. (2011) Economic burden of occupational injury and illness in the United States. *Milbank Quarterly* **89**(4):728-772. DOI:10.1111/j.1468-0009.2011.00648.x.

Liberman, M.C., Epstein, M.J., Cleveland, S.S., Wang, H. and Maison, S.F. (2016) Toward a Differential Diagnosis of Hidden Hearing Loss in Humans. *Journal of Public Library of Science (PLoS One)* **11**(9):e0162726. DOI: 10.1371/journal.pone.0162726.

Lie, A., Skogstad, M., Johannessen, H.A., Tynes, T., Mehlum, I.S., Nordby, K., Engdahl, B. and Tambs, K. (2016) Occupational noise exposure and hearing: A systematic review. *International Archives of Occupational and Environmental Health Journal* **89**(3):351-372. DOI:10.1007/s00420-015-1083-5.

Liu, C.C., Young, L.H., Yu, T.Y., Bao, B.Y. and Chang, T.Y. (2016a) Occupational Noise Frequencies and the Incidence of Hypertension in a Retrospective Cohort Study. *American Journal of Epidemiology* **184**(2):120–128.

Liu, J., Xu, M., Ding, L., Zhang, H., Pan, L., Liu, Q., Ding, E., Zhao, Q., Wang, B., Han, L., Yang, D. and Zhu, B. (2016b) Prevalence of hypertension and noise-induced hearing loss in Chinese coal miners. *Journal of Thoracic Disease* **8**(3):422-429.

Liu, Y. and Tanaka, H. (2002) Overtime work, insufficient sleep, and risk of non-fatal acute myocardial infarction in Japanese men. The Fukuoka Heart Study Group. *Journal of Occupational and Environmental Medicine* **59**:447–451.

Löfstedt, H., Hagström, K., Bryngelsson, I., Holmström, M. and Rask-Andersen, A. (2017) Respiratory symptoms and lung function in relation to wood dust and monoterpene exposure in the wood pellet industry. *Upsala Journal of Medical Sciences* **122**(2):78-84. DOI:10.1080/03009734.2017.1285836.

Luce, B., Gérin, M., Leclerc, A., Morcet, J., Brugère, J. and Goldberg, M. (1993) Sinonasal cancer and occupational exposure to formaldehyde and other substances. *International Journal of Cancer* **53**(2):224-231.

Luce, D., Leclerc, A., Bégin, D., Demers, P.A., Gérin, M., Orlowski, E., Kogevinas, M., Belli, S., Bugel, I., Bolm-Audorff, U., Brinton, L.A., Comba, P., Hardell, L., Hayes, R.B., Magnani, C., Merler, E., Preston-Martin, S., Vaughan, T.L., Zheng, W. and Boffetta, P. (2002) Sinonasal cancer and occupational exposures: a pooled analysis of 12 case-control studies. *Cancer Causes Control* **13**(2):147-157.

Lynch, E.D. and Kil, J. (2005) Compounds for the prevention and treatment of noise-induced hearing loss. *Elsevier* **10**(19):1263-1332.

Malchaire, J. (2000) Strategy for prevention and control of the risks due to noise. *Occupational and Environmental Medicine* **57**:361–369.

Malchaire, J. and Piette, A.A. (1997) Comprehensive strategy for the assessment of noise exposure and risk of hearing impairment. *Annals of Occupational Hygiene* **41**(4):467-484.

Mandryk, J., Alwis, K.U. and Hocking, A.D. (1999) Work-related symptoms and dose-response relationships for personal exposures and pulmonary function among woodworkers. *American Journal of Industrial Medicine* **35**(5):481-490.

Mandryk, J., Alwis, K.U. and Hocking, A.D. (2000) Effects of personal exposures on pulmonary function and work-related symptoms among sawmill workers. *Annals Occupational Hygiene* **44**(4):281-289.

Martin, F.N. (1986). *Introduction to Audiology*. 3rd ed. Upper Saddle River: Prentice-Hall, Inc.

Martin, F.N. (1998) *Exercises in Audiometry.* Boston*:* Allyn and Bacon, Inc.

Martin, M. (1997) *Speech Audiometry*. 2nd ed. California: Singular Publishing Group.

Matsui, J.I., Parker, M.A., Ryals, B.M. and Cotanche, D.A. (2005) Regeneration and replacement in the vertebrate inner ear. *Drug Discovery Today* **10**(19):1307-1312.

McBride, I. and Williams, S. (2001) Audiometric notch as a sign of noise induced hearing loss. *Occupational and Environmental Medicine* **58**(1):46–51.

Mersereau, H. (n.d.) *Moving beyond noise induced hearing loss.* Canada: Cape Breton University.

Metidieri, M.M., Rodrigues, H.F., Filho, F.J., Ferraz, D.P., Neto, A.F. and Torres, S. (2013) Noise Induced Hearing Loss (NIHL): Literature review with a focus on occupational medicine. *International archives of otorhinolaryngology* **17**(2):208–212.

Mizoue, T., Miyamoto, T. and Shimizu, T. (2003) Combined effect of smoking and occupational exposure to noise on hearing loss in steel factory workers. *Occupational and Environmental Medicine* **60**:56-59.

Morata, T.C., Dunn, D.E., Kretschmer, L.W., Lemasters, G.K. and Keith, R.W. (1993) Effects of occupational exposure to organic solvents and noise on hearing. *Scandinavian Journal of Work, Environment and Health* **19**(4):245-254. DOI:10.5271/sjweh.1477.

Mosendane, T., Mosendane, T. and Raal, F. J. (2008) Shift work and its effects on the cardiovascular system. *Cardiovascular Journal of Africa* **19**(4):210–215.

Münzel, T., Gori, T., Babisch, W. and Basner, M. (2014) Cardiovascular effects of environmental noise exposure. *European Heart Journal* **35**(13):829–836.

Muscat, J.E. and Wynder, E.L. (1992) Tobacco, alcohol, asbestos, and occupational risk factors for laryngeal cancer. *Cancer* **69**(2):244-2251.

Nagai, M., Hoshide, S. and Kario, K. (2010) Sleep duration as a risk factor for cardiovascular disease - a review of the recent literature. *Current Cardiology Reviews* **6**(1):54–61.

Nandi, S.S. and Dhatrak, S.V. (2008) Occupational noise-induced hearing loss in India. *Indian Journal of Occupational and Environmental Medicine* **12**(2):53-56.

National Cancer Institute. (2008) *Wood dust*.Available from: https://www.cancer.gov/about-cancer/causes-prevention/risk/substances/wood-dust. (Access on 13 February 2021).

National Cancer Institute. (2018) *cancer causing substances in the environment*. Available from: https://www.cancer.gov/about-cancer/causes-prevention/risk/substances. (Accessed on 13 October 2019).

National Cancer Institute. (2019) *Paranasal sinus and nasal cavity cancer treatment.* Available from:https://www.cancer.gov/types/head-and-neck/patient/adult/paranasal-sinus-treatment-pdq. (Accessed on 30 January 2021).

National Centre for Chronic Disease Prevention and Health Promotion (NCCDPH). (2020) *Heart disease and stroke.* Available from: https://www.cdc.gov/chronicdisease/pdf/factsheets/Heart-Disease-Stroke-factsheet-H.pdf. (Accessed on 27 March 2021).

National Institute for Occupational Safety and Health (NIOSH). (1998) *Criteria for a recommended standard: Occupational noise exposure.* Cincinnati, Ohio: NIOSH.

National Toxicology Program (NTP). (2016) *Wood dust: report on carcinogens.* 14th ed. Triangle Park, NC: NIOSH.

National Toxicology Program (NTP). (2000) *Final report on carcinogens: Background document for wood dust.* Available from: https://ntp.niehs.nih.gov/ntp/roc/content/profiles/wooddust.pdf.(Accessed on 10 March 2021).

Navas-Acien, A., Guallar, E., Silbergeld, E.K. and Rothenberg, S.J. (2007) Lead exposure and cardiovascular disease - a systematic review. *Environmental Health Perspectives* **115**: 472–482.DOI:10.1289/ehp.9785.

Navy and marine corps public health center. (2021) *Industrial hygiene field operations manual. Chapter 4 – Exposure assessment strategies.* [s.l.: s. n.].

Neitzel, R., Andersson, M.E and Andersson, E.E. (2016) Comparison of multiple measures of noise exposure in paper mills. *Annals of Occupational Hygiene* **60**(5): 581–596. DOI:10.1093/annhyg/mew001.

Nelson, D.I., Nelson, R.Y. , Concha-Barrientos, M. and Fingerhut, M. (2005) The global burden of occupational noise-induced hearing loss. *American Journal of Industrial Medicine* **48**(6):446-458. DOI:10.1002/ajim.20223.

Nissen, S.E., Tuzcu, E.M., Libby, P., Thompson, P.D., Ghali, M., Garza, D., Berman, L., Shi, H., Buebendorf, E. and Topol, E.J. (2004) CAMELOT investigators. Effect of antihypertensive agents on cardiovascular events in patients with coronary disease and normal blood pressure: The CAMELOT study - A randomized controlled trial. *Journal of the American Medical Association* **292**(18):2217-2225.

Nurminen, T. (1995) Female noise exposure, shift work, and reproduction. *Journal of Occupational and Environmental Medicine* **37**(8):945-950. DOI:10.1097/00043764-199508000-00010.

Noise Induced Hearing Loss Regulations. (2003) *Noise induced Hearing Loss Regulations, 2003 promulgated under the OHS Act, 1993 (Act No. 85 of 1993) as amended*. Government Gazette 24967: 318-327. Pretoria: Government Printer.

Nylander, L.A. and Dement, J.M. (1993) Carcinogenic effects of wood dust: Review and discussion. *American Journal of Industrial Medicine* **24**(5):619-647.

Occupational Health Clinics for Ontario Workers (OHCOW). (2012) *Submission to Ministry of Labour regarding 2012 notice of proposal to adopt new/revised OEL or listings for hazardous chemical substances*. [s.l.]: [s.n.].

Occupational Health Clinics for Ontario Workers (OHCOW).(2016) *Preventing occupational diseases by preventing exposures.* [s.l.]: [s.n.].

Odibo, A.A., Nwaogazie, I.L., Achalu, E.I. and Ugbebor, J.N. (2018) Effects of Safety Intervention Practices among Selected Sawmill Workers in Sawmills in Delta State, Nigeria. *International Journal of Health, Safety and Environments* **4**(2):218-235.

Ogungbe, A.S. and Amosu, W.C. (2018) Impact of noise pollution on the hearing capability of industrial workers in Logos State, Southwest, Nigeria. *Journal of American Sciences* **14**(4):56-64. DOI:10.7537/marsjas140418.09.

Olayinka, O.S. (2013) Development of noise map for Ilorin metropolis, Nigeria. *International Journal of Environmental Studies* **70**(4): 503-514.

Olsen, J.H., Jensen, S.P., Hink, M., Faurbo, K., Breum, N.O. and Jensen, O.M. (1984) Occupational formaldehyde exposure and increased nasal cancer risk in men. *International Journal of Cancer* **34**(5):639-644. DOI:10.1002/ijc.2910340509.

Otoghile, B., Onakoya, P.A. and Otoghile, C.C. (2019) Effects of Occupational noise on quality of life. *International Journal of Otorhinolaryngology - Head and Neck Surgery* **4**(5):1142-1146.

Otoghile, B., Onakoya, P.A. and Otoghile, C.C. (2018) Auditory effects of noise and its prevalence among sawmill workers. *International Journal of Medicine and Medical Sciences* **10**(2):27-30.

Owoyemi, M.J., Falemara, B.C. and Owoyemi, A.J. (2017) Noise pollution and control in mechanical processing wood industries. *Biomed. Stat. Inf.* **2**(2):54e60.

Partinen, M., Putkonen, P.T.S., Kaprio, J., Koskenvuo, M. and Hilakivi, I. (1982) Sleep Disorders in Relation to Coronary Heart Disease. *Acta Medica Scandinavia* **211**(S660):69-83.Available from: https://doi.org/10.1111/j.0954-6820.1982.tb00362.x. (Accessed on 14 February 2021).

Passchier-Vermeer, W. and Passchier, W.F. (2000) Noise exposure and public health. *Environmental Health Perspective* **108**(1):123-131.

Peter, R. and Siegrist, J. (1999) Chronic psychosocial stress at work and cardiovascular disease: The role of effort–reward imbalance. *International Journal of Law and Psychiatry* **22**(5-6):441–449.

Peter, R. and Siegrist, J. (2000) Psychosocial work environment and the risk of coronary heart disease. *International Archives of Occupational and Environmental Health* **73**:S41–S45.DOI:10.1007/pl00014625.

Peter, R., Alfredsson, L., Knutsson, A., Siegrist, J. and Westerholm, P. (1999) Does a stressful psychosocial work environment mediate the effects of shift work on cardiovascular risk factors? *Scandinavian Journal of Work*, *Environment and Health* **25**(4):376-381. DOI:10.5271/sjweh.448.

Peters, C.E., Ge, C.B., Hall, A.L., Davies, H.W. and Demers, P.A. (2015) CAREX Canada: an enhanced model for assessing occupational carcinogen exposure. *Occupational and Environmental Medicine* **72**: 64–71.

Picard, M., Girard, S.A., Simard, M., Larocque, R., Leroux, T. and Turcotte, F. (2008) Association of work-related accidents with noise exposure in the workplace and noise-induced hearing loss based on the experience of some 240,000 person-years of observation. *Accident Analysis and Prevention* **40**(5):1644-1652.

Pouryaghoub, G., Mehrdad, R. and Mohammadi, S. (2007) Interaction of smoking and occupational noise exposure on hearing loss: A cross-sectional study. *BioMed Central Public Health* **7**:137-139. DOI:10.1186/1471-2458-7-137.

Proto, A.R., Zimbalatti, G. and Negri, M. (2010) The measurement and distribution of wood dust. *Journal of Agricultural Engineering* **41**(1):25-31.

Psaty, B.M., Heckbert, S.R., Atkins, D., Siscovick, D.S., Koepsell, T.D., Wahl, P.W., Longstreth Jr, W.T., Weiss, N.S., Wagner, E.H., Prentice, R. and Furberg, C.D. (1993) A Review of the Association of Estrogens and Progestins with Cardiovascular Disease in Postmenopausal Women. *Archives of Internal Medicine* **153**(12):1421–1427.

Psaty, B.M., Heckbert, S.R., Koepsell, T.D., Siscovick, D.S., Raghunathan, T.E., Weiss, N.S., Rosendaal, F.R., Lemaitre, R.N., Smith, N.L., Wahl, P. W. (1995) The risk of myocardial infarction associated with antihypertensive drug therapies. *Journal of the American Medical Association* **274**(8):620-625.

Puskar, M.A., Fergon, S.M., Harkins, J.M. and Hecker, L.H. (1992) Gravimetric determination of airborne dust by using a filter cartridge inside a closed-face, 37-mm closed-face cassette. *Annals of Industrial Hygiene Association Journal* **53**:692–698.

Puttonen, S., Härmä, M. and Hublin, C. (2010) Shift work and cardiovascular disease-pathways from circadian stress to morbidity.*Scandinavian Journal of Work, Environment and Health* **36**(2): 96-108. DOI:10.5271/sjweh.2894.

Pyykkö, I., Starck, J., Färkkilä, M., Hoikkala, M., Korhonen, O. and Nurminen, M. (1981) Hand-arm vibration in the aetiology of hearing loss in lumberjacks. *British Journal of Industrial Medicine* **38**(3):281-289. DOI:10.1136/oem.38.3.281.

Rabinowitz, P.M. (2000) Noise-Induced Hearing Loss. *American Family Physician* **61**(9):2759-2760.

Ragland, D.R., Winkleby, M.A., Schwalbe, J., Holman, B.L., Morse, L., Syme, S.L. and Fisher, J.M. (1987) Prevalence of Hypertension in Bus Drivers. *International Journal of Epidemiology* **16**(2): 208-214.

Ramachandran, G. (2008) Toward Better Exposure Assessment Strategies—The New NIOSH Initiative. *Annals of Occupational Hygiene* **52**(5): 297–301.

Rappaport, S.M., Kromhout, H. and Symanski, E. (1993) Variation of exposure between workers in homogeneous exposure groups. *Annals of Industrial Hygiene Association Journal* **54**:654-662.

Ratnasingam, J., Ioras, F., Tadin, I., Wai, L.T. and Ramasamy, G. (2014) Respiratory effects in woodworkers exposed to wood and wood coating dust: A regional evaluation of South East Asia Countries. *Journal of Applied Sciences* **14**(15):1763-1768.

Ratnasingam, J., Natthondan, V., Ioras, F. and McNulty, T. (2010) Dust, Noise and Chemical Solvents Exposure of Workers in the Wooden Furniture Industry in South East Asia. *Journal of Applied Sciences* **10**(140):1413-1420.

Robinson, T., Whittaker, J., Acharya, A., Singh, D. and Smith, M. (2015) Prevalence of noise-induced hearing loss among woodworkers in Nepal: A pilot study. *International Journal of Occupational and Environmental Health* **21**(1):14–22.

Rockwell, T.H. (1983) Personal vs. area noise exposure monitoring. *National Safety News* **1**:90-93.

Rosengren, A., Anderson, K. and Wilhelmsen, L. (1991) Risk of coronary heart disease in middle-aged male bus and tram drivers compared to men in other occupations: A Prospective Study. *International Journal of Epidemiology* **20**(1):82-87.

Rosenlund, M., Berglind, N., Pershagen, G., Hallqvist, J., Jonson, T. and Bellander, T. (2006) Long-term exposure to urban air pollution and myocardial infarction. *Epidemiology* **17**(4):383-390. DOI: 10.1097/01.ede.0000219722.25569.

Rybak, L.P. and Whitworth, C.A. (2005). Ototoxicity: therapeutic opportunities. *Drug Discovery Today* **10**(19):1313-1321. DOI:10.1016/S1359-6446(05)03552-X.

Saejiw, N., Chaiear, N. and Sadhra, S. (2009) Exposure to wood dust and its particle size distribution in a rubberwood sawmill in Thailand. *Journal of Occupational and Environmental Hygiene* **6**(8):483-490. DOI:10.1080/15459620902967065.

Sam, W.Y., Anita, A.R., Hayati, K.S., Haslinda, A. and Lim, C.S. (2017) Prevalence of Hearing Loss and Hearing Impairment among Small and Medium Enterprise Workers in Selangor, Malaysia. *Sains Malaysiana* **46**(2):267–274.

Samuelsson, O., Wilhelmsen, L., Andersson, O.K., Pennert, K. and Berglund, G. (1987) Cardiovascular Morbidity in Relation to Change in Blood Pressure and Serum Cholesterol Levels in Treated Hypertension: Results from the Primary Prevention Trial in Göteborg, Sweden. *Journal of the American Medical Association* **258**(13):1768–1776. DOI:10.1001/jama.1987.03400130082038.

Spear, J. (no date) *Industrial hygiene exposure assessments: "worst-case" vs. random sampling*. Available from: https://www.jespear.com/wp-content/uploads/2018/02/Industrial-Hygiene-Exposure-02.21.18.pdf. (Accessed on 16 July 2021).

Sardón, M.A., Chamorro, A.J., García, I.H., Sena, H.I., Rodero, H.M., Herrera, C.H., Marcos, M.M. and Canelo, J.A.M. (2015) Association between Occupational Exposure to Wood Dust and Cancer: A Systematic Review and Meta-Analysis. *Journal of Public Library of Science* (*PLoS ONE*) **10**(7):e0133024.

Sattelmair, J., Pertman, J., Ding, E.L., Kohl, W., Haskell, W. and Lee, I. M. (2011) Dose response between physical activity and risk of coronary heart disease: A meta-analysis. *Circulation* **124**(7):789–795.

Sbihi, H., Davies, H.W. and Demers, P.A. (2008) Hypertension in noise-exposed sawmill workers: cohort study. *Occupational and Environmental Medicine* **65**(9): 643-646.

Scheeper, B., Kromhout, H. and Boleij, J.S. (1995) Wood-dust exposure during wood-working processes. *Annals of Occupational Hygiene* **39**(2): 141-154.

Schernthaner, G., Lotan, C., Baltadzhieva-Trendaflova, E., Ceponis, J., Clodi, M., Ducena, K., Goncalvesova, E., Guja, C., Honka, M., Janež, A., Lalić, N., Lehmann, R., Nyolczas, N., Pauklin, P., Rynkiewicz, A., Sergienko, I. and Duvnjak, L.S. (2018) Unrecognised cardiovascular disease in type 2 diabetes: is it time to act earlier?. *Cardiovasc Diabetol* **17**(145):1-6. DOI:https://doi.org/10.1186/s12933-018-0788-7.

Schulte, P.A., Wagner, G.R., Ostry, A., Blanciforti, L.A., Cutlip, R.G., Krajnak, K.M., Luster, M., Munson, A.E., O'Callaghan, J.P., Parks, C.G., Simeonova, P.P. and Miller, D.B. (2007) Work, obesity, and occupational safety and health. *American Journal of Public Health* **97**(3):428–436.

Schulz, E., Jansen, T., Wenzel, P., Daiber, A. and Münzel, T. (2008) Nitric oxide, tetrahydrobiopterin, oxidative stress, and endothelial dysfunction in hypertension. *Antioxidants and redox signaling* **10**(6):1115-1126.

Scientific Committee on Occupational Exposure Limits (SCOEL).(2003) *Recommendation from the scientific committee on occupational exposure limits: risk assessment for wood dust.* SCOEL/SUM/102. [s.l.:s.n.].

Seidman, M.D. and Standring, R.T. (2010) Noise and quality of life. *International Journal of Environmental Research and Public Health* **7**(10):3730-3738.

Selander, J., Nilsson, M.E., Bluhm, G., Rosenlund, M., Lindqvist, M., Nise, G. and Pershagen, G. (2009) Long-term exposure to road traffic noise and myocardial infarction. *Epidemiology* **20**(2):272-279. DOI:10.1097/EDE.0b013e31819463bd.

South Africa, Hazardous Chemical Substance Regulations. (1995) *Hazardous Chemical Substances Regulations; Government Notice R:1179*. Republic of South Africa. Department of Labour. Pretoria: Government Printer.

South African National Standard (SANS 10083). (2013) *The code of practice for the measurement and assessment of occupational noise for hearing conservation purpose.* Pretoria:SABS.

South African Department of Labour. Circular Instruction No. 171. (2001) *The determination of permanent disablement resulting from hearing loss caused by exposure to excessive noise and trauma*.Available on: https://www.gov.za/sites/default/files/gcis_document/201409/22296.pdf.(Accessed on 10 October 2022).

Shackleton, S. and Piney, M.D. (1984) A comparison of two methods of measuring personal noise exposure. *Annals of Occupational Hygiene* **28**:373-390.

Shargorodsky, J. Curhan, G.C. and Farwell, W.R. (2010) Prevalence and Characteristics of Tinnitus among US Adults. *The American Journal of Medicine* **123**(8):711-718.

Sheiner, E.K., Sheiner, E., Hammel, R.D., Potashnik, G. and Carel, R. (2003) Effect of Occupational Exposures on Male Fertility: Literature Review. *Industrial Health* **41**:55–62.

Shrestha, A. and Shiqi, M. (2017) Occupational Noise Exposure in Relation to Hypertension: A Cross-sectional Study in the Steel Factory. *Occupational Medicine and Health Affairs- Open Access* **5**(3):266-687. DOI:10.4172/2329-6879.1000266.

Siegrist, J., Peter, R., Junge, A., Cremer, P. and Seidel, D. (1990) Low status control, high effort at work and ischemic heart disease: Prospective evidence from blue-collar men. *Social Science and Medicine* **31**(10):1127-1134.

Siemiatycki, J., Richardson, L., Straif, K., Latreille, B., Lakhani, R., Campbell, S., Rousseau, M. and Boffetta, P. (2004) Listing Occupational Carcinogens. *Environmental Health Perspectives* **112**(15):1447–1459.

Siew, S.S., Martinsen, J.I., Kjaerheim, K., Sparén, P., Tryggvadottir, L., Weiderpass, E. and Pukkala, E. (2017) Occupational exposure to wood dust and risk of nasal and nasopharyngeal cancer: A case-control study among men in four nordic countries-With an emphasis on nasal adenocarcinoma. *International Journal of Cancer* **141**(12):2430-2436. DOI:10.1002/ijc.31015.

Siew, S.S., Kauppinen, T., Kyyrönen, P., Heikkilä, P. and Pukkala, E. (2012) Occupational exposure to wood dust and formaldehyde and risk of nasal, nasopharyngeal, and lung cancer among Finnish men. *Cancer Management and Research* **4**:223–232. DOI:10.2147/CMAR.S30684.

Siripanish, S. (2013) *Health risk assessment associated with wood dust exposure and risk management for incense and joss stick workers, Roi-et Province Thailand*. (*Master Thesis*). Thailand: Chulalongkorn University.

Simpson, J.C., Niven, R.M., Pickering, C.A., Fletcher, A.M., Oldham, L.A. and Francis, H.M. (1998) Prevalence and predictors of work related respiratory symptoms in workers exposed to organic dusts. *Occup Environ Med* **55**(10):668-672. DOI: 10.1136/oem.55.10.668.

Skogstad, M., Johannessen, H.A., Tynes, T., Mehlum, I.S., Nordby, K. C. and Lie, A. (2016) Systematic review of the cardiovascular effects of occupational noise. *Occupational Medicine* **66**:10–16. DOI:10.1093/occmed/kqv148.

Sliwinska-Kowalska, M. and Davis, A. (2012) Noise induced hearing loss. *Noise Health* **14**(61):274-280.

Smailyte, G. (2012) Cancer incidence among workers exposed to softwood dust in Lithuania. *Occupational and Environmental Medicine* **69**: 449-451.

Smith, G.D., Ben-Shlomo, Y., Beswick, A., Yarnell, J., Lightman, S. and Elwood, P. (2005) Cortisol, testosterone, and coronary heart disease: prospective evidence from the Caerphilly study. *Circulation* **112**(3): 332–340.

Smith, T.W. and Ruiz, J.M. (2002) Psychosocial influences on the development and course of coronary heart disease: current status and implications for research and practice. *Journal of Consulting and Clinical Psychology* **70**(3):548-568.

Soteriades, E.S., Smith, D.L., Tsismenakis, A.J., Baur, D.M. and Kales, S.N. (2011) Cardiovascular Disease in US Firefighters: A Systematic Review. *Cardiology in Review* **19**(4):202-215. DOI: 10.1097/CRD.0b013e318215c105.

Spee, T., van de Rijdt-Van Hoof, E., van Hoof, W., Noy, D. and Kromhout, H. (2007) Exposure to wood dust among carpenters in the construction industry in The Netherlands. *The Annals of Occupational Hygiene* **51**(3):241–248.

Sprinzl, G.M. and Riechelmann, H. (2010) Current trends in treating hearing loss in elderly people: a review of the technology and treatment options - a mini-review. *The Journals of Gerontology* **56**(3):351-358. DOI:10.1159/000275062.

Sriwattanatamma, P. and Breysse, P. (2000) Comparison of NIOSH noise criteria and OSHA hearing conservation. *American Journal of Industrial Medicine* **37**:334–338.

Stamler, J., Stamler, R., Neaton, J.D., Wentworth, D., Daviglus, M.L., Garside, D., Dyer, A.R., Liu, K. and Greenland, P. (1999) Low risk-factor profile and long-term cardiovascular and noncardiovascular mortality and life expectancy: Findings for 5 large cohorts of young adult and middle-aged men and women. *Journal of The American Medical Association* **282**(21):2012-2018.

Stansfeld, S.A. and Matheson, M.P. (2003) Noise pollution: non-auditory effects on health. *British Medical Bulletin* **68**(1):243–257.

Starck, J., Pyykkö, I. and Pekkarinen, J. (1988) *Effect of smoking on sensory neural hearing loss*. In Claussen, C.F., Kirtane, M.V. and Schlitter, K. (eds). *Vertigo, nausea, tinnitus and hypoacusia in metabolic disorders*. Amsterdam: Elsevier Publishing Co.

Starck, J., Toppila, E. and Pyykkö, I. (1999) Smoking as a risk factor in sensory neural hearing loss among workers exposed to occupational noise. *Acta Oto-Laryngologica* **119**(3):302-305. DOI:10.1080/00016489950181288.

Stokholm, Z.A., Bonde, J.P., Christensen, K.L., Hansen, A.M. and Kolstad, H.A. (2013) Occupational noise exposure and the risk of hypertension. *Epidemiology* **24**:135–142.

Straumfors, A., Olsen, R., Daae, H.L., Afanou, A., McLean, D., Corbin, M., Mannetje, A. T', Ulvestad, B., Bakke, B., Johnsen, H.L., Douwes, J. and Eduard, W. (2018) Exposure to wood dust, microbial components and terpenes in the Norwegian Sawmill industry. *Annals of work Exposures and Health* **62**(6): 674-688.

Suadicani, P., Hein, H.O. and Gyntelberg, F. (2012) Occupational noise exposure, social class, and risk of ischemic heart disease and all-cause mortality—a 16-year follow-up in the Copenhagen Male Study. *Scandinavian Journal of Work, Environment and Health* **38**:19–26.

Sussell, A., Periakaruppan, P. and Burr, G. (2005) *NIOSH health evaluation: ikens hardwood floor services Madison, Wisconsin*. Available from: https://elcosh.org/record/document/1272/d000767.pdf. (Accessed on 04 March 2019.

't Mannetje, A., Kogevinas, M., Luce, D., Demers, P.A., Bégin, D., Bolm-Audorff, U., Comba, P., Gérin, M., Hardell, L., Hayes, R.B., Leclerc, A., Magnani, C., Merler, E., DipStat, A.T. and Boffetta, P. (1999) Sinonasal cancer, occupation, and tobacco smoking in European women and men. *American Journal of Industrial Medicine* **36**(1):101-107.DOI:10.1002(sici)1097-0274(199907)36:1<101:aid-ajim14>3.0.co;2-a.

Talbott, E., Helmkamp, J., Mathews, K., Kuller, L., Cottington, E. and Redmond, G. (1985) Occupational noise exposure, noise-induced hearing loss and the epidemiology of high blood pressure. *American Journal of Epidemiology* **121**(4):501–514.

Talbott, E.O., Findlay, R.C., Kuller, L.H., Lenkner, L.A., Matthews, K.A., Day, R.D. and Ishii, E.K. (1990) Noise-induced hearing loss: a possible marker for high blood pressure in older noise-exposed populations. *Journal of Occupational Medicine* **32**(8):690-697.

Tang, X., Bai, Y., Duong, A., Smith, M.T., Li, L. and Zhang, L. (2009) Formaldehyde in China: Production, consumption, exposure levels, and health effects. *Environment International* **35**(8):1210–1224. DOI: 10.1016/j.envint.2009.12.002.

Taskinen, H.K., Kyyrönen, P., Sallmén, M., Virtanen, S.V., Liukkonen, T.A., Huida, O., Lindbohm, M.L. and Anttila, A. (1999) Reduced fertility among female wood workers exposed to formaldehyde. *American Journal of Industrial Medicine* **36**(1):206-212. DOI:10.1002/(sici)1097-0274(199907)36:1<206: aid-ajim29>3.0.co;2-d.

Thayer, J.F., Yamamoto, S.S. and Brosschot, J.F. (2010) The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. *International Journal of Cardiology* **141**(2):122-131.

Thepaksorn, P., Koizumi, A., Harada, K., Siriwong, W. and Neitzel, R.L. (2019) Occupational noise exposure and hearing defects among sawmill workers in the south of Thailand. *International Journal of Occup. Safety and Ergonomics* **25**(3):458-466.

Themann, C. L. and Masterson, E. A. (2019) Occupational noise exposure: A review of its effects, epidemiology, and impact with recommendations for reducing its burden. *The Journal of the Acoustical Society of America* **146**(5):3879-3905.

Thepaksorn, P., Thongjerm, S., Incharoen, S., Siriwong, W., Harada, K. and Koizumi, A. (2017) Job safety analysis and hazard identification for work accident prevention in para rubber wood sawmills in southern Thailand. *Journal of Occu. Health* **59**:542-551.

Timmins, P. and Granger, O. (2010) *Occupational noise-induced hearing loss in Australia: overcoming barriers to effective noise control and hearing loss prevention.* Canberra, ACT: Safe Work Australia.

Tobin, E.A., Ediagbonya, T.F., Okojie, O.H. and Asogun, D.A. (2016) Occupational exposure to wood dust and respiratory health status of sawmill workers in South-South Nigeria. *Journal of Pollution Effects and Control* **4**(1):1-154. DOI:10.4172/2375-4397.1000154.

Tomei, G., Fioravanti, M., Cerratti, D., Sancini, A., Tomao, E., Rosati, M.V., Vacca, D., Palitti, T., Di Famiani. M., Giubilati, R., De Sio, S. and Tomei, F. (2010) Occupational exposure to noise and the cardiovascular system: A meta-analysis. *Science of the Total Environment* **408**(4):681-689.

Torre, T., Cruickshanks, K.J., Klein, B.E., K., Klein, R. and Nondahl, D.M. (2005) The association between cardiovascular disease and cochlear function in older adults. *Journal of Speech, Language, and Hearing Research* **48**(2):473-481.

Tu, N.C., Bobarnac Dogaru, G.B. and Friedman, R.A. (2017) Noise-induced hearing loss: An update. *Internal Medicine Review* **3**(7):1-10.

Ugbebor, J.N. and Yorkor, B. (2015) Assessment and evaluation of noise pollution levels in selected sawmill factories in Port Harcourt, Nigeria. *International Journal on Emerging Technologies* **6**(2):01-08.

Universal Forestry Product. (2010) *Material Safety Data Sheet: Wood dust.* Available from: http://blogs.nwic.edu/rocketteam/files/2011/09/wooddust-165.pdf. (Accessed on 13 March 2019).

Vallières, E., Pintos, J., Parent, M.E. and Siemiatycki, J. (2015) Occupational exposure to wood dust and risk of lung cancer in two population-based case–control studies in Montreal, Canada. *Environmental Health* **14**:1-1. Available from:https://doi.org/10.1186/1476-069X-14-1. (Accessed on 28 March 2020).

van Kempen, E. and Babisch, W. (2012) The quantitative relationship between road traffic noise and hypertension: a metal-analysis. *Journal of Hypertension* **30**(6):1075-1086. DOI:10.1097/HJH.0b013e328352ac54.

van Kempen, E.E.M.M., Kruize, H., Boshuizen, H.C., Ameling, C.B., Staatsen, B.A.M. and de Hollander, A.E.M. (2002) The Association between Noise Exposure and Blood Pressure and Ischemic Heart Disease: A Meta-analysis. *Environmental Health Perspectives* **110**(3):307–317. DOI:10.1289/ehp.02110307.

Vasan, R.S., Beiser, A., D'Agostino, R.B., Levy, D., Selhub, J., Jacques, P.F., Rosenberg, I.H., Wilson, P.W. (2003) Plasma homocysteine and risk for congestive heart failure in adults without prior myocardial infarction. *Journal of the American Medical Association* **289**(10):1251-1257. DOI:10.1001/jama.289.10.1251.

Vaughan, T.L. and Davis, S. (1991) Wood Dust Exposure and Squamous Cell Cancers of the Upper Respiratory Tract. *American Journal of Epidemiology* **133**(6):560–564.

Vaughan, T.L., Stewart, P.A., Teschke, K., Lynch, C.F., Swanson, G.M., Lyon, J.L. and Berwick, M. (2000) Occupational exposure to formaldehyde and wood dust and nasopharyngeal carcinoma. *Journal of Occupational and Environmental Medicine* **57**(6):376-384. DOI:10.1136/oem.57.6.376.

Venners, S.A., Wang, X., Chen, C., Wang, L., Chen, D., Guang, W., Huang, A., Ryan, L., O'Connor, J., Lasley, B., Overstreet, J., Wilcox, A. and Xu, X. (2004) Paternal smoking and pregnancy loss: A prospective study using a biomarker of pregnancy. *American Journal of Epidemiology* **159**(10):993-1001.

Verbeek, J.H., Kateman, E., Morata, T.C., Dreschler, W.A. and Mischke, C. (2012) Interventions to prevent occupational noise-induced hearing loss. *Cochrane Database of Systematic Reviews* **10**(1):1465-1858.

Vermal, D.K., Demers, C., Finkelstein, M., Shaw, D., Kurtz, L., Verma, P. and Welto, T. (2007) Occupational exposure to chemical, biological, and physical agents in Ontario sawmill and veneer/plywood plants. Research Report Workplace Safety and Insurance Board Research Grant #02001.1-102.Available from: https://www.researchgate.net/publication/267821918.(Accessed on 23 June 2019).

Virkkunen, H., Härmä, M., Kauppinen, T. and Tenkanen, L. (2006) The triad of shift work, occupational noise, and physical workload and risk of coronary heart disease. *Journal of Occupational and Environmental Medicine* **63**:378–386.

Virkkunen, H., Härmä, M., Kauppinen, T. and Tenkanen, L. (2007) Shift work, occupational noise and physical workload with ensuing development of blood pressure and their joint effect on the risk of coronary heart disease. *Scandinavian Journal of Work, Environment & Health*, **33**:425–434.

Virkkunen, H., Kauppinen, T. and Tenkanen, L. (2005) Long-term effect of occupational noise on the risk of coronary heart disease. *Scandinavian Journal of Work, Environment and Health* **31**(4):291-299. DOI:10.5271/sjweh.885.

Virtanen, S.V. and Notkola, V. (2002) Socioeconomic inequalities in cardiovascular mortality and the role of work: a register study of Finnish men. *International Journal of Epidemiology* **31**:614–621.

Vyas, M.V., Garg, M.X., Iansavichus, A.V., Costella, J., librarian, I., Donner, A., Laugsand, L.E., Janszky, I., Mrkobrada, M., Parraga, G. and Hackam, D.G. (2012) Shift work and vascular events: systematic review and meta-analysis. *The British Medical Association Journal* **345**:e4800. DOI:10.1136/bmj.e4800.

Wang, D., Zhou, M., Li, W., Kong, W., Wang, Z., Guo, Y., Zhang, X., He, M., Guo, H., and Chen, W. (2018) Occupational noise exposure and hypertension: The Dongfeng-Tongji Cohort Study. *Journal of the American Society of Hypertension* **12**(2):71-79. DOI: 10.1016/j.jash.2017.11.001.

Wang, P.D. and Lin, R.S. (2001) Coronary heart disease risk factors in urban bus drivers. *Journal of Public Health* **115**:261–264.

Weber, M.A., Schiffrin, E.L., White, W.B., Mann, S., Lindholm, L.H., Knerson, J.G., Flck, J.M., Carter, B.L., Materson, B.J., Ram, C.V., Cohen, D.L., Cadet, J.C., Jean-Charles, R. R., Taler, S. Kountz, D., Townsend, R., Chalmers, J., Ramirez, A.J., Bakris, G.L., Wang, J., Schutte, A.E., Bisognano, J.D., Touyz, R.M., Sica, D. and Harrap, S.B. (2013) Clinical Practice Guidelines for the Management of Hypertension in the Community. A Statement by the American Society of Hypertension and the International Society of Hypertension. *Journal of Hypertension* **31**:1-13.

Weiderpass, E. and Labrèche, F. (2012) Malignant tumors of the female reproductive system. *Safety and Health at Work Journal* **3**(3):166-180.

Whooley, M.A., de Jonge, P., Vittinghoff, E., Otte, C., Moos, R., Carney, R.M., Ali, S., Dowray, S., Na, B., Feldman, M.D., Schiller, N.B. and Browner, W.S. (2008) Depressive symptoms, health behaviors, and risk of cardiovascular events in patients with coronary heart disease. *Journal of the American Medical Association* **300**(20):2379-2388.

Willich, S.N., Wegscheider, K., Stallmann, M. and Keil, T. (2006) Noise burden and the risk of myocardial infarction. *European Heart Journal* **27**(3):276–282.

Wilt, T.J., Bloomfield, H.E., MacDonald, R., Nelson, D., Rutks, I., Ho, M., Larsen, G., McCall, A., Pineros, S. and Sales, A. (2004) Effectiveness of statin therapy in adults with coronary heart disease. *Archives of Internal Medicine* **164**(13):1427-36.

Winkleby, M.A., Jatulis, D.E., Frank, E. and Fortmann, S.P. (1992) Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. *American Journal of Public Health* **82**(6):816–820.

WorkSafe New Zealand. (2019) *Workplace exposure standard (WES) review: Wood dust*. New Zealand : WorkSafe New Zealand.

World Health Organisation (WHO). (1995a) Wood dust. *IARC monographs on the evaluation of carcinogenic risks to humans* **62**:35–37.

World Health Organisation (WHO). (2012) *Global estimates on prevalence of hearing loss*. Geneva: WHO.

World Health Organisation (WHO). (2015) *Hearing loss due to recreational exposure to loud sounds: A review.* Geneva: WHO.

World Health Organisation (WHO). (1995b) *Wood dust and formaldehyde, IARC monographs on the evaluation of carcinogenic risks to humans*. Geneva: WHO.

World Health Organisation (WHO). (1997) *IARC Monographs on the evaluation of carcinogenic risks to humans. wood dust and formaldehyde.* Geneva: WHO.

World Health Organisation (WHO). (2011) *Burden of disease from environmental noise: Quantification of healthy life years lost in Europe.* Europe: WHO.

Wultsch, G., Nersesyan, A., Kundi, M., Wagner, K., Ferk, F., Jakse, R. and Knasmueller, S. (2015) Impact of exposure to wood dust on genotoxicity and cytotoxicity in exfoliated buccal and nasal cells. *Mutagenesis* **30**(5):701–709.

Wynder, E.L., Covey, L.S., Mabuchi, K. and Mushinski, M. (1976) Environmental factors in cancer of the larynx: a second look. *Cancer* **38**(4):1591-1601.

Yamanaka, M.W. (2000) *An investigation of wood dust exposure in Alberta sawmills. (Master Thesis)*. Canada: University of Alberta, Edmonton.

Youldena, D.R., Cramba, S.M, Petersb, S., Porcedduc, S.V., Møllerd, H., Fritschib, L. and Baadea, P.D. (2013) International comparisons of the incidence and mortality of Sino nasal cancer. *Cancer Epidemiology* **37**: 770–779.

Yu, M.C. and Yuan, J.M. (2002) Epidemiology of nasopharyngeal carcinoma. *Seminars in Cancer Biology* **12**(6):421-429. DOI:10.1016/s1044579x02000858.

Chapter 3

Evaluation of occupational exposure to wood dust among sawmill workers within the Gert Sibande District Municipality

This Chapter has been published in an *Annals of Agricultural and Environmental Medicine*.

Rathipe, M. and Raphela, F. S. (2022) Evaluation of occupational exposure to wood dust among sawmill workers within the Gert Sibande District Municipality, South Africa. *Annals of Agricultural and Environmental Medicine* **29**(4): 483–488. https://doi.org/10.26444/aaem/152745.

Abstract

Wood dust is regarded as one of the human carcinogens due to an increased risk of nasal and sinonasal cancer. This study was conducted in two sawmill factories to measure and determine the time-weighted average (TWA) exposure level of wood dust and compare the results with the South African occupational exposure limit (OEL). Personal and area respirable and total inhalable wood dust samples were collected at the sawmills using calibrated Giliair-3 personal air sampling pump (Sensidyne, U.S.A.). Data were analysed using Microsoft Office Excel 2019 Analysis Tool pak for a summary of descriptive statistics. Both the geometric means and standard deviation as well as minimum and maximum values were calculated. The geometric mean=GM (geometric standard deviation=GSD) for personal respirable wood dust at sawmill A was 0.9(4.8) mg/m^3, while at sawmill B was 0.57(0.75) mg/m^3. The GM(GSD) for personal total inhalable wood dust at sawmill A was 0.37(0.94) mg/m^3 while at sawmill B was 1.19(16.91) mg/m^3. Besides that, the GM(GSD) for area respirable wood dust at sawmill A was 0.13(0.09) mg/m^3 while at sawmill B was 0.8(0.6) mg/m^3. Likewise, the GM(GSD) for area total inhalable wood dust at sawmill A was 0.13(0.16) mg/m^3 while at sawmill B was 0.54(0.55) mg/m^3. The results for majority of the samples were below the OEL. Workers smoking tobacco or cigarettes should be encouraged to stop smoking since smoking, especially when associated with exposure to wood dust, may increase the risk of respiratory health symptoms.

Keywords: Wood dust, occupational exposure, human carcinogen

3.1. Introduction

According to Alwis (1998), wood is the most important renewable natural resource and furthermore, Global Environmental Fund (2013) emphasised that about 1 700 million cubic metres of wood are harvested each year for industrial purposes. Nearly 69% of wood utilised worldwide belongs to the softwood group, while 58% of wood used for making fuel belongs to the hardwood group (IARC, 1995). The utilisation of wood by different wood-related industries vary among countries, regions and type of wood products. Wood is processed for a wide variety of use. The composition and substance, which may affect its properties are described in detail elsewhere (IARC, 2012). According to Kauppinen *et al*. (2006), more than 3.6 million people are exposed to wood dust worldwide and the highest exposure levels have been reported in the furniture and cabinet making industries. Wood dust exposure in the furniture manufacturing industry may be due to exposure to solvents and formaldehyde in glues and surface coatings.

Sawmill processes generate wood dust particles of different sizes, concentrations and compositions (Alwis, Mandryk and Hocking, 1999a). The majority of wood dust fractions are contributed by the dust particles with a diameter greater than 10 micrometers (µm), which get stuck in the nasal passage (Mandryk, Alwis and Hocking, 1999; SCOEL, 2003). This, in turn, makes inhalable mass sampling the most suitable method to predict the risk of nasal cancer (Lee *et al*., 2011). The inhalable particulate matter (IPM) sampling is used for personal monitoring of wood dust since concentration and size distribution of wood dust differ by position as a result of presence of local source (Alwis, 1998; Lee *et al*., 2011). Respirable dust sampling is used when health concerns are asthma for timber processing *Thuja plicata,* iroko wood or *Fraxinus Americana* (Alwis, 1998; Shamssain, 1992).

Wood dust monitoring has been conducted since 1970, even though the level of exposure may have been higher in the past due to the non-existence or less efficiency of the local exhaust ventilation and other measures to control dust (Kauppinen *et al*., 2006). Woodworking machines have increased greatly since the introduction of the industrial revolution. An increase in the demand for sawn softwood or hardwood products has resulted in companies building faster machinery to expedite production.

As a result, finer dust particles are being emitted now more than in the past. The introduction of engineering controls in 1950 by some industries has lessened exposure among workers considerably. However, the engineering controls, even if they are properly maintained, may not be 100% effective in reducing exposure below the OEL because the sander can generate dust levels that may be more difficult to control (Davies and Henderson, 2009).

Sawmill workers are exposed to wood dust particles of different sizes which may cause respiratory health effects (Kulkarni, Karigoudar and Aithala, 2016). An assessment of the health effects of exposure among humans and the interpretation of the concentration of the measured results may be difficult to undertake due to the variation in the wood dust particles, which differ in physical or chemical properties (Szewczyńska and Pośniak, 2017). A sampling of a known volume of air into a sampling medium is used to determine the concentration of wood dust particles in the air or workers' breathing zone to demonstrate if the workers' exposure to inhalable or respirable dust is adequately controlled (HSE, 2000). The main objective of the presented study was to determine the occupational exposure levels to wood dust among workers in the timber processing sawmills in the Gert Sibande District Municipality in Mpumalanga Province, South Africa.

3.2. Materials and methods

3.2.1. Location and sampling site

The study was conducted at the two sawmill factories located within the Gert Sibande District Municipality in Mpumalanga Province, South Africa. The sawmills were selected according to their size, the number of employees, location and wood type being processed. At the sawmill factory, the logs are taken from the log yard to the conveyor belts using bell machines. The rough bark is removed by the debarker and the bark is sold or used to fuel the kilns. Furthermore, the logs are then sawn into boards or planks by the head rig saw and then they are sorted and stacked before being transported to the kilns to dry. Some adjustments are made to the dried timber and they are sorted according to their grades.The planks are then wrapped and packed in the warehouse for distribution.

The areas covered for total inhalable dust sampling at the sawmills include the knotty pine and profile house, the door house, the dry mill, the wet mill and the saw shop. Other areas covered were the door house next to the first profile cutter, the green chain next to the first chipper, the knotty pine and profile house next to the first profile cutter, the door house next to the first staffer machine, the door house next to second profile cutter and the knotty house next to the second profile cutter.

3.2.2. Study population and selection of participants

This was a cross-sectional study conducted among sawmill workers who were classified according to their job titles, which included chipper operator, unscramble operator, log operator, welder, stopper operator, bell driver, profile cutter, log frame operator, general worker, trim saw operator, staffer operator, door cutter, house keeper and grader. The type of sampling strategy used was based on the random selection of 34 workers who were observed to be closer to the sources of wood dust emission and monitored for respirable and total inhalable wood dust, as per the NIOSH sampling strategy.

3.2.3 Validity and reliability

The validated sampling method used was adapted from previous studies (Alwis, 1998; Martin and Zalk, 1998; Osman and Pala, 2009; Schlünssen *et al*., 2001; Thepaksorn, 2016; Thepaksorn *et al*., 2017a; Thepaksorn, Fadrilan-Camacho and Siriwong, 2017b; IARC, 1995; Thepaksorn *et al.*, 2019; Thetkathuek *et al.*, 2010; Yamanaka *et al.*, 2009). A simple random sampling was employed to ensure validity of the study and minimise selection bias. The samples were representative of worker's exposure. Errors in measurement, instrumental errors, equipment failure and taking of incorrect readings were rectified by following the instrument operating instructions as well as the specifications of the manufacturer. Pre and post calibration of instruments were done before and after the measurements. The instruments were within the validity period during the sampling period and battery checks were done. The operating conditions of the instruments such as humidity and temperature were checked against the operating requirements of the manufacturer. The validated sampling pumps were used for the study and errors were reduced by taking many measurements. All data collection tools were cross-checked at the end to ensure all the information was collected correctly.

The checking of validity and consultative of missing data was performed separately to correspond with codes. The appropriate time for placing the instruments were selected. The leakage check was done on the sampling train. Some filters like cellulose nitrate filter can show excessive weight change due to moisture absorption and other types of filter such as polyvinyl chloride (PVC) or Teflon can show excessive static build-up (Davies and Henderson, 2009; HSE, 2015). The mixed cellulose esters (MCE) filters were used as they do not have these disadvantages

Each sampling sheet was completed with date of sampling, company code, model and serial number of the pump, date of calibration, employee code, job title, work performed and any abnormal activities. Filter holder and sample number, air flow rate and the time of initiation sampling were recorded. Filter holder and the filter media were labelled with suitable sample numbers and caution was taken to ensure the equipment did not hamper with worker's task or movement.

3.2.4. Piloting of the instrument

Pre-test sampling with suitable flow rate and measurement time to evade filter overload was achieved using calibrated electronic flow meter to keep flow rate within ±5%. Sample filters were weighed using calibrated Citizen CX265 (Serial Number:457854/16) (Citizen, 2009) and worker's exposure was calculated.

3.2.5. Sampling procedure

Personal and area respirable and total inhalable wood dust exposures were monitored at the sawmills using Gilair-3 personal air sampling pumps. The flow rates of the portable pumps were calibrated to 2 litres per minute (l/min) using the Gillian Gilibrator-2 (Sensidyne, U.S.A.) for total inhalable dust and 1.7l/min for respirable wood dust according to the NIOSH method (HSE, 2015; NIOSH, 2015; Sesidyne, 2008; Sesidyne, 2012). The sampling heads for the total inhalable dust viz. 37 mm filter cassettes were fitted with 37 mm diameter filter (Mixed Cellulose of Esther (MCE), with pore size of 0.8µm and for respirable dust sampling, a 10 mm nylon cyclone was used.Thirty-four study subjects (16 for respirable and 18 for total inhalable wood dust exposure) were included in the study according to the NIOSH guideline (Davies and Henderson, 2009; Drolet and Beauchamp, 2013; Eller and Cassinelli, 1994; European Standard, 1995;

Harmse, 2000; Leidel, Busch and Lynch, 1977; NIOSH, 1994). Before placing the instrument on the workers, the purpose and procedure of the study were explained. Then, the consent forms were completed (see Appendix C). The Gilair-3 personal air sampling pumps were attached to the exposed workers' belt with the tubing running at the back or sides. The sampling head was attached to the workers' upper chest or collar within their breathing zone (within 30 cm from their nose or mouth) and the pump was switched on to run for eight hours. Furthermore, the sampling heads for area monitoring were securely attached to a stable platform at the head height as near as possible to the source of emission of airborne contaminants and away from any obstructions or fresh air inlets (HSE, 2015; HSE, 2000). The workers who were provided with pumps, were monitored constantly while performing their tasks to ensure that the equipment was operating effectively. The pumps were removed from the workers at the end of the working shift and the field sheets for wood dust were completed (see Appendix F and G). The collected samples were stored in a safe place before being transported to the laboratory for analysis. A sketch showing points in areas with dust levels above 5mg/m^3 was created (see Appendix H).

Ethical approval (Clearance No.: UFS-HSD2019/2236/3006) was obtained from the Health Science Research Ethics Committee of the Free State University. Permission to conduct the study was granted by the managers in charge of the sawmills, and the participants gave consent to take part in the study. Participation in the study was voluntary and participants were allowed to withdraw at any time.

3.2.6 Data analysis

Data were analysed using Microsoft Office Excel 2019 Analysis Tool pak to obtain a summary of the descriptive statistics. The geometric means, standard deviations, minimum and maximum values were calculated. The data were not normally distributed, hence the Mann-Whitney U test was used to test the significance of the differences between the values in sawmill A and B. A significance level of 0.05 was used.

3.3. Results

3.3.1. Personal respirable and total inhalable wood dust exposure levels

The summary statistics of personal respirable and total inhalable wood dust exposure levels from sawmills A and B are shown in Table 3.1. A total of 16 respirable and 18 inhalable wood dust samples were obtained at the sawmills to determine personal wood dust exposure levels as displayed in Table 3.1. The mean (SD) personal respirable wood dust result at sawmill A was 3.4(5.1) mg/m^3 with a geometric mean (GSD) of 0.9(4.8) mg/m^3 while at sawmill B was 0.85(0.80) mg/m^3 with a geometric mean (GSD) of 0.57(0.75) mg/m^3. The results ranged from 0.09 to 13.57 mg/m^3 at sawmill A and 0.14 to 2.25 mg/m^3 at sawmill B. The mean (SD) personal total inhalable wood dust result at sawmill A was 0.72(1.01) mg/m^3 with a geometric mean (GSD) of 0.37(0.94) mg/m^3 while at sawmill B was 9.54(17.82) mg/m^3 with a geometric mean (GSD) of 1.19(16.91) mg/m^3. The results showed that the exposure levels ranged from 0.10 to 3.03 mg/m^3 at sawmill A and 0.01 to 57.1 mg/m^3 at sawmill B. As a result, a non-significant difference was observed when the wood dust exposure levels of sawmills A and B were compared.

Table 3.1. A summary of the statistics of personal respirable and total inhalable wood dust exposure levels at sawmills A and B

Sawmill	Wood dust type	Number of samples	GM mg/m^3	GSD	Median	Range	Mean (SD) mg/m^3	Min	Max	*p*-value*
Sawmill A	Respirable	8	0.881	4.763	1.205	13.48	3.4(5.09)	0.09	13.57	0.87
Sawmill B	Respirable	8	0.570	0.75	0.425	2.11	0.85(0.75)	0.14	2.25	
Sawmill A	Total inhalable	8	0.374	0.940	0.245	2.93	0.72(1.01)	0.10	3.03	0.14
Sawmill B	Total inhalable	10	1.189	16.905	1.84	57.09	9.54(17.82)	0.01	57.1	

* Mann-Whitney U Test

3.3.2. Area respirable and total inhalable wood dust exposure levels

A summary of the statistics of area respirable and total inhalable wood dust exposure levels from sawmills A and B is illustrated in Table 3.2. Six samples for area respirable and 11 samples for total inhalable wood dust exposure were obtained at the sawmills to determine the background concentration of wood dust in the workroom air. The mean (SD) value for area respirable wood dust level at sawmill A was 0.16(0.13) mg/m^3 with a geometric mean (GSD) of 0.13(0.09) mg/m^3, while at sawmill B it was 0.96(0.69) mg/m^3 with a geometric mean (GSD) of 0.8(0.6) mg/m^3. The wood dust exposure levels ranged from 0.07 to 0.25 mg/m^3 at sawmill A and 0.37 to 1.96 mg/m^3 at sawmill B. Regarding the area total inhalable wood dust exposure levels, the mean (SD) value was 0.2(0.18) mg/m^3 at sawmill A with a geometric mean (GSD) of 0.13(0.16) mg/m^3, whereas at sawmill B it was 0.68(0.61) mg/m^3 with a geometric mean (GSD) of 0.54(0.55) mg/m^3. The results indicate that the area exposure levels for total inhalable dust ranged from 0.05 to 0.4 mg/m^3 at sawmill A and 0.29 to 1.89 mg/m^3 at sawmill B.

Table 3.2. A summary of the statistics of area respirable and total inhalable wood dust results at sawmills A and B

Sawmill	Wood dust type	Number of samples	GM mg/m^3	GSD	Median	Range	Mean (SD) mg/m^3	Min	Max	*p*-value*
Sawmill A	Respirable	2	0.132	0.09	0.16	0.18	0.16(0.13)	0.07	0.25	0.13
Sawmill B	Respirable	4	0.799	0.50	0.75	1.59	0.96(0.69)	0.37	1.96	
Sawmill A	Total inhalable	5	0.132	0.164	0.1	0.35	0.2(0.18)	0.05	0.4	0.055
Sawmill B	Total inhalable	6	0.537	0.554	0.49	1.6	0.68(0.61)	0.29	1.89	

* Mann-Whitney U Test

The proportion of samples for personal respirable and total inhalable wood dust exposure levels below or above the action level and occupational exposure limit is depicted in Table 3.3. The results indicate that 78% of the samples for total inhalable and 88% of the samples for personal respirable dust, respectively were below both the 2.5 mg/m^3 action level and 5 mg/m^3 occupational exposure limit (OEL). Furthermore, 13% of the personal respirable wood dust samples were above both the action level and OEL, while only one sample for personal total inhalable dust was above the action level but below the 5 mg/m^3 OEL. However, 17% of the total inhalable dust samples were above both the action level and OEL.

Table 3.3. Proportion of samples for personal respirable and total inhalable wood dust exposure levels below or above the action level and occupational exposure limit

Sawmill	**Wood dust type**	**N**	**No. of samples < 2.5 mg/m^3 Action Level**	**No. of samples ≥ 2.5 and < 5mg/m^3**	**No. of samples ≥5mg/m^3 OEL**
Sawmill A	Respirable	n=8	6		2
Sawmill B	Respirable	n=8	8		
	Total	**14**	**88% (n=14)**		**13% (n=2)**
Sawmill A	Total inhalable	n=8	7	1	
Sawmill B	Total inhalable	n=10	7		3
	Total	**18**	**78% (n=14)**	**6% (n=1)**	**17% (n=3)**

The proportion of samples for area respirable and total inhalable wood dust exposure levels below the action level are indicated in Table 3.4. All the samples for area respirable and total inhalable dust levels were below both the action level of 2.5 mg/m^3 and 5 mg/m^3 OEL at both sawmills.

Table 3.4. Proportion of the samples for area respirable and total inhalable wood dust exposure levels below or above the action level and occupational exposure limit

Sawmill	**Wood dust type**	**N**	**No. of samples <2.5mg/m^3 Action Level**	**No. of samples ≥ 2.5 & < 5mg/m^3**	**No. of samples ≥5mg/m^3 OEL**
Sawmill A	Respirable	n=2	2		
Sawmill B	Respirable	n=4	4		
	Total	**6**	**100% (n=6)**	**0**	**0**
Sawmill A	Total inhalable	n=5	5		
Sawmill B	Total inhalable	n=6	6		
	Total	**11**	**100% (n=11)**	**0**	**0**

3.3.3 Eight-hour TWA exposure levels for personal respirable wood dust from sawmills A and B

Table 3.5 demonstrates the TWA exposure levels of 16 samples for personal respirable wood dust measured at sawmills A and B (8 samples at each sawmill). The highest TWA wood dust exposure level was 13.57 mg/m^3 recorded from a destacker, while the lowest was 0.09 mg/m^3 recorded from a housekeeper in sawmill A. The highest TWA wood dust exposure level was 2.25 mg/m^3 recorded from a skaff planer while the lowest was 0.14 mg/m^3 obtained from finger-joint employee at sawmill B.

Table 3.5. Eight-hour TWA exposure levels for personal respirable wood dust from sawmills A and B

Sawmill A				**Sawmill B**			
Sample no.	**Participant job title**	**Sampling duration** (min)	**TWA 8-hour Concentration** (mg/m^3)	**Sample no.**	**Participant job title**	**Sampling duration** (min)	**TWA 8-hour Concentration** (mg/m^3)
1	Housekeeper	482	0.09	1	Housekeeper	489	1.89
2	Stacker operator	482	1.74	2	Chipper operator	511	0.38
3	Assistant planer operator	475	9.12*	3	Chipper operator	535	0.46
4	Cutter operator	498	0.10	4	Finger-joint operator	508	0.14
5	Transverse operator	445	0.16	5	Bell driver	527	0.27
6	Stacker	525	0.67	6	Fuel bin operator	542	0.39
7	Destacker	436	13.57*	7	Boiler operator	518	1.02
8	Optic cut	500	1.74	8	Skaff planer	558	2.25

*Personal respirable wood dust exposure results above 2.5mg/m^3 action level and 5 mg/m^3 OEL

3.3.4 Eight-hour TWA exposure levels for personal total inhalable wood dust from sawmills A and B

The results in Table 3.6 show the TWA exposure levels of 18 samples for personal total inhalable wood dust measured at sawmills A and B (8 samples at sawmill A and 10 samples at sawmill B). The highest TWA wood dust exposure level was 3.03 mg/m^3 recorded from a shaver operator, while the lowest was 0.10 mg/m^3 recorded from a planer at sawmill A. The highest TWA wood dust exposure level was 57.1 mg/m^3 recorded from sawdust remover while the lowest was 0.01 mg/m^3 recorded from debarker operator and profile cutter at sawmill B. The sawdust extractor operators recorded the highest wood dust exposure level of 11.10 and 19.2 mg/m^3, respectively.

Table 3.6. Eight-hour TWA exposure levels for personal total inhalable wood dust from sawmills A and B

Sawmill A				**Sawmill B**			
Sample no.	**Participant job title**	**Sampling duration** (min)	**TWA 8-hour Concentration** (mg/m^3)	**Sample no.**	**Participant job title**	**Sampling duration** (min)	**TWA 8-hour Concentration** (mg/m^3)
1	Boiler operator	535	0.16	1	Profile cutter	490	1.40
2	Front-end loader	575	1.25	2	Debarker	483	0.40
3	Planer	564	0.10	3	Door cutter	471	1.69
4	Frame operator	585	0.19	4	Sawdust extractor operator	540	11.10*
5	Housekeeper	535	0.28	5	Shaving remover	507	2.47
6	Trim–saw operator	580	0.21	6	Sawdust remover	478	57.1*
7	Shaver operator	498	3.03**	7	Sawdust extractor operator	488	19.2*
8	Shaver operator	546	0.56	8	Debarker	475	0.01
				9	Baam saag machine operator	491	1.99
				10	Profile cutter	469	0.01

*Personal total inhalable wood dust exposure results above 2.5 mg/m^3 action level and 5 mg/m^3 OEL

**Personal total inhalable wood dust exposure results above 2.5 mg/m^3 action level but below 5 mg/m^3

3.3.5 Eight-hour TWA exposure levels for area respirable wood dust from sawmills A and B

Table 3.7 represents the TWA exposure levels for six (6) area respirable wood dust samples measured at sawmills A and B (two samples at sawmill A and four samples at sawmill B). The lowest TWA exposure level of 0.07 mg/m^3 was recorded at a finger joint machine, while 0.25mg/m^3 was recorded at the green chain at sawmill A. The lower TWA exposure level of 0.37 mg/m^3 was recorded at the wet mill next to saw machine 2, while the lower TWA exposure level 1.96 mg/m^3 was recorded at the dry mill next to opticut machine 1 at sawmill B. All the exposure levels were below the 2.5mg/m^3 action level and 5mg/m^3 OEL.

Table 3.7. Eight-hour TWA exposure levels for area respirable wood dust levels from sawmills A and B

Sawmill A				**Sawmill B**			
Sample no.	**Area/place**	**Sampling duration** (min)	**TWA 8-hour Concentration** (mg/m^3)	**Sample no.**	**Area/place**	**Sampling duration** (min)	**TWA 8-hour Concentration** (mg/m^3)
1	Finger joint machine	455	0.25	1	Dry mill next to profile cutter machine 1	510	0.79
2	Green chain	452	0.07	2	Dry mill next to opticut machine 1	505	1.96
				3	Dry mill next to opticut machine 2	520	0.71
				4	Wet mill next saw machine 2	485	0.37

3.3.6 Eight-hour TWA exposure levels for area total inhalable wood dust from sawmills A and B

Table 3.8 depicts the TWA exposure levels of 11 samples for personal total inhalable wood dust measured at sawmills A and B (five samples for total inhalable wood dust at sawmill A and six at sawmill B). The lower TWA wood dust exposure level of 0.40 mg/m^3 was recorded from knotty pine and profile and dry mill, while the lowest TWA exposure level of 0.05 mg/m^3 was recorded from the door house and saw shop at sawmill A. The lower TWA wood dust exposure level of 0.29 mg/m^3 was recorded from door house nearby profile cutter 2, while the highest exposure level of 1.89 mg/m^3 was recorded from door house nearby profile cutter 2 at sawmill B. All the exposure levels were below the action level of 2.5mg/m^3 and 5mg/m^3. OEL.

Table 3.8. Eight-hour TWA exposure levels for area total inhalable wood dust from sawmills A and B

Sawmill A				**Sawmill B**			
Sample no.	**Area/ place**	**Sampling duration** (min)	**TWA 8-hour Concentration** (mg/m^3)	**Sample no.**	**Area/ place**	**Sampling duration** (min)	**TWA 8-hour Concentration** (mg/m^3)
1	Knotty pine and profile	545	0.40	1	Door house next to profile cutter 1	468	1.89
2	Door house	559	0.05	2	Green chain next to chipper machine 1	464	0.43
3	Dry mill	575	0.40	3	Knotty pine and profile	510	0.55
4	Wet mill	559	0.10	4	Door house next to staffer machine 1	497	0.62
5	Saw shop	580	0.05	5	Door house nearby profile cutter 2	488	0.29
				6	Knotty pine next to profile cutter 2	529	0.30

3.4. Discussions

This study was conducted to determine the occupational exposure levels to wood dust among workers at the timber processing sawmills in the Gert Sibande District Municipality. The results of 16 samples of personal respirable wood dust exposure obtained at sawmills A and B ranged from 0.09 to 13.57 mg/m^3 and from 0.14 to 2.25 mg/m^3, respectively. Furthermore, the results of two personal respirable wood dust samples (9.12 and 13.53 mg/m^3) at sawmill A exceeded both the action level of 2.5 mg/m^3 and the OEL of 5 mg/m^3. Moreover, the two personal respirable wood dust samples were higher than the 5 mg/m^3 permissible exposure limit (PEL) set by OSHA and the 1 mg/m^3 recommended exposure limit set by NIOSH and ACGIH TLV. These exposure levels were lower than the exposure level of 31.75 mg/m^3 as reported by Osuchukwu, Osuchukwu and Eko (2015) at a sawmill factory in Sweden. Fourteen personal respirable wood dust samples at sawmills A and B were well below both the action level of 2.5 mg/m^3 and the OEL of 5 mg/m^3. The geometric mean of respirable dust at sawmill A was 0.88 mg/m^3 and 0.57 mg/m^3 at sawmill B. The study conducted at sawmill factories of New South Wales in Australia, reported the geometric mean (GSD) of 0.33 (2.2) mg/m^3 for respirable dust (Alwis, Mandryk and Hocking, 1999b). Kalliny *et al.* (2008) performed related study at a sawmill factory in USA, reported a geometric means of 1.44 (2.67), 0.35 (2.65) and 0.18(2.54) mg/m^3 for inhalable, thoracic as well as respirable dust. Wood dust has some irritation and allergenic properties and exposure to higher concentrations may cause an increased risk for upper and lower respiratory symptoms as well as airway irritations.

The results of the 18 personal total inhalable wood dust samples obtained from sawmills A and B ranged from 0.10 to 3.03 mg/m^3 and 0.01 to 57.1 mg/m^3, respectively. Furthermore, the time weighted average (TWA) for one personal total inhalable wood dust sample measured at sawmill A was 3.03 mg/m^3 and it exceeded the action level of 2.5 mg/m^3 but was below the OEL of 5 mg/m^3. Moreover, the TWAs of the three personal total inhalable wood dust samples at sawmill B were 11.10, 19.2 and 57.1 mg/m^3 and exceeded both the action level of 2.5 mg/m^3 and the 5 mg/m^3 OEL. The TWAs for the remaining 14 personal total inhalable wood dust samples at sawmills A and B were well below both the action level and OEL. The mean exposure level for the total inhalable dust at sawmill A was higher (0.72 mg/m^3) than the mean exposure level of 0.2 mg/m^3 reported by Cormier, Merlaux and Duchaine (2000) at a Canadian sawmill

factory. The mean exposure levels of 0.5 mg/m^3 and 0.57 mg/m^3 were reported by Chan-Yeung, Giclas and Henson (1980) as well as Ahman *et al*. (1996) at sawmill factories. Halpin *et al.* (1994a) and Halpin *et al*. (1994b) also reported the geometric mean of 0.7 mg/m^3 for personal total dust concentration, which is similar to the mean TWAs for personal total inhalable wood dust concentrations measured at sawmill A in the present study.

The findings of the present study discovered that the mean exposure level of the total inhalable wood dust at sawmill B was 9.54 mg/m^3, which is higher than 1.4 mg/m^3 and 1.42 mg/m^3 as previously reported by Hessel *et al.* (1995) and Schlünssen *et al.* (2002). Similarly, Holness *et al.* (1985) as well as Mandryk, Alwis and Hocking (2000) reported the mean values of 1.5 mg/m^3 and 1.53 mg/m^3, which are below the mean value for the total inhalable dust exposure level measured at sawmill B. Furthermore, Holmstrom and Wilhelmsson (1988) as well as Goldsmith and Shy (1988) reported the mean exposure levels of 1.65 mg/m^3 and 2 mg/m^3. Other studies by Andersen, Andersen and Solgaard (1977) and Pisaniello, Connell and Muriale (1991) reported the mean exposure levels of 2.2 mg/m^3 and 3 mg/m^3, while the mean exposure level of 3.75 mg/m^3 was reported in a study by Jacobsen, Schlünssen and Schaumburg (2008). The higher exposure levels at sawmill B may be due to dust extraction systems that were inefficient and the fact that workers were working close to the machines within an enclosed cabin with limited supply of air circulation. The geometric mean for the total inhalable wood dust exposure level at sawmill A was 0.37 mg/m^3, which is below the geometric mean (GSD) of 1.44 (2.67) mg/m^3 recorded by Kalliny *et al*. (2008) in wood processing plants across the United States of America. In another study the personal wood dust exposure measurements were collected by the Workers' Compensation Board of British Columbia, Canada where the recorded geometric mean was 0.72 (3.49) mg/m^3 (Hall, Teschke and Davies, 2002). The geometric mean (GSD) of 1.0 (2.7) mg/m^3 was reported in a study by Demers, Teschke and Davies (2000) and 0.5 (3.1) mg/m^3 by Teschke, Demers and Davies (1999) in a lumber mill environment. A study that was conducted by Scarselli, Binazzi and Ferrante (2008) to investigate the occupational exposure levels to wood dust at an Italian wood processing factory reported a geometric mean (GSD) of 1.0 (1.6) mg/m^3, which is lower than that recorded at sawmill B in the present study.

The mean values of exposure level for area respirable wood dust at sawmills A and B were 0.16 and 0.96, respectively, and are below the mean exposure level of 0.33 mg/m^3 reported by Tobin *et al.* (2016). In addition, the mean values of exposure level for area total inhalable wood dust at sawmill A and B were 0.2 and 0.68, respectively, and are below the mean exposure level of 1.39 mg/m^3 reported in the same study of Tobin *et al.* (2016). It can be concluded that knowledge about the sawmill workers' exposure to wood dust in this setting has been determined in this study. This will assist the sawmill industry to implement measures to reduce workers' exposure. The high wood dust exposure levels were observed among workers operating the planer, destacker, saw dust remover, saw dust extractor, which were due to the LEV design, hood fitted far away from source. The dust lamp and the smoke tubes can be used to check the effectiveness of the LEV system. Proper maintenance of the local exhaust ventilation (LEV) and regular testing on exposure is essential to mitigate exposure.

3.5. Conclusion

Exposure to wood dust can cause asthma, lung function impairment or pulmonary function, upper respiratory problems and eye, skin and throat irritation (Occupational Health Clinic of Ontario Worker (OHCOW), 2016). Wood dust is classified as either hard or soft wood (WorkSafe New Zealand, 2019). Most studies and nations, are using OEL for wood dust, total dust, hardwood and softwood of 5mg/m^3 measured as either total or inhalable dust (Scheeper, Kromhout and Boleij, 1995). ACGIH have set OEL for soft wood at 5mg/m^3 (Diwe *et al*., 2016). Thailand and United State, OSHA have 5mg/m^3 OELs for reparable wood dust (Siripanish, 2013; OHCOW, 2016). South Africa, Britain and European Union (EU) have 5mg/m^3 OEL for hardwood dust and South Africa anticipate to revise this limit to 2 mg/m^3 with 5 mg/m^3 OEL for softwood dust (Chamber and Nunes, 2016; Simpson *et al.*,1999). When OEL for a specific chemical is not available, exposure to total inhalable dust must be kept below 10 mg/m^3 OEL and 5 mg/m^3 OEL for respirable dust (South Africa, Hazardous Chemical Substance Regulation, 1995). Contrary to that, Health and Safety Executive (SHE) (2020) specified that, if softwood dust might be mixed with hardwood, the OEL applicable for hardwood dust must applied to all wood dust present in the mixture. However, hardwood dust is classified as human carcinogen while softwood is a sensitiser and a suspected human carcinogen (ACGIH, 2012; Spee *et al*., 2007).

This study highlighted the exposure levels for wood dust among workers in sawmill factories. Although the measurements for most of the samples were below the action level and OEL, there is a need to implement safety measures to protect the workers against the highest exposure levels recorded in other areas within the sawmill factories. To our knowledge, this is the first study to investigate the exposure levels of wood dust among workers in sawmill factories in the Gert Sibande Municipality of South Africa. Future studies should be conducted within sawmill factories in other provinces of South Africa.

3.6. References

Ahman *et al*. (1996) Work related impairment of nasal function in Swedish woodwork teachers. *Journal of Occupational and Environmental Medicine* **53**: 112-117.

ACGIH. (2012) *Threshold limit values (TLVS) for chemical substances and physical agents and the biological exposure indices (BEIs).* Cincinnati, Ohio: ACGIH.

Alwis, K.U., Mandryk, J. and Hocking, A.D. (1999a) Dust exposures in the wood processing industry. *American Industrial Hygiene Association Journal* **60**:641-646.

Alwis, K.U., Mandryk, J. and Hocking, A.D. (1999b) Exposure to Biohazards in Wood Dust: Bacteria, Fungi, Endotoxins, and (1→3)-β-D-Glucans. *Occupational Environmental Hygiene* **14**:598–608.

Alwis, K.U. (1998) *Occupational exposure to wood dust.* (*Ph.D. Thesis*). New South Wales: Australia.

Andersen, H.C., Andersen, I. and Solgaard, J. (1977) Nasal cancers, symptoms and upper airway function in woodworkers. *The British Journal of Industrial Medicine* **34**:201-217.

Chan-Yeung, M., Giclas, P.C. and Henson, P.M. (1980) Activation of complement by plicatic acid, the chemical compound responsible for asthma due to western red cedar (*Thuja plicata*). *Journal of Allergy and Clinical Immunology* **65**: 333-337.

Chamber, P. and Nunes, E. (2016) Work-related asthma among workers in the wood-processing industry: A review. *Current Allergy & Clinical Immunology* **29**(2): 110-117.

Cormier, V., Merlaux, A. and Duchaine, C. (2000) Respiratory health impact of. working in sawmills in eastern Canada. *Arch Environ Health* **55**(6):424–430

Citizen. (2009) *Operating Instruction:Analytical balances.*Available from: https://www.manualslib.com/download/824925/Citizen-Cx-165.html.(Accessed on 12 April 2021)

Davies, B. and Henderson, J. (2009) *W501 Student manual measurements of hazardous chemical substances*. Australia: University of Wollongong.

Demers, P.A., Teschke, K. and Davies, H.W. (2000) Exposure to dust, resin acids, and monoterpenes in softwood lumber mills. *American Industrial Hygiene Association (AIHAJ)* **61**:521-528.

Diwe *et al*. (2016) Occupational hazards, safety and hygienic practices among timber workers in a South Eastern State, Nigeria. *Occupational Diseases and Environmental Medicine* **4**(1): 63-71.

Drolet, D. and Beauchamp, G. (2013) *Sampling guide for air contaminants in the workplace*. 8th ed. Québec: IRSST.

Eller, P.M. and Cassinelli, M.L. (1994) *NIOSH manual of analytical methods*. 4th ed. Cincinnati, Ohio: Diane publishing.

European Standard. (1995) *Guidance for assessment of exposure to chemical agents for comparison with limit values and measurement strategy, EN 689*. London: BSI.

Global Environmental Fund. (2013) *Africa will import not export wood*. Available from: https://www.criterionafrica.com/wp-content/uploads/2017/06/Africa-will-Import-not-Export-Wood.pdf. (Accessed on 12 April 2021).

Goldsmith, D.F. and Shy, C.M. (1988) Respiratory health effects from occupational exposure to wood dusts. *Scandinavian Journal of Workers' Environmental Health* **14**:1-15.

Hall, A.H., Teschke, K. and Davies, H. (2002) Exposure levels and determinants of softwood dust exposures in BC lumber mills, 1981–1997. *Am. Ind. Hyg. Assoc. J.* **63**:709–714.

Halpin *et al.* (1994a) Respiratory symptoms, immunological responses, and aeroallergen concentrations at a sawmill. *Occup. Environ. Med* **51**: 165-172.

Halpin *et al*. (1994b) Extrinsic allergic alveolitis and asthma in a sawmill worker: Case report and review of the literature. *Occup. Environ. Med* **51**: 160-164.

Harmse, L. (2000) Implementing an air monitoring programme. *Occupational Health SA* **6**(2):14-17.

South Africa, Hazardous Chemical Substance Regulations. (1995) *Government Notice R:1179 Hazardous Chemical Substances Regulations*. Pretoria: Government Printer.

HSE. (2000) *MDHS 14/3: General methods for sampling and gravimetric analysis of respirable and inhalable dust, methods for the determination of hazard substances*. London: HSE Books.

HSE. (2015) *MDHS 14/4: General method for sampling and gravimetric analysis of respirable, thoracic and inhalable aerosols.* London: HSE.

HSE. (2020) *EH40/2005 workplace exposure* limit. London: TSO.

Hessel *et al*. (1995) Lung health in sawmill workers exposed to pine and spruce. *Chest* **108**: 642–646.

Holmstrom, M. and Wilhelmsson, B. (1988) Respiratory symptoms and pathophysiological effects of occupational exposure to formaldehyde and wood dust.

Scand. J. Work Environ. Health **14**: 306-311.

Holness *et al*. (1985) Respiratory function and exposure-effect relationships in wood dust-exposed and control workers. *Journal of Occupational and Environmental Medicine* **27**: 501-506.

IARC. (2012) *Metal, arsenic, dust and fibres. IARC monographs on the evaluation of carcinogenic risks to humans, Volume 100C*. Lyon, France: WHO.

IARC. (1995) *Wood dust and formaldehyde, IARC monographs on the evaluation of carcinogenic risks to humans, Volume 62.* Lyon, France: WHO.

Jacobsen, G., Schlünssen, V. and Schaumburg, I. (2008) Longitudinal lung function decline and wood dust exposure in the furniture industry. *Eur Respir J* **31**:334–42.

Kalliny *et al*. (2008) A survey of size-fractionated dust levels in the US wood processing industry. *J. Occup. Environ. Hyg* **5**:501-510.

Kauppinen *et al.* (2006) Occupational exposure to inhalable wood dust in the Member States of the European Union. *Annals of Occupational Hygiene*.

Kulkarni, C.M., Karigoudar, M.H. and Aithala, M. (2016) Measurement of wood dust particle size by optical microscopy technique and long-term effect on sawmill workers: A random study. *Asian Journal of Pharmaceutical and Clinical Research* **9**:308-311.

Lee, T., Harper, M. and Slaven, J.E. (2011) Wood dust sampling: field evaluation of personal samplers when large particles are present. *Ann Occup Hyg* **55**(2):180-191. DOI:10.1093/annhyg/meq075

Leidel, N.A., Busch, K.A. and Lynch, J.R. (1977) *Occupational exposure sampling strategy manual*. Cincinnati, U.S.A: NIOSH.

Martin, J.R. and Zalk, D.M. (1998) Comparison of total dust/inhalable dust sampling methods for the evaluation of airborne wood dust. *Applied occupational and environmental hygiene* **13**(3): 177-182.

Mandryk, J., Alwis, K.U. and Hocking, A.D. (2000) Effects of personal exposures on pulmonary function and work-related symptoms among sawmill workers. *Annals Occupational Hygiene* **44**: 281-289.

Mandryk, J., Alwis, K.U. and Hocking, A.D. (1999) Work-related symptoms and dose-response relationships for personal exposures and pulmonary function among woodworkers. *American Journal of Industrial Medicine* **35**:481–490.

National Toxicology Program (NTP). (2016) Wood *dust: report on carcinogens*. 14th ed. Triangle Park, NC: NIOSH.

NIOSH. (2015) *Particulates not otherwise regulated, total 0501*. 5th ed. Cincinnati, Ohio: NIOSH.

NIOSH. (1994) *Particulates not otherwise regulated, respirable 0600*. 4th ed. Cincinnati, Ohio: NIOSH.

Occupational health clinics for Ontario workers (OHCO). (2012) *Submission to Ministry of Labour regarding 2012 notice of proposal to adopt new/revised OEL or listings for hazardous chemical substances*. [s.l.]: [s.n.].

Osman, E. and Pala, K. (2009) Occupational exposure to wood dust and health effects on the respiratory system in a minor industrial Estate in Bursa/Turkey. *International Journal of Occupational Medicine and Environmental Health* **22**:43 – 50.

Osuchukwu, N.C., Osuchukwu, E.C. and Eko, J.E. (2015) Occupational exposure to wood dust in Calabar Municipality, Cross River State, Nigeria. *International Journal of Science and Research (IJSR)* **4**:1414-1420.

Pisaniello, D.L., Connell, K.E. Muriale, L. (1991) Wood dust exposure during furniture manufacture results from an Australian survey and considerations for threshold limit value development. *American Industrial Hygiene Association Journal* **52**:485-492.

Scarselli, A., Binazzi, A. and Ferrante, P. (2008) Occupational exposure levels to wood dust in Italy, 1996-2006. *Occup. Environ. Med.* **65**:567-574.

Scheeper, B., Kromhout, H. and Boleij, J.S. (1995) Wood-dust exposure during wood-working processes. *Annals of Occupational Hygiene* 39(2): 141-154.

Schlünssen *et al*. (2001) Wood dust exposure in the Danish furniture industry using conventional and passive monitors. *Annals of occupational hygiene* **45**(2): 157-164.

Schlünssen *et al*. (2002) Respiratory symptoms and lung function among Danish woodworkers. *J. of Occup. Environ. Med* **44**:82-98.

SCOEL. (2003) *Recommendation from the scientific committee on occupational exposure limit: Risk assessment for wood dust.* [s.l.: s.n.]

Sensidyne. (2008) *Gilian Gillibrator 2 calibration system operations and service manual*. Florida, USA: Sensidyne, LP.

Sensidyne. (2012) *Gilair-3 and Gilair-5 air sampling systems operational manual.* Florida, USA: Sensidyne, LP.

Shamssain, M. H. (1992) Pulmonary function and symptoms in workers exposed to wood dust *Thorax* **47**:84-87.

Spee *et al*. (2007) Exposure to wood dust among carpenters in the construction industry in Netherlands. *Annals of Occupational Hygiene* **51**(3): 241–248.

Siripanish, S. (2013) *Health risk assessment associated with wood dust exposure and risk management for incense and joss stick workers, Roi-et Province Thailand*. (*Master Thesis*). Thailand: Chulalongkorn University.

Simpson *et al.* (1999) Comparative personal exposures to organic dusts and endotoxin. *Annals of Occupational Hygiene* **43**(2): 107–115.

Szewczyńska, M. and Pośniak, M. (2017) Assessment of occupational exposure to wood dust in the Polish furniture industry. *Pub Med* **68**:45-60.

Teschke, K., Demers, P.A. and Davies, H.W. (1999) Determinants of exposure to inhalable particulate, wood dust, resin acids, and monoterpenes in a lumber mill environment. *Annals of Occupational Hygiene* **43**:247-255.

Thepaksorn, P. (2016) *Occupatioal exposure and health hazards in para rubber wood sawmill in Trang Province, Thailand. (Master Thesis)*. Thailand: Chulalong University.

Thepaksorn, P., Thongjerm, S. Incharoen, S. (2017a) Job safety analysis and hazard identification for work accident prevention in para rubber wood sawmills in southern Thailand. *Journal of Occupational Health* **59**:542–551.

Thepaksorn, P., Fadrilan-Camacho, V.F.F. and Siriwong, W. (2017b) Respiratory symptoms and ventilatory function defects among Para rubber wood sawmill workers in the South of Thailand. *Human and Ecological Risk Assessment*: *An International Journal* **23**:788-797. DOI:10.1080/10807039.2017.1285221.

Thepaksorna *et al*. (2019) *Occupational hazard exposures and health risks at wooden toys industry in Southern Thailand*. *Human and Ecological Risk Assessment: An International Journal*. DOI:10.1080/10807039.2019.1656053.

Thetkathuek, A., Yingratanasuk, T. and Demers, P.A. (2010) Rubber wood dust and lung function among thai furniture factory workers. *International Journal of Occupational and Environmental Health* **16**: 69-74.

Tobin *et al.* (2016) Occupational Exposure to Wood Dust and Respiratory Health Status of Sawmill Workers in South-South Nigeria. *J Pollut Eff Cont* **4**:1-154.

WorkSafe New Zealand. (2019) *WES review: Wood dust*. New Zealand: WorkSafe.

Yamanaka *et al.* (2009) Wood dust level in Alberta sawmills. *Archives of Environmental and Occupational Health* **64**(4): 270-277.

Chapter 4

Assessment of occupational exposure to noise among sawmill workers in the timber processing factories

This Chapter has been published in the *Journal of Applied Artificial Intelligence*.

Rathipe M. & Raphela F.S. (2022) Assessment of Occupational Exposure to Noise among Sawmill Workers in the Timber Processing Factories, *Applied Artificial Intelligence*, 36(1): e2110696-3144, https://doi.org/10.1080/08839514.2022.2110696.

Abstract

Noise levels of 85dB(A) or higher in the workplace can increase the risk of developing noise-induced hearing loss (NIHL). Sawmill employees are constantly exposed to noise levels exceeding 85dB(A). Consequently, when they are one metre apart, they must raise their voices when communicating. The purpose of this study was to measure and determine the time-weighted average (TWA) occupational noise exposure levels in two timber processing factories and compare the findings with the noise rating limit. Personal and area noise surveys were conducted using calibrated SV104IS noise dosimeters (SVANTEK, Poland) and a type 1 sound level meter (Soundpro SE/DL, U.S.A.). Data were analysed using Microsoft Office Excel 2019 Analysis Tool Pak to obtain a summary of descriptive statistics. The geometric means, standard deviations, minimum and maximum values were calculated. The *T*-test was used to compare the mean time-weighted averages of noise exposure from sawmills A and B. A significance level of 0.05 was used. The geometric means, standard deviations, minimum and maximum values were calculated. The geometric mean (GSD) for area noise level at sawmill A was 90.05(8.02) dB(A), while it was 90.14(7.94) dB(A) at sawmill B. Furthermore, at sawmill A, the geometric mean (GSD) for personal noise exposure level was 92.26(4.35) dB(A), while at sawmill B, it was 92.24(2.65) dB(A).

The findings revealed that sawmill employees were exposed to high noise levels above the 85dB(A) noise rating limit for a longer duration and were at a moderate to high risk of suffering from NIHL.

Keywords: Occupational exposure, noise, sawmill workers, NIHL

4.1. Introduction

High levels of noise in the environment is one of the most common global occupational health hazard (Nandi, and Dhatrak, 2008; Nelson *et al.*, 2005; Rabinowitz, 2000; Themanna and Masterson, 2019). Workers in the mining, construction, manufacturing and agricultural sector are exposed to high noise levels which may impair their hearing (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004; Gerges, Sehrndt and Parthey, n.d.; Nelson *et al.*, 2005; Nandi and Dhatrak, 2008; Tikka *et al.,* 2017). Previous studies have indicated that exposure to loud noise for a longer duration can damage the hair cells of the cochlear in the inner ear leading to irreversible sensorineural hearing loss (Azizi, 2010; Basner *et al*., 2014; Nandi and Dhatrack, 2008; Hong *et al.*, 2013).

Ahmed *et al*. (2001) conducted a noise survey at the factories in the Eastern Province of Saudi Arabia to determine the levels of occupational exposure to noise among the employees. They reported that the overall noise levels recorded at the two factories ranged from 72 to 102 dB(A) and 75% of the workers were exposed to noise levels above the 85 dB(A) recommended exposure level (REL) of the National Institute for Occupational Safety and Health (NIOSH). In the said study 25% of workers were exposed to noise levels above the 90 dB(A) permissible exposure limit (PEL) established by the Occupational Safety and Health Administration (OSHA). Furthermore, 61% of workers exposed to noise level above 85 dB(A) were reported to have never used the hearing protective devices (HPDs) while 38% had hearing impairment that was 8-fold higher than that found in the non-exposed subjects.

More than 30 million workers in the United States of America (USA) and 4 to 5 million workers in Germany are exposed to high noise levels which is defined by the World Health Organisation (WHO) as hazardous noise (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004). The noise levels generated at the sawmills vary greatly with the activities being performed and the type of equipment being used (Hong, 2005). Normally the levels of exposure exceed the lower exposure action value of 80 dB(A) and the upper exposure action value of 85 dB(A) at which the use of HPD is mandatory and the 87 dB(A) occupational exposure limit that takes into account the attenuation level of the HPD (European Commission, 2020; May, 2000).

Dost (1974a) carried out a noise survey at the California lumber mill and reported the highest average noise level of 106 dB(A) for the tail sawyer, 107 dB(A) for the chipper tender and 115 dB(A) for the planner machine that were above the upper exposure action value of 85 dB(A) and the second paper reported the highest noise levels of 104.2 dB(A) for the planer and 104.5 dB(A) for the tail sawyer (Dost,1974b). In another study conducted by Ayaz (1991) at the Pakistanian sawmills, an average noise levels ranging from 90 to 113 dB(A) was reported. It was reported that the sawmill workers were at a considerable highest risk of suffering from hearing disability and noise induced occupational health disorders than their counter parts in other countries. Although, a noise survey conducted by Ruedy *et al*. (1976) reported the noise levels ranging from 91 to 109 dB(A) for the sawmill machines. likewise, continuous exposure to noise levels above 85 dB(A) is the leading cause of NIHL (Cantley *et al.*, 2015; Hong *et al*., 2013; Phillips, Henrich and Mace, 2010; Rabinowitz, 2000). This is a bilateral hearing loss with an audiometric notch at the frequencies of 3, 4 and 6 kHz with a recovery at 8 kHz (Chang *et al*., 2011; Leensen, van Duivenbooden and Dreschler, 2011; Le *et al.* 2017; May, 2000). This audiometric notch deepens and slowly advances towards lower frequencies if noise exposure continues (Rösler, 1994).

It has been reported that the use of ototoxic drugs such as aminoglycosides, ototoxic and non-ototoxic chemical substances such as toxic solvents in paint or organophosphate pesticides as well as aging, smoking cigarette, heat exposure, diabetes, rheumatoid arthritis and exposure to high noise level above 85 dB(A) can increase the risks of developing NIHL (Ferrite and Santana, 2005; Gan, Davies and Demers, 2011; Hong *et al.*, 2013; Mizoue, Miyamoto and Shimizu, 2003; Phillips, Henrich and Mace, 2010; Pouryaghoub, Mehrdad and Mohammadi, 2007). Exposure to high noise level can also increase risk of auditory and non-auditory health effects such as annoyance, sleep disturbance and cardiovascular health etc. (Basner *et al.*, 2014; Chang *et al.*, 2011; Chang *et al*., 2013; Driscoll, Mikl and Burgess, 2009; Münzel, *et al.*, 2014; Sbihi, Davies and Demers, 2008; Stansfeld and Matheson, 2003). Moreover, prolonged exposure to noise levels above 85 dB(A) can also elevate systolic and diastolic blood pressure in males, thus increasing the risk of hypertension (Chang *et al.*, 2013; de Souza, Périssé and Moura, 2015; Driscoll, Mikl and Burgess, 2009; Sbihi, Davies and Demers, 2008). Males loose more hearing than females due to no adherence to the regulations and use of HPDs (Lie *et al.*, 2016; Nelson *et al.*, 2005).

The South African government had set a noise rating limit of 85 dB(A) for an 8hours period, their action plan includes a stricter enforcement of legislation and better implementation of hearing loss prevention program to reduce noise levels at the sawmills. Five-hundred (500) OHS inspectors has been employed to offer specialised advice on good practise and information on employers to reduce NIHL. South Africa government is also rolling out awareness programs to reduce noise exposure at the sawmill through designing and modification of machinery and isolation and enclosure of noise sources as well as control of noise exposure along the path through separation of workers as well as the use of HPDs. There is a growing concern about the high noise levels at the timber processing factories and the prevalence of NIHL which may have a detrimental impact on workers' health and quality of life (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004; Picarda *et al*., 2008; Suter, 2002; May, 2000). Many countries have set occupational exposure limits of 85 and 90 dB(A) for an 8-hour period. The choice of the standards is based on the ethical, social, political and economic factors (Fisnea and Oktenb, 2013; I-INCE-97-1, 1997; Lester *et al.*, 2001; NIOSH, 1998). To date, few studies have been conducted in South Africa at the timber processing factories to investigate the level of occupational exposure to noise. This study was conducted to determine the TWA occupational exposure to noise in the two timber processing factories and compare the results with the noise rating limits.

4.2. Material and methods

4.2.1. Location and sampling sites

Two sawmill factories in the Gert Sibande District Municipality of Mpumalanga, South Africa served as the research sites. The sawmills were selected based on their size, number of employees, location and the type of processing machinery. The door house room, finger joint at the machine area, knotty pine and profile door, saw shop, workshop, dry mill, boiler house, green chain and wet mill were sampled for area noise evaluation at sawmills A and B.

4.2.2. Study population and selection of participants

Only sawmill workers who had been in continuous employment for a minimum period of six months participated in the study. The participants were selected according to their occupations. Chipper operators, unscramble operators, log operators, welders, stopper operators, bell drivers, profile cutters, log frame operators, general workers, trim saw operators, staffer operators, profile cutters, door cutters, bell drivers, housekeepers and grader operators were the participants of the study.

4.2.3 Validity and reliability

The validated sampling method used was selected based on similar studies published in renowned journals and relevant studies (Ahmed *et al.*, 2001; Boateng and Amedofu, 2004; Davies *et al.*, 2009; Koehncke *et al.,* 2003; Thepaksorn *et al.*, 2017a; Thepaksorn *et al.,* 2017b). A simple random sampling was employed to ensure validity of the study and minimise selection bias. Samples were representative of worker exposure. Errors in measurement, instrumental errors, equipment failure and taking of incorrect readings were rectified by following the instrument operating instructions as well as the specifications of the manufacturer. Pre and post calibration of the device were done before and after the measurements. The SLM and the acoustic calibrator were calibrated externally by a SANAS accredited laboratory. The calibration accuracy of the SLM before and after sampling was within 1.0dB and was valid. All the checks were satisfactory and all measurements were valid. Climatic conditions such as humidity, air movement and temperature did not have any influence on the obtained measurement results. Random errors were reduced by taking many readings. Great care was taken not to expose the instruments to extreme temperatures and vibrations during measurement.

The integrating Soundpro SE/DL sound level meter (SLM) was set to A-weighting scale, 80 dB threshold, 85 dB criterion, 3-dB exchange rate (Franz, 2007; Anthony, 2016; Tingay and Robinson, 2014), and impulse time-weighting (SANS, 2013), and calculated the average $L_{Req,\,T}$ for all microphone positions in accordance with Equation 1 to determine $L_{Req,\,T}$ for the entire area (SANS, 2013; ISO, 2009). The results were averaged arithmetically and presented as overall area monitoring results for each mill. Averaged measurements were normalised to an 8-hour shift using Equation 2. According to Driscoll, Mikl and Burgess (2009), Franz (2007), Goelzer, Hansen and Sehrndt (2001), NIOSH (1989) and Starck, Toppila and Pyykko (2003), integrating SLM uses a pre-programmed exchange rate equivalent to the slow setting used for steady noise, while the fast response is used for a more variable or rapidly changing sound. The SV104IS noise dosimeters were set at a 3-dB exchange rate and 85-dBA criterion (SANS, 2013). According to NIOSH (1989), the choice of a fast or slow response on the dosimeter does not affect the computed noise dose or TWA when using the 3-dB exchange rate. The means were calculated as logarithmic means which denotes the actual value for an overall exposure dose calculated as an 8-hour TWA equivalent (ISO, 1979; ISO, 1990; SANS, 2013).

An appropriate time for placing the instruments was selected. Data for area monitoring were pre-tested to verify if the results from selected workshops differed. The microphone for SLM was directed towards the noise source and the windshield was used where necessary. All data collection tools were cross-checked at the end to ensure that all the information was collected correctly. The checking of validity and consultation of missing data were performed individually on data to correspond with the codes. The appropriate time for placing the instruments was selected appropriately. Each sampling sheet was completed with date of sampling, company code, model and serial number of the dosimeter, the employee code; the job title; the work performed and any other abnormal activities and the time of initiation and stop sampling was recorded. The dosimeter was calibrated and rechecked to ensure that it was still within the calibration limit. Lastly, equipment was stored safely for transportation.

4.2.4 Piloting of the instrument

Pre-test measurements with suitable calibration and sampling duration to avoid taking incorrect readings was achieved by using calibrated Casella CEL 120/2 acoustic calibrator to maintain calibration status within ± 0.5dB (Casella, 2022). The readings were taken at the workshops to determine possible differences in the measurement results. The average noise exposure levels were calculated.

4.2.5. Sampling procedure for personal noise monitoring

A cross-sectional survey was conducted whereby personal noise exposure results were monitored at the sawmills using calibrated SV104IS noise-dosimeters (SVANTEK, Poland). Twenty-two consented participants (12 at sawmill A and 10 at sawmill B) were randomly selected. Before placing the dosimeters on the workers, the purpose of the study and the procedures to be followed were explained to them. Thereafter, the consent forms and the personal noise exposure recording sheets were completed. The dosimeters were attached on the mid top of the participants' shoulders approximately 10 cm (0.10m) on the most exposed ear with the microphone just about a few centimetres above the shoulder (SANS, 2013; SVANTEK, 2015; SVANTEK, 2016). The dosimeters were switched on to run for duration of eight hours. Windshields were used to cover the microphones and care was taken to avoid exposing the dosimeters to any vibration while sampling. The workers who wore the dosimeters were constantly monitored while performing their tasks to ensure that the equipment was operating effectively. The dosimeters were removed from the workers at the end of the shift and the personal noise exposure recording sheets were completed and the instrument was switched off (see Appendix F).

Ethical approval (clearance number: UFS-HSD2019/2236/3006) was obtained from the Health Science Research Ethics Committee of the University of the Free State. Permission to conduct the study was granted by the managers in charge of the sawmill factories and the participants gave consent to participate in the study. Participation in the study was voluntary and the participants were allowed to withdraw at any time.

4.2.5. Sampling procedure for area noise survey

Eleven areas were evaluated for noise using a calibrated type 1 integrating sound level meter at each sawmill (Soundpro SE/DL, U.S.A.). The instrument was calibrated before and after sampling using Casella CEL-120/2 sound level calibrator and the calibration remained within the acceptable range of ± 0.5dB during calibration. The meter was attached on a tripod stand, with the microphone positioned at the ear height or hearing zone about 1.5 m above the ground and 1.2 m away from the reflecting surfaces (SANS, 2013; TSI Incorporated, 2018). The number of area measurements selected deviated from the recommended minimum number of three in view of the practical constraints. Each individual measurement was taken over a long duration to be representative of the exposure levels in each task or area. The measurement results were recorded on the area noise exposure recording sheet (see Appendix I). A sketch of the noise map showing points of noise exposures in each area and task below 82 and above 85dB(A) was created (see Appendix J).

4.2.6. Data analysis

Data were analysed using Microsoft Office Excel 2019 Analysis Tool Pak to obtain a summary of descriptive statistics. The geometric means, standard deviations, minimum and maximum values were calculated. The *T*-test was used to compare the mean time-weighted averages of noise exposure from sawmills A and B. A significance level of 0.05 was used.

4.3. Results

4.3.1. Area noise exposure levels

The summary statistics of area noise exposure levels from sawmills A and B are shown in Table 4.1. The mean (SD) for area noise exposure level at sawmill A was 90.42(8.41) dB(A) with a geometric mean (GSD) of 90.05(8.02) dB(A). Similarly, the mean (SD) for area noise exposure level at sawmill B was 90.5(8.33) dB(A) with the geometric mean (GSD) of 90.14(7.94) dB(A). As shown in Table 4.1, the results ranged from 75.9 to 103.5 dB(A) at sawmill A and 75.8 to 103.1 dB(A) at sawmill B.

Table 4.1: Summary statistics of area noise exposure levels at sawmills A and B

Sawmill	Noise type	Number of areas sampled	GM dB(A)	GSD	Median	Range	Mean (SD) dB(A)	Min	Max	*p*-value*
Sawmill A	Area noise survey	11	90.054	8.023	92.5	27.6	90.4(8.41)	75.9	103.5	0.982
Sawmill B	Area noise survey	11	90.143	7.939	92.9	27.3	90.5(8.33)	75.8	103.1	

**T*-test

4.3.2 Personal noise exposure levels

Summarised statistics of personal noise exposure levels from sawmills A and B are illustrated in Table 4.2. The results indicate that the mean (SD) for personal noise exposure level at sawmill A was 92.36(4.54) dB(A) with a geometric mean (GSD) of 92.26(4.35). Correspondingly, the mean (SD) for personal noise exposure level was 92.28(2.79) dB(A) at sawmill B with a geometric mean (GSD) of 92.24(2.65) dB(A). Furthermore, the results ranged from 86.3 to 101.2 dB(A) at sawmill A and 88.3 to 96.9 dB(A) at sawmill B.

Table 4.2: Summary statistics of personal noise exposure levels at sawmills A and B

Sawmill	Noise type	Number of personnel samples	GM dB(A)	GSD	Median	Range	Mean (SD) dB(A)	Min	Max	*p*-value*
Sawmill A	Personal noise	12	92.258	4.351	91.65	14.9	92.36(4.54)	86.3	101.2	0.961
Sawmill B	Personal noise	10	92.242	2.648	92.15	8.6	92.28(2.79)	88.3	96.9	

**T*-test

Table 4.3 presents the proportion of samples for personal noise exposure levels that are either below or above the action level and noise rating limit. The TWA exposure level for all 22 samples recorded at the sawmills were above both the action level of 82 dB(A) and noise rating limit of 85 dB(A) but below the 105 dB(A) noise level.

Table 4.3: The proportion of samples for personal noise exposure levels below or above the action level and noise rating limit from the sawmills

Sawmill	**Type of noise**	**N**	**<82dB(A)**	**≥ 82 & <85**	**≥85 & <105 dB(A)**
sawmill A	Personal noise	n=12			12
Sawmill B	Personal noise	n=10			10
	Total	22	0	0	100% (n=22)

The results in Table 4.4 display the proportion of samples for area noise exposure levels that are either below or above the action level and noise rating limit. The noise levels of 14 samples recorded at the sawmills exceeded both the action level of 82 dB(A) and the noise rating limit of 85 dB(A) but were below 105 dB(A). The TWA noise exposure levels for four samples exceeded the action level but were below the noise rating limit. Furthermore, the TWA noise exposure levels for the other four samples were below both the action level and noise rating limit.

Table 4.4: The proportion of samples for area noise exposure levels below or above the action level and noise rating limit at the sawmills

Sawmill	**Type of noise**	**N**	**<82dB(A)**	**≥ 82 & <85**	**≥85 & <105 dB(A)**
Sawmill A	Area noise	n=11	2	2	7
Sawmill B	Area noise	n=11	2	2	7
	Total	22	18% (n=4)	18% (n=4)	64% (n=14)

4.3.3 Eight-hour TWA personal noise exposure levels

The results in Table 4.5 show the eight-hour TWA personal noise exposure levels. A total of 22 samples were obtained from sawmills A and B (12 samples at sawmill A and 10 at sawmill B). The highest exposure level of 101.2 dB(A) was recorded from a general worker, whereas the lowest exposure of 86.3 dB(A) was recorded from a stopper operator at Sawmill A. At sawmill B, the highest exposure level of 96.9 dB(A) was recorded from a grader operator, while the lowest exposure level of 88.3 dB(A) was recorded from a profile cutter machine operator at sawmill B.

Table 4.5: Eight-hour TWA personal noise exposure levels from sawmills A and B

Sawmill A			**Sawmill B**		
Sample no.	**Participant job title**	**$L_{Req, 8h}$ dB(A)**	**Sample no.**	**Participant job title**	**$L_{Req, 8h}$ dB(A)**
1	Chipper operator	99.9*	1	Grader operator	92.5*
2	Unscramble operator	92.1*	2	Staffer operator	92.9*
3	Log operator	89.9*	3	Grader operator	96.9*
4	Welder	89.0*	4	Profile cutter machine operator	88.3*
5	Stopper operator	86.3*	5	Door cutter	89.5*
6	Trim saw operator	89.3*	6	Trim saw operator	91.8*
7	Bell driver	93.6*	7	Bell driver	93.6*
8	Bell driver	95.4*	8	Housekeeper	89.7*
9	Profile cutter operator	88.3*	9	Chipper operator	96.2*
10	Log frame operator	91.2*	10	Grader	91.4*
11	General worker	101,2*			
12	Trim saw operator	92.1*			

*Personal noise exposure levels above 82 dB(A) action level and 85 dB(A) noise rating limit

4.3.4 Average Eight-hour TWA area noise exposure levels

The results in Table 4.6 represent the average $L_{Req,\ 8h}$ area noise exposure levels. A total of 22 samples were recorded. The highest exposure level of 103.5 dB(A) was recorded at a chipper machine next to the dry mill, conversely, the lowest exposure of 75.9 dB(A) was recorded in the workshop at sawmill A. Similarly, the highest exposure level of 103.1 dB(A) was recorded chipper machine next to the dry mill and the lowest exposure level of 75.8 dB(A) was recorded in the workshop at sawmill B.

Table 4.6: Average eight-hour TWA area noise exposure levels from sawmills A and B

Sawmill A				**Sawmill B**			
Sample No.	**Area/place**	**No. of Position**	**Average $L_{Req,8h}$ dB(A)**	**Sample No.**	**Area/place**	**No. of Position**	**Average $L_{Req,8h}$ dB(A)**
1	Door house	5	91.7*	1	Door house	5	91.9*
2	Finger joint machine	4	92.5*	2	Finger joint machine	4	92.9*
3	Knotty pine and profiles house	4	97.9*	3	Knotty pine and profiles house	4	98.0*
4	Saw extractor machine next to door house	1	80.4	4	Saw extractor machine next to door house	1	80.8
5	Saw shop	4	83.4**	5	Saw shop	4	83.6**
6	Workshop	3	75.9	6	Workshop	3	75.8
7	Dry mill	3	97.6*	7	Dry mill	3	97.7*
8	Boiler house	4	84.1**	8	Boiler house	4	84.2**
9	Green chain	3	93.5*	9	Green chain	3	93.5*
10	Wet mill	5	94.1*	10	Wet mill	5	94.0*
11	Chipper machine next to dry mill	1	103.5*	11	Chipper machine next to dry mill	1	103.1*

***Area noise levels above 85 dB(A) noise rating limit
**Area noise levels above 82dB(A) action level but below 85 dB(A) noise rating limit

4.4. Discussions

This study was conducted to investigate the TWA noise exposure levels at two sawmill factories. All the measurements for personal noise exposure levels obtained at the sawmills were at or above the NIOSH REL, that is 85 dB(A) but below 105 dB(A) where instant NIHL may occur. This finding contradicts the results of Neitzel, Seixas and Camp (1999) who found that the mean OSHA TWA exposure level from 338 samples was 82.8 dB(A) ± 66.8 dB(A), while the mean NIOSH/ISO TWA exposure level from 174 samples was 89.7 dB(A) ± 66.0 dB(A). In addition, 40% of the OSHA TWAs noise exposure levels exceeded the 85dB(A) NIOSH REL and 13% exceeded the 90 dB(A) OSHA PEL. Likewise, 43% of OSHA TWAs noise exposure levels of the 338 (12.7%) exceeded the 90 dB(A) OSHA PEL and 135 of the 338 (39.9%) exceeded the OSHA action levels of 85 dB(A). In contrast, the noise exposure level of 82% of the 174 NIOSH/ISO TWAs exceeded 85 dB(A) and 45.3% surpassed the 90 dB(A) OSHA PEL.

At sawmill A, the stopper operator had the lowest personal noise exposure at 86.3 dB(A), while the general worker had the highest noise level at 101.2dB(A). Furthermore, the lowest noise level of 88.3 dB(A) was recorded from the profile cutter machine operator, in contrast, the highest noise level of 96.9dB(A) was recorded from the grader operator at sawmill B. These findings negate the results of Thepaksorn *et al.* (2017a) who reported the lowest personal noise exposure level of 88.43 dB(A) from a worker performing vacuuming and wood preservation processes and 88.43dB(A) from an employee in the grading, packaging and storage department. Furthermore, Thepaksorn *et al.* (2017a) reported the highest personal noise exposure level of 94.4 dB(A) from a sawing lumber operator. The mean (SD) personal noise exposure level at sawmill A was 92.36(4.54) dB(A), while at sawmill B was 92.28(2.79) dB(A). Twenty-three percent of the samples for personal noise levels obtained at the two sawmills were above the 85 dB(A) NIOSH REL and 77% were above the 90 dB(A) OSHA PEL. These findings are inconsistent with the study by Davies *et al.* (2009) who reported the mean (L_{eq},8hr) personal noise exposure level of 91.7 dB(A). In the said study, only 4 samples out of the 52 jobs that were sampled were below the 85 dB(A) NIOSH REL, while 28 jobs had a mean exposure level above the 90 dB(A) OSHA PEL and four jobs had a mean exposure level above 100 dB(A).

Koehncke *et al*. (2003) conducted a similar study at Alberta sawmills in Canada and found that 10% of personal noise exposure measurements were below the 85 dB(A) level of the Alberta 8-hour exposure limit, while 27% of the samples were at or above 95 dB(A). They reported that 82% of the samples for personal noise exposure level obtained at the sawmills at or above 95 dB(A) were obtained from the planer infeed operator, while 62% of the samples were obtained from the planermen. In the present study, 18% of the samples for personal noise levels at or above 95 dB(A) were obtained from the general worker [101.2 dB(A)], grader operator [96.9 dB(A)], chipper operator [96.2 dB(A)] and bell driver [95.4 dB(A)]. Furthermore, 23% of the samples were above 85 dB(A) and 59% were above 85 dB(A) but below 95 dB(A).

The lowest area noise level recorded at sawmill A was 75.9 dB(A) from the workshop, conversely, the highest was 97.9dB(A) recorded at the knotty pine and profile door. The area noise level of 97.6dB(A) was recorded from the dry mill. Similarly, the lowest noise level of 75.8 dB(A) was recorded from the workshop at sawmill B, while the highest noise level of 98.0dB(A) was recorded at the knotty pine and profile door. The area noise level of 97.7dB(A) was recorded at the dry mill. These findings are below the 130 dB(A) average noise level recorded by Ratnasingam *et al.* (2010) in a study conducted at a rough milling section of the wooden furniture industry in South East Asia. Furthermore, noise levels ranging from 85 to 110 dB(A) were recorded by Antonio *et al.* (2013) in Italian sawmills while Vermal *et al*. (2010) recorded noise levels between 55 and 117 dB(A) at the Ontario sawmills. In the present study, 36% of the samples for area noise levels obtained at the sawmills were below the 85 dB(A) NIOSH REL and the ACGIH TLV, whereas 64% of the samples were above the 90 dB(A) OSHA PEL. These findings are inconsistent with those of Thepaksorn *et al*. (2017a) who reported the lowest average noise level of 86.0 dB(A) recorded in the grading, packaging and storage department and the 88.4 dB(A) highest average noise level recorded at the area for sawing of the lumber into sheets.

Choudhari, Dhote and Patil (2011) carried out a similar study at sawmills and reported the noise level of 90 dB(A) from the silc machine, 108 dB(A) from the chain saw and 101 dB(A) for the planer machine. Ugbebor and Yorkor (2015) reported the results for area noise levels measured during a monitoring exercise at Rumuosi sawmill ranging from 88.0 to 94.1 dB(A) with a mean (SD) of 92.49±1.91 dB(A). The measured noise levels at Mile 3 were reported to range from 84.4 to 94.2 dB(A) with a mean (SD) of

92.44±3.41 dB(A). Furthermore, the field measurement results at Mile 1 ranged from 66.2 to 94.3 dB(A) with a mean (SD) of 92.0±9.55 dB(A). These aforementioned results differ from the findings of the present study because the highest area noise level recorded at sawmill A was 103.5 dB(A) at the chipper machine next to the dry mill and 103.1 dB(A)at sawmill B. Moreover, these findings contradict those of Ratnasingam *et al.* (2010) who reported an average noise level of 150 dB(A) from the sawmill moulder at the rough mill section and the 110dB(A) noise level from the high speed router at the machine section.

Aremu, Aremu and Olukanni (2015) conducted a study at sawmill factories and reported the background noise level ranging from 58.1 to 64.86 dB(A), while the machine equivalent noise level recorded ranged from 81.1 to 112.3 dB(A). The maximum noise level for a combination of machine operations ranged from 105.6 to 121.7 dB(A) and 73% of the measurements obtained were above the 85 dB(A) NIOSH REL. The results of the said study differ from the findings of the present study because only 64% of the area noise samples obtained were above the 85 dB(A) NIOSH REL. However, Boateng and Amedofu (2004) conducted a similar study at the printing mill, corn mill and sawmill and reported the noise level above the 85 dB(A) noise rating limit at the corn mills and sawmills, while the average noise level measured at the printing mill was 85 dB(A). A high proportion of employees at the corn mills and sawmills and a few at the printing mill reported having experienced some form of NIHL. Moreover, in the said study, a highly significant correlation was found between the noise exposure levels, duration of exposure and the development of NIHL among employees at the corn mills and sawmills except at the printing mill.

Ebe *et al*. (2019) in their study reported that the noise levels at sawmills ranged from 96.15 to 101.65 dB(A) at Ogbosisi and 93.19 to 94.96 dB(A) at an industrial market in Umuonyeali Mbieri. Ugwoha, Momoh and Arusuraire (2016) performed a similar study and reported the background noise levels ranging from 70.58 to 79.70 dB(A) at Mile 3, Mile 1 and Rumuosi sawmill. The noise levels recorded from the machines ranged from 89.76±0.09 to 100.49±0.20dB(A) at Mile 3, 89.81±0.13 to 97.00±0.46 dB(A) at Mill 1 and 89.76±0.07 to 100.10±0.53 dB(A) at Rumuosi sawmill. In addition, Robinson *et al.* (2015) conducted a cross-sectional study among 124 woodworkers (88 carpenters and 36 sawyers) using pure-tone audiometry between the frequencies of 0.5 and 8 kHz to ascertain participants' hearing status and assess noise levels at

selected workplaces. They reported that 31% of the carpenters and 44% of the sawyers met the 7% criteria for NIHL, while 17% met the WHO criteria for hearing impairment. The recorded noise levels at various workplaces ranged from 71.2 to 93.9 dB(A) and were inconsistent with the results of the present study with a range of 86.3 to 101.2 dB(A) at sawmill A and 88.3 to 96.9 dB(A) at sawmill B.

Daniell *et al*. (2006) contend that most companies give limited or no attention to noise controls and rely primarily on HPDs to prevent hearing loss, yet 38% of employees do not utilise HPDs regularly. Neitzel, Seixas and Camp (1999) caution that depending solely on the use of HPDs is not a recommended approach in the real industry because empirical studies indicate that its usage rates are less than 50%. The HPDs that were used by the participants in the present study had an NRR of 25 dB and the minimum NRR for the real world was estimated to be 9 dB when using the derating formula. The NIOSH and OSHA recommended a 50% derating factor depending on the type of HPDs subject to fit test (NIOSH, 1998). Daniell *et al.* (2006) conducted a study in sawmills to assess the effectiveness of using HPDs among sawmill employees. They found that the use of HPDs was high when hearing conservation programmes (HCP) were mostly complete, indicating that the under-use of HPDs was, in some instances, caused by incomplete or inadequate company efforts.

Mandryk, Alwis and Hocking (2000) conducted a study in Australian sawmills and discovered that the prevalence of frequent headaches among sawmill workers was significantly higher among the dry mill and green mill staff as compared to the control group. In addition, Aremu, Aremu and Olukanni (2015) indicate that the most prominent health complaints reported by sawmill staff were tinnitus (96.6%), headache (86.6%) and hearing loss (71.9%). Thepaksorn *et al*. (2017b) discovered a 22.8% prevalence of NIHL and stated that male employees reported having a significantly increased risk of NIHL than female workers. In view of the aforesaid, Mandryk, Alwis and Hocking (2000) assert that it is unlikely that frequent headaches are caused by noise exposure because all the participants wore HPDs during their shifts. However, Thepaksorn *et al*. (2017b) state that about 25% of the employees were trained on the proper use of PPE but half of them never or rarely wore PPE while working. The following are reasons given for the non-utilisation of PPE: discomfort (58%); a lack of knowledge of HPDs (42%); and communication barriers (25%). These results differ from the present study because the participants in the exposed group (87%) reported

that they always wear PPE while on site and the participants in the control group (75%) reported that they wear PPE sometimes. The reason for not using PPE in the exposed group (48%) was due to its unavailability. All the participants in the control group stated other reasons.

Chadambuka, Mususa and Muteti, (2003) conducted a study at a mine clinic among 169 employees aged between 19 and 63 years who were tested for NIHL in both ears. Their findings revealed that the employees experienced NIHL due to working in noisy environments (53.2%), intermittent but very loud sounds (5.9%) and improper use of HPDs (40.8%). About 140 (82.8%) of the employees reported using HPDs because they were always exposed to noise and 13 (7.5%) reported using HPDs whenever they entered a noise zone. One hundred and sixteen (68.6%) of the workers reported using earplugs, while 53 (31.4%) used earmuffs and about 160 (94.7%) of workers reported that they were trained on the use of HPDs. Moreover, the noise levels recorded were 94 dB(A) at the plant processing area, 102 dB(A) at the underground mining area and 103 dB(A) at the underground workshop. Sixty-two workers (36.7%) reported having NIHL and the usage of hearing protectors was high among those exposed to noise levels above 85 dB(A) and 95 dB(A); the self-reported use of HPDs was 84% for 73% of the time (Davies *et al*., 2009).

Presently, most sawmill workers are not enrolled in effective hearing loss conservation programmes (HCP) due to the nature of their work and the workplace locations. The percentage of NIHL can be lowered through a broader approach including noise exposure prevention, identification of noise sources, periodic audiological evaluation of those working in noise zones and engineering control backed up by using fit-tested HPDs. The procedure adopted can include noise measurements, an audiometric evaluation and an assessment of clinical history. The secretion of catecholamine can decrease when workers wear hearing protection against noise exposure (Stansfeld and Matheson, 2003).

4.5. Conclusion

Sawmill workers were exposed to noise levels above 82 dB(A) action level and 85dB(A) noise rating (Noise Induced Hearing Loss Regulations, 2003). Consequently, they are at a moderate to high risk of developing NIHL. Furthermore, exposure to noise levels above the action level and noise rating limit can cause auditory and non-auditory health effects such as cardiovascular disease, psychological stress and hypertension (Neitzel, Andersson and Andersson, 2016; Neitzel, Fligor and World Health Organisation, 2017). In addition, exposure to noise levels above 75 dB(A) among men may increase the risks of coronary heart disease (CHD), while exposure to noise levels between 75 and 80 dB for less than 20 years may increase the risks of ischemic heart disease (IHD) (Dzhambov and Dimitrova, 2016; Eriksson, 2019). In this study, the noise exposure levels among sawmill workers have been determined and this information is necessary to assist the sawmill industry to implement control measures to mitigate exposure.

4.6. References

Ahmed *et al.* (2001) Occupational noise exposure and hearing loss of workers in two plants in eastern Saudi Arabia. *Annals of Occupational Hygiene* **45**:371-380.

Anthony, R.T. (2016) *Noise monitoring*. Available from:https://hwc.public-health.uiowa.edu/wp-content/uploads/2016/05/3D-Renee-Anthony-Conducting-Noise-Exposure-Assessments.pdf. (Accessed on 14 March 2023).

Antonio *et al*. (2013) The assessment of the sawmill noise. *Journal of Agricultural Engineering* **155**:768-772.

Aremu, A.O., Aremu, A.S. and Olukanni, D.O. (2015) Assessment of noise pollution from sawmill activities in Ilorin, Nigeria. *Nigerian Journal of Technology* **34**:72-79.

Ayaz, M. (1991) Noise hazard in sawmilling industry in Pakistan. *Pakistan Journal Forest* **41**:187–194.

Azizi, M. (2010) Occupational noise induced hearing loss. *International Journal of Occupational and Environmental Medicine* **1**:116-123.

Basner *et al*. (2014) Auditory and non-auditory effects of noise on health. *Lancet* **383**:1325–1332.

Boateng, C.A. and Amedofu, G.K. (2004) Industrial noise pollution and its effects on the hearing capabilities of workers: A study from saw mills, printing presses and corn mills. *African Journal of Health Sciences* **11**:55-60. DOI: 10.4314/ajhs.v11i1.30778.

Cantley, L.F., Galusha, D. and Cullen, M.R. (2015) Association between ambient noise exposure, hearing acuity, and risk of acute occupational injury. *Scandinavian Journal of Work, Environment and Health* **41**:75–83.

Chadambuka, A., Mususa, F. and Muteti, S. (2013) Prevalence of noise induced hearing loss among employees at a mining industry in Zimbabwe. *Afr Health Sci*. **13**(4):899-906. DOI:10.4314/ahs.v13i4.6.

Chang, T., Hwang, B. and Liu, C. (2013) Occupational Noise Exposure and Incident Hypertension in Men: A Prospective Cohort Study. *American Journal of Epidemiology* **177**: 818–825.

Chang, T.Y., Liu, C.S. and Huang, K.H. (2011) High-frequency hearing loss, occupational noise exposure and hypertension: a cross-sectional study in male workers. *Environ Health* **10**: 2 – 8. Available from: https://doi.org/10.1186/1476-069X-10-35. (Accessed on 14 March 2021).

Choudhari, V. P., Dhote, D. S. and Patil, C. R. (2011) Assessment and control of sawmill noise. International Conference on Chemical, Biological and Environmental Sciences (ICCBES). *Bangkok* **1**(1):299– 303.

Casella. (2022) *Casella CEL-120 acoustic calibrators user guide*. Available from: https://manuals.plus/cassella/cel-120-acoustic-calibrators-manual.pdf. (Accessed on 20 March 2021).

Concha-Barrientos, M. Campbell-Lendrum, D. and Steenland, K. (2004) *Occupational noise: Assessing the burden of disease from work-related hearing impairment at national and local levels*. Geneva: WHO.

Daniell, W.E., Swan, S.S. and McDaniel, M.M. (2006) Noise exposure and hearing loss prevention programmes after 20 years of regulations in the United States. *Occupational and Environmental Medicine* **63**: 343-351.

Davies *el al*. (2009) Occupational noise exposure and hearing protector use in Canadian Lumber Mills. *Journal of Occupational and Environmental Hygiene* **6**:32-41. DOI:10.1080/15459620802548940.

de Souza, T.C.F., Périssé, A.R.S. and Moura, M. (2015) Noise exposure and hypertension: investigation of a silent relationship. *BMC Public Health* 15:328 (the last page number is missing) Available from: https://doi.org/10.1186/s12889-015-1671-z. (Accessed on 14 March 2021).

Dost, W. A. (1974a) Noise levels in soft wood lumber mills. *Forest Products Journal* **24**:25–27.

Dost, W. A. (1974b) Sawmill noise at the operating level. *Forest Products Journal* **24**:13–17.

Driscoll, D.P.P.E., Milk, K. and Burgess, M. (2009) *W503 Student manual noise: measurements and its effects*. British: British Occupational Hygiene Society (BOHS).

Dzhambov, A.M. and Dimitrova, D.D. (2016) Occupational Noise and Ischemic Heart Disease: A Systematic Review. *Noise Health* **18**(83):167–177.

Ebe, T.E., Njoku-Tony, R.F. and Emereibeole, E.I. (2019) Assessment of noise exposure levels of sawmill workers at Ogbosisi and Mbieri Timber and Allied Industrial Market in Owerri, Imo State. *International Journal of Advanced Research (IJAR)* **7**(1): 889-893.

Eriksson, H. (2019) *Work-related cardiovascular disease*. (*P.h.D. Thesis*). Sahlgrenska: University of Gothenburg.

European Commission. (2020) *Exposure to physical hazards*: *Directive 2003/10/EC-Noise of 6 February 2003 on the minimum health and safety requirements regarding the exposure of workers to the risks arising from physical agents (noise) (17 individual Directive within the meaning of Article 16(1) of Directive 89/391/EEC)*. Available from: https://eur-lex.europa.eu/LexUriServ/LexUriServ.do?uri=OJ:L:2003:042:0038:0044:EN:PDF. (Accessed on 4 April 2021)

Ferrite, S. and Santana, V. (2005) Joint effects of smoking, noise exposure and age on hearing loss. *Journal of Occupational Medicine* **55**:48–53.

Fisnea, A. and Oktenb, G. (2017) Noise exposure of underground coal miners in Turkey. *Noise Control Engineering Journal* **61**:553- 566.

Franz, M. (2007) Handbook on mine occupational hygiene measurements. Available from:https://mhsc.org.za/sites/default/files/public/publications/Handbook%20Pages%20124-246.pdf. (Accessed on 12 June 2022).

Gan, W.Q., Davies, H.W. and Demers, P.A. (2011) Exposure to occupational noise and cardiovascular disease in United States:National Health and Nutrition Examination Survey 1999–2004. *Occupational & Environmental Medicine* **68**:183-190.

Gerges, S.N.Y., Sehrndt, G.A. and Parthey, W. (n.d.) *Noise sources*. Germany: Federal Institute for Occupational Safety and Health.

Goelzer, B., Hansen, C.H. and Sehrndt, G. (2001) *Occupational exposure to noise evaluation, prevention and control*.Available from: https://edocs.tib.eu/files/e01fn02/346867487.pdf. Accessed on 4 April 2021).

Hong, O, Kerr F.M.J. and Poling, G. (2013) Understanding and preventing noise-induced hearing loss. *Disease-a-Month* **59**:110–118.

I-INCE. (1997) *Final report, technical assessment of upper limits on noise in the workplace*. I-INCE Publication 97-1, noise/news international (1997), 203-216.

ISO. (1990) *Acoustics — Determination of occupational noise exposure and estimation of noise-induced hearing impairment, ISO-1999*. Geneva: ISO.

ISO. (1979) *Acoustics — Guidelines for the measurement and assessment of exposure to noise in a working environment, ISO-9612*. Geneva: ISO.

ISO. (2009) *Acoustics — Determination of occupational noise exposure — Engineering method, ISO 9612:2009(E)*. 2nd ed. Geneva:ISO.

Koehncke *et al*. (2003) An investigation of noise levels in Alberta sawmills. *American Journal of Industrial Medicine* **43**(2):156–164.

Lahav, A. and Skoe, E. (2014) An acoustic gap between the NICU and womb: a potential risk for compromised neuroplasticity of the auditory system in preterm infants. *Frontiers in Neuroscience* **8**:1-8. DOI:10.3389/fnins.2014.00381.

Le, T.N., Straatman, L.V.and Lea, J. (2017) Current insights in noise-induced hearing loss: a literature review of the underlying mechanism, pathophysiology, asymmetry, and management options. *J Otolaryngol Head Neck Surg* **46**:1-15.

Leensen, M.C.J., Van Duivenbooden, J.C. and Dreschler, W.A. (2011) A retrospective analysis of noise-induced hearing loss in the Dutch construction industry. *Int Arch Occup Environ Health* **84**:577–590.

Lester, H., Malchaire, J. and Arbey, H.S. (2001) *Strategies for noise surveys*. In Goelzer, B., Hansen, C.H. and Sehrndt, G.A. (eds.). *Occupational Exposure to Noise Evaluation, Prevention and Control*. Geneva: WHO.

Lie, A., Skogstad, M. and Johannessen, H.A. (2016) Occupational noise exposure and hearing:A systematic review. *International Archives of Occupational and Environmental Health* **89**:351–372. DOI: 10.1007/s00420-015-1083-5.

Mandryk, J., Alwis, K.U. and Hocking, A.D. (2000) Effects of personal exposures on pulmonary function and work-related symptoms among sawmill workers. *Annals of Occupational Hygiene* **44**:281-289.

May, J.J. (2000) Occupational hearing loss. *American journal of industrial medicine* **37(1)**: 112-120. Doi:10.1002/(sici)1097-0274(200001)37:1112::aid-ajim93.0co;2-#.

Mizoue, T., Miyamoto, T. and Shimizu, T. (2003) Combined effect of smoking and occupational exposure to noise on hearing loss in steel factory workers. *Occupational and Environmental Medicine* **60**:56-59.

Münzel, T., Gori, T. and Babisch, W. (2014) Cardiovascular effects of environmental noise exposure. *European Heart Journal* **35**:829-836.

Nandi, S.S. and Dhatrak, S.V. (2008) Occupational noise-induced hearing loss in India. *Indian Journal of Occupational and Environmental Medicine* **12**: 53-56.

Neitzel, R., Andersson, M.E. and Andersson, E.E. (2016) Comparison of multiple measures of noise exposure in paper mills. *Annals of Occupational Hygiene* **60**:581–596. DOI:10.1093/annhyg/mew001.

Neitzel, R. and Fligor, B. (2017) Determination *of risk of noise-induced hearing loss due to recreational sound: A review*. Geneva, Switzerland: WHO.

Neitzel, R., Seixas, N.S. and Camp, J. (1999) An assessment of occupational noise exposures in four construction Trades. *American Industrial Hygiene Association Journal* **60**: 807–817

Nelson *et al*. (2005) The global burden of occupational noise-induced hearing loss. *American Journal of Industrial Medicine* **46**: 446-458.

NIOSH. (1998) *Criteria for recommended standard. Occupational noise exposure.* USA: NIOSH

NIOSH. (1997) *Health hazard evaluation: Noise and hearing loss 1986 - 1997*. Available from: https://www.cdc.gov/niosh/docs/99-106/pdfs/99-106.pdf?id=10.26616/NIOSHPUB99106. (Accessed on 29 April 2021).

Phillips, S., Henrich, V.C. and Mace, S.T. (2010) Prevalence of noise-induced hearing loss in student musicians. *International Journal of Audiology* **49**:309-316.

Picarda, M., Girard, S.A. and Simard, M. (2008) Association of work-related accidents with noise exposure in the workplace and noise-induced hearing loss based on the experience of some 240,000 person-years of observation.*Accident Analysis Prevention* **40**:1644-1652.

Pouryaghoub, G. Mehrdad, R. and Mohammadi, S. (2007) Interaction of smoking and occupational noise exposure on hearing loss: A cross-sectional study. *BMC Public Health* **7**:137-139. DOI:10.1186/1471-2458-7-137.

Rabinowitz, P.M. (2000) Noise-Induced Hearing Loss. *Am Fam Physician* **61**(9):2759-2760.

Ratnasingam *et al*. (2010) Dust, noise and chemical solvents exposure of workers in the wooden Furniture Industry in South East Asia. *Journal of Applied Sciences* **10**:1413-1420.

Robinson, T., Whittaker, J. and Acharya, A. (2015) Prevalence of noise-induced hearing loss among woodworkers in Nepal: a pilot study. *International Journal of Occupational and Environmental Health* **21**:14-22.

Rösler, G. (1994) Progression of hearing loss caused by occupational noise. *Scandinavian Audiology* **23**:13-37. DOI:10.3109/01050399409047483.

Ruedy, T.C., Lamb, F.M. and Johnson, J.A. (1976) Noise survey of a small wood products company. *Forest Products Journal* **26**:38–44.

SANS 10083. (2013) The measurement andassessment of occupational noise for hearing conservation purposes. Pretoria: Government printer.

Sbihi, H., Davies, H.W. and Demers, P.A. (2008) Hypertension in noise-exposed sawmill workers: A cohort study. *Occupational & Environmental Medicine* **65**:643-646.

Stansfeld, S.A. and Matheson, M.P. (2003) Noise pollution: non-auditory effects on health. *British Medical Bulletin* **68**(1):243–257.

Noise Induced Hearing Loss Regulations. (2003) *Noise induced hearing loss regulations, GNR.307. Government Gazette No. 24967*. Pretoria: Government Printer.

Starck, J., Toppila, E. and Pyykko, I. (2003) Impulse noise and risk criteria. *Noise Health* **5**:63-73.

Suter, A.H. (2002) *Noise, standards and regulations*. *ILO Encyclopaedia of occupational health and safety*. Available from: http://www.ilocis.org/en/default.html. (Accessed on 4 March 2021)

SVANTEK. (2016) *SV104IS user's manual*. Warsaw, Poland: Svantek.

SVANTEK. (2015) *SV104 & SV104IS noise dosimeters*. Warsaw, Poland: Svantek.

Themanna, C. and Masterson, E.A. (2019) Occupational noise exposure: A review of its effects, epidemiology, and impact with recommendations for reducing its burden. *The Journal of the Acoustical Society of America* **146**:3879-3905.

Thepaksorn *et al*. (2017a) Job safety analysis and hazard identification for work accident prevention in para rubber wood sawmills in southern Thailand. *J. Occup. Health* **59**:542-551. DOI:10.1539/joh.16-0204-CS.

Thepaksorn *et al*. (2017b) Occupational noise exposure and hearing defects among sawmill workers in the south of Thailand. *International Journal of Occupational Safety and Ergonomics* **25**(3):458-466. DOI:10.1080/10803548.2017.1394710.

Tikka *et al*. (2017) *Interventions to prevent occupational noise-induced hearing loss*. Cochrane database of systematic reviews, Issue 7. Art. No.: CD006396. DOI: 10.1002/14651858.CD006396.pub4

Tingay, J. and Robinson, D. A (2014) *Practical comparison of occupational noise standards*. United Kingdom: Cirrus Research plc.

TSI Incorporated. (2018) *Soundpro integrated sound level meters' series SE/DL user manual*. Available from:https://data2.manualslib.com/pdf6/139/13866/1386535-3m/spse211.pdf?a65b6207429d9156382228b449f117a3&take=binary. (Accessed on 4 March 2021).

Ugbebor, J.N. and Yorkor, B. (2015) Assessment and Evaluation of Noise Pollution Levels in Selected Sawmill Factories in Port Harcourt, Nigeria. *International Journal on Emerging Technologies* **6**:01-08.

Ugwoha, E., Momoh, Y. and Arusuraire, F.E. (2016) Assessment of noise pollution in selected sawmills in Port Harcourt. *Int. Journal of Engineering Research and Application* **6**:20-25.

Vermal *et al*. (2010) Occupational health and safety issues in Ontario sawmills and veneer/plywood plants: A pilot study. *J Environ Public Health* **1**: 526-487.

Chapter 5

Hearing loss and respiratory symptoms among sawmill workers of the timber processing factories within the Gert Sibande District Municipality

This Chapter has been submitted for publication in the *Journal of BMC Public Health.*

ISSN 1471 – 2458

Abstract

Wood dust is linked with respiratory symptoms while exposure to extreme noise is linked with NIHL. The objective of the study was to assess prevalence of hearing loss and respiratory symptoms and use of PPE among sawmill workers. A comparative cross sectional study comprising of 137 exposed workers and 20 unexposed randomly selected workers was undertaken from January to March 2021. The respondents completed a semi-structured questionnaire on hearing loss and respiratory symptoms. Discrete data are represented as percentages and tables. The *t*-test was used to compare continuous variables, while the chi-square test was used to test categorical responses. Fisher's test was employed when the anticipated number was below 5. A significance level of 0.05 was used.

Chest pains or shortness of breath were the most common respiratory symptoms among unexposed (50%) and exposed group (44%). Tinnitus (ringing in the ears) was the most common conditions or symptoms of hearing loss among exposed (50%) and unexposed group (33%) while ear infections was also common among unexposed (66.67%) and exposed group (21.43%). Exposed group (86.86%) reported always wear PPE than unexposed (75%) who wear it sometimes. Exposed group (48.48%) did not wear PPE consistently due to not being available than unexposed (100%) who reported other reasons

Prevalence of chest pains or shortness of breath among the unexposed was higher than exposed while tinnitus (ringing in the ears) among the exposed was higher than the unexposed and ear infections among the unexposed was higher than the exposed. The findings suggest that sawmill owners should take steps to lower the level of exposure to noise and wood dust at the sawmill to help protect workers' health.

Key words: Wood dust, respiratory symptoms, noise, sawmill workers, hearing loss

5.1. Introduction

A timber processing factory or lumber mill is a workplace where fresh logs or dry timber is processed (Beiler's Sawmill, n. d; Guyana Forestry Commission, 2012). Fresh logs are normally processed at the wet mill (green chain), while kiln-dried timber is usually processed at the dry mill (Mandryk, Alwis and Hocking, 2000). These processes are normally performed in an open shed or enclosure where the supply of natural ventilation is a main problem (Tobin *et al*., 2016). Likewise, the use of old machines and poor repair of plant and LEV systems are some of the major factors contributing to excessive noise at the wet and dry mills (Adeoye *et al*., 2014; Judd and Janice, 2004). Kitcher *et al*. (2014) performed a comparative cross-sectional study among 101 market mill workers and 103 small-scale traders in the city of Accra, Ghana. They reported that 24% of mill workers' had more symptoms of hearing loss compared with 8% of controls, even though 55% of mill workers showed more knowledge of the health effects of noise on health than 52% of controls. Yet, only 5% of the mill workers were reported to use HPDs. There was a presence of notable sensorineural hearing loss at mean threshold frequencies of 2, 3, and 4 kHz among the mill workers compared with the control. The incidence of NIHL in the right ear was 25% higher in mill workers compared with 5% in controls. Contrary to that, Adeoye *et al*. (2014) carried out a similar comparative cross-sectional analytical study among 50 randomly selected sawmill workers and 50 controls in Osun State, Nigeria. They reported that the incidence of respiratory symptoms such as cough (60%), phlegm production (46%), and sneezing (54%), among sawmill workers, were significantly higher than 20% cough, 10% phlegm production, and 18% sneezing among controls. This finding was supported by an earlier comparative cross-sectional study conducted by Mandryk, Alwis and Hocking (2000) who reported a higher incidence of cough, phlegm, nasal secretion, runny nose, eye irritation, and frequent headaches among sawmill workers compared with control.

Shamssain (1992) reported that the incidence of nasal symptoms (49%), cough (43%) and phlegm production (15%) were higher among the exposed than the control group. It was specified that the frequency of respiratory problems such as cough and nasal symptoms increased with the number of years of employment. Ugheoke, Wahab and Erhabor (2009) reported that the prevalence of cough (35%), sputum production (52%), breathlessness (8%) and wheezing (3%) among sawmill workers was higher than the control. Respiratory problems such as coughs, phlegm production, dyspnea, wheezing, and a runny nose have been found worldwide to be more common among exposed sawmill workers compared with control workers (Ige and Onadeko, 2000; Mandryk, Alwis and Hocking, 2000; Okwari *et al*., 2005; Ugheoke, Wahab and Erhabor, 2009). Substantial relationships have also been established between wood dust and decline in lung functions among sawmill workers (Anderson and Meade, 2014; Douwes *et al*., 1992; Douwes *et al*., 2000; Hayes et al., 1986a; Hernberg *et al*., 1983; Mandryk, Alwis and Hocking, 2000; Shamssain, 1992).

Inhaling wood dust may increase the incidence of non-asthmatic chronic airflow obstruction (NACAO), occupational asthma (OA), extrinsic allergic alveolitis (EAA) (hypersensitivity pneumonitis), organic dust toxic syndrome (ODTS) and simple chronic bronchitis (SCB) (mucus hypersecretion) (Enarson and Chan-Yeung, 1990; Malmberg, 1990; Mandryk, Alwis and Hocking, 1999; Ratnasingam *et al*., 2014; Shamssain, 1992). Occupational asthma, NACAO, and respiratory problems were linked with allergic and non-allergic softwood exposure (Demers, Teschke and Kennedy, 1997). Fransman *et al*. (2003) and Vendal *et al.* (1986) and Chan-Yeung *et al*. (1980) indicated that after 10 years' exposure to western red cedar, symptoms of asthma are more prevalent. Western red cedar is associated with a decrease in lung function impairment, leading to NACAO (Noertjojo *et al*.,1986). Wood dust exposure is also linked with dermal, respiratory, and nasal cancers (Demers *et al*., 1995; Demers *et al*., 2000; Kauppinen *et al*., 2006; Hayes *et al*., 1986a; Hayes *et al*., 1986b; Leclerc *et al*., 1994; Vaughan *et al*., 2000).

Likewise, nearly 30 million workers worldwide are exposed to ototoxic chemicals such as chemotherapeutic agents comprising of cisplatin, nitrogen mustard, furosemides, and salicylates, which are harmful to their ears and dangerous to hearing, while 22 million are exposed to noise considered dangerous by the WHO (Hammer, Swinburn and Neitzel, 2014; Rabinowitz, 2000). Noise levels above 85 dB(A) can cause auditory effects such as temporary changes in hearing, tinnitus, and NIHL, as well as non-auditory effects such as annoyance, stress, hypertension, and heart disease (Concha-Barrientos, Campbell-Lendrum and Steenland, 2004; Clark and Bohne, 1999; Nelson *et al*., 2005). There is a robust relationship between exposure to noise levels above 85 dB(A) and NIHL and diastolic blood pressure measurement above 90 mmHg (Talbott *et al*., 1985). Noise exposure is also linked with high blood pressure and ischemic heart disease, even though there are limited epidemiologic studies (van Kempen *et al*., 2002). Noise exposure causes temporary or permanent hearing loss, which has a negative impact on workers' health and well-being (Fernández *et al*., 2009). Other health outcomes consist of lack of attentiveness, annoyance, exhaustion, headaches, and sleep disruption (Nandi and Dhatrak, 2008). Dost (1974a) carried out a noise survey at a California lumber mill and reported the highest average noise levels of 106 dB(A) for the tail sawyer, 107 dB(A) for the chipper tender, and 115 dB(A) for the planner machine that were beyond the upper exposure action value of 85 dB(A), and the second paper reported the maximum noise levels of 104.2 dB(A) for the planer and 104.5 dB(A) for the tail sawyer (Dost, 1974b).

In another study, Ayaz (1991) conducted a noise survey at the Pakistani sawmills and reported average noise levels ranging from 90 to 113 dB(A). It was reported that sawmill workers were at a significantly higher risk of suffering from hearing disabilities and noise-induced occupational health disorders than sawmill workers in other countries. Similarly, continuous exposure to noise levels above 85 dB(A) is the leading cause of NIHL (Cantley *et al*., 2015; Hong *et al*., 2013; Phillips, Henrich and Mace, 2010; Rabinowitz, 2000). This is a bilateral hearing loss with an audiometric notch at frequencies of 3, 4, and 6 kHz with a recovery at 8 kHz (Chang *et al*., 2011; Chang *et al*., 2013; Leensen, van Duivenbooden and Dreschler, 2011; Lester *et al*., 2001; Lie *et al*., 2016; May, 2000; Rabinowitz, 2000). This audiometric notch deepens and slowly advances towards lower frequencies of 0.5, 1, and 2 kHz if noise exposure continues (Rösler, 1994). NIHL occurs mostly at frequencies of 3 to 6 kHz, with the largest effect at 4 kHz (Nandi and Dhatrak, 2008; Pouryaghoub, Mehrdad and Mohammadi, 2007).

It has been reported that the use of ototoxic drugs such as aminoglycosides, ototoxic and non-ototoxic chemical substances such as toxic solvents in paint or organophosphate pesticides, as well as aging, smoking cigarettes, heat exposure, diabetes, rheumatoid arthritis, and exposure to high noise levels above 85 dB(A) can increase the risks of developing NIHL (Ferrite and Santana, 2005; Gan, Davies and Demers, 2011; Mizoue, Miyamoto and Shimizu, 2003; Phillips, Henrich and Mace, 2010; Pouryaghoub, Mehrdad and Mohammadi, 2007). Cigarette smoking and exposure to excessive noise increase the risks of NIHL (Cruickshanks *et al*., 1998; Ferrite and Santana, 2005; Mizoue, Miyamoto and Shimizu, 2003; Palmer *et al*., 2004; Robert, 2008). Information about the prevalence of hearing loss such as tinnitus, temporary and permanent threshold shifts, acoustic trauma, and permanent sensorineural hearing loss, as well as respiratory problems such as cough, catarrh, breathing difficulty, wheeziness, and nasal symptoms among sawmill workers of the timber processing factories, is lacking in South Africa, particularly at the Gert Sibande District Municipality. The purpose of the study was to evaluate the occurrence of hearing loss and respiratory problems among sawmill workers within the Gert Sibande Municipality in Mpumalanga Province, South Africa.

5.2. Methodology

5.2.1. Setting of the study

The study was conducted at the sawmill factories located within the Gert Sibande District Municipality in Mpumalanga Province, South Africa. A comparative cross-sectional study comprising 137 exposed and 20 unexposed workers randomly selected using simple random sampling was undertaken at the sawmill factories. Furthermore, the study was conducted from January to March 2021. The estimated sample size for the study was 236 based on the Cochran formula for determining sample size (Adeoye *et al*., 2014; Kitcher *et al*., 2014; Israel, 1990; Lemeshow *et al*.,1990; Rosner, 2010). There were 12 sawmill factories located within the Gert Sibande District municipality. Out of 12 sawmills, two were the largest, while 10 were medium-sized. The study focused on large sawmills because they were more active than medium sawmills. These criteria were chosen because large mills have more employees than medium or small mills, and their operations represent a typical sawmill enterprise within the Gert Sibande District. The two largest sawmills were selected based on their size, location of the sawmills, and type of wood being processed (Alwis, 1998; Alwis, Mandryk *et al*., 1999; Alwis, Mandryk and Hocking, 1999; Koehncke, 1999; Yamanaka, 2000).

The types of trees being processed were predominantly pine, mostly Pinus radiata, Pinus patula, Pinus elliottii, and others.

5.2.2. Participants

Sawmill workers were grouped into exposed and unexposed groups. The exposed group consisted of operators, packers, receivers, artisans, stackers, feeders, general workers, and cleaners, while the unexposed group consisted of administrative staff (office) and maintenance workers whose jobs did not expose them to wood dust or noise (Alwis, 1998). The inclusion criteria involved all consenting females and men aged between 18 and 65 years who have been employed for more than six months and who were willing to participate, while those excluded were workers who did not give consent or were not willing to participate, and those with less than six months' experience at the time of the study.

5.2.3. Validity and reliability

The questionnaire was developed based on similar research studies published elsewhere (Adeoye *et al.*, 2014; Kitcher *et al.*, 2014; Mandryk, Alwis and Hocking, 2000; Tobin *et al.*, 2016; Judd and Janice, 2004; Shamssain, 1992; Rongo *et al.,* 2002; Löfstedt *et al.*, 2017; Schlünssen *et al.*, 2002; Thepaksorn *et al.,* 2013; Thepaksorn *et al.*, 2017). The question was adopted from previous research and literature to suit the needs of the sawmill industry.

5.2.4 Piloting of the questionnaire

The questionnaire was piloted to six workers in each mill and workshop. The participants of the pilot study consisted of a stacker, an operator, a general worker, and three office staff. This was done to check for the clarity of the questions, the validity of the content, and the reliability of the questionnaire. Feedback from the pilot study was incorporated into the final questionnaire. Participants of the pilot study were excluded from the main study. The questionnaire was reviewed by experts in the field of occupational hygiene to determine its applicability to the sawmill industry.

Furthermore, it was pre-tested for content validity with the expectation of obtaining the same responses from all respondents. Closed-ended questions were used to reduce bias and improve the tendency to produce the same results.

5.2.5. Data collection

Data gathering was accomplished using a self-administered questionnaire. The questionnaire was adapted and modified from a Medical Research Council questionnaire on chronic bronchitis and noise exposure (Bellia *et al*., 2003; Medical Research Council, 1996; Morata and Little, 2002; Venables *et al*., 1993). Section A of the questionnaire included questions about participants' socio-demographic characteristics such as age, gender, education, *et cetera*. Sections B, C, and D sought to collect data about work-related information such as working hours, job title, work experience, safety training, previous dusty job, and health-related information regarding smoking and respiratory symptoms. Section E included questions about the use of PPE (Ahmed and Abdullah, 2012; Alwis, 1998; Thepaksorn *et al*., 2017; Osman and Pala, 2009; Milanowski *et al*., 2002; Thepaksorn *et al*., 2018). Questionnaire was written in English and interpreted into Isizulu and Afrikaans.

The first step of data collection began with obtaining permission from the Faculty of Health Sciences as well as from the management of the two sawmill factories. Subsequently, meetings were held with participants to clarify the aim and purpose of the study and the distribution of information documents and consent forms, as well as the collection of signed consent forms. Next, the questionnaires were administered to sawmill workers. The data were collected with care to prevent any harm while upholding privacy and confidentiality. Moreover, the anonymity of the respondents was guaranteed by omitting their names from the questionnaire. When the respondents did not understand any terminology, the researcher explained using plain language. The questionnaires were completed during lunch breaks at the sawmills. The participants took about 20 minutes to complete the questionnaires, and data were collected over five days at each worksite. The researcher collected the completed questionnaires for capture. Thereafter, the questionnaires were cross-checked to ensure that all the necessary information was completed correctly. Finally, data were coded and stored electronically in a Microsoft Excel spreadsheet and later analysed.

5.2.6. Ethical considerations and permission

Ethical approval (clearance number: UFS-HSD2019/2236/3006) was obtained from the Health Science Research Ethics Committee of the University of the Free State. Permission to conduct the study was granted by the managers in charge of the sawmill factories and the participants gave consent to participate in the study. An information letter was sent together with the questionnaire stated that if the subjects answered and returned the questionnaire it meant they had given informed consent. Participation in the study was voluntary and participants were allowed to withdraw at any time without any penalty. To ensure confidentiality, anonymity of participants was maintained

5.2.7 Data analysis

Data was analysed using the Microsoft Excel 2019 analysis Toolpak for a summary of descriptive statistics. Discrete data are represented as percentages and tables. The *t*-test was used to compare continuous variables, while the chi-square test was used to test categorical responses. Fisher's test was employed when the anticipated number was below 5. A significance level of 0.05 was used.

5.3. Results

The study targeted 236 participants, but only 157 completed and returned questionnaires, giving a response rate of 67%. Table 5.1 shows the summary statistics for the demographic characteristics of the respondents. Fifty-three percent (53%) of the exposed group were males, 48% were females, and 60% of the unexposed group were males, 40% were females. The exposed had a mean (SD) age of 34,25 (23), while the unexposed had a mean (SD) age of 5. (4,97). The majority of the exposed group (41%) was 40 to 49 years old, while 55% of the unexposed group was 30 to 39 years old. Ninety-two percent (92%) of those exposed and 70% of those not exposed were black. The majority of the exposed group (68%) and 75% of the unexposed group were single. Furthermore, 42% of the exposed group had secondary, while 50% of the unexposed group had matric. The majority of the unexposed group appears to be more educated (had matric and tertiary) than the exposed (who had secondary and matric).

Table 5.1.: Demographic characteristics of the participants

	Exposed group (n=137)	**Unexposed group** (n=20)	***p*-value**
Variable	**n (%)**	**n (%)**	
Gender/sex			
Females	65 (47.45)	8 (40)	0.533[a]
Males	72 (52.55)	12 (60)	
Age group (years)	mean(SD)=34,25(23)	mean(SD)=5(4,97)	
20 to 29	16 (11.68)	2 (10)	0.047[c]
30 to 39	52 (37.96)	11 (55)	
40 to 49	56 (40.88)	7 (35)	
50 to 59	13 (9.49)	0	
Ethnicity/race			
Black	127 (92.70)	14 (70)	0.002[a]*
White	10 (7.30)	6 (30)	
Marital status			
Single	93 (67.88)	15 (75)	0.817[b]
Married	34 (24.82)	5 (25)	
Divorced	8 (5.84)	0	
Widowed	2 (1.46)	0	
Level of education			
None	4 (2.92)	0	0.077[c]
In-house	3 (2.19)	0	
Primary	8 (5.84)	0	
Secondary	58 (42.34)	2 (10)	
Matric	47 (34.31)	10 (50)	
Tertiary	17 (12.41)	8 (40)	

[a]Chi-Square test; [b]Fisher's exact test; [c]Student t-test; *Significant at p<0.05

The work-related data of participants is presented in Table 5.2. A high proportion of the exposed group (30%) were general workers, while 70% of the unexposed group had other job titles. The majority of those exposed (46%) and those who were not exposed (30%) had previously worked in a dusty environment. Ninety-three percent (93%) of those exposed and 100% of those not exposed reported having received safety training. Fourty-four percent (44%) of those exposed and 45% of those not exposed had work experience ranging from three to ten years. Fifty-five percent (55%) of exposed workers work 9 hours a day (45 hours a week), while 55% of unexposed

workers work 8 hours a day (40 hours a week). The exposed group differs from the unexposed group by previous dusty job and the number of years of employment. Even though the exposed group worked fewer hours than the unexposed.

Table 5.2.: Work-related information of the participants

	Exposed group (n=137)	**Unexposed group** (n=20)	***p*-value**
Variable	**n (%)**	**n (%)**	
Job title			
Operator	35 (25.55)	0	n/a
Packer	8 (5.84)	0	
Receiver	2 (1.46)	0	
Artisan	4 (2.92)	0	
Stacker	31 (22.63)	0	
Feeder	1 (0.73)	0	
Supervisor	5 (3.65)	0	
General worker	41 (29.93)	0	
Cleaner	2 (1.46)	0	
Other	8 (5.84)	20 (100)	
Previous dusty jobs			
Yes	63 (45.99)	6 (30)	0.0132[c*]
No	74 (54.01)	14 (70)	
Received safety training			
Yes	127 (92.7)	20 (100)	0.363[b]
No	10 (7.3)	0	
Working experience (years)			
1 to 2	48 (35.04)	4 (20)	0.058[c]
3 to 10	60 (43.8)	9 (45)	
11 to 20	25 (18.25)	6 (30)	
21 to 30	4 (2.92)	1 (5)	
Woking hours			
8 hrs	59 (43.07)	11 (55)	0.152[c]
9 hrs	75 (54.74)	9 (45)	
10 hrs	3 (2.19)	0	

N/A: Not assessed; [a]Chi-Square test; [b]Fisher's exact test; [c]Student t-test; *Significant at p<0.05

The results in Table 5.3 show the prevalence of respiratory symptoms and health-related information reported by respondents. The exposed group reported respiratory symptoms comprising of phlegm (61%), chest pains or shortness of breath (44%), cough (13%), nose and throat irritations (8%) and chest-related illness (3%) while the unexposed group reported chest pains or shortness of breath (50%), cough (10%), and nose and throat irritations (5%). Fifty percent (50%) coughs among the exposed group took place in the morning, 22% day and 28% night while 50% unexposed group reported to occurred in the morning and night. In addition, 61% of the coughs among the exposed were reported to last between 1 to 3 days, 17% between 3 to 5 days, and 22% more than 5 days, while 100% of the coughs in the unexposed group were reported to last between 1 to 3 days. Two percent (2%) of the exposed group reported health-related conditions or diseases comprising of heart failure, chest problems, and pneumonia, while 1% reported pulmonary tuberculosis, and 10% of the unexposed group reported chest-related problems. The unexposed group reported more respiratory symptom for chest pains or shortness (50%) of breath than the exposed group (44.44%).

Table 5.3.: Respiratory symptoms and health-related information the participants

	Exposed group (n=137)	**Unexposed group** (n=20)	***p*-value**
Variable	**n (%)**	**n (%)**	
Do you suffer from nose and throat irritations?			
Yes	11 (8.03)	1 (5)	0.421[c]
No	126 (91.97)	19 (95)	
Do you cough?			
Yes	18 (13.14)	2 (10)	0.371[c]
No	119 (86.86)	18 (90)	
When do you cough?			
Morning	9 (50)	1 (50)	0.0270[c*]
During	4 (22.22)	0	
Night	5 (27.78)	1 (50)	
How long the cough last?			
1 to 3	11 (61.11)	2 (100)	0.110[c]
3 to 5	3 (16.67)	0	
More than 5 days	4 (22.22)	0	
Do you produce phlegm?			
Yes	11 (61.11)	0	0.0700[c]
No	7 (38.89)	2 (100)	
Do you suffer from chest pains/shortness of breath when coughing?			
Yes	8 (44.44)	1 (50)	0.0153[c*]
No	10 (55.56)	1 (50)	
Have you ever suffered from chest related illness in the past?			
Yes	4 (2.92)	0	0.4052[c]
No	133 (97.08)	20 (100)	
Have you had similar illness like this in the past?			
Yes	11 (8.03)	0	0.4217[c]
No	126 (91.97)	20 (100)	
Do you suffer from one of the following health conditions?			
Heart failure/problem	3 (2.19)	0	0.3838[c]
Pneumonia	2 (1.46)	0	
Pulmonary tuberculosis	1 (0.73)	0	
Chest related problem	3 (2.19)	2 (10)	
None of the above	128 (93.43)	18 (90)	

[a]Chi-Square test; [b]Fisher's exact test; [c]Student t-test; *Significant at p<0.05

The smoking status and other health-related information of the respondents are shown in Table 5.4. Eighteen percent (18%) of the exposed group were smokers who smoked more than 25 packets of cigarettes per week (56%) compared to 45% of the unexposed group who smoked more than 25 packets of cigarettes per week (100%). Sixty-eight percent (68%) of the exposed group and 11% of the unexposed group reported planning to reduce smoking, and 60% of the exposed group and 11% of the unexposed group reported requiring assistance to quit smoking. Eighty-five percent (85%) of those exposed and 100% of the unexposed reported undergoing medical fitness test. Sixteen percent (16%) of the exposed and 15% of the unexposed reported their latest medical test report requiring follow-ups with the doctor on health-related conditions (55% and 100%, respectively). The exposed group differs from the unexposed group in terms of smoking habits. There appear to be fewer smokers among the exposed (18%) than the unexposed (45%). This could be attributed more males outnumbering females and may be hesitant to quit smoking due to addiction

Table 5.4.: Smoking related information of the participants

	Exposed group (n=137)	**Unexposed group** (n=20)	***p*-value**
Variable	**n (%)**	**n (%)**	
Do you smoke?			
Yes	25 (18.25)	9 (45)	0.0161[b*]
No	112 (81.75)	11 (55)	
What type of substance do you smoke?			
Cigarette	19 (76)	9 (100)	0.478[b]
Dagga	1 (4)	0	
Other	5 (20)	0	
How many cigarette packets you smoke per week?			
10 packets	0	0	0.0139[b*]
15 packets	3 (12)	0	
20 packets	6 (24)	1 (11.11)	
25 packets	2 (8)	3 (33.33)	
More than 25 packets	14 (56)	5 (55.56)	
Are you planning to stop smoking?			
Yes	17 (68)	1 (11.11)	0.0056[b*]
No	8 (32)	8 (88.89)	
Do you need assistance to stop smoking?			
Yes	15 (60)	1 (11.11)	0.009[b*]
No	10 (40)	8 (88.89)	
Does the company subject you to medical fitness test?			
Yes	117 (85.4)	20 (100)	0.078[b]
I don't know	20 (14.6)	0	
Does your medical test report require follow up?			
Yes	22 (16.06)	3 (15)	0.000[b*]
No	31 (22.63)	17 (85)	
Don't know	84 (61.31)	0	
Follow up relate to the following			
Hearing loss	8 (36.36)	0	0.634[b]
Heart disease	2 (9.09)	0	
Other health condition	12 (54.55)	3 (100)	

[a]Chi-Square test; [b]Fisher's exact test; [c]Student t-test; *Significant at p<0.05

The use of PPE by participants is reflected in Table 5.5. Ear plugs were the most commonly worn PPE by the exposed group (96%) and the occasionally worn PPE were respiratory masks (12%) and safety glasses (9%), while helmets, ear plugs, and safety boots were the most commonly worn PPE by the unexposed group (25%), and and the least worn PPE being protective gloves (5%). PPE was worn regularly by 87% of the exposed group than 75% of the unexposed group who wear it sometimes. The exposed group appears to wear PPE more often than the unexposed group due to the fact that they are mostly exposed to noise and wood dust than the unexposed group. The reason given by the exposed group for not wearing PPE was a lack of availability (48%), while 100% of the unexposed group gave other reasons.

Table 5.5.: Utilisation of PPE among the participants

	Exposed group (n=137)	**Unexposed group** (n=20)	***p*-value**
Variables	**n (%)**	**n (%)**	
Type of PPE worn			
Helmet	130 (94.89)	5 (25)	1[a]
Ear plugs	131 (95.62)	5 (25)	1[a]
Safety glass	12 (8.76)	4 (20)	1[a]
Respiratory mask	16 (11.68)	0	1[a]
Protective gloves	119 (86.86)	1 (5)	1[a]
Overalls	103 (75.18)	0	1[a]
Apron	45 (32.85)	0	1[a]
Safety boots	99 (72.26)	5 (25)	1[a]
How often do you wear PPE?			
Always	119 (86.86)	0	0.000[b*]
Most of the times	8 (5.84)	0	
Sometimes	9 (6.57)	15 (75)	
Never	1 (0.73)	5 (25)	
Why you do not wear protection constantly?			
Not available	16 (48.48)	0	0.006[b*]
Uncomfortable	5 (15.15)	0	
Make difficult breathing	5 (15.15)	0	
Can't hear properly	4 (12.12)	0	
Other	3 (9.09)	3 (100)	

[a]Chi-Square test; [b]Fisher's exact test; [c]Student t-test; *Significant at p<0.05

The prevalence of hearing loss among participants is shown in Table 5.6. Tinnitus (ringing in the ears) was the most prevalent condition or symptom of hearing loss among the exposed group (50%) than the unexposed group (33.33%), and ear infections was the most prevalent condition or symptom of hearing loss among the unexposed group (66.67%) than the exposed group (21.43%). Four percent (4.38%) of the exposed group and 4.46% of the unexposed group reported a history of hearing loss. Ninety-six percent (96%) of the exposed group reported wearing ear plugs, compared to 25% of the unexposed group, but the exposed group reported a higher percentage of tinnitus. This could be explained by the fact that 48% of PPE was reported as unavailable. Lack of training on proper inserting of ear plugs into the ear canal can be another factor if earmuffs are not used, as the majority of those exposed are between the ages of 40 and 49, and their hearing will deteriorate as their age increases.

Table 5.6.: Prevalence of hearing loss symptoms among participants

Variables	**Exposed group**	**Unexposed group**	***p*-value**
Do you experience the following health related conditions?			
Ringing in the ear	21 (50)	1 (33.33)	0.036[c*]
Ear infection	9 (21.43)	2 (66.67)	
Rupture ear drum	7 (16.67)	0	
Ear injury	5 (11.9)	0	
Do you have any history of hearing loss?			
Yes	6 (4.38)	1 (4.46)	1[b]
No	131 (95.62)	19 (95.54)	

[a]Chi-Square test; [b]Fisher's exact test; [c]Student t-test; *Significant at p<0.05

5.4. Discussions

This study investigated incidence of hearing loss and respiratory problems among sawmill workers of the timber processing factories within the Gert Sibande District Municipality. Majority of the exposed 72(53%) and unexposed group 12(60%) were males. This was related to Shamssain (1992) findings who reported (77 males and 68 female) exposed and (77 males and 75 females unexposed) and Adeoye *et al*. (2014) who reported 50 male sawmill workers and 50 control subjects and Thepaksorn *et al.* (2013) who reported exposed group comprising of 115 male workers and 134 male unexposed but differ with that of Kitcher *et al.* (2014) who reported one female and 100 males for the study group and 68 females and 38 males control and Thepaksorn *et al*. (2017) who reported 304(44.30%) males exposed and 64(9.30%) females unexposed. This might be caused by high level of physical work required to move the timber at the sawmill. The lower female population ascend from timber yard cleaners. Fourth-two percent (42%) majority of the exposed had secondary education while 50% of unexposed had matric. Majority of unexposed appears to be more educated (matric and tertiary) than exposed (secondary and matric). This was similar to Thepaksorn *et al.* (2017) study who reported 31.10% secondary education for the exposed compared to 9.28% higher degree for the unexposed and Adeoye *et al*. (2014) who reported secondary 27(54%) for sawmill workers compared to 22(44%) first degree for the control but differ with 74.78% higher degree for exposed compared with 42.53% higher degree for unexposed as reported by Thepaksorn *et al*. (2013). Since majority of employees in the current study had attained secondary, they will understand basic training on safety measures as compared to those with primary education. Educational level is essential during hazardous awareness on respiratory symptoms and NIHL

A high proportion of exposed group in the present study were general workers (30%) and operators (26%) while the unexposed were other job titles (100%). This finding were inconsistent with Mong'are (2013) who reported 50.2% majority of respondents in their study were wood packer and stacker while 4% was supervisors or managers. It was reported that majority of workers who were less educated were involved in manual operations and were uninformed on safety procedures. Forty-four percent (44%) majority of exposed group and 45% unexposed group had working experience of between 3 to 10 years. This was consistent with Adeoye *et al.* (2014) who reported

64% sawmill workers and 40% control (LG workers) who spent less than 10 years on their job. Adeyemi (as cited on Mong'are, 2013) indicated sawmill workers who had worked for longer period are more experienced than those with 2 to 5 years. Contrary to that Rusca *et al.* (2008) indicated that junior labours had higher incidence of rhinitis, wheezing and bloodshot eyes than senior labours. Exposure to 85dB(A) NIOSH REL over 40 years increase risk of developing NIHL by 8%, while 80dB(A) action level increase risk by 1% while 90dB(A) OSHA PEL worsens the risk by 25% (NIOSH, 1998) even though contrary to that Eriksson *et al*. (1996) indicated that employees with less than five years of exposure have higher risk of suffering from bronchial reactivity. Thepaksorn *et al.* (2019) indicated that workers with more than 25 years have higher risk of developing NIHL than those with 25 years or lesser. NIHL is more prevalent on workers with 14 years of experience while exposure between 4 to 8 years results in development of hearing impairment (Ighoroje, Marchie and Nwobodo, 2004).

The most prevalent respiratory symptoms among the exposed group were phlegm (61%), shortness of breath (44%) and cough (13%) while the unexposed group were shortness of breath (50%) and cough (10%). The prevalence of phlegm (61%) among the exposed was higher than that reported by Chan-Yeung *et al.* (1973), Vedal *et al.* (1986), Osman and Pala (2009), Al-Neaimi, Gomes and Lloyd (2001) and Adeoye *et al*. (2015). Incidence of shortness of breath (44%) among exposed and 50% unexposed was greater than that of Holness *et al.* (1985), Chan-Yeung *et al*. (1984), Vedal *et al*. (1986) and Adeoye *et al.* (2015). These inconsistencies may be attributed by high levels of wood dust and kind of tree that was processed (Shamssain, 1992). These findings were closely related with that of Tobin *et al.* (2016) who established prevalence of respiratory problems comprising of cough (47%), catarrh (50%), cough (5%), chest tightness (10%), chest pain (6%) and breathing difficulty (8%). They indicated that incidence of respiratory problems was higher among exposed than unexposed. However, incidence of cough (13%) among exposed and 10% unexposed in the current study was lower than 43% reported by Osman and Pala (2009) and 30% by Al-Neaimi, Gomes and Lloyd (2001) and 30% by Mandryk, Alwis and Hocking (1999) and 60% by Adeoye *et al*. (2015) and 34% by Ige and Onadeko (2000).

Osman and Pala (2009) reported that workers with more than 10 years of experience are at high risk of suffering from nasal and eye symptoms than those with fewer years of experience. Contrary in that, Milanowski *et al*. (2002) reported that eye, skin and nasal problems were not prevalent on furniture employees with 5years of exposure. In the said study, it was reported that incidence of respiratory symptoms among workers increased with duration of exposure. The incidence of shortness of breath (44%) among exposed and 50% unexposed was higher than 8% reported by Al-Neaimi, Gomes and Lloyd (2001). Wood dust exposure can cause dermatitis and allergic respiratory infections. Sawdust can cause eye irritation, nasal dryness, eye and nose irritations as well as frequent dryness (Holness *et al*., 1985).

The predominance of cough during sunrise (50%), daytime (22%) and sunset (28%) reported by exposed group and 50% morning and night reported by unexposed group was higher than occurrence of coughs during sunrise (40%) and daytime or sunset (43%) reported by Shamssain (1992) among exposed workers and differ with that of Al-Neaimi, Gomes and Lloyd (2001) who reported incidence of coughs during sunrise (40%), daytime and sunset (43%) that were higher in the exposed than control. The exposed group reported higher prevalence of respiratory symptoms than unexposed group, except 50% of chest pain or shortness of breath reported that was higher than 44% reported by the exposed group. This can be attributed by poor use of respiratory masks because 12% of the exposed group reported wearing respiratory masks. In addition, the duration of exposure working in the dusty area can be another factor. Douwes *et al.* (1992) reported that asthma, cough symptoms, nose and eye irritations were considerably higher among exposure than control while Mandryk, Alwis and Hocking (2000) reported that employees at the green chain had higher incidence of wheeze, nasal congestion, sneeze, sinusitis, constant pain in the head, flu-like problems, chronic bronchitis, eye and throat irritations than dry mill employees.

The unexposed group in the present study had more smokers (45%) than exposed (18%). This was similar to Thepaksorn *et al.* (2013) who reported 30% smokers among exposed than 47% among unexposed but differ with Theparksen *et al.* (2017) who reported 27% smokers among exposed and 1% among unexposed. This may be attributed by 60% males that were more than 40% females and may be reluctant to quit smoking due to addiction. Hessel *et al.* (1995) indicated that significant elevation

of age and smoking increase risk of dyspnea, chest pain and wheezing whereas non-significant elevation of age and smoking increase prevalence of coughing, catarrh, breathlessness, dyspnea and chest pain. Jacobsen *et al.* (2008) stated that fresh wood dust exposure is linked with cough, occupational asthma, asthmatic symptoms, chronic bronchitis, acute and chronic lung function impairments. Douwes *et al*. (1992) indicated that exposure to endotoxin may cause respiratory symptoms. People exposed to organic dust may have high incidence of respiratory problems as a result of smoking cigarettes and being exposed to dry dust (Simpson *et al.*, 1998). Chang-Yeung *et al.* (1980) reported the highest chest related symptoms and lower pulmonary function test on the current smokers than non-smokers or ex-smokers.

The most frequently worn PPE reported by the exposed in the present study was ear plugs (96%) while unexposed reported helmet, ear plugs and safety boots (25%). These findings were consistent with that of Feder *et al.* (2017) who reported 80% of workers wearing HPD and were inconsistent with that of Osman and Pala (2009) who reported safety goggles (11%) and dust masks (17%) as the most frequently worn PPE. Likewise, respiratory masks (12%) was the least worn PPE reported by the exposed group while the unexposed group reported safety gloves (5%). PPE such as FFP 2 dust masks and eye protection (safety goggles) are poorly worn during wood production and are often considered disturbing, uncomfortable and making difficulty in breathing (Yahya *et al*., 2013). Fatusi and Erhabor (1996) reported that 58% of workers in their study reported PPE cause uncomfortable feelings while 42% lack awareness on use of PPE and 25% feels the device create barriers in communication. The findings were consistent with that of Alwis *et al.* (1998) who indicated that 90% of workers observed were not wearing suitable respirators approved for dust and only 10% of the workers were observed wearing approved respirators for dust. Mandryk, Alwis and Hocking (2000) reported that all the workers observed in their study were not wearing respirators. They indicated that 50% of the workers who used the respirators wore them sometimes during the shift.

Fatusi and Erhabor (1996) reported 20% of the employees observed were wearing protective masks. They reported cough, sputum production and chest pain as the most prevailing respiratory problems amongst employees. Furthermore, most of exposed group had higher incidence of skin irritation, conjunctivitis and hearing difficulties than unexposed. This finding were consistent with that of the present study, because 12% of the exposed workers reported using respiratory mask to prevent exposure to dust and majority of the workers were observed wearing cloth masks during the shift. According to Health and Safety Executive (2004), nuisance dust masks or cloth masks do not provide reliable protection against wood dust. Ugheoke, Ebomoyi and Iyawe (2006) stated that, less than 5% of sawmill workers in their study were wearing PPE and safety standards were not practiced or enforced. Their findings discovered that respiratory symptoms especially chest pain and sputum production were common among sawmill workers.

Eighty-seven (87%) of the exposed group reported wearing PPE constantly (always) while 75% of unexposed group reported wearing it sometimes even though PPE safety signs are displayed at respiratory or hearing zones. However, the reason for not wearing PPE by the exposed was reported to being not available (48%) while 100% of unexposed indicated other reasons. The exposed group appear to wear PPE more often than unexposed due to the fact they are mostly exposed to noise and dust. Osagbemi, La-Kadri and Aderibigbe (2010) indicated that 20% of sawmill workers in their study were wearing PPE. They reported that most of perceived occupational health hazards in the sawmill were wood dust (28%) and noise (26%). Thepaksorn *et al.* (2018) stated that employees are expected to use HPDs based on risk recognition and teaching. However, HPDs instruction was inversely linked with age (years) and length of experience. According to defence stimulus theory, workers who anticipate risks and take precautionary measures are more likely to protect themselves. Thepaksorn *et al.* (2019) reported 65% workers using HPDs despite the fact that 70% were men than 65% women. Consistent use of HPD were higher on workers with 1 year of employment (75%) as well as those in sawmilling (70%). Eighteen percent (18%) respondents thought use of PPE can avert hazards and required employers to provide them with durable PPE even though usage of PPE was 10% with the highest being those wearing coveralls.

Osagbemi, La-Kadri and Aderibigbe (2010) study revealed low use of PPE among sawmill workers. Reasons given were unavailability because employer rarely provide PPE and when provided, they are not durable. This is in agreement with Osagbemi, La-Kadri and Aderibigbe (2010) previous studies that have reported similar findings. Provision of appropriate and durable PPE is the employers' responsibility. Previous research studies by Neitzel *et al.* (1999) and Otoghile *et al.* (2019) reported 50% usage of HPD and usage of HPDs alone was not recommended to control noise. It has been reported that 13% and 64% of construction workers surveyed reported using ear muffs and ear plugs respectively and 22% reported not using HPDs (Suter, 2002). Ahmed *et al*. (2012) reported 61% of workers in their study who had never used any form of HPDs. In addition, Davies (2008) indicated that 17% of workers in their study never used HPD while 83% used the devices regularly even though none of them used the device consistently while Edelson *et al.* (2009*)* indicted that 80% of workers reported either almost never or almost always using HPDs.

Tinnitus (ringing in the ears) and ear infections were the two conditions or symptoms of hearing loss that were common in the exposed group (50%) and unexposed group (66%), respectively. A history of hearing loss was reported by 4% of the exposed group and 5% of unexposed group. According to Alwis *et al*. (1998), exposed group were far more likely to have an ear infection than controls. The reason for high occurrence of NIHL among exposed might be attributed by 48% PPE that was reported of being unavailable. Thepaksorn *et al.* (2019) discovered that men had a 23% higher frequency of NIHL than women, and that 50% of the study's participants never (or very never) wore HPDs.Additionally, 34% of workers reported hearing impairment related to noise, even though only 11% sought medical attention, and 37% had hearing impairment, despite the fact that 83% of individuals with hearing impairment had more than six years of work experience. Furthermore, 97% of the exposed group were aware that noise exposure can cause deafness, but only 4% and 1% of them often wear HPD.

Sawmilling is the noisiest operation and can damage individual hearing, leading to tinnitus and NIHL if proper protections are not applied (Davies *et al*., 2009). Mong'are (2013) argues that, noise reduction at the source, route, and worker might be the achievable goal, but machine isolation, accurately positioned barriers, and double-walled enclosures have been accepted in most wood processing industries in

developed countries. This drastically reduced noise emissions from an average of 98 to 86 dB(A), even though exposure may still be above 85 dB(A), which must be complemented by the use of HPD. The use of a hearing aid by those working in a designated noise zone should not be permitted (NSSA, 2009). Thepaksorn *et al.* (2019) in another study reported a higher incidence of NIHL among sawing workers than office workers. However, Vermal *et al*. (2010) reported that 80% of sawmills had a NIHL prevention program, in contrast to 33% that have respiratory protection.

5.5. Conclusion

Currently, there are no published studies on hearing loss and respiratory problems among sawmill workers at the timber processing factories, particularly at the Gert Sibande District Municipality in Mpumalanga Province. Globally, 16% of cases of disabling hearing loss in old ages are caused by exposure to noise beyond 85 dB(A) (Nelson *et al.*, 2005). Few sawmills had put in place sawdust extraction system and this has subsequently help reduce dust. More studies are required to probe the prevalence of hearing loss and respiratory problems among sawmill workers in the timber processing factories (Nandi and Dhatrak, 2008). This study provides new evidence that exposure to wood dust and noise increases risk of developing chest pains or shortness of breath, tinnitus and ear infections and the prevalence of chest pains or shortness of breath was higher among the unexposed than exposed while tinnitus among among exposed was higher than unexposed and ear infections among the unexposed was higher than exposed. Exposed group reported always wear PPE than unexposed who wear it sometimes. Exposed did not wear PPE consistently due to not being available than unexposed who reported other reasons. Since smoking when associated with exposure to wood dust and noise may increase risk of respiratory symptoms and development of high frequency hearing loss. Workers smoking cigarettes should be encouraged cut dawn smoking to help protect developing a more severe effects in the future (Tao *et al.*, 2013; Paschoal and de Azevedo, 2009; Mwaiselage *et al.*, 2005; Zeleke, Moen and Bråtveit, 2010). The results suggest that sawmill owners should implement controls to protect workers' health (Black, Dilworth and Summers, 2007; Driscoll, Milk and Burgess, 2009; Davies and Henderson, 2009a; Davies and Henderson, 2009b; Hirst, 2010).

5.6. References

Adeoye *et al.* (2014) Respiratory Symptoms and peak expiratory flow among sawmill workers in South West Nigeria. *Journal of Environmental and Occupational Science* **3**(3):141-145.

Adeoye *et al*. (2015) Awareness of Occupational hazards and health problems among sawmill workers in Osun state, Nigeria. *International Journal of Research* **2**(1):1-14.

Ahmed, H.O. and Abdullah, A.A. (2012) Dust exposure and respiratory symptoms among cement factory workers in the United Arab Emirates. *Ind Health* **50**: 214-222.

Al-Neaimi, Y.I., Gomes, J. and Lloyd, O. (2001) Respiratory illness and ventilator function among workers at a cement factory in a rapid developing country. *Occupational Medicine* **51**: 367- 373.

Alwis, K.U. (1998) *Occupational Exposure to Wood Dust*. (*P.h.D. Thesis)*. University of Sydney, Department of Public Health and Community Medicine.

Alwis, K.U., Mandryk, J. and Hocking, A.D. (1999) Exposure to Biohazards in Wood Dust: Bacteria, Fungi, Endotoxins, and (1→3)-Beta-D-Glucans. *Applied Occupational and Environmental Hygiene* **14**: 598-608. DOI: 10.1080/104732299302404

Alwis *et al*. (1999) Dust Exposures in the Wood Processing Industry. *American Industrial Hygiene Association Journal* **60**: 641-646.

Anderson, S.E. and Meade, B.J. (2014) Potential health effects associated with dermal exposure to occupational chemicals. *Environ Health Insights* **8**:51-62.

Ayaz, M. (1991) Noise hazard in sawmilling industry in Pakistan. *Pakistan Journal Forest* **41**:187–194.

Beiler's Sawmill. (n. d.) *How does a sawmill work*?. [S.l.: S.n.].

Bellia *et al*. (2003) Questionnaires, spirometry and PEF monitoring in epidemiological studies on elderly respiratory patients. *Eur Respir J Suppl* **40**: 21s-27s. DOI: 10.1183/09031936.03.00402303.

Black, N., Dilworth, M. and Summers, N. (2007) Occupational Exposure to Wood Dust in the British Woodworking Industry in 1999/2000. *Ann. Occup. Hyg* **51**(3): 249–260.

Cantley *et al*. (2015) Association between ambient noise exposure, hearing acuity, and risk of acute occupational injury. *Scandinavian Journal of Work, Environment and Health* **41**: 75–83.

Chang *et al.* (2013) Occupational Noise Exposure and Incident Hypertension in Men: A Prospective Cohort Study. *American Journal of Epidemiology* **177**: 818–825.

Chang *et al*. (2011) High-frequency hearing loss, occupational noise exposure and hypertension: a cross-sectional study in male workers. *Environ Health* **10**: 2 – 8.

Chan-Yeung *et al*. (1973) Occupational asthma and rhinitis due to Western red cedar (*Thuja Plicata*). *Am Rev Respir Dis* **108**(5):1094–1102.

Chan-Yeung *et al*. (1980) Epidemiologic health survey of grain elevator workers in British Columbia. *Am Rev Respir Dis* **121**:329-338.

Chan-Yeung *et al.* (1984) Symptoms, pulmonary function, and bronchial hyperreactivity in western red cedar workers compared with those in office workers. *Am Rev Respir Dis* **130**(6):1038–1041.

Clark, W.W. and Bohne, B.A. (1999) Effects of noise on hearing. *JAMA* **281**: 1658-1659. DOI: 10.1001/jama.281.17.1658.

Concha-Barrientos, M., Campbell-Lendrum, D. and Steenland, K. (2004) *Occupational noise: Assessing the burden of disease from work-related hearing impairment at national and local levels*. Geneva: WHO.

Cruickshanks *et al.* (1998) Cigarette Smoking and Hearing Loss: The Epidemiology of Hearing Loss Study. *JAMA* **279**: 1715–1719. DOI:10.1001/jama.279.21.1715.

Davies, B. and Henderson, J. (2009a) *W501 student manual measurements of hazardous chemical substances*. Australia: University of Wollongong.

Davies, B. and Henderson, J. (2009b) *W505 student manual control of hazardous substances*. Australia: University of Wollongong.

Davies *et al*. (2009) Occupational noise exposure and hearing protector use in Canadian lumber mills. *J Occup Environ Hyg* **6**(1):32-41.

Davies, R.R. (2008) What Do We Know about Hearing Protector Comfort?. *Noise Health* **10**(40): 83-89

Demers *et al*. (1995) Wood dust and sino-nasal cancer: Pooled reanalysis of twelve case-control studies. *American journal of industrial medicine* **28**:151-166.

Demers, P.A., Teschke, K. and Kennedy, S.M. (1997) What to do about softwood? A review of respiratory effects and recommendations regarding exposure limits. *Americn journal of industrial medicine* **31**(4): 35-398.

Demers *et al*. (2000) Exposure to dust, resin acids, and monoterpenes in softwood lumber mills. *American Industrial Hygiene Association (AIHAJ)* **61**:521-528.

Dost, W.A. (1974a) Noise levels in soft wood lumber mills. *Forest Products Journal* **24**:25-27.

Dost, W.A. (1974b) Sawmill noise at the operating level. *Forest Products Journal* **24**:13–17.

Douwes *et al*. (2000) Worker exposures to airborne dust, endotoxin and β-1,3)-glucan in two New Zealand sawmills. *American journal of industrial medicine* 38(4):426-430.

Douwes *et al*. (1992) Asthma and other respiratory symptoms in New Zealand pine processing sawmill workers. *American Journal of Industrial Medicine* **8**: 608–615.

Driscoll, D.P.P.E., Milk, K. and Burgess, M. (2009) *W503 student manual noise: measurements and its effects*. UK: BOHS.

Edelson *et al.* (2009) Predictors of Hearing Protection Use in Construction Workers, *The Annals of Occupational Hygiene* **53**(6): 605–615.

Enarson, D.A. and Chan-Yeung, M. (1990) Characterization of health effects of wood dust exposures. *Am J Ind Med* **17**:33-8. DOI:10.1002/ajim.4700170107.

Eriksson *et al*. (1996) Terpene exposure and respiratory effects among sawmill workers. *Scandinavian journal of work, environment & health* **1**:182-190.

Fatusi, A. and Erhabor, G. (1996) Occupational health status of sawmill workers in Nigeria. *Journal of the Royal Society of Health* **116**(4): 232-236.

Feder *et al.* (2017) Prevalence of hazardous occupational noise exposure, hearing loss, and hearing protection usage among a representative sample of working canadians. *J Occup Environ Med* **59**(1):92-113.

Fernández *et al*. (2009) Noise exposure of workers of the construction sector. *Applied Acoustics* **70**: 753-760.

Ferrite, S. and Santana, V. (2005) Joint effects of smoking, noise exposure and age on hearing loss. *Journal of Occupational Medicine* **55**:48–53.

Fransman *et al*. (2003) Respiratory symptoms and occupational exposure in New Zeaand Plywood mill workers. *Annals of occupational hygiene* **47**(4):27-295 .

Gan, W.Q., Davies, H.W. and Demers, P.A. (2011) Exposure to occupational noise and cardiovascular disease in the United States: The National Health and Nutrition Examination Survey 1999–2004. *Occupational & Environmental Medicine* **68**:183-190.

Guyana *Forestry Commission.* (2012) *Code of practice for wood processing facilities: sawmills and lumberyards*. [S.l.: S.n.].

Hammer, M.S., Swinburn, T.K. and Neitzel, R.L. (2014) Environmental noise pollution in the United States: developing an effective public health response. *Environ Health Perspect* **122**(2):115-119. DOI:10.1289/ehp.1307272.

Hayes *et al*. (1986a) Cancer of the nasal cavity and paranasal sinuses, and formaldehyde exposure. *Int J Cancer* **37**:487-492.

Hayes *et al*. (1986b) Wood-related occupations, wood dust exposure, and sinonasal cancer. *American Journal of Epidemiology* **124**(4): 569–577.

Health and Safety Executive (HSE). (2004) S*election of respiratory protection equipment suitable for use with wood dust. Working sheet No. 14, SHE Information Sheet.* UK: HSE.

Hernberg *et al*. (1983) Nasal and Sino nasal cancer: Connection with occupational exposures in Denmark, Finland and Sweden. *Scandinavian Journal of Work, Environment and Health* **9**:315-326.

Hessel *et al*. (1995) Lung Health in Sawmill Workers Exposed to Pine and Spruce. *Chest* **108**(3):642-646.

Hirst A. (2010) *W201 Basic principles in occupational hygiene*. UK: OHTA.

Holness *et al*. (1985) Respiratory function and exposure-effect relationships in wood dust-exposed and control workers. *J Occup Med* **27**(7):501–506.

Hong *et al*. (2013) Understanding and preventing noise-induced hearing loss. *Disease-a-Month* **59**: 110–118.

Ige, O.M. and Onadeko, O.B. (2000) Respiratory symptoms and ventilatory function of the sawmillers in Ibadan, Nigeria. *Afr J Med Med Sci* **29**(2):101-104.

Ighoroje, A.D.A., Marchie, C. and Nwobodo, E.D. (2004) Noise-induced hearing impairment as an occupational risk factor among Nigerian Traders. *Nigerian Journal of Physiological Sciences* **9**(1):14-19. DOI**:** 10.4314/njps.v19i1.32630.

Israel, G.D. (1990) Determining sample size. Gainesville: University of Florida.

Jacobsen *et al*. (2008) Longitudinal lung function decline and wood dust exposure in the furniture industry. *Eur Respir J* **31**: 334–342.

Judd, H.M. and Janice, K.W. (2004) Safety in the wood products industry. *Forest Products Journal* **54**(10):8-18.

Kadam, P. and Bhalerao, S. (2010) Sample size calculation. *Int J Ayurveda Res* **1**(1):55-57. DOI: 10.4103/0974-7788.59946.

Kauppinen *et al*. (2006) Occupational Exposure to Inhalable Wood Dust in the Member States of the European Union. *Annals of Occupational Hygiene* **50**: 549–561.

Kitcher *et al*. (2014) Occupational haring loss of market mill workers in the city of Accra, Ghana. *Noise health* **16**(70): 183-188.

Koehncke, N. (1999) *An investigation of noise levels in Alberta sawmills*. (*Master Thesis)*. University of Alberta, Department of Public Health Sciences.

Leclerc *et al*. (1994) Sino nasal Cancer and Wood Dust Exposure. *American Journal of Epidemiology* **140**:340-349.

Leensen, M.C.J., van Duivenbooden, J.C. and Dreschler, W.A. (2011) A retrospective analysis of noise-induced hearing loss in the Dutch construction industry. *Int Arch Occup Environ Health* **84**: 577–590.

Lemeshow *et al*. (1990) *Adequacy of Sample Size in Health Studies*. USA: Wiley.

Lester, H., Malchaire, J. and Arbey, H.S. (2001) *Strategies for noise surveys*. In Goelzer, B., Hansen, C.H. and Sehrndt, G.A. (eds.). *Occupational Exposure to Noise Evaluation, Prevention and Control*. Geneva: WHO.

Lie *et al.* (2016) Occupational noise exposure and hearing: a systematic review. *International Archives of Occupational and Environmental Health* **89**:351–372. DOI: 10.1007/s00420-015-1083-5.

Löfstedt *et al*. (2017) Respiratory symptoms and lung function in relation to wood dust and monoterpene exposure in the wood pellet industry. *Ups J Med Sci* **122**(2):78-84.

Malmberg, P. (1990) Health effects of organic dust exposure in dairy farmers. *Am J Ind Med* **17**(1):7-15. DOI: 10.1002/ajim.4700170104.

Mandryk, J., Alwis, K.U. and Hocking, A.D. (1999) Work-related symptoms and dose-response relationships for personal exposures and pulmonary function among woodworkers. *American Journal of Industrial Medicine* **35**:481-490.

Mandryk, J., Alwis, K.U. and Hocking, A.D. (2000) Effects of Personal Exposures on Pulmonary Function and Work-related Symptoms Among Sawmill Workers. *Annals Occupational Hygiene* **44**: 281-289.

May, J.J. (2000) Occupational hearing loss. *American Journal of Industrial Medicine* **37**: 112-120.

Medical Research Council. (1996) Questionnaire on respiratory symptoms approved by Medical Research Council's Committee on Research into Chronic Bronchitis (MRCCRB). Available from: https://www.ukri.org/wp-content/uploads/2021/11/MRC-291121-QuestionnaireOnRespiratorySymptoms1966.pdf. (Accessed on 16 July 2021).

Milanowski *et al*. (2002) Work-related symptoms among furniture factory workers in Lublin region (Eastern Poland). *Ann Agric Environ Med* **9**: 99–103.

Mizoue, T., Miyamoto, T. and Shimizu, T. (2003) Combined effect of smoking and occupational exposure to noise on hearing loss in steel factory workers. *Occupational and Environmental Medicine* **60**: 56-59.

Mong'are, O.R. (2013) *Assessment of occupational safety and health status in selected sawmilling industries in Nakuru, Kenya*. (*Master Thesis).* Kenya: Kenyatta University.

Morata, T.C. and Little, M.B. (2002) Suggested guidelines for studying the combined effects of occupational exposure to noise and chemicals on hearing. *Noise Health* **4**:73-87.

Mwaiselage *et al*. (2005) Respiratory symptoms and chronic obstructive pulmonary disease among cement factory workers. *Scand J Work Environ Health* **31**(4):316-*323.*

Nandi, S.S. and Dhatrak, S.V. (2008) Occupational noise-induced hearing loss in India. Indian *Journal of Occupational and Environmental Medicine* **12**: 53–56.
DOI: 10.4103/0019-5278.43260.

Neitzel *et al*. (1999) An Assessment of Occupational Noise Exposures in Four Construction Trades. *American Industrial Hygiene Association Journal* **60**:807–817.

Nelson *et al*. (2005) The global burden of occupational noise-induced hearing loss. *American Journal of Industrial Medicine* **48**(6):446-458.

NIOSH. (1998) *Criteria for a recommended standard, occupational noise exposure, Revised Criteria*. USA: NIOSH.

Noertjojo *et al*. (1996) Western red cedar dust exposure and lung function: A dose-response relationship. *American Journal of Respiratory and Critical Care Medicine* **154**(4): 968-973.

National Social Security Authority (NSSA). (2009) *National report on occupational diseases*. Available from: http://www.nssa.org.zw. (Accessed on 10 October 2022).

Okwari *et al*. (2005) Lung function status of workers exposed to wood dust in timber markets in Calabar, Nigeria. *Afr J Med Med Sci* **34**:141-145.

Osagbemi, G.K., La-Kadri, R.T. and Aderibigbe, S.A. (2010) Awareness of occupational hazards, health problems and safety measures among sawmill workers in North Central Nigeria. *TAF preventive medicine bulletin* **9**(4):1-2.

Osman, E. and Pala, K. (2009) Occupational exposure to wood dust and health effects on the respiratory system in a minor industrial estate in Bursa/Turkey. *International Journal Occupational Medicine and Environmental Health* **22**: 43-50.

Otoghile *et al*. (2019) Effects of Occupational Noise on Blood Pressure. *Global Journal of Health Science, Canadian Centre of Science and Education* **11**:1-63.

Palmer *et al*. (2004) Cigarette smoking, occupational exposure to noise, and self-reported hearing difficulties. *Occupational and Environmental Medicine* **61**: 340-344.

Paschoal, C.P. and de Azevedo, M.F. (2009) Cigarette smoking as a risk factor for auditory problems. *Braz J Otorhinolaryngol* **75**(6):893-902.

Phillips, S., Henrich, V.C. and Mace, S.T. (2010) Prevalence of noise-induced hearing loss in student musicians. *International Journal of Audiology* **49**:309-316

Pouryaghoub, G., Mehrdad, R. and Mohammadi, S. (2007) Interaction of smoking and occupational noise exposure on hearing loss: a cross-sectional study. *BMC Public Health* **7**:137-139. DOI:https://doi.org/10.1186/1471-2458-7-137.

Rabinowitz, P.M. (2000) Noise-induced hearing loss. *Am Fam Physician* **61**(9):2759-2760.

Ratnasingam *et al*. (2014) Respiratory effects in woodworkers exposed to wood and wood coatings dust: a regional evaluation of South East Asian Countries. *Journal of Applied Sciences* **14**: 1763-1768.DOI: 10.3923/jas.2014.1763.1768.

Robert, D.A. (2008) The burdens of age-related and occupational noise-induced hearing loss in the United States. *Ear and Hearing* **29**: 565-577.

Rongo *et al*. (2002) Respiratory symptoms and dust exposure among male workers in small-scale wood industries in Tanzania. *Journal of Occupational and Environmental Medicine* **44**(12): 1153–1160.

Rösler, G. (1994) Progression of hearing loss caused by occupational noise. *Scandinavian Audiology* **23**:13-37. DOI: 10.3109/01050399409047483.

Rosner, B. (2010) *Fundamentals of Biostatistics*. 7th ed. Salt Lake City: Utah.

Rusca *et al*. (2008) Effects of bioaerosol exposure on work-related symptoms among Swiss sawmill workers. *International Archives of Occupational and Environmental Health* **81**(4):415-421.

Schlünssen *et al*. (2002) Respiratory symptoms and lung function among Danish woodworkers. *J. of Occup. and Environ. Med* **44**:82-98.

Shamssain, M.H. (1992) Pulmonary function and symptoms in workers exposed to wood dust. *Thorax* **47**:84-87.

Simpson *et al*. (1998) Prevalence and predictors of work related respiratory symptoms in workers exposed to organic dusts. *Occup Environ Med* **55**(10):668-672. DOI: 10.1136/oem.55.10.668.

Suter, A.H. (2002) Construction Noise: Exposure, Effects, and the Potential for Remediation; A Review and Analysis. *AIHA Journal* **63**:768–789.

Talbott *et al*. (1985) Occupational noise exposure, noise induced hearing loss, and the epidemiology of high blood pressure. *American Journal of Epidemiology* **121**(4): 501–514.

Tao *et al*. (2013) Effect of cigarette smoking on noise-induced hearing loss in workers exposed to occupational noise in China. *Noise Health* **15**(62):67-72. DOI: 10.4103/1463-1741.107159.

Thepaksorn *et al.* (2017) Respiratory symptoms and ventilatory function defects among Para rubber wood sawmill workers in the South of Thailand. *Human and Ecological Risk Assessment: An International Journal* **23**(4):788-797. DOI: 10.1080/10807039.2017.1285221.

Thepaksorn *et al*. (2019) Occupational noise exposure and hearing defects among sawmill workers in the south of Thailand. *International Journal of Occupational Safety and Ergonomics* **25**(3):458-466.

Thepaksorn *et al*. (2013) Respiratory symptoms and patterns of pulmonary dysfunction among roofing fiber cement workers in south of Thailand. *J Occup Health* **55**(1):21-28.

Thepaksorn *et al*. (2018) Relationship between noise-related risk perception, knowledge, and the use of HPDs among para rubber wood sawmill workers. *Safety and Health at Work* **9**(1): 25-29.

Tobin *et al*. (2016) Occupational exposure to wood dust and respiratory health status of sawmill workers in South-South Nigeria.*J Pollut Eff Cont* **4**:1-154. DOI:10.4172/2375-4397.1000154.

Ugheoke AJ, Ebomoyi MI, Iyawe VI. (2006) Influence of smoking on respiratory symptoms and lung function indices in sawmill workers in Benin City, Nigeria. *Nigerian Journal of Physiological Sciences* **21**:1-2.

Ugheoke, A.J., Wahab, K.W. and Erhabor, G.E. (2009) Prevalence of respiratory symptoms among sawmill workers in Benin city, Nigeria. *Int J Trop Med* **4**:1-3.

van Kempen *et al*. (2002) An association between noise exposure and blood pressure and ischemic heart disease: A meta-analysis. *Environmental Health Perspectives* **110**(3):307–317.

Vaughan *et al.* (2000) Occupational exposure to formaldehyde and wood dust and nasopharyngeal carcinoma. *Occup Environ Med* **57**:376-384.

Vedal *et al*. (1986) Symptoms and pulmonary function in western red cedar workers related to duration of employment and dust exposure. *Arch Environ Health* **41**(3):179–183.

Venables *et al*. (1993) Respiratory symptoms questionnaire for asthma epidemiology: validity and reproducibility. *Thorax* **48**: 214-219. DOI: 10.1136/thx.48.3.214.

Vendal *et al.* (1986) Symptoms and pulmonary function in western red cedar workers related to duration of employment and dust exposure. *Achieves of Environmental Health: An international Journal* **41**(3):179-183.

Vermal *et al*. (2010) Occupational health and safety issues in Ontario sawmills and veneer/plywood plants: A pilot study. *J Environ Public Health* **1**: 526-487.

Yahya *et al.* (2016) Satisfaction study of using hearing protection device among sawmill workers in Kuantan, Malaysia. *Journal of International Pharmaceutical Research* **8**(1):50-56.

Yamanaka, M.W. (2000) *An investigation of wood dust exposure in Alberta sawmills. (Master Thesis)*. Canada: University of Alberta, Edmonton.

Zeleke, Z.K., Moen, B.E. and Bråtveit, M. (2010) Cement dust exposure and acute lung function: A cross shift study. *BMC Pulmonary Medicine* **10**:1-19. DOI:10.1186/1471-2466-10-19.

Chapter 6: Conclusion and recommendations

6.1. Introduction

Inhaling sawdust beyond 1 mg/m^3 can cause respiratory symptoms and sinonasal cancer while exposure to 0.5 mg/m^3 can cause occupational astha (American Conference of Governmental Industrial Hygienists (ACGIH), 2005). Likewise, exposure to chronic noise above 85dB(A) noise rating limit can cause permanent hearing loss (Rabinowitz, 2000). A health risk assessment has confirmed that exposure to 85 dB(A) NIOSH REL over 40-years escalates risk of developing NIHL by 8%, while exposure to 90dB(A) OSHA PEL escalates risk by 25% and 80dB(A) lower exposure action value increase risk by 1% (NIOSH, 1998). Similarly, exposure to noise levels at or above 75dB(A) may also increase risks of coronary heart disease in men while exposure to noise levels between 75 to 80 dB(A) for less than 20 years, could increase risk of ischemic heart disease (Eriksson, 2019; Dzhambov and Dimitrova, 2016; Neitzel, Andersson and Andersson, 2016; Passchier-Vermeer and Passchier, 2000). A respiratory protection and hearing loss conservation programme should be established at the sawmill factories and its components should include health risk assessment, audiometric and pulmonary function test, selection, fit test and use of RPD and HPDs, education and training about occupational asthma, sinonasal cancer and NIHL (Ahmed, Dennis, Badran, Ismail, Ballal, Ashoor and Jerwood, 2001; Davies and Henderson, 2009a;b; Driscoll, Mikl and Burgess, 2009; Franks, Stephenson and Merry, 1996; Gerges, Vedsmand, Lester and Bat-kartellet, 1995; Hirst, 2010; HSE, 2013; NIOSH, 1990; SANS, 2013; United Kingdom, 2005).

6.2. General discussions

The background, rationale for the study, problem statement, aim and objectives of the study, hypotheses, significance and scope of the study were outlined in chapter 1. A review of literature about noise and sawdust and probable health outcomes were presented in chapter 2. The findings regarding levels of wood dust exposure were outlined in chapter 3. Overall, the geometric mean(GM) or geometric standard

deviation(GSD) for personal respirable wood dust at sawmill A was 0.9(4.8) mg/m^3, while at sawmill B was 0.57(0.75) mg/m^3. The GM(GSD) for personal total inhalable wood dust at sawmill A was 0.37(0.94) mg/m^3, while at sawmill B was 1.19(16.91) mg/m^3.Likewise, the GM(GSD) for area respirable wood dust at sawmill A was 0.13(0.09) mg/m^3 while at sawmill B was 0.8(0.6) mg/m^3. In addition, the GM(GSD) for area total inhalable wood dust at sawmill A was 0.13(0.16) mg/m^3 while at sawmill B was 0.54(0.55) mg/m^3. Most wood dust results were below the OEL. The results about occupational noise were presented in Chapter 4. The GM(GSD) for area noise result at sawmill A was 90.05(8.02) dB(A) while at sawmill B was 90.14(7.94) dB(A). The GM(GSD) for personal noise exposure at sawmill A was 92.26(4.35) dB(A) while at sawmill B was 92.24(2.65) dB(A), respectively. Majority of the noise exposure levels were above noise rating limit. The findings about hearing loss and respiratory symptoms associated with sawdust and noise were presented in chapter 5. The prevalence of shortness of breath or chest pain among unexposed was higher than exposed while tinnitus (ringing in the ears) among exposed was higher than unexposed and ear infections among unexposed was higher than exposed.

6.3. Research limitation

The study was restricted to 137 exposed and 20 unexposed workers due to logistics, resource limitations and budgetary constraints. The study used a cross-sectional design with simple random sampling. A cross-sectional study was selected to examine current knowledge and practices on use of personal protective clothing among the participants without any follow up (Kadam and Bhalerao, 2010; Setia, 2016). The sample size was very small for comparison purpose and to make the findings plausible. Consequently, these findings should be interpreted with caution. There were some differences in the filter cassettes used that have been considered. It is difficult to directly measure wood dust using inhalable dust sampling and compare findings with OEL without following methods applicable to your country including sampling method and physical or chemical properties of wood dust at each sawmill.

The symptoms reported in this study were based on self-reported health issues of hearing loss and respiratory symptoms from the questionnaire, which may lead to misclassification of disease or symptoms and underestimation of exposure. It is important to link hearing or respiratory symptoms with a specific job or task and the specific type of wood used with health outcomes. In addition, area results for wood dust were compared with OEL and all the results were below OEL. Previous studies have indicated that area results for wood dust do not always provide an accurate result for personal exposure. Data associated with alcohol intake, hypertension and other potential confounders were not investigated. Moreover, lung function tests and audiometric examinations were not undertaken to link the exposure and/or use of PPE with the reported work-related symptoms or exposure.

6.4. Future studies

To the knowledge of researcher, this was the first study to investigate wood dust and noise exposure among workers in the sawmill factories within the Gert Sibande Municipality. Future studies should be conducted at the timber processing factories in other provinces to determine if the results are related. Future studies should integrate dose response correlation between personal exposure with work-related symptoms. Studies should be carried out to examine the efficiency of LEV systems used in the woodworking process for appropriate repairs, modification or design. A detailed investigation of LEV systems as well as effectiveness of use of RPDs and HPDs in sawmills should be done. Research is required to examine an association between ototoxic or neurotoxic chemicals and the risk of hearing loss from exposures to these chemicals alone or in combination with noise. The findings will be useful for setting up a strategy for targeted control. A detailed study on the effects of personal habits such as smoking and ototoxic drugs should be investigated since smoking cigarette or taking medicinal drugs when associated with exposure to noise and wood dust can increase risk of respiratory symptoms or development of high frequency hearing loss.

6.5. Recommendations

Majority of wood dust results reported in the present study were below 5mg/m^3 OEL. However, implementation of engineering and administrative controls supplemented by use of appropriate fit-tested RPDs is recommended in areas where exposures were above OEL. In addition, majority of the noise exposure results were above 85 dB(A) noise rating limit. The implementation of engineering controls at the source, routes and workers is vital to reduce worker exposure while workers should constantly wear PPE in noise zones irrespective of shorter duration. The recommendations presented in this chapter are a summary of the controls that address the findings presented in chapter 3, 4 and 5.

The following recommendations should be implemented at sawmill factories to reduce workers' exposure to sawdust:

- LEV captor hood should be fitted on machines not far from source to capture dust and prevent it being airborne and egular inspection and maintenance of the LEV.
- Fresh logs to be sawed while wet and dry timbers to be processed in an open building with supply of good ventilation to allow dust to disperse.
- A wet method should be used to suppress dust to avoid being airborne.
- The duration and number of workers performing dusty work should be reduced to lower the exposure.
- The distance between dust sources and workers should be increased to lower dust generation from source towards workers
- Employees in respiratory zones to undergo medical surveillance frequently for early detection of underlying diseases, reporting to workman's compensation fund and early treatment.
- Wearing RPD in respiratory zone to be made compulsory irrespective of short time spent in the area.
- The use of compressed air to remove sawdust to be avoided as it blows more dust making it airborne.
- Proper selection, fit-testing, training, instruction, information, supervising, observation and coaching workers on correct use, care and maintenance of RPD to reduce risks from exposure to sawdust

It is recommended that the following measures be implemented at sawmill factories to reduce sawmill workers' exposure to high noise levels by:

- Regular maintenance of noisy machinery to reduce noise emissions.
- Reducing fall height of timber during stacking by avoiding dropping timber few centimeters above surface
- Avoid metal to metal contact of conveyor by balancing rotating parts
- Keeping dull blades of saw sharp and balance tools to reduce vibration
- Cutting logs while wet as dry hardwood create more noise
- Isolating noise sources in an enclosure by placing barrier between noise sources and workers.
- Increase distance between noise source and workers to decrease exposure
- Relocating exposed worker to other areas with low noise levels.
- Avoid operating noisy machines simmateniously at any time as the noise level will be much lower compared with operating lots of machines that will emit more noise levels.
- Reduce time and number of workers exposed at a given time in addition to wearing HPDs.
- Wearing of HPDs on noise zones to be made compulsory to prevent NIHL.
- Educating and training workers on health risk of exposure and control measure to be implemented including selection, fit test, proper use, care and maintenance of HPD.
- Workers to undergo audiometric tests regularly for early detection of NIHL, reporting if workers have more than 10% percentage loss of hearing (PHL) from baseline audiogram and prevention of NIHL.
- Reducing operating speed of machines
- Absorption of sound by covering correct surfaces with sound absorbent material.
- Policy on buy quiet equipment that generate low noise levels

6.6. Conclusion

The study established that workers were unprotected from extreme noise and wood dust of different size, concentration and composition. This study confirmed previous findings by Shamssain (1992) who reported that inhaling sawdust can increase risk of respiratory symptoms which escalates with duration of exposure. The aim of the study was to measure and determine the time-weighted average (TWA) occupational exposure levels to wood dust and noise and compare results with noise rating limit and OEL and to evaluate hearing loss and respiratory symptoms and use of PPE among sawmill workers within the Gert Sibande District Municipality. Majority of wood dust results were below South African OEL, while majority of the noise exposure results were above noise rating limit (H_a accepted and H_0 rejected). A non-significant difference on the levels of exposure to noise was observed when comparing the results of sawmill A to B (H_0 accepted and H_a rejected). Similarly, a non-significant difference on the levels of exposure to wood dust was observed when comparing the results of sawmill A to B (H_0 accepted and H_a rejected). Occupational exposure to wood dust and noise was associated with respiratory symptoms such as chest pains or shortness of breath and hearing loss conditions or symptoms such as tinnitus (ringing in the ears) and ear infections (H_a accepted and H_0 rejected). The exposed group reported always wear PPE than unexposed who wore it sometimes. The exposed did not wear PPE consistently due to not being available than unexposed who reported other reasons

6.7. References

American Conference of Governmental Industrial Hygienists (ACGIH). (2005) *Wood dusts*. Cincinnati, Ohio: ACGIH.

Ahmed, H.O., Dennis, J.H., Badran, O., Ismail, M., Ballal, S.G., Ashoor, A. and Jerwood, D. (2001) Occupational noise exposure and hearing loss of workers in two plants in eastern Saudi Arabia. *Annals of Occupational Hygiene* **45**(5):371-380.

Driscoll, D.P., Mikl, K. and Burgess, M. (2009) *W503 student manual noise: measurement and its effects.* Australia: University of Wollongong.

Dzhambov, A.M. and Dimitrova, D.D. (2016) Occupational noise and ischemic heart disease: a systematic review. *Noise Health* **18**(83):167–177.

Davies, B. and Henderson, J. (2009a) *W501 student manual measurements of hazardous chemical substances*. Australia: University of Wollongong.

Davies, B. and Henderson, J. (2009b) *W505 student manual control of hazardous substances*. Australia: University of Wollongong.

Eriksson, H. (2019 *Work-related cardiovascular disease*. (*P.h.D. Thesis*). Gothenburg: University of Gothenburg.

Franks, J. R., Stephenson, M. R and Merry, C.J. (1996) *Preventing occupational hearing loss: A practical guide*. Cincinnati, Ohio: NIOSH.

Gerges, S.N.Y., Vedsmand, L., Lester, H. and Bat-kartellet, K. (1995) Personal measures and hearing conservation. *Occupational exposure to noise: evaluation, prevention and control*. Geneva: World Health Organization (WHO).

Health and Safety Executive (HSE). (2013) *Respiratory protective equipment at work: A practical guide, HSG53*. 4th ed. London: HSE.

Hirst, A. (2010) *W201 Basic principles in occupational hygiene.* UK: OHTA.

Neitzel, R., Andersson, M.E. and Andersson, E.E. (2016) Comparison of multiple measures of noise exposure in paper mills. *Ann of Occ Hyg* **60**(5):581–596.

National Institute for Occupational Safety and Health (NIOSH). (1990) *Proceedings: Best practices in hearing loss prevention*. Washington, D.C: NIOSH.

National Institute for Occupational Safety and Health (NIOSH). (1998) *Criteria for a recommended standard, occupational noise exposure, revised criteria*. USA: NIOSH.

Passchier-Vermeer, W. and Passchier, W.F. (2000) Noise exposure and public health. *Environmental health perspectives* **108**(1):123-131.

Rabinowitz, P.M. (2000) Noise induced hearing loss. *Am Fam Physician* **61**(9): 2759-2760.

South African National Standard (SANS 10083). (2013) *The code of practice for the measurement and assessment of occupational noise for hearing conservation purpose.* Pretoria: SABS

Setia, M.S. (2016) Methodology Series Module 3: Cross-sectional Studies. *Indian J Dermatol* **61**(3):261-264. DOI: 10.4103/0019-5154.182410.

Shamssain, M.H. (1992) Pulmonary function and symptoms in workers exposed to wood dust. *Thorax* **47**(2):84-87.DOI:10.1136/thx.47.2.84.

Kadam, P. and Bhalerao, S. (2010) Sample size calculation. *Int J Ayurveda Res* **1**(1): 55-57.DOI:10.4103/0974-7788.59946

United Kingdom. (2005) The control of noise at work regulations.Available from: http://www.legislation.gov.uk/uksi/2005/1643/pdfs/uksi_20051643_en.pdf. (Accessed on 6 November 2018).

Appendix A: Ethical approval for the study

UNIVERSITY OF THE FREE STATE
UNIVERSITEIT VAN DIE VRYSTAAT
YUNIVESITHI YA FREISTATA

UFS·UV
HEALTH SCIENCES
GESONDHEIDSWETENSKAPPE

Health Sciences Research Ethics Committee

17-Jun-2020

Dear Mr Moeletsi Rathipe

Ethics Clearance: **Evaluation of occupational exposure to wood dust and noise among sawmill workers in the Gert Sibande District Municipality**
Principal Investigator: **Mr Moeletsi Rathipe**
Department: **Environmental Health Sciences - CUT**
APPLICATION APPROVED

Please ensure that you read the whole document

With reference to your application for ethical clearance with the Faculty of Health Sciences, I am pleased to inform you on behalf of the Health Sciences Research Ethics Committee that you have been granted ethical clearance for your project.

Your ethical clearance number, to be used in all correspondence is:**UFS-HSD2019/2236/3006**

The ethical clearance number is valid for research conducted for one year from issuance. Should you require more time to complete this research, please apply for an extension.

We request that any changes that may take place during the course of your research project be submitted to the HSREC for approval to ensure we are kept up to date with your progress and any ethical implications that may arise. This includes any serious adverse events and/or termination of the study.

A progress report should be submitted within one year of approval, and annually for long term studies. A final report should be submitted at the completion of the study.

The HSREC functions in compliance with, but not limited to, the following documents and guidelines: The SA National Health Act. No. 61 of 2003; Ethics in Health Research: Principles, Structures and Processes (2015); SA GCP(2006); Declaration of Helsinki; The Belmont Report; The US Office of Human Research Protections 45 CFR 461 (for non-exempt research with human participants conducted or supported by the US Department of Health and Human Services- (HHS), 21 CFR 50, 21 CFR 56; CIOMS; ICH-GCP-E6 Sections 1-4; The International Conference on Harmonization and Technical Requirements for Registration of Pharmaceuticals for Human Use (ICH Tripartite), Guidelines of the SA Medicines Control Council as well as Laws and Regulations with regard to the Control of Medicines, Constitution of the HSREC of the Faculty of Health Sciences.

For any questions or concerns, please feel free to contact HSREC Administration: 051-4017794/5 or email EthicsFHS@ufs.ac.za.

Thank you for submitting this proposal for ethical clearance and we wish you every success with your research.

Yours Sincerely

Dr. SM Le Grange
Chair : Health Sciences Research Ethics Committee

Health Sciences Research Ethics Committee
Office of the Dean: Health Sciences
T: +27 (0)51 401 7795/7794 | E: ethicsfhs@ufs.ac.za
IRB 0001 1992; REC 230408-011; IORG 0010096; FWA 00027947
Block D, Dean's Division, Room D104 | P.O. Box/Posbus 339 (Internal Post Box G40) | Bloemfontein 9300 | South Africa

Appendix B: Proof of language editing

CONFIRMATION LETTER FOR EDITING

Mr M. Koai
35 Winterland
WILLOWS
Cell nr : 082 309 7723
Email : mojalefakoai@gmail.com

TO: **Mr M. Rathipe**

Department: **Life Sciences**

Project Title: **Evaluation of occupational exposure to wood dust and noise among sawmill workers in the Gert Sibande District Municipality**

I wish to confirm that I have edited this study for the aforesaid student.

Yours sincerely

Mr M Koai

Appendix C: Consent to participate in research

Project title: **Evaluation of occupational exposure to wood dust and noise among sawmill workers in the Gert Sibande District Municipality (GSDM)**

You have been asked to participate in a research study and you have studied the information letter about this study.

Kindly note that your participation in this research is voluntary and you will not be penalised or lose benefits if you refuse to participate or decide to terminate participation.

If you agree to participate, you will be given a signed copy of this document as well as the information letter, which is a written summary of the research.

You may contact Mr Moeletsi Rathipe at: (+27) 738 3696 at any time if you need some clarity regarding the research study or alternatively the Secretariat of the Ethics Committee of the Faculty of Health Sciences at University of Free State (UoF) may be contacted at: (+27) 51 4052 812 if you have questions about your rights as a research subject.

The research study, including the above information has been verbally described to me. I understand what my involvement in the study means and i voluntarily agree to participate.

…… ……..

Signature of researcher Date

…… ……..

Signature of participant Date

…… ……

Signature of translator Date

(Where applicable)

Appendix D: Informational document/letter

Research topic: Evaluation of occupational exposure to noise and wood dust among sawmill worker's in the Gert Sibande District Municipality

Dear participants

I, Rathipe Moeletsi, a student pursuing a Master's Degree in Health Science in Environmental Health (MHscEH) at the Central University of Technology, Free State (CUT, FS). I am the principal researcher in this study and i am conducting a study on the evaluation of occupational exposure to noise and wood dust among sawmill workers in the Gert Sibande District Municipality. The study is purely an academic exercise and it forms part of the researcher work towards the award of a Master Degree in Environmental Health. This study aimed at investigating the health effects associated with exposure to wood dust and noise and to provide strategies for monitoring the exposure. The prevention of respiratory health symptoms such as cough, phlegm, wheezing, chest tightness and breathlessness among sawmill workers will help reduce exposure to wood dust not only in South Africa but also in other parts of the countries that will be using the same method. You are invited to take part in this study. This academic study will be beneficial to both employees and the company in raising awareness on noise and dust exposure.

Possible risk: There is no possible risk associated with this study but we anticipate some discomfort during the interviewing process given the sensitivity of some questions. You may feel uncomfortable answering those questions or you may not know the answer to a particular question. You are free to skip any questions you are not comfortable answering.

Possible benefits: There are no direct or indirect benefits to the participants of the study. However, the information provided will contribute to the overall knowledge on occupational exposure to wood dust and noise in the sawmilling. This is a voluntary participation study and you have the right to refuse to participate or withdraw your participation in this study. During the questionnaire completion process, you can choose not to answer any question that you do not want to answer. Additionally, you are at liberty to withdraw from the study or stop the process at any time. However, we will encourage you to participate and complete the questionnaire and participate in noise and dust monitoring since your opinions are very important in helping us to understand occupational exposure to occupational noise and wood dust in the sawmill at the Gert Sibande District Municipality.

Confidentiality: Information provided will be held absolute confidentially and data collected in this study are strictly for the research purpose and will be stored in electronically will a password protection. Access will be limited to ensured dissemination of findings from the study since participants will not be identified by their names**.** You may withdraw from the study at any time without any penalties.

Participation is voluntary. There are no costs payable to you to take part in the study.

For further information, /reporting of the study-related issues, you may contact the researcher:

Name: Moeletsi Rathipe
Cell: (+27) 737 383 694
Email: rathipemoeletsi819@gmail.com

Appendix E: Questionnaire

EVALUATION OF OCCUPATIONAL EXPOSURE TO WOOD DUST AND NOISE AMONG SAWMILL WORKERS IN THE GERT SIBANDE DISTRICT MUNICIPALITY

This is an independent research project of the Central University of Technology, Free State. The purpose of this project is to identify problems associated with exposure to wood dust and noise in your company and to make recommendations to improve the current situation. The information provided on the questionnaire will be kept confidential. Your name and company will remain anonymous throughout the entire project and after completion the project.

Instructions

Please complete the questionnaire by putting "X" next to the relevant answer where applicable and by writing answers in the lines provided.

SECTION A: Biographical information

1. What is your gender?

Male	
Female	

2. What is your age? ……..... years

3. What is your race or ethnic group?_____

Black	
White	
Other, specify:	

4. What is your marital status?_____

Single	
Married	
Divorced	
Widowed	
Other, specify	

5. What is your highest level of education?

None	
In-house training	
Primary	
Secondary	
Matric	
Tertiary	

SECTION B: Work-related information

6. Were you previously employed in a dusty job?

Yes	
No	

7. What is your current job title? _____

Operator	
Packer	
Receiver	
Artisan	
Stacker	
Feeder	
Supervisor	
General worker	
Cleaner	
Other, specify	

8. How long have you been in this position? _____years

9. Have you ever received training about the operations within the sawmill factory?

Yes	
No	

SECTION C: Health-related information

10. Have you ever suffer from nose or throat irritation in past three months?

Yes	
No	

11. Do you usually cough?

Yes	
No	

11.1. If yes, to question (11) do you cough in the morning, during the day or at night?

Morning	
During	
Night	

11.2. How long does the cough last?

1-3 days	
3-5 days	
5 days and more	

11.3. Do you produce phlegm's from your chest when coughing?

Yes	
No	

11.4 Do you suffer from chest pains or shortness of breath when coughing?

Yes	
No	

12. Have you suffered from chest illness in the past three months?

Yes	
No	

13. Have you had more than one illness like this in the past three years?

Yes	
No	

14. Have you ever been told by the doctor that you had one of the following conditions?

No.	Condition	YES	NO
14.1	Heart failure		
14.2	Bronchitis		
14.3	Bronchial asthma		
14.4	Pneumonia		
14.5	Pulmonary tuberculosis		
14.6	Other chest problem		
14.7	None of the above		

15. Do you smoke?

Yes	
No	

15.1. If yes, to question (15), which of the following do you smoke? _____

Cigarette	
Dagga	
Other, specify	

15.2 If cigar, tobacco or other, how many do you smoke per day? _____ or per week? _____

15.3 If you smoke, are you planning to cut down smoking?

Yes	
No	

15.4 If yes to question (15.3), do you require any assistance to quit smoking?

Yes	
No	

SECTION D: Work related questions

16. How many hours do you work daily? _____ and weekly?_____

17. Does the company subjected you to a medical fitness test?

Yes	
No	
Don't know	

17.1 If yes to question (17), does the medical fitness test cover spirometry (lung function test), chest x-ray and audiometric test (hearing test)?_____

Yes	
No	
Don't know	

17.2. If yes to question (17.1), when was the latest test conducted? _____Day/month/year

18. Did your medical report from the occupational medical doctor/nurse require follow ups?

Yes	
No	
Don't know	

18.1 If yes, to question (18), was it related to the following?_____

Hearing loss	
Cancer	
Heart disease	
Other, specify	

19.Do you know that Occupational related diseases such as noise induce hearing loss or cancer contracted while at work are compensateable in terms of the Compensation for Occupational Injuries and Diseases Act No. 130 of 1993 (as amended) and that the employer/occupational medical doctor must report the disease by forwarding the WCL1 form to the Provincial Director (Workman's Compensation Fund) within 14 days for your claim to be processed and enjoy the benefits of the fund?

Yes	
No	

SECTION E: Personal Protective Clothing (PPE)

20. What type of PPE do you wear?_____

Helmet	
Ear plugs	
Safety glass	
Respiratory mask	
Protective gloves	
Overall	
Apron	
Safety boots	
Other, specify	

20.1. How often do you wear your PPE?

Always	
Sometimes	
Never	

20.2. How often do you wear dust mask and ear plugs?

Always	
Most of the time	
Sometimes	
Never	

20.3. If never, why don't you utilise the dust mask and ear plugs?_____

They are not available.	
They are uncomfortable.	
I have a difficulty breathing.	
I can't hear signals	
Other reasons, specify	

20.4. Do you wear the following devices when working on dust/noise areas on a daily basis?

No.	Safety device	YES	NO
20.4.1	Respiratory mask		
20.4.2	Ear plugs		
20.4.3	Safety glass		
20.4.4	Protective gloves		
20.4.5	Overalls		
20.4.6	Apron		
20.4.7	Safety boots		
20.4.9	Other, specify		

20.5. Do you have a history of hearing loss?

Yes	
No	

20.6. Do you wear hearing aid(s)?

Yes	
No	

20.7. If yes, to question (20.6) on which ear?

Left ear	
Right ear	
Both ears	

20.8. Have you ever experienced the following health conditions in the past twelve months?

No.	Health condition	YES	NO
20.8.1	Ringing in ears		
20.8.2	Ear infection		
20.8.3	Ruptured eardrum		
20.8.4	Ear injury		
20.8.5	Other, specify		

Thank you for your assistance

Appendix F: Field sheet for wood dust and noise

Company/Facility code:_____ Area/location:_____
Sample by:_____ Date:_____
Temperature:_____ Altitude(pressure):_____

Chemical to be monitored:_____ Lab analysis required:_____
Operation to be monitored/task performed:_____
Maximum Risk Employee (MRE):_____Group sampling:_____HEG:_____

Sampling media type: _____
Type of tubing used to connect the pump to the sampling head:_____
Clip-on holder for attaching the filter holder on worker breathing zone:_____
Belt or any attaches to which the sampling pump was secured:_____
Cyclone used: _____
Sample number:_____ Model and pump number:_____
Employee name or Coy No.:_____ Job title/Designation:_____
Leak test before and after:_____

Type of sample
Personal:_____ Breathing zone:_____ Zonal/Area:_____
Operating conditions:_____

Engineering control
LEV system:_____ Natural ventilation:_____ Enclosure:_____
Dust suppression:_____Automation:_____ Demarcation of noise/respiratory zones:_____ Other:_____

HPD/RPD
HPD/RPD provided_____ HPD/RPD worn correctly and consistently_____
HPD/RPD Type_____Brand_____Reduction/protection factor_____
Hearing/respiratory programme in place?_____
Supplier instructions and information_____

Shift:
Day_____ Afternoon_____Night_____ From_____ To_____

Calibration
Location:_____ by:_____ Date:_____
Time on:_____ Time off:_____Total time (min):_____
Pre-calibration:_____Post-calibration:_____Average flow rate (l/m):_____
Deviation within ± 5% (Flow rate after - before ÷by flow rate before measurement) x 100:_____
Volume = flow rate x time÷1000:_____m^3

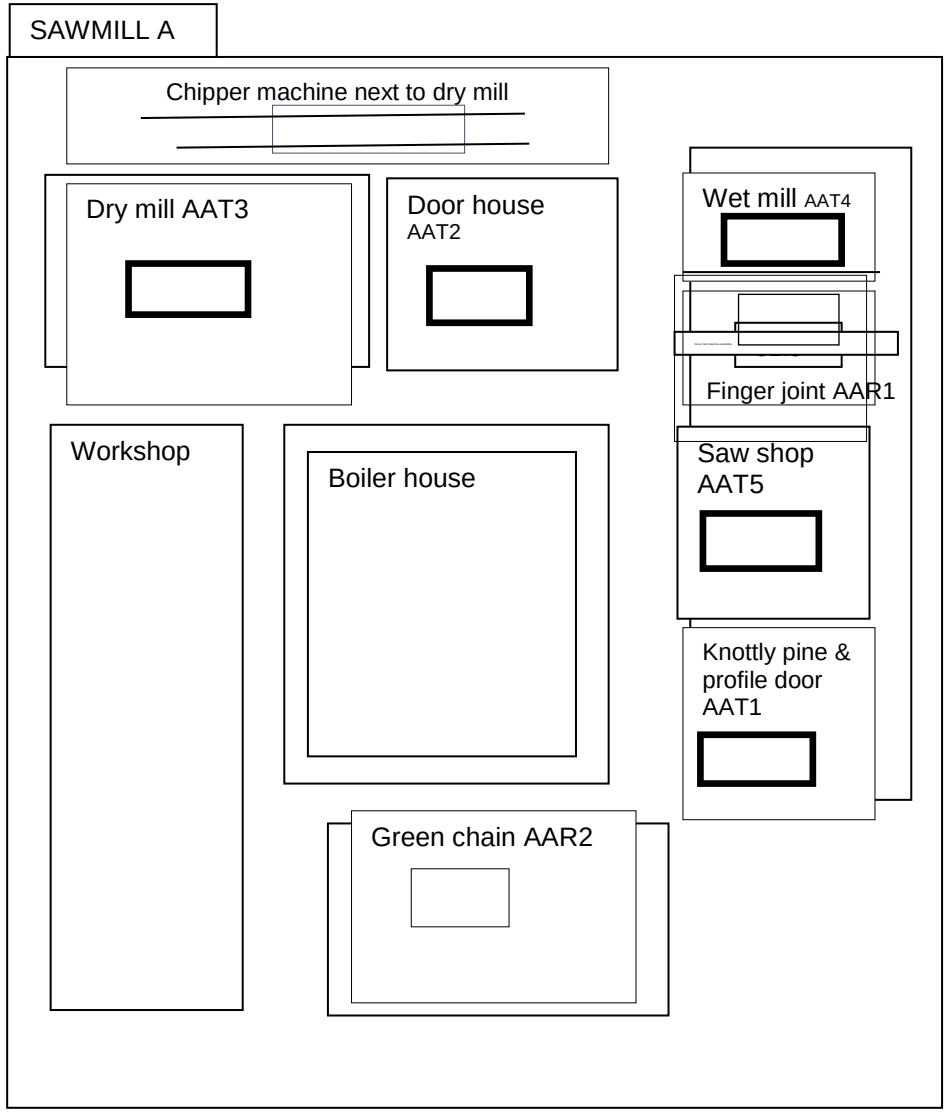
Sampling/analytical method used_____
Evidence of accuracy:_____
Remarks, possible interference and action taken: _____
Results of sample analysis or instrument reading:_____
Employee exposure (average - 8hours or 15 minutes):_____sample number:_____

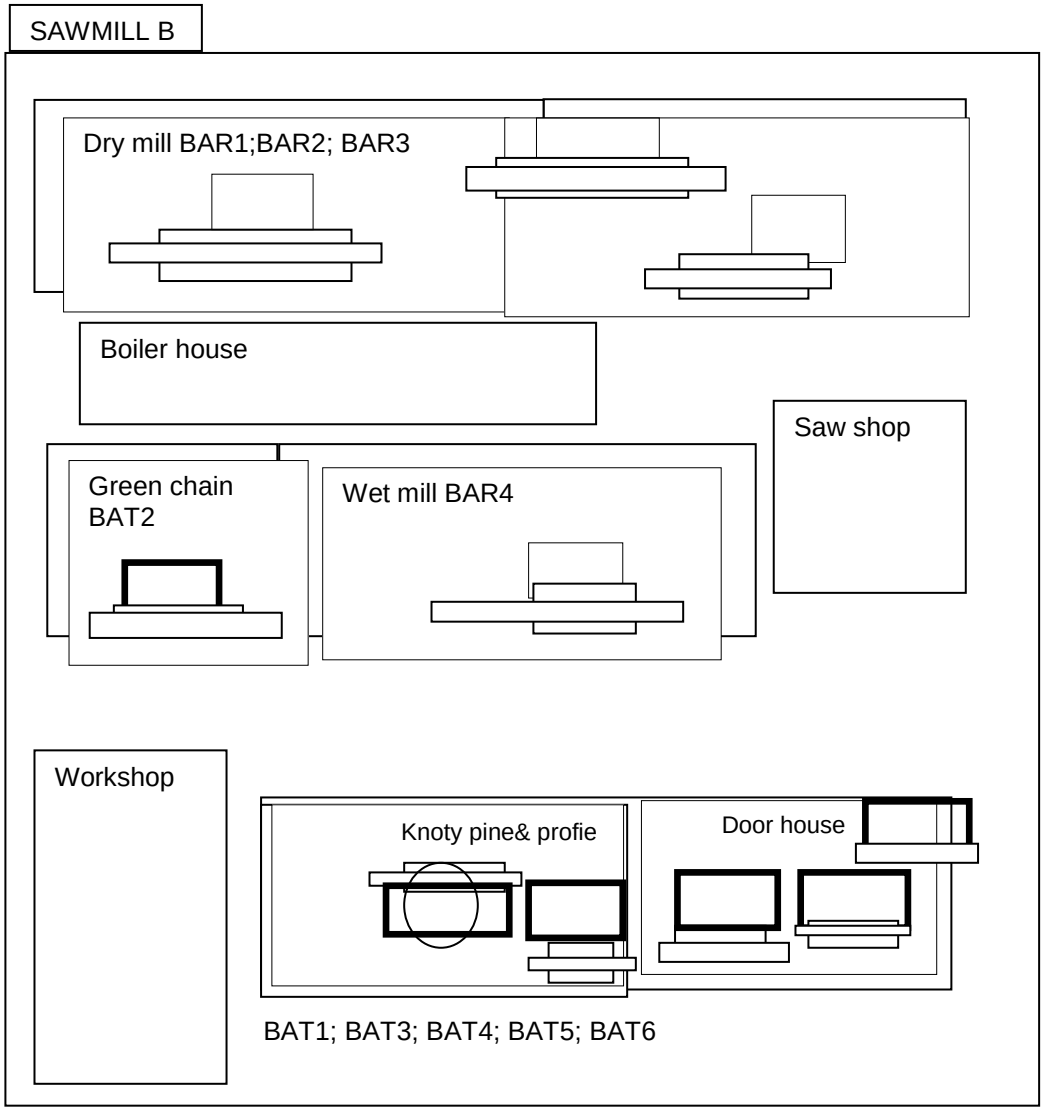
Appendix G: Sample sheet for wood dust monitoring

Company code/work area:_____ Section/Department:_____Contact person and Tell No:_____
Sampled by:_____ Date:_____

Job title of participant/ Area Sampled	Occupation/work Performed/Location of a Sampling Train	Sampler Details		Average Flow rate (l/m)	Total Time (min)	Volume of air Sampled (litres)	TWA-E8heV Con.	TWA – OEL	Observation and comment
		Sample No:	Pump S/N:						

Appendix H: Floor plan of area wood dust results at sawmill A and B





LEGEND

Area respirable wood dust results below 2.5 action level and 5mg/m^3 OEL

Area total inhalable wood dust results below 2.5 action level and 5mg/m^3 OEL

AAR Area respirable wood dust results at sawmill A
AAT Area total inhalable wood dust at sawmill A
BAR Area respirable wood dust results at sawmill B
BAT Area total inhalable wood dust results at sawmill B

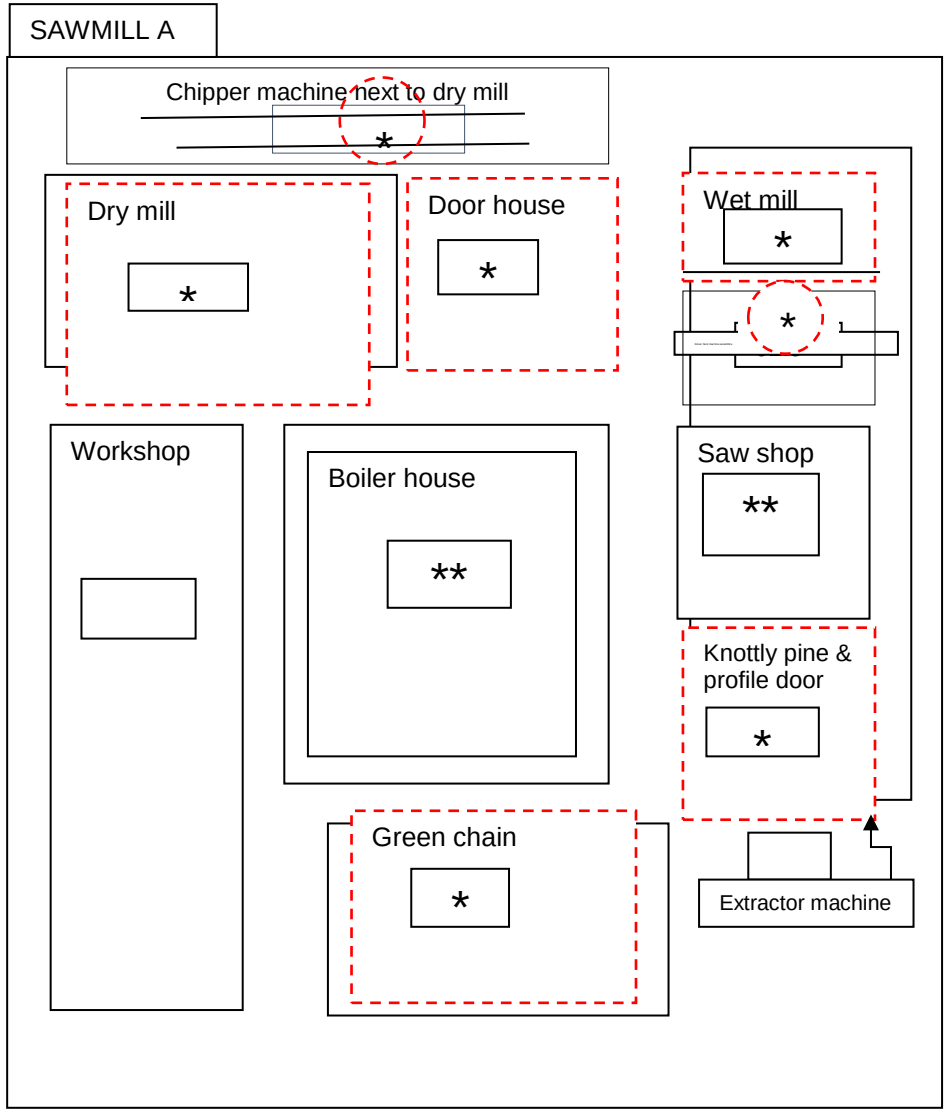
Appendix I: Sample sheet for noise evaluation

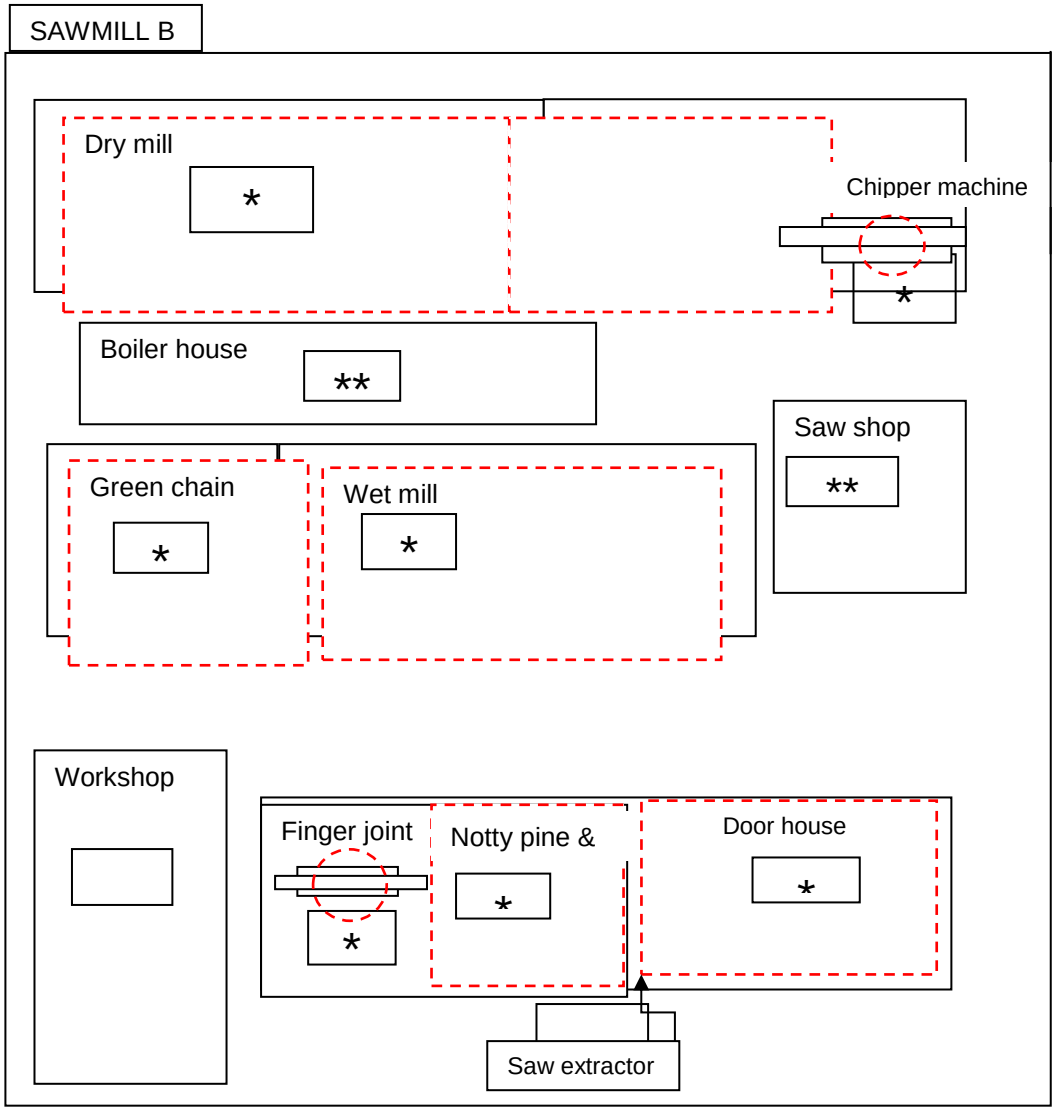
Company code/ Workplace:_____ Section/department:_____Contact person and Tell No:_____
Sampled by:_____ Date:_____

Job title of participant./ area evaluated	Occupation/task perform/ measuring Point ($L_{Req,T}$) dB(A)	Dosimeter Serial No.	Personal	Area	Statutory Limit 85 dB(A)	Observation and comment/Sources of exposure
			Measured *L*Req,8h dB(A)	Average noise rating (*L*Req,T), dB(A)		

255

Appendix J: Floor plan of area noise results at sawmill A and B

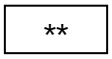




LEGEND


Average noise rating levels ≥85 dB(A) demarcated as noise zones

Average noise rating levels ≥85dB(A) noise rating limit (NRL)



Average noise rating level ≥82dB(A) action level but <85dB(A) NRL


Noise reading taken 1m away from machinery beyond 85dB(A) NRL

Appendix K: Letters of request to conduct the study

Joubert St/Merino Trust Building
Ermelo
2350
06 August 2019

The Chief Executive Officer
Maswati Phambile Investments (PTY) LTD
Farm Rinkink
Lothair, 2370
Tell: 017 845 1098/9

Dear Sir/Madam

Request to conduct a research study at Maswati Phambile Sawmill, Lothair

I, Rathipe Moeletsi with student Number: 210015942 hereby requests consent to conduct a research study at Maswati Phambile located at Farm Rinkink, Lothair, Mpumalanga

The title of the study is **evaluation of occupational exposure to wood dust and noise among sawmill workers in Gert Sibande District Municipality**

The study is purely an academic exercise and it forms part of my research work towards the award of a Masters of Health Science Degree. In this study, wood dust and noise exposure will be assessed in different departments (log yard to final distribution) in the mill utilising instruments. Workers will be required to complete a questionnaire to gather information on the knowledge of exposure to wood dust and noise in the mill.

The name(s) of the companies and participants involved in the study will not be included in the research report. The statistical results obtained will be used for writing the manuscripts and the dissertation. Thank you very much for your time and consideration, for any information regarding the project, you can contact me at (073) 738 3694 or alternatively on this email: moeletsi.rathipe@labour.gov.za.

I trust that you will consider the above request favourably.

Yours Faithfully

Rathipe Moeletsi
OHS Inspector
Department of Labour: Ermelo
Cell: 073 738 3694 Tel: 017 819 7632
e-mail: moeletsi.rathipe@labour.gov.za

Kindly indicate your approval by marking the appropriate box below and acknowledge receipt

[X]	[ ]
APPROVED	NOT APPROVED
M J van Wyk	8/8/2019
Signature of CEO/Manager	Date

Appendix L: Letters of request to conduct the study

Joubert St/Merino Trust Building
Ermelo
2350
16 July 2019

The Chief Executive Officer
York Timbers (Pty) Ltd
Jessievale, N17 Oshoek Road, Ermelo, 2351
Tell: +27 17 845 1337

Dear Sir/Madam

Request to conduct a research study at York Timbers (Pty) Ltd Sawmill

I, Rathipe Moeletsi with student Number: 210015942 hereby requests consent to conduct a research study at York Timbers (Pty) Ltd Sawmill located at Jessievale, N17 Oshoek Road, Ermelo, 2351

The title of the study is **evaluation of occupational exposure to wood dust and noise among sawmill workers in Gert Sibande District Municipality**

The study is purely an academic exercise and it forms part of my research work towards the award of a Masters of Health Science Degree. In this study, wood dust and noise exposure will be assessed in different departments (log yard to final distribution) in the mill utilising instruments. Workers will be required to complete a questionnaire to gather information on the knowledge of exposure to wood dust and noise in the mill.

The name(s) of the companies and participants involved in the study will not be included in the research report. The statistical results obtained will be used for writing the manuscripts and the dissertation. Thank you very much for your time and consideration, for any information regarding the project, you can contact me at (073) 738 3694 or alternatively on this email: moeletsi.rathipe@labour.gov.za.

I trust that you will consider the above request favourably.

Yours Faithfully

Rathipe Moeletsi
OHS Inspector
Department of Labour: Ermelo
Cell: 073 738 3694 Tel: 017 819 7632
e-mail: moeletsi.rathipe@labour.gov.za

Kindly indicate your approval by marking the appropriate box below and acknowledge receipt

☐	☐
APPROVED	NOT APPROVED
……	……
Signature of CEO/Manager	Date