Occupational risk factors for ischaemic heart disease

Differences among males and females in the Māori and general populations of New Zealand

A thesis with publications presented in partial fulfilment of the requirements for the degree of

Doctor of Philosophy

in

Public Health (epidemiology)

at Massey University, Wellington, New Zealand

Lucy Anne Barnes

2021

ABSTRACT

Occupation is a poorly characterised risk factor for cardiovascular disease, a leading cause of death worldwide, with females and indigenous peoples underrepresented in research. This thesis assessed associations between occupation/occupational exposures and cardiovascular risk factors and ischaemic heart disease (IHD), for males and females of the Māori and general populations of New Zealand.

Two previous New Zealand Workforce Surveys (NZWS) in the general (NZWS; n=3003; 2004-2006) and Māori (Māori NZWS; n=2107; 2009-2011) populations were linked to Ministry of Health data in Statistics New Zealand's Integrated Data Infrastructure. Age-adjusted logistic regression was used to assess associations between occupational groups and cardiovascular risk factors (high blood pressure, high deprivation, high cholesterol, diabetes, smoking and obesity). Cox proportional hazard regression, adjusted for age, deprivation and smoking, was used to assess associations between ever working in an occupational group and incident IHD, over a 7-14 year follow-up period. Associations with occupational exposures were also assessed.

Cardiovascular risk factors were disproportionately experienced by blue-collar workers, particularly *plant and machine operators and assemblers* and *elementary workers.* In contrast, *professionals* were less likely to experience risk factors. Similarly, having ever worked as a *plant and machine operator and assembler* or *elementary worker* was positively associated with IHD for Māori females. Having ever worked as a *technician and associate professional* was inversely associated with IHD. Associations were not consistent between Māori and the general population or between males and females, with differences observed for *clerks, agriculture and fishery workers* and *sales and service workers*.

Occupational exposure to dust, tools that vibrate, smoke or fumes, oils and solvents, and high frequency exposure to repetitive tasks, loud noise, working at high speeds and awkward grip or hand movements, common in blue-collar occupations, was positively associated with IHD. Associations were not consistent across all populations, with no positive associations observed among Māori males.

In conclusion, this thesis shows a role of occupation in IHD risk, presenting an opportunity for IHD interventions, particularly for blue-collar occupations. However, findings suggest that occupational risk factors cannot be generalised across all populations and future research and intervention development therefore requires consideration of sex and ethnicity.

“Science and everyday life cannot and should not be separated.”

- *Rosalind Franklin*

Acknowledgments

In 2017, I embarked on this journey at the Centre for Public Health Research (CPHR) and nearly 4 years later, I feel like a different person – older, wiser and significantly more tired. There have been many ups and downs, doubts and triumphs, but above all else, lessons learned, skills gained and friendships made. I know this experience has shaped who I am and how my career will unfold for the better. I feel extremely grateful to have been able to complete this journey and it would not have been possible without the incredible team and support network I have.

Firstly, I would like to thank my primary supervisor, Jeroen Douwes. Thank you for taking a chance on a young immunologist and trusting she would be able to complete a 'data analysis heavy' Ph.D. in a different field. I couldn't have done this without your enthusiasm, guidance and support. Secondly, I would like to thank my other supervisors, Amanda Eng and Andrea 't Mannetje. Amanda, thank you for your patience and advice as I have come to you with thousands of questions over the years. Andrea, you are the queen of clever ideas; thank you for constantly elevating my work and your words of encouragement.

I would also like to thank the other members of the CVD team. Hayley Denison and Marine Corbin, I am so grateful for your friendship and help every step of the way. Stephen Haslett, although your statistical feedback sometimes presented challenges, I believe my thesis is much better for your guidance, thank you. Dave McLean, thank you for your advice and encouragement along the way. Thank you also to my co-investigators, Rod Jackson and Lis Ellison-Loschmann, for providing new perspectives and useful advice on my papers. Finally, a big thank you to all the interviewers and the participants involved in the original collection of the survey data, without your work my thesis would never have existed.

Thank you to all of the staff at CPHR for your kindness and friendship, you have all made me feel incredibly welcome. I would especially like to thank Nat and Hils who have been my ‘work mums’ throughout my Ph.D. and have always looked after me. I am grateful to have shared an office with fellow PhDers, celebrating successes and sharing each other’s pain; thank you to Hajar, Grace, Nia and Hana, for the support, friendship, laughter and dreams about the future. Dani, thank you for your support and camaraderie, especially in the last few months. Good luck to all of you, I wish you all the best for your future adventures.

Thank you also to my incredible friends; Georgia, Alice, Kate, Aleshia, Melissa and Jess. Thanks for being an integral part of my world throughout high school and university, I am so grateful for your endless support, patience (I swear I’m finally finished at uni!) and entertainment. India, thank you for being my biggest cheerleader and my dearest Laurel, thank you for being my number one since the beginning of this crazy university adventure, I am so grateful to have had you by my side as we’ve navigated our twenties together. Thank you also to Peter and the Linney family for your love and support, I feel so blessed to have had you as my Wellington family.

Finally, I would like to thank my family. Dad, throughout my university journey you have always been there to help, to soundboard ideas off and talk through any challenges. Mum, you have always been there with a hug, kind words or booking flights home so I could be with you when I needed it most! Thank you both for always encouraging me to do my best and loving me unconditionally. And of course, I would like to thank my sister, Rosie. You have always been my number one support person, by my side at both the darkest and the happiest of times, and I would not be here without you. I am so lucky to have you as my sister.

Statement of authorship

This thesis was produced according to Massey University's 'Thesis with publications' requirements. Chapters 4, 5 and 6 have been written as individual research papers. Consequently, there is some repetition in the introduction, methods and population characteristics sections of those chapters. Each results chapter is set out in the style of the journal to which it has been/will be submitted and they have been written in the first-person plural.

The authors for all of the research papers in this thesis include my Ph.D. supervisors, colleagues and collaborators from different institutes in New Zealand. Chapter 4 has been published in the *Annals of Work Exposures and Health;* Chapters 5 and 6 have been submitted to international journals and are currently under review.

While assisted and supported by my co-authors and supervisors, my contribution was the greatest. I formulated the concept for each chapter, assisted with the data extraction, carried out all of the statistical analyses and prepared the manuscripts before receiving valuable feedback from my co-authors.

Integrated Data Infrastructure Disclaimer

The results in this thesis are not official statistics. They have been created for research purposes from the Integrated Data Infrastructure (IDI), managed by Statistics New Zealand.

The opinions, findings, recommendations, and conclusions expressed in this paper are those of the author(s), not Statistics NZ.

Access to the anonymised data used in this study was provided by Statistics NZ under the security and confidentiality provisions of the Statistics Act 1975. Only people authorised by the Statistics Act 1975 are allowed to see data about a particular person, household, business, or organisation, and the results in this paper have been confidentialised to protect these groups from identification and to keep their data safe.

Careful consideration has been given to the privacy, security, and confidentiality issues associated with using administrative and survey data in the IDI. Further detail can be found in the Privacy impact assessment for the Integrated Data Infrastructure available from www.stats.govt.nz.

Table of contents

List of figures

List of tables

List of abbreviations

ANZSIC	Australia and New Zealand Standard Industrial Classification 1996
BCW	Blue-collar workers
CHD	Coronary heart disease (another term for ischaemic heart disease)
CPHR	Centre for Public Health Research
CVD	Cardiovascular disease
dB	Decibel
ERI	Effort-reward imbalance (job stress model)
HbA1C	Haemoglobin A1C (a measure of blood sugar)
HDL	High-density lipoprotein (cholesterol)
HR	Hazard ratio
ICD	International Classification of Diseases
IDI	Integrated Data Infrastructure
IHD	Ischaemic heart disease
JEM	Job exposure matrix
LDL	Low-density lipoprotein
LTPA	Leisure time physical activity
Māori NZWS	Māori New Zealand Workforce Survey
MI	Myocardial infarction
MoH	Ministry of Health
NHI	National Health Index
NZ	New Zealand
NZDep2006	New Zealand deprivation index 2006
NZSCO	New Zealand Standard Classification of Occupations 1999
NZWS	New Zealand Workforce Survey
OPA	Occupational physical activity
OR	Odds ratio
PR	Prevalence ratio
RR	Relative risk
S	Suppressed data (counts under 6 and associated statistical tests)
SES	Socioeconomic status
SCORE	Systematic Coronary Risk Evaluation
SD	Standard deviation
U.S.A.	The United States of America
WCW	White-collar workers

CHAPTER ONE

Introduction, aims and outline of thesis

1.1 Introduction

Cardiovascular disease (CVD) is the leading cause of death worldwide[1] and accounts for 33% of deaths in New Zealand each year.[2] Atherosclerosis is the most common underlying pathophysiology of CVD and is characterised by a build-up of lipid-laden plaques in the arteries that can lead to vessel occlusion and reduced blood flow to different parts of the body.[3, 4] Decades of research have identified conventional risk factors for CVD and these include non-modifiable factors such age, sex, ethnicity and a family history of CVD. [4, 5] Other potentially modifiable cardiovascular risk factors include: smoking, high blood pressure, high cholesterol, diabetes, socioeconomic status (SES) and obesity (high body mass index (BMI)).[4, 5] Although public health strategies targeting the reduction of these risk factors have had a major impact on the overall morbidity and mortality of CVD,[6, 7] the effectiveness has not been the same for everyone, with females and ethnic minorities often not optimally benefiting from these interventions.[8, 9] Research continues into alternative methods of minimising these known risk factors and identifying additional risk factors.

Previous work has suggested that occupation is a plausible risk factor for CVD and may be a novel avenue for minimising CVD morbidity.[10] Most of the world's population will spend one-third of their adult life at work, and therefore, the physical, organisational, chemical and psychosocial factors in that environment have the potential to affect cardiovascular health. In New Zealand, it has been estimated that 18.9% of CVD mortality in males is attributable to work and 9.1% in females,[11] suggesting that reducing work-related CVD could have a significant impact on overall CVD mortality. Minimising exposure to occupational risk factors presents a unique opportunity for population-level rather than individual-level interventions.

Although research into occupation and CVD dates back to the 1950s, [12] there are significant gaps and a lack of consistency in knowledge, which has complicated the implementation of occupation-based interventions. Literature has highlighted significant differences between blue- and white-collar occupations in the prevalence and distribution of conventional cardiovascular risk factors, such as smoking, SES, BMI, high cholesterol, high blood pressure, diabetes and inflammation markers.[13] In general, blue-collar occupations have been associated with a higher prevalence of CVD risk factors.[14] Further research has found specific associations between some occupations and high CVD risk, many of which are again blue-collar occupations, including factory workers and drivers, although a few white-collar occupations, such as clerks, have also been identified as representing a higher risk.[15] In New Zealand specifically, spinners, weavers and dyers, and plant machine operators and assemblers have been associated with having a higher risk of CVD .[16, 17] Potential causal exposures have been studied to various degrees with differing levels of evidence. These exposures include stress, irregular working hours, noise, physical exertion, sedentary work, pesticides and other chemicals.[18-20] Many of the hypothesised causal pathways associated with these exposures include metabolic changes, weight gain and increased blood pressure, potentially explaining the increased prevalence of cardiovascular risk factors observed in some high-risk occupations.[21-23]

To date, the literature has primarily focused on males. As a greater proportion of the female population currently work compared to several decades ago, extending the research to include women and identifying sex-specific results is increasingly important. Similarly, relatively few studies have focussed on ethnic minorities, despite these groups making up a large proportion of the workforce at risk of occupational ill-health. In New Zealand, associations between occupation and CVD have not previously been studied in Māori, the indigenous peoples of New Zealand, even though Māori have a considerably greater cardiovascular risk factor burden and are twice as likely to die of CVD compared to non-Māori.[24, 25]

The aim of this thesis is to assess the association between occupation and CVD in New Zealand using occupational surveys, one conducted in the general population and the other in the Māori population. Analyses were conducted separately for these surveys as they were undertaken at different time points with different follow-up periods and response rates. In addition, results were further stratified by sex. This allowed ethnic and sex-specific associations to be assessed, which is important as exposure conditions and co-exposures (e.g. systemic racism and gender discrimination) are likely to be different between Māori and non-Māori[26] and males and female.[27] These factors may affect the association between occupational exposure and CVD risk. Also, there are physiological differences between males and females,[28, 29] which also likely affects associations between occupational exposures and CVD risk. Thus, the studies described in this thesis include roughly equal numbers of participants in each subgroup to ensure that the evidence derived from the research is based on the whole workforce including females and Māori. This should ensure that any future interventions resulting from this work are relevant to all members of the workforce.

Although the review of the literature (Chapter 2) covers all forms of CVDs, ischaemic heart disease (IHD) will be the focus of the analyses and discussion as it is the most prevalent CVD in New Zealand (and internationally). Occupational information from the New Zealand workforce surveys was linked to routinely collected health data of the Integrated Data Infrastructure (a longitudinal meta-dataset linking data from governmental agencies). The analyses in this thesis assess the cross-sectional relationship between occupational groups and conventional cardiovascular risk factors and longitudinally assess the association between occupational factors and the development of incident IHD for different populations in New Zealand. However, due to the limited study power of the surveys, differences between groups cannot be formally tested and are instead assessed qualitatively. These findings identify high-risk occupational groups, exposures and relevant cardiovascular factors that may be contributing to IHD risk in New Zealand, extending the evidence base required to develop effective and equitable interventions to reduce IHD in New Zealand (and internationally).

1.2 Aims

The overall objective of this thesis is to assess associations between occupation and IHD for males and females of the Māori and general populations of New Zealand.

The specific aims are to:

I. Assess the prevalence and distribution of conventional cardiovascular risk factors across occupational groups;
II. Assess associations between occupational groups and IHD;
III. Assess associations between occupational exposures and IHD;
IV. Assess whether associations between occupations/occupational exposures and IHD are consistent between males and females;

V. Assess whether associations between occupations/occupational exposures and IHD are consistent for the Māori and general populations of New Zealand.

1.3 Outline of thesis

Chapter One: Introduction, aims and outline of the thesis

This chapter introduces the thesis, providing a brief overview of what is currently known of occupational risk factors for CVD, gaps that exist and the rationale for extending this knowledge. This is followed by the aims and structure of the thesis.

Chapter Two: Background

This chapter provides the background for this thesis by summarising and analysing the current literature focused on CVD and risk factors in an occupational health context. The literature on cardiovascular risk factors and CVD risk by occupation and potential high-risk occupational exposures is reviewed.

Chapter Three: Methods

This chapter outlines the methods used for the collection of data in the NZWS survey and from the Integrated Data Infrastructure and briefly discusses how it is used for the analyses described in chapters 4, 5 and 6.

Chapter Four: The prevalence of cardiovascular risk factors in different occupational groups in New Zealand

In this chapter, the prevalence of conventional cardiovascular risk factors, including diabetes, high blood pressure, high cholesterol, obesity, low SES and smoking, are presented across occupational groups in New Zealand.

Chapter Five: A longitudinal linkage study of occupation and ischaemic heart disease in the general and Māori populations of New Zealand

This chapter presents the analyses focusing on associations between ever working in a range of occupations and industries and incident IHD.

Chapter Six: Ischaemic heart disease and occupational exposures – a longitudinal linkage study in the general and Māori populations of New Zealand

In this chapter, associations between a range of self-reported occupational exposures and incident IHD are described.

Chapter Seven: Discussion and conclusions

This final chapter summarises the main findings of the thesis, reviews the strengths and limitations, and discusses the implications of this research in future work and occupation-based interventions.

References and appendix

This final section presents the literature referenced throughout this thesis and statements of contribution for the publications included in this thesis.

CHAPTER TWO

Background

This literature review presents the literature investigating the associations between occupation, occupational exposures and CVD. The first section briefly discusses CVD and conventional cardiovascular risk factors. Section 2.2 discusses the role of occupation in cardiovascular risk. Section 2.3 evaluates the literature describing the prevalence and distribution of conventional risk factors across occupations. Section 2.4 discusses studies that have identified occupations associated with CVD and Section 2.5 briefly discusses some of the occupational exposures that have been linked to CVD. Section 2.6 summarises the relationship between occupation and CVD and highlights the current knowledge gaps, some of which are addressed by the analyses described in this thesis.

2.1 Cardiovascular disease

2.1.1 Cardiovascular diseases

CVD is the leading cause of death worldwide;[30] in New Zealand, 33% of all deaths each year are due to CVD.[24] The term CVD encompasses a range of diseases including ischaemic heart disease (also known as coronary heart disease (CHD)), cerebrovascular disease and peripheral arterial disease; Table 2.1 contains a list of CVDs as defined by the World Health Organisation (WHO).[31] In New Zealand, IHD accounts for over half of the overall CVD mortality and the estimated prevalence in 2018-2019 was 4.3%.[32]

For the purpose of this literature review, the focus will be on CVDs in general as limiting the focus on IHD would significantly reduce the available literature and thus, limit the ability to make robust conclusions about existing knowledge and gaps in research. However, the subsequent findings and discussion of this thesis will specifically focus on IHD because this is the most prevalent form of CVD in New Zealand.

Table 2.1. Cardiovascular diseases as defined by the World Health Organisation[31]

Cardiovascular disease	Alternative name	Definition
Ischaemic heart disease	Coronary Heart Disease	Disease of the blood vessels supplying the heart.
Cerebrovascular disease	Stroke, transient ischaemic attack	Disease of the blood vessels supplying the brain.
Peripheral artery disease		Disease of the blood vessels supplying the arms and legs.
Rheumatic heart disease		Damage to the heart muscle and heart valves from rheumatic fever, caused by streptococcal bacteria.
Congenital heart disease		Malformations of heart structure existing at birth.
Deep vein thrombosis and pulmonary embolism		Blood clots in the leg veins, which can dislodge and move to the heart and lungs.

The primary pathogenesis of CVDs (other than rheumatic heart disease, congenital heart disease, and deep vein thrombosis and pulmonary embolism) is known as atherosclerosis, a progressive disease characterised by chronic inflammation resulting in a build-up of lipid-laden plaques with a layer of fibrous connective tissue within the arteries.[3] This process is driven by the immune system but is affected by many factors that are not fully understood, with low-density lipoprotein (LDL cholesterol) and endothelial dysfunction thought to be key initiators.[4] The clinical presentation of CVDs includes chronic symptoms such as angina, due to the narrowing of the artery lumen, while plaque rupture may cause an acute event of artery occlusion and restriction of blood and oxygen flow.[4] Identifying risk factors that influence the progression of atherosclerosis or stability of the plaque is key to prevent and reduce CVD mortality and morbidity.

The Framingham Heart study pioneered early research into modifiable and non-modifiable cardiovascular risk factors.[5] Residents of Framingham, Massachusetts, in the United States of America (U.S.A.) between the ages of 30 and 59 years old were monitored for 12 years from 1948 to track potential cardiovascular risk factors. Within the first decade, several risk factors were identified including age, sex, blood pressure, high cholesterol, body weight, diabetes, smoking, lack of physical activity and alcohol consumption. These factors still account for most of the cardiovascular risk worldwide, in both males and females, and all ages.[33]

Public health interventions have primarily targeted behaviour to reduce the prevalence of tobacco smoking, diabetes, high cholesterol and high blood pressure, and this has contributed to half of the reduction in CVD mortality internationally and in New Zealand, while medical therapies account for the other half of the mortality reduction.[6, 7, 34] However, research is increasingly directed at social and environmental factors (e.g. air pollution[35]), which can be targeted at a population level to reduce the prevalence of physiological intermediates (e.g. high blood pressure, high cholesterol, diabetes and inflammation).

2.1.2 Conventional cardiovascular risk factors

Environmental, behavioural and socioeconomic factors may cause physiological changes, such as elevated blood pressure and blood lipids, altered blood glucose regulation and inflammation.[35] The following paragraphs describe some of the conventional cardiovascular risk factors and physiological pathways that lead to increased CVD risk.

Non-modifiable risk factors

Age and family history

The prevalence and duration of exposure to other risk factors accumulate with age and therefore, along with age-related cellular, structural and functional changes occurring in the heart and blood vessels, there is an incremental increase in CVD with age.[36] A family history of CVD is another major risk factor for CVD; if a first-degree relative has suffered a heart attack before the ages of 55 in males or 65 in females, a person is at a significantly greater risk of developing CVD.[37] This is likely due to inherited genetics that may influence lipid metabolism.[38]

Socioeconomic, environmental and behavioural risk factors

Socioeconomic status and environment

In general, people with lower SES have poorer health and this is also the case with CVD, where there are marked socioeconomic disparities[39] with cardiovascular scores (calculated from the number of risk factors) lower among higher socioeconomic groups.[40] Chronic stress, differences in lifestyle and behaviour, and poorer access to healthcare may be contributing factors in the high CVD risk experienced by low socioeconomic groups.[41] Risk associated behaviours are often more common in lower SES groups, for example, smoking is more common among disadvantaged groups and quitting attempts are less likely to be successful.[42]

Environmental factors such as extreme temperatures, the built environment, noise and pollution,[43] are emerging as relevant to CVD, and in this regard, the workplace may also play a critical role. Occupational risk factors may contribute to the physiological changes involved in CVD and/or influence behavioural factors, similar to the effects of socioeconomic and environmental factors.

Smoking, poor diet, alcohol consumption and physical inactivity

Smoking is a well-established risk factor for CVD. The mechanisms by which smoking (cigarette smoking in particular) may contribute to poor cardiovascular health include the following: damaging the endothelium, increasing fatty deposits in the arteries, raising cholesterol levels and promoting inflammation and clotting.[37, 44] Similarly, as well as being associated with weight gain, a poor diet is also associated with lipid and glucose dysregulation, high blood pressure, decreased plaque stability and inflammation.[45] Light alcohol consumption has been associated with a protective effect in CVD as it reduces LDL levels,[46, 47] however, heavier drinking (≥14 drinks per week in women and ≥21 drinks for men) is associated with adverse cardiovascular effects.[48] Excessive alcohol consumption has been linked to high blood pressure, increased lipid levels, promotion of inflammation and endothelial dysfunction.[49] Physical activity is important in the role of preventing CVD as exercise is associated with decreased pro-inflammatory cytokines, improved lipid metabolism and reduced prevalence of other cardiovascular risk factors.[22] Physical inactivity is linked to poor cardiovascular health not only through lack of the aforementioned functions but also through independent mechanisms that may involve reduced endothelial function and arterial stiffness.[50]

Physiological pathways

Metabolic Syndrome

Certain cardiovascular risk factors that lie on the physiological pathway to CVD often occur together and the term commonly used to refer to this is 'metabolic syndrome'. Most definitions of metabolic syndrome include some form of insulin resistance, a measure of obesity, high triglyceride levels, low high-density lipoprotein (HDL) and elevated blood pressure,[51] all of which are also independent risk factors for CVD.

High blood pressure (also referred to as hypertension)

High blood pressure is associated with all-cause and CVD mortality, particularly in combination with other risk factors.[52] Chronic and untreated elevated blood pressure results in end-organ damage, vascular damage and increased risk of an acute cardiac event.[53]

High cholesterol (also referred to as raised blood lipids, hyperlipidaemia or hypercholesterolemia)

High levels of LDL cholesterol are associated with CVD risk as oxidative damage of LDL is a key factor in the immune response of atherosclerosis.[54] High serum levels of triglycerides (a component of lipid levels) have been linked to atherosclerosis, also potentially promoting inflammation.[55] HDL plays a role in the removal of excess cholesterol from the bloodstream and has additional anti-inflammatory and endothelial functions, with low HDL cholesterol regarded as a risk factor for CVD.[56]

Diabetes mellitus (also referred to as raised blood glucose levels or insulin resistance)

Diabetes is the dysregulation of blood glucose and chronically high blood glucose is associated with vascular damage and chronic inflammation, which increase the risk of CVD.[57] Dyslipidaemia and endothelial dysfunction are also often common in diabetics and these factors contribute to the initiation and progression of CVD.[58]

Inflammation

Inflammation is a key component of most cardiovascular risk factors and underlies the initiation, progression and acceleration of heart disease, with inflammatory markers a biomarker of plaque progression and cardiac events.[59] Research is expanding to identify additional factors that may increase inflammation levels, such as chronic infections (e.g. human immunodeficiency virus[60] or periodontal disease[61]), environmental factors (e.g. chemical irritants and alcohol[62]) or the western diet.[63]

Obesity

Excess body fat increases the risk of other risk factors such as high cholesterol and high blood pressure, and is also independently associated with CVD as adipose tissue releases bioactive mediators that can lead to high blood glucose, alterations in lipids, high blood pressure, endothelial dysfunction and promote a pro-inflammatory state.[64] Obesity is likely due to a combination of behaviours, genetics and environmental factors[65] and is often quantified with BMI, waist-to-hip ratio (a measure of abdominal obesity) or body fat measurements.

2.1.3 Cardiovascular disease in males and females

Despite common misconceptions that CVD is a male disease due to the higher incidence rates, CVD is also a significant issue for females and is often underrecognised and undertreated.[66] In New Zealand, IHD is the leading cause of death in females and kills twice as many women than any other single cause.[67] Globally, the rates of CVD mortality have declined since the 1990s, however, the decline has not been as marked for women.[8]

It is hypothesised that oestrogen has a protective effect on cardiovascular health and this is the reason women have a lower risk of CVD before menopause than men.[68] However, after the reduction of oestrogen at menopause, the risk of CVD rises faster in women than men of the same age.[69] In terms of clinical manifestation, men are more likely to develop IHD as a first event, whereas, women are more likely to develop less common forms of CVD, leading to lower rates of recognition, diagnosis and less aggressive treatment in females.[29, 70, 71]

Due to biological differences, females have unique risk factors, such as pregnancy and hormone-related risk factors,[72] and the influence of some conventional risk factors on disease incidence and progression differs between males and females.[73] For example, lifestyle factors including diet and physical exercise may influence cardiovascular systems in a sex-specific manner due to differences in physiology (e.g. hormones).[74] The role of obesity in CVD may be different between sexes due to differences in fat distribution, which also leads to a high prevalence of cardiometabolic risk factors in women after menopause.[75] Evidence suggests that metabolic risk factors may be more detrimental in females, with a higher risk of fatal IHD in women with diabetes than their male counterparts.[76] The association between CVD and both smoking and SES has also been shown to be stronger in females.[77, 78] Although men generally have a greater prevalence of these risk factors, the differences in prevalence diminish with age.[8]

2.1.4 Cardiovascular disease and ethnicity

There are also significant differences in CVD and risk factors between ethnic groups.[79] In New Zealand, Māori are 1.56 times more likely to have IHD than non-Māori[32] and although fatal and non-fatal IHD rates are declining in all New Zealand populations, Māori and Pacific peoples have disproportionately high rates of IHD mortality.[9] IHD mortality is more than twice as high among Māori compared to non-Māori, and Māori are 1.3 times more likely to be hospitalised, with the disparity greatest for Māori females.[24] Furthermore, the prevalence of cardiovascular risk factors (such as smoking, obesity, diabetes and high cholesterol), is greater in Māori.[25, 80, 81] These ethnic differences in CVD prevalence are not solely due to differences in SES,[39] with access to and treatment within the health care systems of New Zealand significantly contributing to health inequities (see Section 2.2.3).[82]

2.2 Occupational risk factors for cardiovascular disease

2.2.1 The role of occupation in cardiovascular disease

According to the WHO, 58% of the world population will spend one-third of their adult life at work,[83] which can have profound effects on health and well-being. In New Zealand, it has been estimated that 19% of CVD mortality may be attributable to work in males and 9% in females, although these estimates are based on overseas data.[11] Furthermore, heart disease is the leading cause of occupational deaths and disability in New Zealand and worldwide.[16, 84] These statistics suggest that using workplace interventions to reduce occupational exposures associated with CVD may be of considerable benefit In New Zealand and internationally. As summarised by Novak et al. (2007),[85] the workplace is a particularly effective setting for heart health interventions as a significant amount of time is spent at work, the working population is large, people of working age can be reached before the disease develops and the ability to influence the workplace environment can be significant.

Previous research suggests physical, biomechanical, organisational and psychosocial occupational exposures may contribute to some of the social inequities in the incidence of cardiovascular risk factors (e.g. physical inactivity, obesity, diabetes, dyslipidemia and high blood pressure),[86] implying that reducing occupational exposures may be a novel way to reduce some cardiovascular inequities. However, before effective and targeted changes can be developed and implemented in the workplace, there must be strong evidence regarding specific high-risk occupations, the potential causal exposures and the underlying mechanisms.

2.2.2 Sex and gender in occupational risk factors for cardiovascular disease

Throughout the literature, the terms sex and gender are often used interchangeably although many studies only collect information on sex. This thesis uses both terms as defined by the WHO. 'Sex' refers to the biological and physiological characteristics that define humans as males or females and will be used in situations where biological differences are being discussed. 'Gender' refers to the socially constructed characteristics of men and women, such as roles, behaviours, activities and attributes; gender can vary between cultures and is not constant over time. Although few studies have assessed the role of gender, the term will be used when discussing the role of gender in the workplace.

In occupational health research, gender is an important factor as it is strongly associated with all components of occupation such as job title, income, exposure to occupational hazards and occupational status. For example, previous research has demonstrated differences in occupational exposures between genders within the same job.[27] Other differences may include discrimination (based on gender), harassment and having to use equipment designed for males. In addition, there are important physiological differences that may affect associations between occupational exposures and CVD. For this reason, analyses that focus on occupational factors and CVD risk are best conducted stratified by sex/gender.

2.2.3 Ethnicity in occupational risk factors for cardiovascular disease

Ethnicity is also a relevant, but often overlooked, factor in occupational health research as there are ethnic differences in occupational distribution[87] and occupational exposures within the same job title.[26] Furthermore, ethnic minorities may experience additional exposures such as racism or discrimination.[88] Racism is a significant underlying issue that contributes to the health and workplace inequities experienced by Māori in New Zealand. In particular, systemic racism affects the availability of socioeconomic opportunities and access to resources such as medical care, housing, income and wealth, and community support.[89] In the workplace, discrimination may occur between workers (e.g. bullying), at the management level resulting in differential treatment (e.g. less pay or fewer promotion opportunities), or at a policy level as health and safety guidelines are often not developed in a culturally sensitive manner. Systemic racism also affects the occupational distribution of Māori, with an overrepresentation of Māori in lower-skilled occupations.[87] Therefore, systemic racism may have significant effects on the association between occupation and CVD risk through a range of mechanisms, including differences in occupational exposures, medical treatment, exposure to unhealthy lifestyle factors (e.g. smoking and alcohol consumption) and stress.[89] Although analyses can account for some of these factors (such as smoking), it is possible that other factors may affect the association between occupational exposure and CVD risk. For this reason, analyses that focus on occupational factors and CVD risk are best conducted stratified by ethnicity.

2.3 Literature review methods

The remainder of the background section will present and analyse the available literature assessing a range of potential occupational risk factors for CVD. This literature was collected from the PubMed and ScienceDirect databases using the search terms presented in Table 2.2. Relevant studies were included if they were published in English (or an English translation was available) and other relevant literature was identified through the reference lists of selected articles.

Table 2.2. Literature search terms

Outcome or exposures	**Terms searched**
Outcome	'Cardiovascular'; 'cardiovascular disease'; 'heart disease'; 'ischaemic heart'; 'ischemic heart'.
Exposures	'Workplace'; 'occupation'; 'occupational group'. Job stress: 'job stress'; 'stress'; 'work stress'; 'effort-reward imbalance'; 'job strain'. Irregular hours: 'irregular hours'; 'long hours'; 'long working hours'; 'shift work'; 'shiftwork'; 'night shift'; 'nightshift'. Noise Physical activity: 'sedentary'; 'sitting'; 'standing'; 'physical demands'; 'physical exertion'; 'lifting'. Other exposures: 'chemicals'; 'pesticides'; 'lead'; 'fumes'; 'diesel'; 'dust'; 'particulate matter'.

2.4 Occupation and conventional cardiovascular risk factors

Assessing the prevalence of conventional cardiovascular risk factors across occupational groups provides insight into potential intermediates, and hence, the physiological pathways, that may link occupational risk factors or occupation-associated behaviours with CVD risk. Table 2.2 presents studies that have assessed the prevalence of CVD risk factors across occupational groups. The paragraphs following the tables discuss these studies, with many studies comparing white-collar workers (WCW) and blue-collar workers (BCW).

Table 2.3. Studies evaluating the distribution of cardiovascular risk factors across multiple occupations

Authors, year	Occupational groups	Risk factors	Study design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Pereira et al., 1998[90]	Professional/skilled; partly skilled; unskilled; and homemaker.	Insulin, glucose, triglycerides, HDL cholesterol, LDL cholesterol, blood pressure, BMI and waist-to-hip ratio.	Cross-sectional	In 1992 a questionnaire along with blood tests and physical measurements were taken.	Mauritius	2,795 (35-54 years old) 1,338 males 1,457 females	Results were stratified by sex.	Not included	In *unskilled* men, age-adjusted means were lower for insulin and glucose, cholesterol and triglycerides, and blood pressure, while HDL was significantly higher. Trends were not consistent across occupational statuses in females, but *homemakers* showed the poorest risk factor profiles.
Nakamura et al., 2000[91]	BCW and WCW.	Waist circumference, waist-to-hip ratio, total cholesterol, BMI, triglycerides, blood pressure, smoking and blood pressure.	Cross-sectional	Blood and physical measurements were taken at a 1993 annual check-up with a questionnaire completed.	Japan	1,145 653 males (mean age 32.6) 492 females (mean age 26.3)	Results were stratified by sex.	Not included	After adjusting for age, male BCW had a significantly higher waist-to-hip ratio and blood pressure than WCW. BCW were also more likely to smoke. In females, BCW had significantly higher BMIs and waist-to-hip ratios than WCW.
Rosvall et al., 2000[92]	High-level nonmanual; medium-level nonmanual; low-level nonmanual; skilled manual worker; and unskilled manual worker.	BMI, HDL cholesterol, LDL cholesterol, blood pressure, diabetes and smoking.	Cross-sectional	Baseline exams and lab tests were taken between 1991 and 1996.	Sweden	4,176 (46-68 years old) 1,713 males 2,463 females	Results were stratified by sex.	Not included	In general, the prevalence of BMI, high LDL, low HDL, high blood pressure and current smoking increased with decreasing occupational status. These trends were statistically significant in both males and females.

Authors, year	Occupational groups	Risk factors	Study design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Gallo, 2003[93]	Clerical; BCW; WCW; and not employed.	BMI, blood pressure, LDL cholesterol, HDL cholesterol, triglycerides, smoking and fasting glucose.	Cross-sectional	Questionnaires, blood tests and physical measurements were completed between 1983 and 1985.	U.S.A.	362 (42-50 years old)	Only females	Not included	BCW and *clerical workers* had significantly higher BMIs compared to WCW.
Engstrom et al., 2006[94]	High-level nonmanual; medium-level nonmanual; low-level nonmanual; skilled manual worker; unskilled manual worker; and other.	Inflammation-sensitive proteins	Cross-sectional	Physical examinations and blood tests were completed between 1974 and 1984, and occupation was obtained through linkage to the National Census.	Sweden	6,065 (28-61 years old)	Only men	Not included	There were higher concentrations of inflammatory proteins found in manual workers
Ainy and Azizi, 2007[95]	Working women and housewives.	BMI, waist circumference, waist-to-hip ratio, blood pressure, triglycerides and total cholesterol.	Cross-sectional	Questionnaires, blood tests and physical measurements were collected from 1991 and 2001.	Iran	566 working women and 561 age-matched housewives (mean age 35.0)	Only females	Not included	Mean BMI, waist circumference, waist-to-hip ratio, blood pressure, triglycerides and total cholesterol were all significantly higher in *housewives* than *working women*. Only 9% of *working women* had 4-6 risk factors, compared to 17% of *housewives*.

Authors, year	Occupational groups	Risk factors	Study design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Sanchez Chaparro et al., 2011[96]	Agriculture; construction; industry; and service. Blue and white-collar occupational groups.	Cardiovascular risk SCORE (based on presence of diabetes, total cholesterol, LDL and blood pressure).	Cross-sectional	Routine health check-ups (including a questionnaire, physical examination and laboratory tests) from 2004 and 2004.	Spain	309,955 workers (16- 74 years) 77.6% males 27.4% females	Results were stratified by and adjusted for sex.	Not included	The highest prevalence of high cardiovascular risk (i.e. a high number of risk factors), was found in the *agricultural* sector and *industry*, for both sexes. After adjustment for age, sex and area of residence, *agriculture* and *construction* were significantly associated with increased risk of high cardiovascular risk SCOREs. Age-adjusted prevalence showed an increased prevalence of high cardiovascular risk SCOREs in blue-collar occupations overall.
Allman-Farinelli et al., 2010[97]	Managers & administrators; professionals; tradespersons; advanced clerical & service workers; intermediate clerical & sales & services workers; intermediate production & transport; elementary clerical sales & service workers; and labourers.	BMI	Cross-sectional	All information was collected in the Australian National Health survey in 2005.	Australia	14,618 (20-64 years old) 7,466 males 7,152 females	Results were stratified by sex.	Not included	In males, *associate professionals* and *intermediate production and transport* occupations had a higher risk of being overweight than those unemployed. In females, *professionals*, and those in *management* and *advanced clerical and service* occupations had a lower risk of being overweight, than those unemployed. Adjustment for SES and lifestyle factors attenuated these results.

Authors, year	Occupational groups	Risk factors	Study design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Zimmermann et al., 2010[98]	Directors; professionals; technician & associate professionals; administrators; restaurant & personal services workers; security services; business assistants; agriculture & fishery workers; construction; extraction, metal & machinery workers; graphic design, textile & food; operators & assemblers of machinery; drivers & operators of moving machinery; unqualified service workers; labourers; armed forces; students; home duties; and retirees.	Diet, weight, sedentary work, alcohol and tobacco consumption and high blood pressure.	Cross-sectional	Surveys from 8 consecutive years (2000-2007) were aggregated and analysed.	Spain	106,048 (18 to 64 years old) 7,817 males and 8,231 females	Results were presented separately for each sex.	Not included	Sedentary work and tobacco consumption were the most common risk factors found. *Drivers*, *administrative secretaries* and *directors* had the highest risk of CVD risk factors.

Authors, year	Occupational groups	Risk factors	Study design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Ham et al., 2011[99]	BCW; WCW; and service workers.	Smoking status	Cross-sectional	Data were analysed from the Current Population Survey – Tobacco Use Supplement surveys from 1992 through to 2007.	U.S.A.	106,604 (18-64 years old) 54% male 46% female.	Adjusted for sex	Adjusted for ethnicity	BCW were at a higher risk than WCW for ever-smoking, current smoking and persistent smoking. Of the blue-collar groups, *construction workers* were the most likely to be current smokers.
Davis-Lameloise et al., 2013[100]	Agricultural workers; technicians; managers; and "home duties".	Triglycerides, LDL cholesterol, HDL cholesterol, total cholesterol, blood pressure, fasting plasma glucose, BMI, weight, smoking and waist circumference.	Cross-sectional	In 2004-2006 three cross-sectional surveys were undertaken and included a self-administrated questionnaire, physical measurement and laboratory checks.	Australia	1,001 (25-74 years old) 293 agricultural workers, 123 technicians, 420 managers, and 165 home duties.	Results were presented separately for each sex.	Not included	In the males, *agricultural workers* had lower fasting levels of glucose, higher levels of occupational physical activity, lower alcohol consumption, a healthy diet and the lowest proportion of smokers, compared to the other groups. In females, *managers* had higher HDL cholesterol, lower blood pressure, lower waist circumference and lower self-reported diabetes, compared to the other groups.

Authors, year	Occupational groups	Risk factors	Study design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Lewin et al., 2014[101]	Manufacturing industry; construction; commercial repair of motor vehicles & motorcycles; hotels & restaurants; transport & communications; financial activities; real estate, renting & business services; public administration; education; health & social work; and collective, social, and personal services.	Fat mass index (FMI) and percentage of fat mass.	Cross-sectional	From 2007-2008, data for height and weight were collected at medical examinations, and a Tanita foot-to-foot bioelectrical impedance analyser was used to measure FMI and the percentage of fat mass.	France	6,094 (aged 30-79 years old) Males and females included.	Results were presented separately for each sex.	Not included	In females, there was no significant difference in body fat in any work sector compared to *transport and communications*. In males, those in *construction* had a higher percentage of fat mass than those in *education*.
Strauss et al., 2016[102]	Firefighters and office workers.	Metabolic syndrome: abnormal HDL cholesterol, high blood pressure, waist circumference, BMI, triglycerides and HbA1c.	Cross-sectional	Questionnaires, blood tests and physical measurements were collected at an examination. No date provided.	Germany	97 firefighters (mean age 40.5) 46 office workers (mean age 45.8)	Only males	Not included	Waist circumference was significantly greater in *office workers* compared to *firefighters*, and abnormal metabolic risk factors were more prevalent (although not significant when adjusted for age).

Authors, year	Occupational groups	Risk factors	Study design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Shockey et al., 2016[103]	All occupational groups	Cardiovascular health metrics (CHM): 1) not smoking, 2) being physically active, 3) having normal blood pressure, 4) having normal blood glucose, 5) being of normal weight, 6) having normal cholesterol, 7) eating a healthy diet.	Cross-sectional	Data from a 2013 random-digit-dialled survey was analysed.	U.S.A.	66,609 (≥18 years old) 30,604 males 36,005 females	Adjusted for sex	Adjusted for ethnicity	*Transportation and material moving* employees had the highest adjusted prevalence of 'not ideal' scores for physical activity, blood pressure and BMI. Both *transportation* and *community and social services* employees had the highest prevalence of 2 or fewer CHMs. Adjusted prevalence ratios also showed these two groups were more likely to meet two or fewer CHM compared to the other groups. *Farming, forestry, and fishing*; *production;* and *arts, design and entertainment, sports, and media* employees had the lowest adjusted prevalence of two or fewer CHM compared to the other groups
Nam et al., 2016[104]	Office work; sales and service; agriculture, forestry & fishery; machine fitting & simple labour; and unemployed, housewife, or student.	Metabolic syndrome: increased triglyceride level, decreased HDL cholesterol, raised blood pressure and increased fasting plasma glucose or type 2 diabetes.	Cross-sectional	Data was used from the second year of the sixth Korean National Health and Nutrition Examination Survey, 2014.	Korea	4,303 (≥19 years old) 307 males 276 females	Results were not adjusted or separated by sex.	Not included	*Office workers* had a two-fold increase compared to *agriculture, forestry, and fishery workers*.

Authors, year	Occupational groups	Risk factors	Study design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Akintunde and Oloyede, 2017[105]	Auto technicians and schoolteachers.	Metabolic syndrome: obesity, triglycerides, low HDL cholesterol, high blood pressure and high blood glucose.	Cross-sectional	Surveys, blood tests and physical measurements were conducted from January 2016 till August 2016.	Nigeria	166	Age and sex-matched samples.	Not included	*Auto technicians* had a greater risk of being diagnosed with metabolic syndrome than *schoolteachers*. Impaired blood glucose, hypertriglyceridemia and low HDL were more prevalent in the *auto technicians*. However, high LDL was more prevalent in *schoolteachers*.
Lee and Kim, 2017[106]	Firefighters (compared to other workers).	Metabolic syndrome: central obesity, elevated blood pressure, hyperglycemia, high triglycerides and low HDL cholesterol.	Cross-sectional	Data was used from routine check-ups of firefighters in 2013 and other workers in the Korea National Health and Nutrition Examination Survey.	Korea	257 firefighters and 1,064 other workers (30-59 years old)	Only males	Not included	*Firefighters* had a similar prevalence of metabolic syndrome factors compared to general workers.
Aginsky et al., 2017[107]	BCW: mining, manufacturing and construction. WCW: I.T., banking and retail.	High blood pressure, high cholesterol and obesity.	Cross-sectional	Data was used from six companies' health screening where health measurements were taken. No date provided.	South Africa	603 (mean age 38.2 years old) 308 BCW 295 WCW 66% males 34% females	Results were stratified by sex	Not included	BCW were significantly older, had higher blood pressure and were more likely to develop hypertension, but less likely to have high cholesterol. WCW had higher cholesterol and stress levels.

Authors, year	Occupational groups	Risk factors	Study design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Macdonald et al., 2017[13]	Management; professional; service; sales, office and administrative support; national resources, construction, maintenance; and production, transportation, material moving. 21 more specific occupational groups were also included.	Cardiovascular health (CVH): blood glucose, total cholesterol, blood pressure, BMI, diet quality, physical activity, and smoking. Each risk factor was given a score. The total score of risk factors was calculated for each participant, giving a CVH score of either "optimal", "moderate" or "inadequate".	Cross-sectional	Study participants completed a clinical exam and self-administered questionnaire between 2011 and 2013.	U.S.A.	6,282 black and white Americans, (≥45 years old) 51.9% males 48.1% females	Adjusted for sex	Adjusted for ethnicity	*Managers* and *architects and engineers* had the highest prevalence of optimal CVH, while *sales, office and administrative support* had the lowest.
Prihartono et al., 2018[108]	BCW; WCW; and other.	BMI, high blood pressure and diabetes.	Cross-sectional	Data from the 2007 Indonesian National Health Survey of self-reported cardiovascular risk factors was analysed.	Indonesia	137,378 (40-69 years old) 88,670 males 48,709 females	Does not specify what is adjusted for in the adjusted models.	Not included	Both WCW and BCW were associated with BMI, diabetes, and high blood pressure, with BCW having the highest prevalence ratios for diabetes and BMI.

Authors, year	Occupational groups	Risk factors	Study design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Kim et al., 2018[109]	WCW; BCW; pink collar; and agribusiness, fisheries & low-level labours.	Metabolic syndrome (≥3): abdominal waist obesity, high triglyceride concentration, low HDL cholesterol, high blood pressure or diabetes.	Cross-sectional	Data based on longest held occupation was collected from the Korea National Health and Nutrition Examination Survey 2010-2011.	Korea	679 (≥65 years old)	Only males	Not included	Mean body fat mass was lower in *agribusiness and low-level workers* than *white-collar*, *blue-collar* and *pink-collar*. The risk of metabolic syndrome was higher in *white-collar* and *blue-collar* than *agribusiness and low-level workers*.
Mehrdad et al., 2018[110]	Office work; physical exposure; and chemical exposure.	Metabolic syndrome: waist circumference, diabetes, low HDL cholesterol, hypertriglyceridemia and high blood pressure.	Cross-sectional	Surveys and physical measurements were taken between October 2015 and September 2016.	Iran	3,949 (23-73 years old)	Only males	Not included	The prevalence of metabolic syndrome did not significantly differ between 'job ranks'. There was no adjustment.
Kelsall et al., 2018[111]	Managers; professionals; community & personal service; clerical & administrative; sales workers; technicians & trades; machinery & operators & drives; and labourers. 19 industries.	CVD risk and diabetes risk calculated from age, gender, blood pressure, cholesterol, smoking, blood glucose, family history, smoking, diet, exercise and waist circumference.	Cross-sectional	Participants in the WorkHealth program between 2009 and 2012 completed a self-administrated questionnaire and health check.	Australia	499,965 (≥24 years old) 257,200 males 242,765 females	Results were presented separately for each sex.	Adjusted for indigenous status	In both males and females, the proportion with high diabetes risk was higher in BCW and non-manager WCW, compared to *managers*. The risk was lowest for *professionals*. In males, CVD risk was increased in all groups, except *professionals*, compared to *managers*. In females, *professionals* also had a lower proportion of CVD risk, but *labourers* were the only group in which high CVD risk was greater.

Authors, year	Occupational groups	Risk factors	Study design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Shen et al., 2018[112]	BCW and WCW.	High blood pressure	Cross-sectional	Participants completed a questionnaire and had blood pressure measurements taken between 2009 and 2012.	China	37,856 (aged 18-60 years old) 24,648 males 13,208 females	Results were not adjusted or stratified results by sex.	Not included	The prevalence of hypertension was highest in BCW and lowest in WCW.
Vandersmissen et al., 2020[113]	Health care; government; education; accommodation & food service; distributive trade; manufacturing; services; construction; transport & storage; and other.	Smoking, physical activity, BMI and high blood pressure.	Cross-sectional	Interview and measurements were taken by a nurse or physician in 2018.	Belgium	212,792 (15-69 years old) 55.1% males 44.9% females	Adjusted for sex in some analyses and stratified in others.	Not included	High BMI and high blood pressure were most prevalent in the *transport and storage sector*. Poor health for smoking and physical activity were most prevalent in *construction*. Whereas, the *education sector* had the lowest prevalence of smoking, high BMI, and high blood pressure. The highest cardiovascular risk (multiple risk factors) was observed for *transport and storage and construction*, with the lowest in the *education sector* again. Over time, smoking and physical activity has decreased across all economic sectors, whereas obesity and high blood pressure have increased.

2.4.1 Blue- and white-collar occupations

Direct comparisons of BCW and WCW show that BCW are more likely to have high blood pressure, diabetes, smoke, be overweight and have metabolic syndrome.[91, 93, 96, 99, 107, 111, 112] In a study looking only at BCW, it was estimated that 73% of the workers had at least one CVD risk factor and about 30% had more than three risk factors.[114] Elser et al. (2018) summarised the literature relating to the health (including CVD and cardiovascular risk factors) of women working in blue-collar occupations published from 1990 to 2015 and concluded that female BCW had poorer health compared to other women or male BCW with increased risk for high blood pressure, but mixed findings for obesity.[14]

A 2017 South African cross-sectional study, looking at risk factors in blue- and white-collar occupations using routine health screening information of 603 participants, showed that BCW were 1.72 times more likely to have high blood pressure (Odds ratio (OR) 1.72 (95% CI 1.05-2.81)) but less likely to have high cholesterol (OR 0.50 (0.31-0.81)).[107] Another study in Japan found that the mean waist-to-hip ratio was significantly greater in BCW compared to WCW for males and females, and blood pressure was greater for male BCW compared to WCW.[91] Neither of these studies included ethnicity in their analyses, but they did adjust for age and stratify by sex.

Other studies have compared specific blue-collar and white-collar occupations such as data analysed from the Korean National Health and Examination Survey, which found a two-times greater risk of metabolic syndrome for office workers compared to agricultural and fishery workers (OR 2.01 (1.26-3.22).[104] Although this study was based on 4,303 participants and included both males and females, results were not adjusted (or stratified) for sex or ethnicity. In Nigeria, a cross-sectional study found the prevalence of metabolic syndrome to be greater in auto-technicians compared to age- and sex-matched schoolteachers (8.4% vs. 1.2%, $p < 0.05$).[105]

Transportation is a specific blue-collar occupation that has consistently been associated with a high prevalence of cardiovascular risk factors.[97, 103, 113] Intermediate production and transport work has been associated with increased risk of obesity in males (OR 1.24 (1.03-1.50), although adjusting for socioeconomic factors and health behaviours (e.g. physical activity and diet) attenuated these findings.[97] A large random-digit-dial survey of 66,609 American men and women identified the transportation and material moving sector as having an increased risk of having at least 6 risk factors (Prevalence Ratio (PR) 1.55 (1.25-1.92)) (e.g. smoking, inadequate physical activity, high blood pressure, high blood glucose, high cholesterol, high BMI or poor diet).[103] Results were adjusted for age, sex, ethnicity and education. Taxi drivers have also been investigated with results of one study showing that 99.2% of taxi drivers had at least one cardiovascular risk factor and working more than 10 years as a taxi driver was significantly associated with a higher prevalence of cardiovascular risk factors.[115]

There are other blue-collar occupations consistently associated with a high prevalence of cardiovascular risk factors. For example, the construction sector had the highest prevalence of high cardiovascular risk (defined as having at least three of the following risk factors: smoking, high BMI, physical inactivity and high blood pressure) in a study of 212,792 males and female participants.[113] Differences remained statistically significant after adjusting for age and sex. Other research has highlighted that among BCW, construction workers are most likely to smoke[99] and have a higher fat mass compared to workers in the education sector.[101] A study of Spanish workers found a high cardiovascular risk score (based on the number of risk factors present) in both construction and agriculture work for males and females.[96] In contrast to construction work, results are mixed for agricultural work, with some studies demonstrating a low prevalence of risk factors.[100, 103, 104]

Findings for white-collar occupations are less consistent. In general, WCW seem less likely to smoke or have hypertension.[99, 112] In contrast, WCW has been associated with high cholesterol and being overweight. [105, 107] Generally, “office work” or “non-manager” white-collar occupations are associated with cardiovascular risk factors. Office workers in a South Korean study had a higher risk of metabolic syndrome compared to agriculture, forestry and fishery workers (OR 2.01 (1.26-3.22))[104] and metabolic syndrome was also significantly more prevalent in office workers compared to firefighters in a study of 143 males in Germany (33% vs. 14%, $p<0.05$).[102] A study of 6,282 participants in America found that service and low-status office workers (sales, office and administrative support) had the lowest prevalence of ideal cardiovascular health metrics (based on scores for blood glucose, cholesterol, blood pressure, diet, physical activity and smoking). [13] These analyses were adjusted for sex and ethnicity.

2.4.2 Methodological differences

Some of the variation in results may be due to differences in occupational group classifications; for example, Mehrdad et al. (2018) did not find significant associations using broad occupation classifications with wide-ranging exposures (e.g. office work, physical exposure and chemical exposure).[110] Some studies use blue- and white-collar categories, while others included a pink-collar category (working in a care-oriented field) or look at a much broader range of groups. These differences make it hard to generalise the evidence as they are assessing different occupational classifications. Nonetheless, this is often unavoidable as separating broad occupational groups into smaller classifications is often not feasible unless studies have very large sample sizes.

The reference group also varies across studies, with some studies using a specific occupational group as the reference, while others compare results for one occupational group to all participants else who do not work in that occupation. When using a specific occupation as a reference group, it is important to ensure it has enough participants and as occupation distribution significantly differs between males and females, it may be necessary to use different reference occupations for each sex. Again, depending on the sample size of the study, this may not always be possible.

Measurement of cardiovascular risk factors is another source of variation. The majority of studies utilised physiological measurements for health conditions such as diabetes, high blood pressure, high cholesterol and obesity, meaning there is minimal self-report bias. However, others relied on self-reports and Kramer et al. (2012) demonstrated the potential misclassification of self-reports as they found high cholesterol in 27% of participants who self-reported normal cholesterol.[116] Another common method was using routinely collected health data from annual health check-ups or national health surveys, which may be a more reliable measure for metabolic factors than self-reports.

2.4.3 Sex

There is some evidence of sex differences in the prevalence of cardiovascular risk factors in occupational groups. A large Australian study found that compared to managers, non-manager white-collar occupations (e.g. clerical or administrative, community and personal service workers, and sales workers), were more likely to have multiple risk factors (high blood pressure, smoking, diabetes and high cholesterol ratio), however, this was only significant for males.[111] Another study found that male associate professionals were more likely to be overweight (OR 1.34 (1.10-1.63)), while female professionals (OR 0.71 (0.61-0.82)), managers (0.72 (0.56-0.92)) and clerical workers (OR 0.73 (0.58-0.93)) were generally less likely to be overweight.[97] In another study, only female managers had lower blood pressure, higher HDL cholesterol, lower waist circumference and less diabetes than agriculture and fishery workers, technicians or home duties.[100]

2.4.4 Ethnicity

To date, there have been limited comparisons of ethnicity in research evaluating the prevalence of cardiovascular risk factors across occupations or associations between occupation and CVD.

2.5 Occupations and industries associated with cardiovascular disease

Research assessing associations between occupation and CVD dates back to the 1950s when Morris (1953) identified bus drivers as having high CVD risk.[12] Over the years this knowledge has expanded, with an increasing number of studies identifying other occupations that are potentially linked to CVD. Table 2.4 presents studies that assessed cardiovascular risk across a range of occupations/occupational groups or industries. The paragraphs following the table discuss these studies in more detail.

Table 2.4. Studies evaluating CVD risk in different occupational groups

Authors, Year.	Occupations	Type of CVD	Study Design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Haynes and Feinleib, 1980[117]	Housewives; BCW; WCW; and clerical & sales workers.	IHD	Longitudinal	A sample of participants from the Framingham cohort study was interviewed between 1965 and 1967 and followed for 8 years to identify a diagnosis of IHD.	U.S.A.	1317 (aged 45-64 years old) 350 housewives 387 working women 580 men	Results were presented separately for each sex.	Not included	In males, WCW had the highest incidence of IHD. Whereas, in females, *clerical and sales workers* had the highest incidence.
Pearce and Howard, 1985[16]	All occupations	IHD	Age-standardised mortality rates	Age-standardised male mortality based on death registered during 1974-78 and occupation recorded at time of death.	New Zealand	N/A	Males (15-64 years old)	Not included	IHD was the leading cause of death and the occupations identified as having a particularly high risk were *hairdressers and beauticians*; *spinners, weavers and dyers*; *shoemakers and leather goods*; and l*abourers*.
Leigh and Miller, 1998[84]	All occupations	CVD	Cross-sectional	Aggregation and analysis of job-related disease and occupation data from the Bureau of Labor Statistics' Supplementary Data System for 1985-1986.	U.S.A.	N/A	Did not adjust or stratify for sex.	Not included	Heart conditions were the leading cause of occupational deaths and disability. The occupations reporting more heart-related deaths than others were *heavy truck drivers, managers and administrators* and *sales supervisors and proprietors*.

Authors, Year.	Occupations	Type of CVD	Study Design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Aronson et al., 1999[118]	All occupations	IHD	Longitudinal	An occupational surveillance system of the Canadian labour force (established between 1965 and 1971) was linked to mortality records from 1965 to 1991.	Canada	699,440 457,224 males 242,196 females	Results were presented separately for each sex.	Not included	Among males, *taxi drivers and chauffeurs* were identified as having high risk. *Inspectors and foremen* were among the occupations identified as high risk for females.
Calvert et al., 1999[119]	Blue- and white-collar occupations.	IHD	Cross-sectional	Data from the National Occupational Mortality Surveillance system for 1983 to 1992 was used to calculate proportionate mortality ratios (PMR).	U.S.A.	113,396 'White' males 13,955 'Black' males (16-60 years old)	Only males.	Results were presented separately for ethnicity.	In the blue-collar occupations, the highest PMRs were seen in *sheriffs, correctional institution officers, policemen, firefighters* and *machine operators*. In white-collar occupations, *physicians* ('Blacks' only) and *clergy* (both races) were among the occupations with the highest PMR.
Rosvall et al., 2000[92]	High-level nonmanual; medium-level nonmanual; low-level nonmanual; skilled manual worker; and unskilled manual worker.	Carotid intima-media thickness and stenosis.	Cross-sectional	Occupational information was collected between 1991 and 1996, and carotid intima-media thickness was assessed with an ultrasound.	Sweden	4,176 (46-68 years old) 1,713 males 2,463 females	Results were stratified by sex.	Not included	In women, *manual occupations* had increased odds of carotid stenosis compared to women in *high or medium-level non-manual occupations*. The same was not observed in males.

Authors, Year.	Occupations	Type of CVD	Study Design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Muntaner et al., 2001[120]	Professional and non-professional workers in the production, finance and government industries.	CVD mortality	Longitudinal	Population surveys from 1978 to 1985 were linked to the National Death Index to identify CVD mortality for the period 1979 to 1989.	U.S.A.	375,974 (25-65 years old) 212,518 males 163,456 females	Results were stratified by sex.	Adjusted for ethnicity.	In men, the lowest CVD mortality was seen for *professionals* in the finance industry and highest among *non-professional* workers in the production industry. In contrast, in females, *professionals* in the finance sector had the highest CVD mortality.
Malinauskiene et al., 2002[121]	Legislators, senior officials & managers; professionals; technicians & associate professionals; clerks; service & shop & market sales workers; agricultural & fishery workers; craft & related trades workers; plant & machine operators & assemblers; elementary occupations; and armed forces.	Myocardial infarction	Case-control	Participants with non-fatal MI were recruited at Kaunas hospitals and interviewed for occupational information between 1997 to 2000.	Kaunas	448 cases and 1,777 controls (25-64 years old)	Only men	Not included	Compared to *trades workers*, an increased risk of MI was observed in *legislators, senior officials and managers*, *professionals* and *machine operators and assemblers.*

Authors, Year.	Occupations	Type of CVD	Study Design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Gallo, 2003[93]	Clerical; BCW; WCW; and not employed.	Carotid intima-media thickness	Longitudinal	Occupational information was collected via questionnaire between 1983 and 1985. Carotid artery ultrasounds were undertaken 11 years later.	U.S.A.	362 (42-50 years old)	Only females	Not included	*Clerical workers* had a greater carotid intima-media thickness than all of the other groups.
Finkelstein et al., 2004[122]	Construction unions: heavy equipment operators; boilermakers; electricians; insulators; painters; plumbers; and sheet metal.	IHD	Retrospective	Information from construction unions was linked to mortality data in 2000.	Canada	4,104 deaths	Does not specify	Not included	The proportional mortality ratios were highest for *heavy equipment operators* (although not statistically significant).
Engstrom et al. 2006[94]	High-level nonmanual; medium-level nonmanual; low-level nonmanual; skilled manual worker; unskilled manual worker; and other.	Coronary events	Longitudinal	From baseline examinations between 1974 and 1984, men were followed up got 18 years until death or self-reported coronary events.	Sweden	6,065 (aged from 28-61 years old)	Only men	Not included	The incidence of coronary events was much higher in low occupation levels.

Authors, Year.	Occupations	Type of CVD	Study Design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Holmes et al., 2011[17]	All occupations	IHD and other CVDs	Cross-sectional	Occupation was taken from the free text field of mortality records for all male deaths from 2001-2005.	New Zealand	12,713 deaths	Only males	Not included	*Plant machine operators and assemblers* had the highest IHD mortality rates.
Luckhaupt et al., 2014[123]	WCW; Service; and farm & BCW. 19 industries	IHD or stroke	Cross-sectional	Participants were interviewed as a part of the National Health Interview Survey for the period 2008-2012).	U.S.A.	91,331 (aged 18-55 years old) 41,522 males 49,870 females	Adjusted for sex	Did not adjust for ethnicity	*Service* and BCW were more likely to have IHD or stroke compared to white-collar workers. The two industries with a significantly higher prevalence of IHD/stroke were *administrative and support and waste management and remediation services* and *accommodation and food service*.
Fujishiro et al., 2015[124]	Management; professional; service; sales/office & administrative support; and blue-collar occupations.	Carotid artery intima-media thickness and carotid plaque score and shadowing	Longitudinal	Starting from 2000-2002, carotid arteries were assessed at an examination every two years with a mean follow-up of 9.4 years.	U.S.A.	3,109 (45-84 years old) 1,499 males 1,610 females	Results were stratified by sex.	Adjusted for ethnicity	*Professional* men had the lowest plaque scores, men in *management*, *sales/office and administrative support*, *service* and *blue-collar jobs* had higher carotid plaque scores. Physically hazardous jobs were associated with increased plaque scores in females. Associations were only significant at baseline and not in the longitudinal analyses.

Authors, Year.	Occupations	Type of CVD	Study Design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Wada et al., 2016[125]	Service; administrative & managerial; agriculture; construction & mining; transport & machine operation; professional & engineering; manufacturing process; carrying, cleaning, packaging; security; clerk; and sales.	IHD	Cross-sectional	Mortality rates for each occupational group were estimated from data from the Japanese Ministry of Health, Labour and Welfare dataset in 2010.	Japan	23,349,301 (25-59 years old).	Only males	Not included	The highest IHD mortality was seen in *administrative and managerial* and *mining* occupational groups. Compared to *sales*, age-adjusted analyses showed that *service*, *administrative and managerial*, *construction and mining*, *transport and machine operation* and *professional and engineering*, were more at risk for IHD.

Authors, Year.	Occupations	Type of CVD	Study Design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Macdonald et al., 2017[13]	Management, professional; service; sales, office, administrative support; national resources, construction, maintenance; and production, transportation, material moving. 21 more specific occupational groups were also included.	IHD or stroke	Cross-sectional	Cohort study participants completed a clinical exam and self-administered questionnaire between 2011 and 2013.	U.S.A.	6,282 black and white Americans, ≥45 years old 51.9% males 48.1% females	Adjusted for sex	Adjusted for ethnicity	Although not significant, the *service* occupational group *and sales, office and administrative support* had the greatest prevalence ratio for IHD/stroke.
Prihartono et al., 2018[108]	BCW; WCW; and other.	CVD	Cross-sectional	The 2007 Indonesian Nation Health survey was analysed.	Indonesia	137,378 (40-69 years old) 88,670 males 48,709 females	Does not specify what is adjusted for in the adjusted models.	Not included	White-collar and ‘other’ workers were more likely to have been diagnosed with CVD than blue-collar workers.

Authors, Year.	Occupations	Type of CVD	Study Design	Data collection	Country	Subjects	Sex	Ethnicity	Main findings
Zaitsu et al., 2019[126]	Blue-collar, service, professional, and manager within blue-collar, service and white-collar sectors.	IHD	Case-control	Study subjects were patients admitted to the hospital between 1984 and 2016. Each case had 10 controls matched based on sex, 5-year age category, and admission date and hospital. Analyses were based on the longest-held occupation.	Japan	1,128,591 (128,615 CVD cases and 999,976 controls, ≥20 years old) 20.3% of females in controls and 23.8% of females in cases	Some results were presented separately for sex, while others adjusted for sex.	Not included	Compared to BCW in the blue-collar sectors, *managers* and *professionals* in blue-collar and service sectors had elevated risk for IHD.
Kim et al., 2019[127]	White-collar; blue-collar work; and service & sales workers.	CVD	Cross-sectional	Data was used from the 2014 Korea Working Conditions Survey was analysed.	Korea	27,662 (aged 15 years old and above). 50.5% males 49.5% females	Results were not adjusted or stratified by sex.	Not included	Occupation was significantly associated with CVD, with BCW having the highest incidence of CVD.

2.5.1 Blue- and white-collar occupations

An early 1996 review by Tuchsen et al. collating data from four longitudinal and two cross-sectional studies of European men, highlighted occupations such as driving, barbers and hairdressing, warehouse and wholesale staff, laboratory assistants, and radio and telegraph operators as associated with IHD.[128] An updated review from Steenland et al. included the Tushsen review and other literature up to 2000, and summarised occupations associated with CVD.[15] An Australian report produced in 2006 again provided an overview of these occupations but did not expand upon this list.[10] Table 2.5 presents an updated list of occupations based on the original table from Steenland et al. (2000)[15] and summarises the occupations that have been associated with CVD in the literature presented in Table 2.4, which are predominantly male blue-collar occupations as they have been the focus of most studies.

Table 2.5. Occupations that have been associated with cardiovascular disease

Males	Females
Administrative and managerial[125]	Bus drivers[15]
Air traffic controllers[15]	Cleaners[15]
Bakers[15]	Clerical work[117]
Beauticians[16]	Home help[15]
Bus drivers[15]	Inspectors and foremen[118]
Cannery workers[15]	Rubber and plastics workers[15]
Clergy[119]	Sales[117]
Cooks[15]	Self-employed in hotel and catering[15]
Firefighters[15]	Taxi drivers[15]
Fisherman[15]	Unskilled workers in tube, sheet and steel construction[15]
Foundry workers[15]	Waitresses[15]
Hairdressers[15]	
Heavy equipment operators[122]	
Laboratory assistants[128]	
Labourers[16]	
Lorry drivers[15]	
Machine operators[119]	
Mining[125]	
Other uniformed men[128]	
Paper industry workers[15]	
Physicians[119]	
Plant and machine operators and assemblers[17]	
Police officers[15]	
Prison wardens[15]	
Radio and telegraph operators[128]	
Rubber and plastics workers[15]	
Sheriffs[119]	
Ship's deck officers and sea pilots[15]	
Spinners, weavers and dyers[16]	
Shoemakers and leather goods[16]	
Storekeepers[15]	
Taxi drivers[15]	
Waiters[15]	
Warehousemen and storekeepers[15]	

As presented in Table 2.4, studies using the broad classification of BCW and WCW are limited and show mixed results. A higher prevalence of IHD or stroke has been observed in BCW compared to WCW (PR 1.40 (1.19-1.65), adjusted for age and sex) in a large National Health Interview survey in the U.S.A using self-reports of CVD history.[123] Similar results have also been found using the Korea Working Conditions Survey where BCW had the highest crude prevalence of CVD compared to WCW (2.5% vs. 0.8%).[127] A recent systematic review on BCW and women's health reported a consistent increase in CVD risk for women in blue-collar occupations, compared to white-collar work.[14] However, a large cross-sectional study in Indonesia contradicts these findings, concluding that WCW were 1.6 times more likely to be diagnosed with CVD than BCW.[108]

There is a wide range of occupations within blue and white-collar classifications and variability in definitions of these broad groups. An alternative way of classifying the difference between blue- and white-collar workers is using a measure of manual activity, which is generally greater in blue-collar occupations. In the few studies that have used manual activity to classify occupation, the results were more consistent with increased CVD risk in lower-skilled and more manual occupations, particularly for females.[92, 94, 124] An 18-year follow-up study of 6,075 men found that unskilled manual workers had the greatest risk of coronary events over the follow-up period (age-adjusted relative risk (RR) 2.26 (1.6-3.2)).[94] Cross-sectional analyses have linked physically hazardous jobs to subclinical measures of CVD in women, such as carotid stenosis and high coronary calcium plaque scores.[92, 124]

Blue-collar occupations have also been identified in two New Zealand studies assessing associations between occupation and IHD. The initial study from 1985 identified spinners, weavers and dyers, shoemakers and leather goods workers, and labourers as associated with IHD.[16] The other study, published in 2011, identified plant and machine operators and assemblers as high risk for IHD.[17] Both studies were based on the cause of death and occupation recorded at death, and did not account for ethnic differences or adjust for other risk factors such as smoking.

Other blue-collar occupational groups that have been consistently associated with CVD internationally include construction and mining, transport, and machine operation.[125] Specific blue-collar occupations in the literature include drivers,[84, 118] machine operators,[17, 119, 121, 125] and uniformed men (e.g. police or sheriffs).[119] In a 1999 longitudinal study, the Canadian labour surveillance systems were linked to mortality records, and taxi drivers and chauffeurs were identified as having high CVD risk among males, and inspectors and foremen among females.[118]

Despite many occupations previously associated with CVD being blue-collar, there are still some key findings for WCW. Results for the generalised white-collar occupation classification are inconsistent, with some studies showing a decreased risk overall for WCW,[123] while others have shown a greater CVD risk in WCW compared to BCW.[108, 117] This is potentially due to the heterogeneity of these large groups with narrower classifications providing more consistent results; for example, clerical work, in particular, is more consistently associated with increased CVD incidence and carotid intima thickness.[93, 117]

Higher status office jobs such as legislators and managers also show consistent results. A case-control study of males with myocardial infarction (MI) in Kaunas, Lithuania showed that legislators, senior officials and managers, and professionals were more likely to have MI.[121] In another study, the RR of IHD for men working in the administrative and managerial occupational group compared to sales, was estimated to be 2.68 (2.01-2.59). In contrast, less consistent results are observed professionals, another high status white-collar occupational group, which have shown no association with subclinical CVD compared to other occupations[124] but lower CVD mortality risk rates compared to non-professionals.[120]

Studies of service work, despite the ambiguity in definitions, have consistently demonstrated an association with CVD, such as a large cross-sectional study from America, analysing data from males and females of the National Health Survey.[123] Results based on 91,331 participants and adjusted for age, showed that service and BCW were more likely to have IHD or stroke compared to WCW. A similar cross-sectional study of 6,282 Americans telephone interviews, showed that the service occupational group had an elevated prevalence of IHD and stroke.[13]

2.5.2 Methodological differences

Research into associations between occupation or industry and CVD includes a mixture of longitudinal, cross-sectional, case-control and retrospective cohort studies, with the majority being cross-sectional. All study designs provide useful findings; however, longitudinal studies may be more informative for assessment of CVD risk over time as it is a slowly progressing disease with a potential lag time between occupational exposure and clinical manifestation.

Measurement of CVD is variable across studies with some focusing on IHD or subclinical CVD, while others use a more general umbrella CVD term encompassing all coronary events. Identification of the CVD endpoint differs across studies with some using health or death records, while others rely on self-reports of diagnosis. Self-report may introduce recall bias and the use of death records restricts the endpoint to severe CVD resulting in death, whereas CVD can be present and progressing (with symptoms such as angina) before death.

Classification of occupation is often inconsistent across studies both in broad and smaller occupational groupings. Furthermore, most studies have only looked at current or longest-held occupation, with studies lacking information on participant's complete job history, including the duration of all occupations ever worked, which may be due to restrictions of what data was available.

Many studies have not adjusted for SES, which is closely associated with both occupation and cardiovascular risk. Occupational status has long been considered a marker for social gradient, [129] therefore, to determine whether the observed effect in CVD risk is attributable to the occupation exposure itself, controlling for SES would generally be necessary.

2.5.3 Sex

The majority of studies have been conducted among males and this may explain why the number of occupations associated with CVD is larger in males. In studies including both males and females, there are limited analyses that present separate results, though, in the few that do, results are often inconsistent between males and females. For example, the highest incidence of IHD was observed for male WCW, whereas among females, clerical work was associated with the highest IHD incidence, in a 1980 study by Haynes and Feinleib (1980).[117] In a study by Muntaner et al. (2001), men and women working as professionals in the finance sector showed opposing results, with males having the lowest CVD mortality and females having the highest.[120] There is a particular lack of research of females within blue-collar occupations, likely due to the smaller number of female workers in these areas; however, results consistently show increased risks among females in manual occupations.[92, 124]

2.5.4 Ethnicity

There are limited studies comparing associations between occupation and CVD in different ethnic groups. In one of the few studies that have compared associations between ethnic groups, blue-collar occupations such as uniformed men had high IHD mortality rates for both

'Blacks' and 'Whites', while white-collar occupations such as physicians were associated with high IHD mortality for 'Blacks' only.[119]

2.6 Occupational exposures associated with cardiovascular disease

Reviews by Kristensen (1989) and Hwang and Hong (2011), have evaluated the following occupational exposures associated with CVD: job stress, irregular hours, noise, occupational physical activity and chemicals.[18-20] The following paragraphs summarise the latest research and current consensus of evidence for these risk factors, as well as highlighting the hypothesised mechanisms by which they may contribute to CVD.

2.6.1 Job stress

Job stress is one of the most researched occupational exposures concerning CVD risk, with the job strain model (also known as the demand-control model; Figure 2.2) being most widely applied. This model proposes that job strain does not result from a single aspect of the work environment but instead is a consequence of the joint effects of work demands and the range of decision-making freedom available to the worker facing those demands. As outlined by Karasek in 1979, there are four types of jobs based on job demand and control: low control and low demands = passive job; high control and high demands = active job; high control and low job demands = low strain job; low control and high job demands = high strain job.[130]

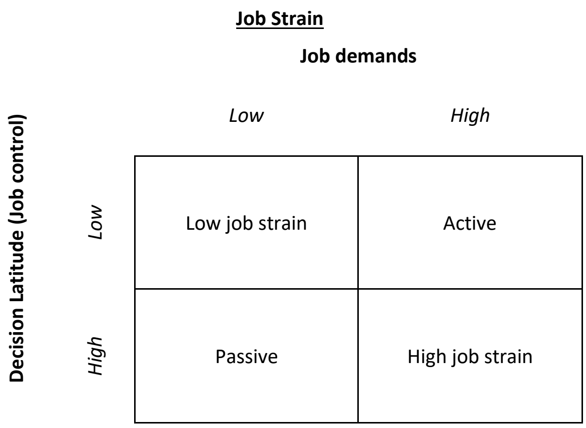

Figure 2.1. Job strain model of job stress developed by Karasek (1979)[130]

The second most commonly used model of job stress is the effort-reward imbalance (ERI) model. In this model, developed by Siegrist in 1996, situations of high cost (e.g. demand or obligations) and low gain (e.g. money, esteem or status) are likely to elicit feelings of anger, depression and being threatened, and may be more likely in occupations with a low level of occupational control such as blue-collar occupations.[131]

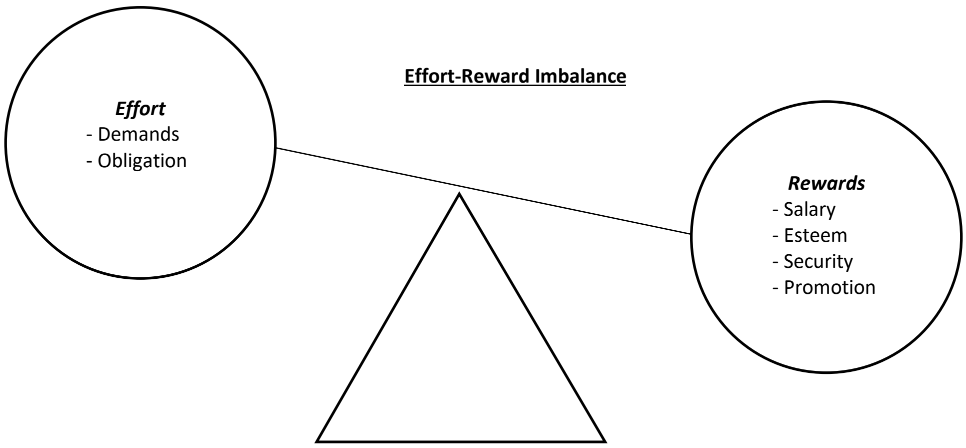

Figure 2.2. **Effort-reward imbalance model of job stress developed by Siegrist (1996)[131]**

In the 2018 literature review by Sara et al., the authors summarised associations with CVD across 23 prospective studies using both job stress models.[21] For the 12 studies with significant findings, RRs ranged from 1.22 (1.01-1.46) to 4.53 (1.43-14.3) for IHD incidence, and between 1.53 (1.08-1.97) to 1.92 (1.51-3.21) for CVD events and mortality; however, 11 studies showed no association.[21] In another meta-analytic review that included both stress models, the age and sex-adjusted RR for high strain compared to low strain was 1.43 (1.15-1.84), and in the ERI model it was 1.58 (0.84-2.97), suggesting job stress is associated with an approximately 50% increase risk of IHD.[132]

Kivimaki and Kawachi (2015) focused on the job strain model and reviewed evidence from over 600,000 men and women.[133] They found a robust association across age groups and different SES backgrounds of general working populations, estimating that the excess risk of CVD for those exposed to job stress is 10-40%. A similar review of 26 prospective studies estimated a hazard ratio (HR) for IHD for those with job strain compared to those without to be 1.3 (1.2-1.5).[134] Other reviews have identified that it is specifically the low control component of the model that is associated with an increase in the risk of all-cause mortality and IHD compared to workers with high job control.[135]

A study among 416 male BCW who were followed up for 6.5 years found that lack of control, such as job insecurity and work pressure, were positively associated with IHD.[136] Other cohort studies, such as the Whitehall II study of civil servants, which used questionnaires to obtain information about job stress and health outcomes, found that men and women with low job control had a higher risk of incident IHD when followed up for an average of 5.3 years (OR 1.93 (1.34-2.77)).[137] Kuper et al. (2003) studied 6,895 males and 3,413 females of the same cohort for an average of 11 years and found that people with low decision latitude (i.e. low job control) and high demands were at the greatest risk of IHD (HR 1.55 (1.24-1.94); adjusted for age, sex and SES).[138] A dose-response was observed when assessing job strain at different phases of follow-up in the Whitehall II study, with more job strain (i.e. job strain reported at multiple times) associated with a greater risk of IHD (One report HR 1.23 (0.90-1.68); two reports HR 1.33 (1.04-1.69).[139] Another large cohort study, the Framingham Heart Study, assessed the effects of job strain on 10-year IHD incidence in 3,039 participants. The results of this study were not consistent with other research and the only significant finding was that women with both high control and high job demands, but not job strain, had a greater risk.[140]

Other work has focused on the ERI model; a multi-cohort study linking 11 prospective cohorts assessed ERI using a questionnaire and monitored incident IHD through national hospital records and self-reports. This study showed that ERI was associated with IHD (HR 1.19 (1.04-1.38)), when adjusted for age, sex and SES.[141] ERI has also been shown to be associated with atherosclerosis as assessed by angiogram in a case-control study,[142] and high blood pressure and intima-media thickness as reported in a systematic review.[143]

Sex

The majority of research has combined males and females and has not stratified by sex; evidence of female-specific associations is therefore rare and inconsistent. In a large study of 22,086 women followed up for 10 years, it was reported that compared to low strain jobs, those with high strain jobs had a greater CVD risk (RR 1.63 (1.28-2.08)) when adjusted for age and ethnicity.[144] In contrast, a large study in nurses that followed 35,038 females for 4 years did not find an association between job strain and IHD.[145]

In the studies that have stratified by sex, it is not clear if associations between job stress and CVD are consistent between males and females. For example, one systematic review did find consistent associations between males and females,[146] while another systematic review of prospective studies found that most significant results were observed in studies considering only men and results for females were less clear.[147]

Differences between males and females in the effect of job stress on CVD are plausible as females often have additional family and household responsibilities, which may affect their perception and/or effect of work stress.[148] Padkapayeva et al. (2018) looked at life and work stress, and showed that the influence of work exposures (e.g. co-worker support, job insecurity, etc.) on life and work stress were different between males and females, with higher levels of support associated with less stress in females; the same was not observed in males.[149] Furthermore, there are potentially physiological differences in the response to work stress between males and females, as females have shown greater changes in heart rate compared to males in response to occupational stress,[150] and have distinctly different hormone profiles.

Ethnicity

There is a lack of studies that have characterised the relationship between job stress and CVD for different ethnic groups, however, there are suggestions that some null results may be due to analyses not accounting for racially diverse groups.[148] Similar to the differences that may exist in the perception of stress between males and females due to other life stresses, cultural differences between ethnicities or differences in the level and cumulative effect of stress by ethnicity due to additional factors such as discrimination, may affect the association between job stress and CVD.

Potential physiological pathways

The mechanisms linking general stress to CVD have been reasonably well researched. The human stress response is a complicated process that aims to maintain homeostasis under stressful conditions by altering immune function, metabolism and the cardiovascular system, to ensure survival.[151] The autonomic nervous system and hypothalamus-pituitary-adrenal axis control the stress response and this is mediated by cortisol. Long-term dysfunction of these two systems can lead to chronically increased cortisol, which may increase blood pressure, elevate cholesterol levels and increase the risk of diabetes and obesity.[152, 153] Evidence seems to support these mechanisms, as job stress has been specifically linked to high blood pressure and type 2 diabetes and lipid disorders using both the job strain and ERI models.[154-156] Stress is also associated with pro-inflammatory responses.[153] Additionally, stress may increase poor health-associated behaviours, such as low physical activity and poor diet,[139] indirectly increasing the prevalence of other risk factors.

2.6.2 Irregular hours

Many jobs require employees to work irregular hours; non-standard hours include working long hours, shift work and night work, all of which may disrupt circadian rhythm, reduce sleep and have been related to CVD.[157]

Long Hours

The definition of long hours has varied significantly across studies, ranging from >38 hours to 60+ hours per week. In 2015 a comprehensive meta-analysis of longitudinal studies, including data from unpublished cohorts and based on 603,838 individuals (from 25 studies), showed an increased risk of incident IHD for those working ≥55 hours per week compared to 35-40 hours (RR 1.13 (1.02-1.26)).[158] In a 2018 meta-analytic update of the 2015 meta-analysis, Virtanen and Kivimaki included the Danish Labour Force Survey and reported a RR of 1.12 (1.03-1.21) for IHD.[159] Case-control and cross-sectional studies have been included in other reviews, with similar results e.g. OR 1.37 (1.11-1.70)[160] for CVD and RR 1.80 (1.42-2.29) for IHD.[161] However, a recent review, published in 2020, concluded that there was only weak evidence of an association between long working hours and CVD due to the large heterogeneity across studies.[162]

Recent studies have linked survey or census information to health records, with many observing no significant associations. In a study by Hannerz et al. (2018), a labour force survey was linked to national registers with data on SES, industry, emigrations, prescriptions, hospital contacts and death, and participants were followed up for an average of 7.7 years. This study showed no statistically significant associations between long hours and IHD.[163] Linking the Italian Census to mortality records for a follow-up period of 5 years also did not provide evidence of an association.[164]

An early case-control study conducted in Asia found a U-shaped relationship with both short and long working hours associated with MI in men (≤7 hours OR 3.07 (1.77-5.32); ≥11 hours OR 2.44 (1.26-4.73)).[165] Similar male case-control studies have been conducted since, with a two-fold increase in the risk of acute MI for those working ≥61 hours compared to 40 hours,[166] and in another study, working more than 60 hours was associated with increased IHD risk compared to 40-48 hours (OR 2.4 (1.5-4.0)).[167]

Sex

The majority of research has been conducted in males, however, within the limited literature focused on females, stronger associations between IHD and long working hours were observed. In a longitudinal study from South Korea, stratification by sex showed a much greater risk of CVD for females compared to males when working ≥50 hours (females HR 2.348 (1.180-4.672); males HR 1.122 (0.797-1.578)).[168] A similar trend was observed in a 5-year follow-up study in Italy, where men working ≥55 hours per week had an HR for IHD of 0.95 (0.86-1.05) and women showed an excess risk (HR 1.28 (0.88-1.87)), although results were not statistically significant.[164]

Ethnicity

Although some studies have included an adjustment for ethnicity, there is a lack of ethnic-specific results for the association between working long hours and CVD. However, there has been some evidence of ethnic differences in mediating factors, such as short sleep duration, which has been shown to be generally increased with increasing professional responsibility in 'Blacks', whereas the opposite relationship was observed for 'Whites'.[169]

Potential physiological pathways

Short sleep duration is a possible mediating factor linking long working hours to CVD, with both short sleep duration and lack of sleep associated with acute MI.[170] Physiological changes such as endothelial dysfunction, changes in lipid and glucose metabolism, sympathetic nervous system activation and immune impairment have all been linked to short sleep duration.[171] Separate studies have also linked short sleep duration to glucose impairment,[171] high blood pressure[172] and increased inflammation.[173] For long working hours specifically, the evidence is inconsistent; however, some studies have linked long working hours to increased waist circumference, reduced HDL and high blood pressure.[174, 175] Working long hours has been linked to diabetes, but only in individuals with low SES,[176] and metabolic syndrome.[177]

Additionally, working long hours also reduces the time available for other activities (e.g. recreational physical activity or socialising) and can lead to increased psychosocial stresses and behavioural changes.[159] For example, a New Zealand study of nurses and midwives showed an association between working longer hours and increasing alcohol consumption.[178]

Shift Work/Night Work

Shift work is a term that encompasses shifts of work outside of the standard hours: night work, evening work and early morning shifts, and rotating or alternating between these and day shifts. All of these shift work definitions have the potential to disrupt the circadian rhythm. A 1986 study showed an elevated IHD risk for shift work (RR 2.2 ($p<0.04$)) when comparing those with 11-15 years of exposure to those with no exposure, independent of age and smoking.[179] Systematic reviews have summarised findings since this early research and have estimated that shift work (all forms) increases CVD risk by about 40%.[180] Updates to this estimate have included a meta-analysis of over 2 million workers from 34 studies, which found an association between shift work and coronary events (including MI and ischaemic stroke), with a risk ratio of 1.24 (1.10-1.39) for all shift work and a risk ratio of 1.4 (1.13-1.76) for nightshift only.[181] When analyses were restricted to prospective cohort studies the risk increased for coronary events (RR 1.32, 95% CI 1.07-1.63). In a 2018 review, 21 studies were analysed and results estimated that the risk of a CVD event was 17% higher among shift workers compared to day workers (95%CI 1.09-1.25) and the risk of IHD morbidity was 26% higher (95%CI 1.10-1.43).[182]

Recent research further suggests that night shift presents the greatest risk for IHD (RR 1.44 (1.10-1.89).[183]

Female nurses have often been the focus of studies as they are commonly exposed to some form of shift work. One prospective study of 79,109 women, which defined night shift as working at least 3 nights a week in addition to days and evenings, found an increased risk (RR 1.51, 1.12-2.03) of IHD among women who had worked night shift for at least 6 years compared to women who had never worked night shifts.[184] The same cohort of nurses was also followed up for 22 years and results demonstrated a statistically significant, but small, elevation in IHD risk with each increase in the duration of exposure to shift work (i.e. <5 years; 5-9 years; and ≥10 years).[185] Death records of female nurses have also shown that compared to day shifts, nurses working night shifts had an increased risk of CVD mortality (HR 1.71 (1.09-2.69)).[186]

The use of prospective studies has allowed the assessment of dose-response relationships. In a meta-analysis of 5 prospective studies, 5 years of shift work exposure was associated with a 5% increase in CVD risk.[187] In another review previously referred to, it was found that after the first 5 years of shift work, there was a 7.1% increase in the risk of CVD event for every additional 5 years of exposure.[182] A 2019 meta-analysis of prospective, retrospective and case-control studies, found a dose-response relationship indicating a 0.9% increase in IHD risk with each 1-year increase in exposure to shift work.[183] A study looking specifically at pulp and manufacturing workers found a longer duration of exposure to shift work was associated with an increased IHD risk and workers with more than 30 years of shift work exposure had the greatest risk.[188]

Compared to other occupational exposures, shift work is one of the most studied hazards; however, the lack of clear definitions for shift work (including variations such as the number of consecutive nights worked, length of shift and rotation shift pattern) in the literature means results are not always consistent. For example, a 2010 longitudinal study that followed 20,142 adults for 22 years, found no significant association between shift work, night time work or daytime work and IHD mortality.[189] A large systematic review of available systematic reviews with meta-analyses concluded there was weak evidence linking shift work to IHD.[162] Another systematic review reported RRs for the association between shift work and IHD ranging from 0.6-1.4, with considerable heterogeneity in the outcome or exposure information and confounder or mediator control.[190]

Sex

Females have been studied in the context of shift work more than other occupational exposures and this is primarily because nursing is both a female-dominated profession and an occupation with a high proportion of shift work. Research has shown that circadian mechanisms influence cardiovascular disease in a sexually dimorphic manner,[191] highlighting the importance of not only including both sexes but also analysing the data for males and females separately.

Ethnicity

To date, there is a lack of both adjustment and stratification of ethnicity in the literature evaluating shift work and CVD. However, there has been some evidence of differences in the effect of shift work on other biological systems. For example, less of a reduction in melatonin (a regulator of circadian rhythms) was observed for Asian women working night shift compared to ‘Whites’.[192] Shift work has also been associated with high blood pressure in ‘Blacks’ (OR 1.35 (1.06-1.72) but not among whites (OR 1.01 (0.85-1.20)).[193]

Potential physiological pathways

The function of the circadian rhythm is to optimise the brain, nervous system, heart and vasculature. Disruption of the circadian rhythm can impair glucose control, lead to poor recovery from work, cause changes in lipids and activate the autonomic nervous system (stress response).[157, 194] Inflammation is a key component of the stress response to shift work and has been linked to night shift work in Japanese workers[195] and increased immune cell counts have also been demonstrated in shift workers.[196] Metabolic changes such as increased insulin and cortisol disruptions have been found in laboratory analyses of circadian misalignment[197] and one study identified cortisol as a mediator between shift work and CVD risk.[198] Evidence also suggests an increased risk of high blood pressure,[199, 200] obesity[201-203] and diabetes[204] in shift workers. Various reviews have highlighted the link between shift work and these risk factors, but the results have not been consistent across all of them, with differences observed between males and females.[205-208]

Disruptions of these circadian rhythms may change an individual's eating pattern and diet, social interactions, increase stress and lead to other behavioural changes such as increased smoking.[180, 209] Additionally, similar to working long hours, shift workers are likely to suffer the consequences of short sleep duration previously outlined.[210] Previous work has also demonstrated that people working non-standard hours are more likely to be exposed to workplace hazards and multiple hazards,[211] suggesting workers may have increased exposure to other high cardiovascular risk exposures compared to standard hours workers. For populations such as Māori, who disproportionately experience occupational exposures,[26] this may further affect CVD risk.

2.6.3 Noise

Occupational noise exposure has been studied since the 1950s and many systematic reviews and meta-analyses have evaluated associations with CVD. One systematic review concluded there was some evidence to suggest an increased risk of IHD for those exposed to >75-80 decibels (dB) for <20 years (based on five studies); self-reported exposure was also associated with an increased risk of IHD.[212] A more recent systematic review that focused solely on longitudinal studies of high quality showed a 34% increased risk (RR 1.34, 1.15-1.56) of CVD.[213] This review concluded that although there was evidence of a link between occupational noise exposure and CVD, the association was strongest for high blood pressure (HR 1.68 (1.10-2.57)). A 2021 systematic review of occupational noise and IHD concluded that there was limited evidence for a causal role for noise in CVD risk.[214]

Recent large prospective studies have used job exposure matrices (JEMs) to estimate noise exposure based on job title and assess the risk of CVD. For example, one study of 5,723 men who were born between 1915 and 1925 and followed until the age of 75 years or until hospital discharge (of IHD/stroke), found that both medium and high noise exposure showed an increased risk of IHD when compared to low noise (HR 1.19, 1.03-1.38 and 1.27, 0.99-1.63, respectively).[215] SES was not adjusted for in these analyses. A similar cohort study found that medium and high noise exposure was associated with increased risk of MI even when adjusting for age, BMI and smoking habits.[216] In both of these longitudinal studies the interaction of additional exposures, such as job strain and cold temperature, were included and results demonstrated an additive effect of multiple exposures.

Long-term noise exposure has been associated with CVD; in a cross-sectional study, previous exposure to loud noise for 1.6-18.8 years was associated with IHD (OR 2.11, 1.25-3.55), whereas results were not significant for exposure of 1.5 years and less.[217] This study adjusted for age, sex, ethnicity and other cardiovascular risk factors. Other studies have found an increase in IHD risk for participants working more than 19 years and another with 18 years of exposure.[218, 219] A separate study identified being exposed for 1-5 years was associated with an increased risk (HR 1.6 (1.1-2.3)) of CVD mortality compared to those never who had never been exposed to workplace noise.[220] However, results are not consistent, with a 16-year follow-up study of men showing no association between ≥5 years of occupational noise exposure and IHD mortality (HR 0.97 (0.71-1.33)). The magnitude of the dose-response relationship is not clear and may also depend on the level of exposure, the amount of time exposed in a day, as well as the years exposed.

Sex

The majority of work has focused on males and in the limited studies that have included both sexes, results were not analysed separately for males and females.

Ethnicity

There are no studies available that have analysed the effect of occupational noise on CVD in different ethnic groups, although a few studies have adjusted for ethnicity.

Potential physiological pathways

The primary explanation for occupational noise affecting the cardiovascular system is that it acts as a stressor, causing annoyance and initiating a stress response within the body, which is related to many downstream physiological processes.[221] Activation of the sympathetic nervous system causes a release of corticosteroids and consequences include an elevation of blood pressure, which has more consistently been linked to occupational noise exposure than any other cardiovascular condition.[222, 223] The release of corticosteroids can lead to endothelial dysfunction, oxidative stress and metabolic abnormalities.[23, 221] However, studies have failed to find evidence of elevated cholesterol in noise-exposed workers,[224, 225] with some exceptions.[226] Another study found that noise exposure during the day affected sleep and hypothesised these disruptions were likely due to elevated cortisol and altered autonomic nervous system activity.[227] Additionally, DNA damage and obesity have also been associated with noise exposure.[228, 229]

2.6.4 Physical activity

Pioneering research into occupational physical activity (OPA) and CVD dates back to the 1950s when J. N. Morris studied CVD incidence in the drivers and conductors of London's double-decker buses and found that sedentary drivers had higher rates of CVD than the physically active conductors.[12] Since then, there has been more research into the association between OPA and cardiovascular health, with inconsistent results, indicating a complex relationship. The following paragraphs will discuss two ends of the OPA spectrum: sedentary work and physical exertion. Evidence suggests a U-shape relationship, with both ends of the spectrum associated with negative cardiovascular outcomes. For example, in a study focused on lifting, both participants with no heavy lifting and more than 40 hours of heavy lifting were more likely to have cardiac events.[230] In another study, a population-based survey of 37,300 men and women was linked to death registries to assess the effect of OPA and metabolic syndrome on CVD mortality; compared to persons lifting/walking at work without metabolic syndrome, those with metabolic syndrome and sedentary work (HR 2.74 (1.82-4.12)) or heavy physical work (HR 3.02 (1.93-4.75)) had increased risk of CVD mortality.[231] These findings suggest the optimal level of OPA may be somewhere in between sedentary behaviour and physical exertion.

Sedentary work

Over the past few decades, there has been a reduction in the physical demands of occupations, with less than 20% of current jobs requiring at least moderate intensity compared to the 1960s where almost half involved moderate physical activity.[232] The consequences of this dramatic increase in sedentary work in many workplaces are only beginning to be understood.

Most of the research into the adverse health effects of sedentary time (commonly characterised as sitting/stationary behaviour with low energy expenditure as opposed to physical inactivity which is defined as a lack of moderate physical activity[233]) have looked at sedentary or sitting behaviour in general, rather than specifically focusing on occupational exposure. Although there is substantial heterogeneity across studies, there has been some evidence of a relationship between sedentary behaviour (not work-related) and CVD.[22, 234] A 2015 systematic review and meta-analysis showed an increased risk of CVD mortality and incidence (HR 1.13 (1.05-1.21) and 1.14 (1.00-1.73), respectively) for those exposed to sedentary behaviour.[235] Another systematic review and meta-analysis of sedentary time estimated a RR of 2.47 (1.44-2.24) for CVD events and 1.90 (1.36-2.66) for CVD mortality.[236] A meta-analysis restricted to prospective studies showed similar results with increased risk of CVD (HR 1.14 (1.09-1.19)) for the highest level of sedentary behaviour (12.5h/day) compared to the lowest (2.5h/day).[237] In a large study of 71,363 men and women, total sitting time was assessed by questionnaire and CVD identified by national registers; results showed more than 10 hours of daily sitting compared to less than 6 hours was associated with MI risk (HR 1.38

(1.01-1.88)) and IHD risk (HR 1.07 (1.01-1.40)).[238] Results in these analyses were adjusted for other cardiovascular risk factors including physical activity in leisure time.

There are limited reviews specifically looking at occupational exposure to sedentary behaviour, with those that exist suggesting only weak evidence of a relationship with health risks (e.g. CVD, cancer, diabetes and all-cause mortality).[239] A pooled analysis of seven British cohorts found that sitting occupations were associated with all-cause mortality but not CVD mortality when followed up for 12.9 years.[240] Nevertheless, workplace interventions targeting sedentary behaviour have shown improvement in cardiovascular risk factors.[241] Increased physical activity among workers is associated with more favourable risk factor profiles compared to sedentary occupations.[242] However, results are not consistent across all studies. Moller et al. (2016) found no difference in IHD risk in sedentary and non-sedentary workers for males or females when analysing self-reported sitting time information from the Danish Work Environment Cohort study linked it to national registers.[243] Other studies have demonstrated that standing is the important component of sedentary behaviours, with a Canadian study concluding that standing occupations were associated with a 2-times increased risk of CVD compared to predominantly sitting occupations using healthcare records over a 12 year follow-up period (HR 2.32 (1.16-4.62)).[244]

An important consideration in the relationship between OPA and CVD is the role of leisure time physical (LTPA) activity. In most of the studies, analyses have adjusted for LTPA to determine whether the relationship between OPA and cardiovascular health is independent of recreational physical activity. However, as evidence suggests that LTPA may be an effect modifier that alters the effect of OPA on CVD risk, adjusting for LTPA may be an over adjustment. In a study by Saidj et al. (2013), both leisure and occupational sitting were associated with elevated CVD risk but the joint effect of both had the greatest effect.[245] In a prospective study it was shown that LTPA was associated with decreased CVD mortality, but the benefit was greatest for those with low or moderate activity at work.[246] A large meta-analysis also demonstrated that high levels of physical activity attenuated the high risk of high total sitting time, for both all-cause mortality and CVD mortality.[247] Therefore, it is potentially incorrect to consider them independent of each other and other methods, such as stratification by the level of LTPA or mediation analyses, may be more appropriate.

Sex

In contrast to some of the other occupational exposures, most of the research in this area already includes both sexes, yet few studies have presented results separately for males and females. However, some analyses have demonstrated statistically significant interactions between sex and sedentary behaviour for cardiovascular risk factors such as high blood pressure, HDL cholesterol and triglycerides.[248]

Ethnicity

Differences in the relationship between sedentary time and waist circumference have been observed between ethnicities[248] and research has demonstrated a modifying role of ethnicity in the association between both physical activity and sedentary behaviour, and diabetes.[249] This suggests there may be differences in the association between occupational sedentary behaviour and CVD for different ethnic groups, however, there is no literature investigating this.

Potential physiological pathways

Sedentary behaviour has been linked to metabolic syndrome,[104, 250] diabetes,[236] high blood pressure[22] and weight gain, particularly for women.[251] In addition to the increased prevalence of conventional cardiovascular risk factors, emerging evidence suggests sedentary behaviour has a direct effect on the cardiovascular system, altering mitochondria, cardiac muscle and artery function.[50] The body rapidly maladapts to inactivity, altering the vasculature and function of arteries, and the downregulated blood flow due to lack of movement potentially leads to endothelial dysfunction and vascular deconditioning.[252, 253]

Physical exertion

The link between physical activity and cardiovascular health has been studied for many years, with results consistently showing that increased physical activity reduces CVD morbidity and mortality[254] as it improves weight management, blood pressure, lipid disorders and inflammation.[22, 255, 256] However, although lack of physical activity is detrimental to our health, it does not mean that all physical activity is beneficial. Increasingly, evidence suggests that high OPA (defined as physical exertion or hard/high physical workload) is associated with adverse health effects, thus having different effects from LTPA. A recent systematic review with meta-analysis concluded that men, but not women, engaging in high OPA compared to low OPA had an increased risk for all-cause mortality (HR 1.18 (1.05-1.34)).[257] Moderate and high levels of LTPA have been associated with a reduced risk of CVD, while moderate and high levels of OPA slightly increased the risk of CVD in a 2013 meta-analysis.[258]

In 2007, Krause et al. showed that high energy expenditure at work, based on the energy requirements of specific activities, was associated with accelerated atherosclerosis progression.[259] Cohort studies have also demonstrated associations using self-reported OPA, such as a nursing study that followed 12,093 nurses for 15 years.[260] In this study, nurses with a higher physical workload had an approximately 40% higher risk of IHD, which remained after adjustment for other CVD risk factors, such as age, LTPA and lifestyle factors. In another study, which linked two cohorts with census information, death and migration records, males from one cohort with moderate physical activity had a greater risk of CVD mortality than those with low physical activity.[261] However, results were not consistent across cohorts, not significant in women and stronger for all-cause mortality. In another cohort study, moderate-to-hard OPA was significantly associated with all-cause mortality and an association was also observed for CVD mortality (HR 1.35 (0.94-1.95)), but this was not statistically significant.[262] In general, the evidence appears to be more consistent for overall health, rather than CVD specifically.

Similar to sedentary behaviour, a major issue when looking at high OPA seems to be the modifying effects of LTPA and physical fitness levels. In a 30-year follow-up study of men, in addition to an association between increasing levels of OPA and increased IHD mortality, the combination of low or moderate fitness and high OPA further increased IHD risk.[263] A similar study looking at both males and females showed that those with high OPA had an increased risk of IHD mortality (HR 1.61 (1.19-2.19)) when adjusted for age and sex; notably, a combination of high OPA and low self-reported cardiorespiratory fitness was associated with a considerably greater risk (HR 10.31 (4.50-23.60)).[264] Likewise, and similar to sedentary occupational behaviour, LTPA also appears to be a modifying factor as findings have shown that although high OPA is a risk factor for IHD at all levels of LTPA, the risk was greatest for those with both high OPA and sedentary LTPA (age-adjusted HR 3.30 (1.87-5.82)).[260] In contrast, the lowest risk was observed in nurses with moderate OPA and vigorous LTPA. Considering fitness and LTPA as effect modifiers may be necessary for fully understanding the consequences of high OPA on cardiovascular health.

Sex

The majority of research in this area has been conducted in males and in the few studies that also include females it has been suggested that associations are strongest for males as there is limited evidence of an association for females.[257, 260, 262] Due to physical differences in the size and strength between men and women, it is likely that the threshold of "high" OPA needs to be different for sexes and this has not been accounted for in previous studies. Furthermore, OPA and LTPA activities have been shown to significantly differ between males and females.[265]

Ethnicity

To date, there has been no ethnic stratification of analyses and limited studies have adjusted for ethnicity. As some studies have reported different levels of exposure to physical factors for different ethnic groups e.g. Māori males reported higher exposure to physical exposures compared to non-Māori in the same jobs,[26] ethnic-specific analyses are necessary.

Potential physiological pathways

The mechanisms through which physical exertion may contribute to cardiovascular health have been summarised by Holtermann et al. (2018), who emphasise that physical activity at work is very different from recreational physical activity.[266] Firstly, OPA is performed at a lower and prolonged intensity rather than the short bursts of LTPA, meaning it does not confer the cardiovascular fitness benefits of LTPA. OPA also elevates heart rate and blood pressure, both of which are risk factors for CVD if prolonged. Finally, without the sufficient recovery time of LTPA, OPA increases inflammation levels rather than having the anti-inflammatory effects of recreational physical activity. Oxidative stress and inflammation have been demonstrated in heavy workers (workers with high physical load).[267]

2.6.5 Other exposures

Some additional occupational exposures have been linked to CVD, but the evidence for these is limited and not as thoroughly investigated as the exposures described above. For this reason, these exposures will only be covered briefly.

Chemicals

The evidence linking specific occupational chemical exposure to CVD is limited and inconsistent. The clearest evidence is for carbon disulphide and nitroglycerin (both are now rare exposures), and carbon monoxide.[268] Other chemicals include benzene, metals (e.g. lead), pesticides, diesel exhaust, dioxins, solvents and combustion products.[269-275] Combustion products, such as the organic chemicals from diesel exhaust, have been shown to affect inflammation levels and endothelial cells.[276] Carbon monoxide potentially blocks the oxygen-transporting capacity of the blood by converting haemoglobin to carboxyhaemoglobin and may increase the risk of an acute IHD event and lower the threshold for angina.[10, 272] Research suggests there may be a dose-response relationship between these chemical exposures and IHD or CVD.[271] The following paragraphs summarise two of the more well-documented chemicals: pesticides and lead.

Pesticides

Pesticides are substances used to protect plants from pests, diseases or weeds, and are typically used in the agriculture sector; chronic exposure is increasingly linked to adverse health outcomes.[277] A recent study of 7557 Japanese-American men from Honolulu assessed associations between pesticide exposure (including organophosphates, organochlorines, insecticides, fumigants and herbicides) CVD.[273] These men were followed up for up to 34 years and CVD diagnosis was identified through hospital surveillance systems. Results showed a positive association between high levels of pesticide exposure (assessed using a JEM) and CVD incidence (HR 1.42, 1.05-1.92) when adjusted for a wide range of other cardiovascular risk factors. The difference in CVD incidence between the varying levels of exposure was most prominent in the first 10 years of follow-up. A cross-sectional study of 7,404 male and female American Hispanics/Latinos found that individuals reporting pesticide exposure (measured by questionnaire) were at risk of IHD (PR 2.25 (1.26-4.01)) when adjusted for age, sex and Hispanic/Latino background.[269] While studies have used the umbrella term of pesticides, there have also been studies specifically linking organophosphates to IHD.[278] The underlying mechanisms potentially include DNA damage, epigenetic modifications, endocrine disruptions, mitochondrial dysfunction and oxidative stress.[279]

Lead

A systematic review evaluating the evidence of the relationship between lead exposure and cardiovascular disease concluded that there is sufficient evidence of a causal relationship between high blood pressure with a possible dose-response effect.[280] Mechanisms underlying the relationship between lead and hypertension include oxidative stress, alterations to the nitric oxide systems and promotion of inflammation.[281] The evidence for an association between lead and CVD is currently insufficient, mostly due to the lack of studies in this area. Despite this, the mechanistic evidence of the effect of lead on high blood pressure, cholesterol and endothelial dysfunction,[282] suggests that an association with CVD may be plausible.

Particulate matter

Particulate matter is a term used to describe all solid and liquid particles in the air, from both natural and industrial sources.[283] Particulate matter has primarily been studied in the context of air pollution and is associated with poor cardiovascular health outcomes.[284] The proposed mechanisms linking particulate matter exposure to cardiovascular health include vascular dysfunction, endothelial dysfunction, systemic inflammation and disturbance of the autonomic nervous system.[285] Occupational exposure to particulate matter has also been linked to inflammation[286] and CVD in a 2010 systematic review of 37 articles.[287] Occupational exposure to dust, a specific form of particulate matter, has also been linked to IHD.[275, 288] There are many forms of dust, and although there is a lack of research across the different types, inorganic dust (asbestos, cement, concrete dust, man-made mineral fibres or quartz) has specifically been associated with IHD mortality (RR 1.13 (1.10-10.16)).[289]

Vibration

There are some suggestions that exposure to vibration may contribute to CVD, particularly acute MI, due to changes in blood pressure, although there are limited studies in females.[290, 291]

Sex

Inclusion of females is rare in the study of these other exposures and stratification by sex is even less common. This is not unexpected as females are rarely employed in occupations commonly exposed to these hazards, making it hard to study large samples of exposed females.

Ethnicity

Ethnic groups other than those of European descent are underrepresented in this research with both a lack of adjustment for ethnicity and stratification by ethnic groups.

2.6.6 Multiple exposures

Occupational exposures often cluster together; for example, shift workers are more likely to be exposed to noise, heat and physical factors.[180] Therefore, in occupations associated with CVD, there may be multiple exposures that may contribute to increased risk. As it is unlikely workers are exposed to hazards in isolation, the interaction between exposures may be relevant. There have only been a few studies that have started to investigate these combined exposures. Virkkunen et al. (2006) looked at the impact of the triad of shift work, physical workload and noise exposure.[292] This study showed that each exposure was related to IHD, but those participants exposed to two or all of these factors had a much greater risk of IHD, with those exposed to three exposures having nearly a two-times greater risk than those exposed to none of these exposures. Another study investigating occupational noise exposure, road traffic and job strain, showed that exposure to all three factors was associated with a greater risk of CVD (OR 2.2 (1.41-3.64)) than when workers were exposed to a single factor.[293] A study of Swedish men found an association between >75dB noise exposure (measured with a JEM) and IHD (age-adjusted HR 1.15 (1.01-1.31)), however, in combination with job strain this risk was even greater (age-adjusted HR 1.80 (1.19-2.73)).[215] Also, noise in combination with other factors such as vibration, chemical agents and shift work, may have additional effects on the cardiovascular system and result in a greater risk of CVD.[294]

2.6.7 Methodological differences

Across all the literature analysing occupational exposures there are differences in how CVD is measured. These methods include self-reports of CVD diagnosis, use of death or health records, and measurement of subclinical CVD, each of which be detecting different stages of disease development. Subclinical measures of CVD can be used as a reflection of early disease development in response to occupational exposure, for example, carotid artery thickness and inflammation markers have been associated with the number of years working in shift work.[295] In contrast, death records only capture severe CVD very late in disease progression. Additionally, some studies have looked at CVD in general or specific forms, such as acute MI or IHD.

Measurement of occupational exposure has been highly variable. Methods used to assess exposure include self-reports, job records, JEMs and physical measurements. The different methods of measuring exposures may mean that studies are assessing different aspects of exposure; for example, self-reports are subjective and affected by individual perception. A study looking at job strain based on the job title found no association between job strain and incident IHD,[296] suggesting that the personal perception of the stress may be an important factor. Similarly, for occupational noise exposure, subjective, self-report may give a better indication of noise annoyance, which potentially has a compounding effect on noise exposure.[297] There is also variation in physical measurements; for example, in the measurement of noise exposure, there are differences in where the noise is being measured (i.e. at the ear or environmental) and what type of noise it is (e.g. impulse or continuous).

The definition of exposures and levels of exposure (i.e. low/moderate/high) are inconsistent across studies. For example, it is not clear if nightshift is comparable to evening shift work, yet many reviews have combined this information. Additionally, although job stress is usually assessed by applying a common theoretical model, the specific questions used to measure job stress vary across studies. Ambiguity may also be an issue for measuring sedentary work and physical exertion. OPA questions often refer to the work in general (e.g. hard/high physical demands/heavy workload) and do not account for the specific activities or amount of time exposed within a day. The frequency of the physical activity, the weight of items, postures and movements involved may all be relevant factors that are not routinely considered.

In terms of study design, there is considerable heterogeneity with a mixture of cross-sectional, longitudinal, case-control and retrospective studies in this area of research. Due to the nature of CVD, the optimum study design is longitudinal as a long follow-up period captures the slowly progressing disease and potential lag time between exposure and outcome. For example, with job stress, a subgroup analysis in a meta-analysis of prospective studies found that IHD risk was only significant in studies with a follow-up period of at least 10 years.[298] Assessment of the duration (years) of exposure may be important as many of these occupational exposures show dose-response trends.

Another source of methodological variation is the adjustment for confounders, with the range of variables highly variable across studies. Factors that have been controlled for in analyses include smoking, alcohol, weight, high blood pressure, diabetes, cholesterol, alcohol consumption, physical activity, family history, SES and other occupational factors. However, the roles of these other factors are not clear, and while some may act as confounders, others may lie on the physiological pathway or be effect modifiers. Therefore, controlling for some of these factors may be an over adjustment. However, although SES is a strong confounder that is generally not considered an intermediate factor on the causal pathway, it is not consistently controlled for in analyses.

2.7 Summary

Figure 2.3 summarises the literature with respect to potential occupational risk factors, behaviours, and physiological pathways that may be underlying associations between occupation and CVD. The level of evidence for each of the occupational factors has been evaluated as weak, moderate or strong. The following paragraphs summarise the different components of Figure 2.3 and current knowledge gaps.

Research design

Study design and populations

There is an overall lack of longitudinal studies with sufficient follow-up periods, and the diversity of the populations assessed is generally limited. The majority of work has been undertaken in males of European descent, with few studies on other ethnicities or with ethnic comparisons, resulting in a distinct lack of understanding of the relationship between CVD and occupation for females and indigenous peoples (and/or other minority populations).

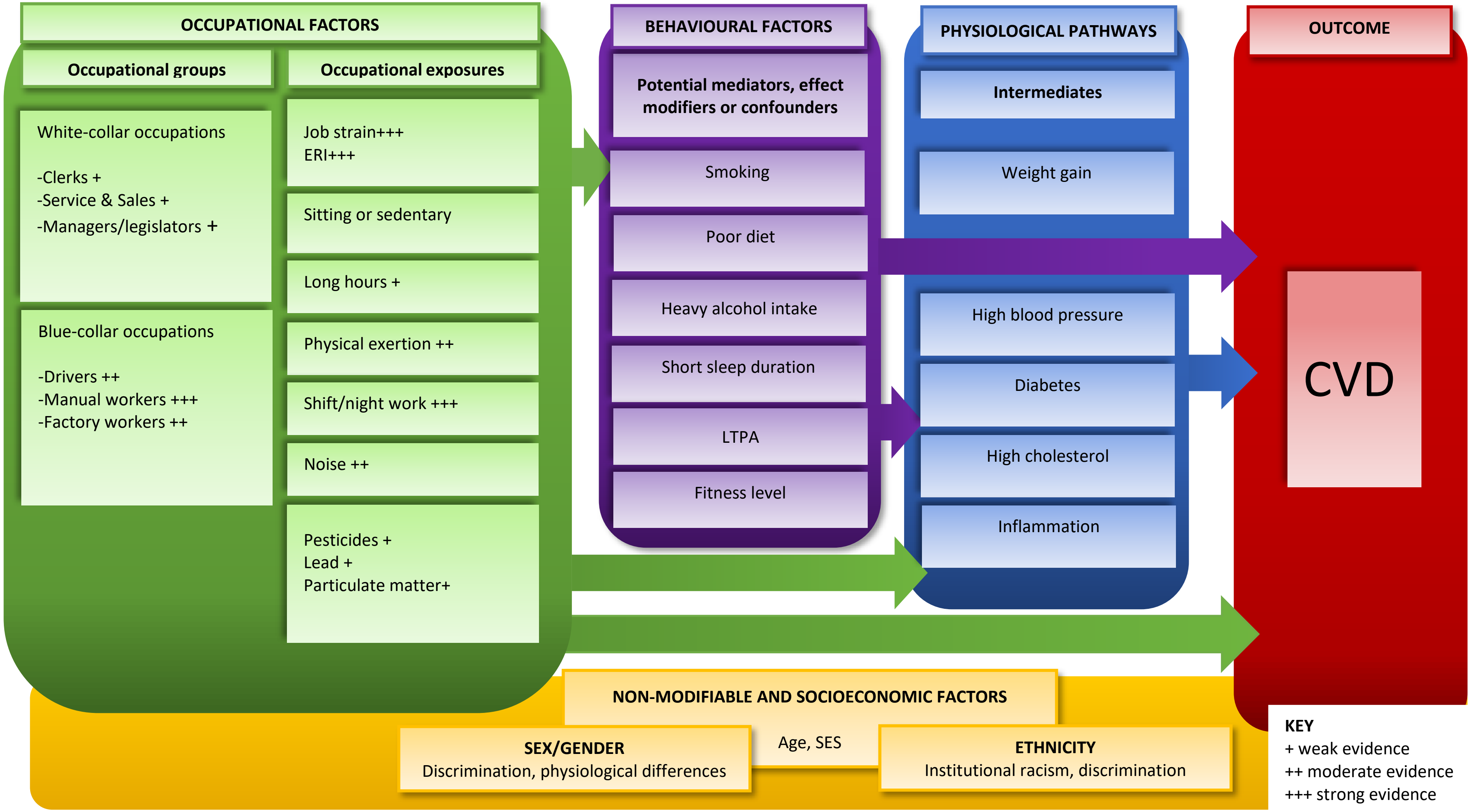

Figure 2.3.The relationship between occupation and cardiovascular disease

Non-modifiable and socioeconomic factors

Age is the most important variable as CVD risk is strongly associated with increasing age and although a few studies have stratified by age group, the majority have adjusted for age in their analyses and it is the most consistently included confounder. However, the age range of participants included varies significantly and as including younger or older workers may influence results, cautious interpretation or sensitivity analyses may be required. Low SES is strongly associated with blue-collar occupations and increased CVD risk, yet SES of any form (e.g. income, economic status, education level) is not routinely included in all analyses.

Sex has been discussed throughout this literature review and it has been consistently highlighted that females are underresearched in the context of occupational health and this presents a major knowledge gap. As there are biological differences between males and females, as well as potential differences in exposure conditions and co-exposures, it is important to consider sex-specific results.

Ethnicity has been even less of a focus in research to date, despite the growing focus of ethnicity and culture in determining CVD risk factors.[299] The workplace exposure conditions and co-exposures may differ across ethnicities, due to factors such as racial discrimination, and therefore, it is important to consider ethnic-specific results.

Occupational factors

Occupation

Although evidence generally indicates a greater burden of conventional cardiovascular risk factors and CVD risk for BCW, there is a lack of information on which specific occupations within this broad occupational group represent a particularly high risk. Furthermore, the broad classification of white-collar occupations yields even less consistent results; as a consequence, it is difficult to determine which specific occupations have a particularly high risk, hampering efforts to reduce occupational CVD through targeted interventions. As noted previously, although it would be desirable to assess associations using more specific occupational groups, this is generally only possible in very large studies, of which there are not many.

Additionally, most studies have looked at current occupation, longest occupation held or occupation at death, with very few accounting for work history or duration of employment. Therefore, there is a gap in the understanding of the effect of prolonged exposure to an occupation and the cumulative effect of employment in multiple occupations.

Occupational exposures

A range of occupational exposures have been linked with CVD, but the evidence is mixed and many occupational exposures have not been extensively researched. There is also a distinct lack of knowledge on the effect of multiple occupational exposures and prolonged exposure.

Behavioural factors

Potential mediators, confounders or effect modifiers

These factors include smoking, consuming a poor diet, high alcohol intake, short sleep duration, LTPA and fitness levels. Most commonly, analyses have adjusted for these factors, which is appropriate for confounders such as smoking, which is consistently associated with occupation and CVD. However, some literature proposes that some of these factors may lie on the causal pathway and play a mediating or effect modifying role. Therefore, controlling for these factors may not be appropriate. Other studies have stratified by these potential mediators, such as fitness level and LPTA, and have found them to be effect modifiers, for example, the adverse effects of physical exertion are more distinct at low fitness and low LPTA.[260, 263] However, few studies have explored these issues (often as a result of requiring much larger sample sizes to be able to do this) and, as noted above, most have simply adjusted the analyses for these factors potentially resulting in biased results. Also, few studies have used alternative statistical methods, such as mediation analyses, to specifically assess the role of mediating factors. It is likely there are also additional mediators or effect modifiers that are not recognised as such.

Physiological pathways

Conventional cardiovascular risk factors, such as obesity, high blood pressure, high cholesterol, diabetes and inflammation, may be on the physiological pathway between occupational exposure and CVD. As such, they may be intermediates rather than confounders (although they could be both). The majority of studies have included models with adjustments for all of these risk factors. However, when considering the proposed causal pathways, it is highly unlikely they act independently and adjusting for these factors may be an overadjustment. To add to the complexity, as the physiological pathways are not conclusively known for occupational exposures, it is not clear which cardiovascular risk factors to treat in which way.

Outcome - CVD

Much of the research relies on self-report or death records, introducing recall bias or restricting the outcome to fatal CVD, respectively. However, more studies are beginning to use health records to reliably detect non-fatal CVD as well. Many studies have looked at CVD in general, some even including stroke. However, the relationship may not be consistent for all CVDs, despite similar pathologies, potentially resulting in a lack of knowledge for specific CVDs, such as IHD.

Conclusion

From the literature summarised in this review, it is clear that the association between occupation and CVD is complex, involving many factors and physiological pathways. The literature demonstrates occupation is important in the context of CVD; however, knowledge about specific occupations, causal exposures and physiological pathways is limited, hampering efforts to reduce occupation-related CVD. Furthermore, the understanding of this association in different populations, such as males and females or in different ethnic groups, is significantly limited.

CHAPTER THREE

Methods

The methods are briefly described in each of the papers (Chapters 4, 5 and 6). This chapter provides a more comprehensive overview of the sources of exposure and health data used in the analyses.

3.1 New Zealand Workforce Survey (NZWS) and the Māori NZWS

The NZWS and the Māori NZWS were conducted by the Centre for Public Health Research (CPHR, Massey University) and are the first and only workforce surveys in New Zealand that assess information on occupational exposures in a random sample of the general and Māori working populations.

3.1.1 Recruitment

Random samples of men and women aged 20-64 years old were selected from the Electoral Roll. The Electoral Roll includes information on age, occupation and Māori ancestry/ethnicity, and is approximately 95% complete. It is the best sample frame available for the research described in this thesis with any bias likely to be small once the data have been age-adjusted.

The NZWS was conducted from 2004-2006 and participants were randomly selected from the 2003 and 2005 Electoral Rolls. The Māori NZWS was conducted from 2009-2011 and equal numbers of participants were selected from both the 2008 General (where Māori descent was indicated) and Māori Electoral Rolls (Māori can choose to be on either).

The invitation to participate in a telephone interview was mailed up to 3 times and non-respondents were contacted by phone when a phone number was available. Individuals did not have to work at the time of interview and were aged between 20 and 64 years. The overall response rate for the NZWS was 37%, with 3003 respondents; the response rate for the Māori NZWS was 29%, with 2107 respondents. Two participants were included in both surveys; therefore, their most recent interview was excluded (i.e. the Māori NZWS). The methodology is described in detail by Eng et al. (2010) and Denison et al. (2018),[26, 300] and the representativeness of the NZWS is described by 't Mannetje et al. (2011).[301] While some groups were underrepresented in the survey sample, such as younger ages, higher deprivation and Māori (in the NZWS), the prevalence of key survey variables (both occupational exposure and health-related variables) were unchanged after standardising to the demographic distribution of the source population, and similar between early and late responses. Therefore, response bias is estimated to be low.

Ethics approval was granted by the Massey University Human Ethics Committee for both surveys (NZWS: WGTN 03/133 and NZWS: MUHEC 08/28) and from the New Zealand Health and Disability Ethics Committees for the linkage of the two surveys (16/NTB/173).

3.1.2 Funding

The general New Zealand Workforce Survey was funded from a Joint Research Portfolio of the Health Research Council, the Accident Compensation Corporation and the Department of

Labour, which issued a Request for Proposals for a study of the burden of occupational ill-health in New Zealand (HRC 04/072) in 2003. The Māori Workforce Survey was funded by the Health Research Council of New Zealand as part of a Programme grant awarded to CPHR, Massey University (HRC 08-041E) in 2008. The linkage component was also funded by the Health Research Council of New Zealand (HRC 16/351).

3.1.3 Lifestyle information

Participants' residential address, available from the Electoral Rolls, was used to determine their meshblock address, which is the smallest unit at which the New Zealand Census reports data (on average, 95 people live in a meshblock), and deprivation index (a census-based index with a relative deprivation score, ranging from 1 to 10, assigned to each geographical meshblock).

In the NZWS, ethnicity was self-identified; participants were asked if they identified as Māori (~16.5% of the New Zealand population), Pacific peoples, European/Pākehā or 'other'. Pākehā are non-Māori and usually of European descent (~ 70% of the New Zealand population).[302] Pacific peoples (~8% of the New Zealand population) with ancestral links to Samoa, Fiji, Cook Islands, Niue and Tokelau were grouped together as the sample was not large enough to analyse each group separately. If a participant identified multiple ethnicities, Māori was prioritised.

The questionnaire also collected information on participants' date of birth and smoking habits, including whether they were ex, current or had never smoked. If participants had smoked, the start and cessation date, along with the number of cigarettes per day were recorded. Participants were also asked to provide their height and weight, which was used to calculate

BMI, an indicator of weight relative to height measured by a person's weight in kilograms divided by the square of height in metres (kg/m^2).

3.1.4 Occupational Information

Participants were asked to provide their current or most recent job title and outline their main tasks. This information was then used to code each job using the New Zealand Standard Classification of Occupations (NZSCO) 1999,[303] which is a hierarchical skills-based classification of occupations. The main activity of the organisation of the job was used to code industry, based on the Australian and New Zealand Standard Industrial Classification (ANZISC) 1996.[304] Lifetime work history of all jobs with a minimum of 6 months employment was also collected. Each record included the date of job commencement and cessation, a description of the main activity of the employer, department and job title, and was coded using NZSCO.

3.1.5 Occupational Exposures

All occupational exposure information collected from participants was based on their current or most recent job. Participants were asked whether the following exposures were present (yes/no): dust; smoke or fumes; gas; oil and solvents; acids or alkalis; and pesticides. Participants were also asked how frequently they were exposed to the following physical factors: awkward and tiring positions; awkward grip or hand movements; lifting; standing; sitting; using tools that vibrate; loud noise; repetitive tasks; working at very high speeds; and working to tight deadlines. The frequency of exposure was measured on a scale from never to always (participants could provide a percentage of time or choose a point on the scale from never, ¼, ½, ¾, or all the time). Participants were also asked about organisational factors, including average hours worked per week and irregular hours (outside 7:00 to 20:30), and the question "In the last 4 weeks, did you work for pay, profit or income for at least 3 hours between midnight and 5 am?" was used to assess participants' exposure to night shift. Job stress was assessed using the question "In general, how do you find your current job?" and measured on a 5-point scale (Not at all stressful, mildly stressful, moderately stressful, very stressful and extremely stressful).

3.2 Health Information

3.2.1 Integrated Data Infrastructure

The health information was obtained from routinely collected health records accessed from the Integrated Data Infrastructure (IDI), a linked longitudinal meta-dataset consisting of social, health and economic data from different Government agencies in New Zealand and is managed by Statistics New Zealand.[305] The IDI has been primarily used by government agencies but is increasingly used by researchers throughout New Zealand.

The 'spine' is the main dataset to which all of the other datasets are linked; it is comprised of tax, births and visa data, and covers 95 % of the 'ever-resident New Zealand population'.[306] It is considered the primary 'person-level' dataset that all other 'person-level' datasets are linked to, allowing analysis across different governmental sectors.[307] These datasets are exactly matched where there are common unique identifiers available or probabilistically linked where there are overlapping personal and demographics variables.[308]

All information within the IDI is considered confidential, requiring permission and training to access as the law requires private information to be protected. All data available in the IDI is de-identified and personal information such as names, addresses and unique identifiers are removed. For this reason, all data released from the IDI is subject to Statistics New Zealand outputting rules, such as random rounding to up to down to the nearest multiple of three for frequency counts and suppression of counts under six, hence, counts and percentages presented throughout this thesis may not add up to 100%

There are four refreshes throughout the year where various datasets are updated with recent information, often extending the date range of the available data. Table 3.1 contains a summary of the refreshes that were used in the analyses of the studies and the date ranges of available data. The September 2018 refresh was used for the analyses in Chapter 4 and the September 2019 refresh was used for the analyses in Chapters 5 and 6.

Table 3.1. Dates of available data for the IDI datasets

	From date (for all refreshes)	To date	
		September 2018 refresh	**September 2019 refresh**
Pharmaceutical	2005	Dec-17	Dec-18
Laboratory claims	2003	Jun-17	Jun-18
Mortality	1988	Dec-15	Dec-16
National non-admitted patient collection	2007	Jun-17	Jun-19
Population cohort demographics and addresses	2004	Jul-18	Jan-19
Publicly funded hospital discharges	1988	Dec-17	Dec-18
Personal details	1842/1876	Jul-2018	Aug-2019
Person overseas spell	1900	Jul-2018	Jul-2019

The mortality dataset, available from 1988 to December 2015, is the only dataset containing cause of death information. The delay in the update of this dataset is due to the matching of the death registrations to the cause of death certificates with additional information on the underlying cause of death obtained from multiple sources (Hospital discharges; the New Zealand Cancer Registry; the New Zealand Transport Agency; Water Safety New Zealand; and from writing letters to certifying doctors, coroners and medical officers). However, the *personal details* dataset can be used to obtain registered deaths without information about cause of death.

3.2.2 Linking survey data to the IDI

Survey data was sent to the Ministry of Health (MoH) to undergo probabilistic matching based on date of birth, sex, family name and first two given names, to identify National Health Index (NHI) numbers for each participant. This NHI number was used to link participants to the MoH datasets in the IDI and then link participants to the IDI spine, allowing identification of participants in other datasets such as *personal details* and *person overseas spell*, to identify participants that died or moved overseas, respectively. There were 70 participants excluded as NHI numbers were unobtainable and 51 participants who could not be linked to the spine; 36 of those participants who were not matched to the spine could be identified in other MoH datasets to determine a date last observed but the remaining 15 participants were excluded.

3.2.3 Pathway to care for IHD patients

As health conditions in this thesis are defined based on interactions with healthcare providers (see below), a brief description of the pathway to care for IHD patients is outlined in the following paragraphs.

Primary prevention of IHD involves lifestyle and therapeutic interventions to minimise the presence of risk factors (e.g. smoking; high cholesterol, high blood pressure and diabetes). If lifestyle changes do not produce satisfactory improvements and/or the metabolic risk factors are severe, medication is also used to treat and manage these conditions. Cardiovascular risk assessment is also recommended for the early management of IHD (from the age of 45 years in men and 55 in women; 15 years earlier in high-risk ethnicities such as Māori).[309] In patients with moderate to high 5-year risk (of experiencing a cardiac event), drug therapy (statins and anti-hypertensives) is strongly recommended, regardless of their specific risk factors.[309] Therefore, pharmaceutical dispensings of medication to lower blood pressure and cholesterol may either indicate the presence of the respective risk factors or the presence of overall high cardiovascular risk.

The diagnosis of IHD specifically is based on clinical findings, such as electrocardiograms, cardiac stress testing or coronary angiograms, which are conducted due to chronic symptoms (e.g. angina) or acute symptoms (e.g. heart attack).[310] Once IHD is established, treatment is aimed at reducing symptoms and disease progression. Anti-angina medication is the primary medication used to manage symptoms and to prevent the progression of disease. It is also common practice in New Zealand for patients to be put on triple therapy; a combination of antiplatelet/anticoagulant, blood pressure-lowering and statin medication.[311] Though not all IHD patients are on triple therapy, most are on some combination of anti-angina, blood pressure lowering and cholesterol lowering medication. Surgical interventions (such as coronary stent placement or bypass surgery) are also used in combination with medication to restore blood flow and prevent future events when severe blockages are present.

3.2.4 Incident IHD

Based on the pathway to care for IHD patients in New Zealand, the definition for an incident case of IHD includes a hospital diagnosis, death record or two or more dispensings of anti-anginal medication within a 12-month period (other medications used in the treatment of IHD were not included due to the ambiguity of reason of use). The IHD definition (Table 3.2) was developed from the PREDICT studies,[312] a previous research paper examining sociodemographic differences in IHD in New Zealand using linked health records[313] and the definition of IHD in the MoH chronic conditions dataset.[314]

Table 3.2 outlines the definitions of the cardiovascular health conditions of interest in the current study. The International Classification of Diseases (ICD) codes presented in the table includes both the 9th and 10th revisions, ICD-9-CMA and ICD-10-AM, to capture diagnoses that have been classified with either version.

Table 3.2. IHD definition

Condition	**Data Source**	**ICD Code or Criteria**	**Details**
IHD	Mortality data	ICD-9-CMA 410, ICD-10-AM I21	Acute myocardial infarction
		ICD-10-AM I22	Subsequent myocardial infarction
	National Minimum Dataset (NMDS): Publicly funded hospital discharges – diagnosis/procedure information	ICD-9-CMA 411, ICD-10-AM I24	Other acute and subacute forms of ischaemic heart disease
		ICD-9-CMA I23	Certain current complications following myocardial infarction (within the 28-day period)
		ICD-9-CMA 413, ICD-10-AM I20	Angina pectoris
		ICD-9-CMA 412, ICD-9-CMA 414, ICD-10-AM I25*	Chronic ischaemic heart disease **ICD-10-AM I25.2 was excluded as it refers to old myocardial infarction and therefore, not incident IHD*
		ICD-9-CMA V45.81	Aortocoronary bypass status
		ICD-9-CMA V45.82	Percutaneous transluminal coronary angioplasty status
		ICD-10-AM Z95.1	Presence of aortocoronary bypass graft
		ICD-10-AM Z95.5	Presence of coronary angioplasty implant and graft
		ICD-9- CMA 36.0x, 36.1x ICD-10-AM 3530400, 3530401, 3530500, 3530501, 3531000, 3531001, 3531002, 3531003, 3531004, 3531005, 3849700, 3849701, 3849702, 3849703, 3849704, 03849705, 3849706, 3849707, 3850000, 3850001, 3850002, 3850003, 3850004, 3850300, 3850301, 3850302, 3850303, 3850304, 9020100, 9020101, 9020102, 9020103, 3863700, 3845619, 3865308, 3850500	Procedures
	Pharmaceutical Data	1577 Glyceryl trinitrate 2377 Isosorbide dinitrate 2836 Isosorbide mononitrate 1292 Nicorandil 1949 Perhexiline maleate	≥2 dispensings within 12 months

3.2.5 Diabetes Mellitus

The diabetes definition was developed using the IDI chronic conditions definition[314] and the Virtual Diabetes Register of New Zealand's criteria (a database monitoring people suspected as having diabetes).[315] The definition of diabetes (Table 3.3) includes diagnoses, laboratory claims and outpatient visits, all relating to diabetes, and two or more dispensings of medication used in the management of diabetes. To exclude participants with gestational diabetes, females with only insulin dispensings within five months before and two weeks after a birth discharge were excluded. Furthermore, female participants that were only dispensed metformin and met no other criteria, were also excluded as this indicates treatment of Polycystic Ovarian Syndrome.

Table 3.3. Diabetes definition

Condition	Data Source	ICD Code or Criteria	Details
Diabetes Mellitus	NMDS: Publicly funded hospital discharges – diagnosis/procedure information	ICD-10-AM E10	Type 1 diabetes mellitus
		ICD-10-AM E11	Type 2 diabetes mellitus
		ICD-10-AM E13	Other specified diabetes mellitus
	Mortality data	ICD-10-AM E14	Unspecified diabetes mellitus
		ICD-10-AM O24.0	Pre-existing diabetes mellitus, Type 1 in pregnancy
		ICD-10-AM O24.1	Pre-existing diabetes mellitus, Type 2 in pregnancy
		ICD-10-AM O24.2	Pre-existing diabetes mellitus, other specified types, in pregnancy
		ICD-10-AM O24.3	Pre-existing diabetes mellitus, unspecified, in pregnancy.
	Pharmaceutical Data All subsidised forms of insulin, oral hypoglycaemics, and glucagon	Insulin lispro Acarbose Glibenclamide Gliclazide Glipizide Insulin neutral Insulin isophane Insulin zinc suspension Metformin hydrochloride Tolazamide Tolbutamide Rosiglitazone Insulin aspart Pioglitazone Insulin glargine Insulin lispro with insulin lispro protamine Insulin glulisine Insulin aspart with insulin aspart protamine Insulin isophane with insulin neutral	≥2 dispensings within 2 years (if only metformin use, must meet other criteria)

3.2.6 High Blood Pressure and High Cholesterol

The definitions of high blood pressure and high cholesterol were based on previous studies using pharmaceutical and administrative records for high blood pressure.[316-318] Two or more dispensings of medication used to treat these conditions or hospital diagnoses with an ICD related to these conditions were used to identify participants (Table 3.4).

Table 3.4. High blood pressure and high cholesterol definitions

Condition	Data Source	ICD Code or Criteria	Details
High Blood Pressure	NMDS: Publicly funded hospital discharges – diagnosis/procedure information	ICD-9-AM 401.0	Malignant essential hypertension
		ICD-9-AM 401.1	Benign essential hypertension
		ICD-9-AM 401.9	Unspecified essential hypertension
		ICD-10-AM I10	Essential (primary) hypertension
		ICD-9-AM 403, ICD-10-AM I12	Hypertensive chronic kidney disease
	Pharmaceutical Data	**ACE inhibitors** Trandolapril Cilazapril with hydrochlorothiazide Enalapril with hydrochlorothiazide Enalapril Cilazapril Quinapril Benazepril Lisinopril with hydrochlorothiazide Lisinopril Perindopril Captopril with hydrochlorothiazide Captopril Quinapril with hydrochlorothiazide Angiotension II antagonists Losartan Candesartan **Beta blockers** Acebutolol Alprenolol Atenolol Labetalol Metoprolol succinate Metoprolol tartrate Nadolol Oxprenolol Pindolol with clopamide Pindolol Propranolol Sotalol Timolol maleate Celiprolol Carvedilol **Calcium channel blockers** Nifedipine Verapamil hydrochloride Felodipine Diltiazem hydrochloride Isradipine Amlodipine **Other AHT**	≥2 dispensings within 12 months

Condition	Data Source	ICD Code or Criteria	Details
		Amiloride Clonidine Clonidine hydrochloride Hydralazine Methyldopa **Thiazides** Acebutolol with hydrochlorothiazide Amiloride with hydrochlorothiazide Atenolol with hydrochlorothiazide Bendrofluazide Chlorothiazide Chlorothalidone Cyclopenthiazide Indapamide Methyclothiazide Methyldopa with hydrochlorothiazide Triamterene with hydrochlorothiazide	
High Cholesterol	NMDS: Publicly funded hospital discharges – diagnosis/procedure information	ICD-9-AM 272.0, ICD-10-AM E780	Pure hypercholesterolemia
		ICD-9-AM 272.4, ICD-10-AM E785	Hyperlipidaemia, unspecified
	Pharmaceutical Data	**Statins** Fluvastatin Atorvastatin Simvastatin Pravastatin Ezetimibe with simvastatin **Other cholesterol-lowering medications** Ezetimibe	≥2 dispensings within 12 months

3.2.7 Follow-up

For the survival analyses (Chapters 5 and 6), participants were followed from the date of interview, which differed across the two surveys (NZWS 2004-2007; Māori NZWS 2009-2011). Participants with IHD prior to interview were excluded and others that moved overseas or died of non-IHD related causes were identified through immigration and mortality datasets and censored from that time point. The end of follow-up was 31 December 2018 as this is the last date a hospital diagnosis could be identified for those that were not lost to follow-up or did not have an incident IHD event.

3.3 Statistical analyses

This section briefly describes the sample size and statistical methods used to address the aims of this thesis. Further details about these analyses are presented in Chapters 4, 5 and 6.

3.3.1 Survey samples

In total, there were 3003 NZWS participants and 2105 Māori NZWS participants in the original surveys. After a 98% matching rate to MoH data, there were 2968 NZWS participants and 2072 in the NZWS Māori survey linked to health records in the IDI.

3.3.2 Aim 1 (Chapter 4)

Logistic regression, adjusted for age group, was used to assess associations between current or most recent occupational group at the time of interview and conventional cardiovascular risk factors (high deprivation, ever-smoked, obesity, diabetes, high blood pressure and high cholesterol).

3.3.3 Aim 2 (Chapter 5)

Cox proportional hazards regression was used to assess associations between ever working in a particular occupational group or industry, using occupational history, and incident IHD. Results were adjusted for age group, high deprivation and smoking status.

3.3.4 Aim 3 (Chapter 6)

Cox proportional hazards regression was used to assess associations between occupational exposures at the time of interview and incident IHD. Results were adjusted for age group, high deprivation and smoking status.

3.3.5 Aim 5 (Chapters 4, 5 and 6)

All analyses were stratified by sex to determine whether associations between occupational risk factors and IHD were consistent for males and females.

3.3.6 Aim 6 (Chapters 4, 5 and 6)

The general population survey (NZWS) and Māori population survey (Māori NZWS) were analysed separately throughout all analyses to determine whether associations between occupational risk factors and IHD were consistent for the Māori and general populations.

CHAPTER FOUR

The prevalence of cardiovascular risk factors in different occupational groups in New Zealand

Lucy A Barnes, Amanda Eng, Marine Corbin, Hayley J Denison, Andrea t' Mannetje, Stephen Haslett, Dave McLean, Rod Jackson, Jeroen Douwes.

Objectives: Although cardiovascular disease (CVD) risk has been shown to differ between occupations, few studies have specifically evaluated the distribution of known CVD risk factors across occupational groups. This study assessed CVD risk factors in a range of occupational groups in New Zealand, stratified by sex and ethnicity.

Methods: Two probability-based sample surveys of the general New Zealand adult population (2004-2006; n=3003) and of the indigenous peoples of New Zealand (Māori; 2009-2011; n=2107), for which occupational histories and lifestyle factors were collected, were linked with routinely collected health data. Smoking, body mass index, deprivation, diabetes, high blood pressure and high cholesterol were dichotomised and compared between occupational groups using age-adjusted logistic regression.

Results: The prevalence of all known CVD risk factors was greater in the Māori survey than the general population survey, and in males compared to females. In general, for men and women in both surveys, *plant and machine operators and assemblers* and *elementary workers* were more likely to experience traditional CVD risk factors, while *professionals* were less likely to experience these risk factors. *Clerks* were more likely to have high blood pressure and male *agricultural and fishery workers* in the general survey were less likely to have high cholesterol, but this was not observed in the Māori survey. Male Māori *trades workers* were less likely to have high cholesterol and were less obese, while for the general population survey, this was not observed.

Conclusion: This study showed differences in the distribution of known CVD risk factors across occupational groups, as well as between ethnic groups and males and females.

Annals of Work Exposures and Health 2020 64: 645-658[319]

The manuscript which appears here differs slightly from the final version published in the Annals of Work Exposures and Health.

4.1 Introduction

Cardiovascular disease (CVD) is a leading cause of death worldwide.[30] Known risk factors for CVD include behavioural factors such as smoking, physical activity, unhealthy diet and obesity; metabolic risk factors including high blood pressure, impaired glucose control and raised lipid levels; and socio-demographic risk factors such as age, sex, ethnicity, deprivation, as well as family history.[320]

Previous studies have reported marked differences in CVD risk between occupational groups.[10, 16, 111] In particular, elevated risks of CVD have been shown for bus drivers, police officers, hairdressers, sales and low-status office workers, [13] community and social service employees,[103] spinners, and shoemakers and leather goods workers.[11, 16, 111, 123] Differences in metabolic CVD risk factors, such as high blood pressure, diabetes and high cholesterol have also been observed between occupational groups,[13, 105, 107] although results have been mixed. For example, metabolic risk factors are highly prevalent in sedentary occupations, such as office workers, [102, 321] and other occupations like auto-technicians.[105] Blue-collar workers (BCW), typically engaging in manual labour, may be at the greatest risk, with the strongest associations observed for high blood pressure.[107, 111, 112] Results for white-collar workers (WCW; i.e. non-manual occupations) are less consistent, although an increased risk of obesity and high cholesterol has been reported.[107]

Determinants contributing to these differences may include occupation-associated behavioural and demographic factors (e.g. smoking, diet, deprivation), but it has been suggested that specific workplace exposures may also play a role, such as noise, job strain/stress, occupational physical activity and chemical exposures.[10, 19, 20] Characterising the distribution of traditional CVD risk factors such as smoking, obesity and socioeconomic status (SES), and metabolic risk factors including high blood pressure, diabetes and high cholesterol, across occupational groups is an important first step in elucidating the complex mechanisms through which occupation may contribute to CVD risk, but the literature on this is limited. In particular, few studies have described the prevalence of risk factors across multiple occupational groups, and even less have focused on sex and ethnic differences, despite evidence that CVD risk factors differ between males and females, and between ethnic groups.[28, 39, 80] Potential causal exposures may also differ between ethnicities and males/females even within the same occupation,[26, 27] thus providing further reason for taking into account ethnic and sex differences.

The aim of this study was to assess the distribution of traditional CVD risk factors across occupational groups in New Zealand, stratified by sex and ethnicity. Data from two previous New Zealand workforce surveys (one in the general population and one in the Māori population, the indigenous peoples of New Zealand) was linked to routinely collected health data, to describe the distribution of smoking, obesity and SES, and metabolic risk factors including high blood pressure, diabetes and high cholesterol, across occupational groups

4.2 Methods

Recruitment

This linkage study used demographic and occupational data from two previous workforce surveys, one in the general population i.e. the New Zealand Workforce Survey (NZWS)[300] and one in the indigenous Māori population i.e. the Māori NZWS.[26] In the NZWS, a random proportionally stratified, systematic and self-weighted sample of people aged 20-64 years was selected from the New Zealand Electoral Roll and interviewed between 2004 and 2006. The Electoral Roll is ~95% complete and any bias in the source population is therefore likely to be small. An invitation to participate in a telephone interview detailing the objectives of the study was mailed up to three times, and non-responders were contacted by phone where a phone number was available. In total, 3003 participants (response rate 37%) completed the survey. The methods are described in more detail in Eng et al. (2010). The same methodology was used for the Māori NZWS. The total sample comprised of 2107 participants (response rate 29%) aged 20-64 years randomly selected from the 2008 and 2010 general and Māori Electoral Rolls (Māori can choose to register on either) and interviewed over the 2009-2011 period. The Māori NZWS methods are described in more detail in Denison et al. (2018).[26]

We have previously assessed possible response bias from the low survey response rates for both surveys but found this to be small.[26, 301] Two participants were included in both surveys and we, therefore, excluded their most recent interview (i.e. the Māori NZWS).

All participants provided written consent, and the study conformed to the principles embodied in the Declaration of Helsinki. Ethics approval was granted by the Massey University Human Ethics Committee for both surveys (NZWS –WGTN 03/133, Māori NZWS – MUHEC 08/28) and the linkage study was granted approval by the Health and Disabilities Ethics Committees (16/NTB/173).

Study questionnaire

The questionnaire used in both surveys included questions about lifetime work history, current workplace exposures, and demographic and lifestyle factors. Participants were asked if they identified as Māori, Pacific Islander, European/Pākehā (non-Māori, usually of European descent) or other. Pacific peoples are composed of people from several Pacific Island states including Fiji, Samoa, Tonga, Cook Islands, Niue and Tokelau. If a participant identified with multiple ethnicities, this was classified into a single ethnicity, which prioritised Māori, followed by Pacific Islander, other and European/Pākehā.

CVD risk factors collected via questionnaire

All jobs were coded using the New Zealand Standard Classification of Occupations (NZSCO) 1999, which is a hierarchical skills-based classification with nine major occupational groups.[303] BCW were defined as the NZSCO groups 6-9: *agricultural and fishery workers*; *trades workers*; *plant and machine operators and assemblers*; and *elementary occupations*. WCW were defined as the NZSCO groups 1-5: *legislators, administrators and managers*; *professionals*; *technicians and associate professionals*; *clerks*; and *service and sales workers*. Age at interview was categorised as follows: 20-34; 35-44; 45-54; and 55+ years. Deprivation was assessed using the New Zealand Deprivation Index 2006 (NZDep2006), which is a census-based index with a relative deprivation score (ranging from 1-10) assigned to each geographical meshblock based on place of residence recorded on the Electoral Roll. The distribution of deprivation index was presented in quintiles, but for subsequent logistic regression, NZDep2006 was dichotomised combining NZDep2006 scores 9-10 (people living in neighbourhoods of high deprivation) and scores 1-8 (people living in neighbourhoods of lower deprivation). Participants provided their height and weight at the time of interview and this was used to calculate their Body Mass Index (BMI), which was grouped into four categories based on the World Health Organisation guidelines.[322] It was dichotomised using 30 kg/m^2 as the cut-point for obesity for use in logistic regression.

Participants stated whether they were current, ex- or never smokers; if they had smoked, they provided the years of commencement and cessation and the number of cigarettes smoked per day. Pack-years were calculated from the number of cigarettes smoked per day divided by 20, multiplied by the number of years the person smoked. Participants who selected ex- or current smoker were categorised as having ever smoked. As current smoking can be subject to recent changes in smoking habits and the pack-years variable was incomplete due to missing information, dichotomous ever/never smoking was used to evaluate smoking status (reanalyses using incomplete pack-years did not alter the analyses; data not shown).

CVD risk factors obtained from linked health records

To enable data linkage, probabilistic matching was used based on date of birth, sex, family name and first two given names, to identify participants' National Health Index (NHI) number, which resulted in 98% of survey respondents being successfully matched. The routinely collected health data was obtained from Ministry of Health (MoH) records within the Integrated Data Infrastructure (IDI), which is a longitudinal meta-dataset linked at the individual level, consisting of de-identified data from different government agencies and managed by Statistics New Zealand.[305, 307] The health records included mortality records, public hospital diagnoses, outpatient visits, laboratory claims and pharmaceutical dispensing. The IDI has a central spine to which individual datasets (including health data) are linked based on data from the Inland Revenue Department and births and visa applications. This covers almost 95% of New Zealand's ever-resident population.[306, 307]

Using a definition similar to previous studies,[316, 317] participants who had ever had high blood pressure were identified if they had two or more pharmaceutical dispensings of anti-hypertensive medication within a 12 month period (from 2005 to December 31st 2017); or if there was a record of a public hospital discharge (primary healthcare information is not available in the IDI and private hospital admission data are inconsistent and incomplete) with a high blood pressure related ICD code (ICD-9-AM 401.0; ICD-9-AM 401.1; ICD-9-AM 401.9; ICD-10-AM I10; ICD-9-AM 402, ICD-10-AM I11; ICD-9-AM 403/4, ICD-10-AM I12/3) (from 1988 to June 30th 2017).

Similarly, participants who had ever had high cholesterol were identified through the pharmaceutical data if they had two or more dispensings within a 12 month period (from 2005 to December 31st 2017) of medication used to lower cholesterol,or a public hospital discharge with a high cholesterol related ICD code (ICD-9-AM 272.0, ICD-10-AM E780; ICD-9-AM 272.4, ICD-10-AM E785).

Identification of participants who had ever had diabetes was largely based on the algorithm developed as part of the New Zealand Virtual Diabetes Register (VDR), a national register providing annual estimates of diabetes prevalence to inform policy and service planning.[315] Specifically, participants were classified as having diabetes if the pharmaceutical data (from 2005 to December 31st2017) indicated two or more dispensings for any subsidised form of insulin, oral hypoglycaemic or glucagon within a two year period, or had a public hospital discharge (from 1988 to June 30th 2017) or death record (up to December 2015) with a diabetes related ICD code (ICD-10-AM E10; ICD-10-AM E11; ICD-10-AM E13; ICD-10-AM E14; ICD-10-AM O24.0; ICD-10-AM O24.1; ICD-10-AM O24.2; ICD-10-AM O24.3). Laboratory claims (from 2003 to June 30th 2017) of four or more glycated haemoglobin tests (HbA1c) and two or more albumin creatinine ratio tests within a two year period, and outpatient visits with codes for diabetes education or diabetes retinal screening (from 2007 to June 30th 2017) were used to identify additional diabetes patients. If women had diabetes-related laboratory claims limited to the 9 months before a birth discharge, or insulin dispensing limited to the 5 months before and two weeks after a birth discharge, this was interpreted as gestational diabetes and therefore not included as having diabetes. More details about the available date ranges of the data sources and criteria of these definitions are presented in Supplementary Tables S4.3 and S4.4.

Statistical Analysis

All analyses were stratified by survey and sex as the surveys were completed at different time periods and in different populations, and there is evidence of sex-specific differences in CVD risk.[28] Occupational information was missing for 21 participants and NHI numbers unobtainable for 70, leaving a total anonymised sample sizes of 2968 in the NZWS sample and 2070 in the Māori NZWS sample.

As noted in the introduction, the main purpose of this paper is to describe the distribution of traditional CVD risk factors across occupational groups in New Zealand, but as this is affected by age we conducted logistic regression to allow adjustment for this. In particular, for each of the 9 major occupational groups (based on last reported occupation), we calculated prevalence odds ratios (OR) and 95% confidence intervals[323] for deprivation (dichotomised), obesity (dichotomised), ever smoking, high blood pressure, diabetes and high cholesterol, one factor at a time. Analyses were adjusted for age group at interview. For each specific occupational group, all other occupation groups were used as the reference group. We also conducted analyses using a fixed reference group (i.e. NZSCO 2: *professionals*) and the results were very similar (data not shown), indicating that the analysis is robust and independent of the method used. As similar studies used all other groups/not working in an occupational group as reference groups,[13, 97] we presented results for this method. The same analyses were conducted for the two-digit sub-major occupational groups in the NZSCO hierarchical classification. These results are presented in Supplementary Tables S4.5 – S4.9.

As the Māori and general population surveys were conducted at different time periods, we did not formally test whether statistical differences existed between surveys; instead, where pronounced differences were observed, this is reported in the results without providing p-values.

In compliance with the confidentiality requirements of Statistics New Zealand's IDI, all frequencies have been rounded to the nearest multiple of three and percentages have been calculated from the rounded counts (hence the number of total participants in each table may vary slightly from the total number listed above and percentages may not add up to exactly 100%). The statistical tests were performed on the unrounded counts and have been presented in their unmodified form. All counts under six and the ORs derived from these are suppressed.

4.3 Results

Population characteristics

The prevalence of CVD risk factors was higher in the Māori survey; participants were older and were more likely to be living in neighbourhoods of high deprivation, have ever smoked and be in the obese BMI category (>30Kg/m^2). Participants in the Māori survey also had a higher prevalence of metabolic risk factors such as diabetes, high blood pressure and high cholesterol (Table 1). Furthermore, the prevalence of these risk factors was significantly greater in males than females in both surveys, with the exception of high blood pressure in Māori (Chi-squared tests; $p< 0.05$). In the NZWS survey, participants were more likely to work in white-collar occupations, whereas in the Māori NZWS, they were more likely to work in blue-collar occupations (Table 4.1).

Socio-demographic and behavioural risk factors

In general, across all cohorts, ORs were higher for the blue-collar occupational groups than the white-collar occupational groups (Table 4.2). *Plant and machine operators and assemblers* were more deprived and obese and were more likely to have ever smoked; this was particularly the case in males (ORs ranging from 1.41 to 2.38 across these risk factors in the males; p<0.05). *Elementary workers* were consistently more deprived (ORs ranging from 1.69 to 3.87; p<0.05) and female *elementary workers* were more likely to have ever smoked (ORs ranging from 2.44 to 2.99; p<0.01); NZWS females in this group were also more likely to be obese (OR 2.01; p<0.05).

Professionals across all groups were less deprived (ORs ranged from 0.43 to 0.68, p<0.05) and less likely to have ever smoked (ORs ranging from 0.45 to 0.65, p<0.05), and males in the NZWS were less obese (OR 0.51; p<0.01). Both NZWS males and Māori females in the *legislators, administrators, and managers* group were less likely to live in neighbourhoods of high deprivation (males OR 0.50, females OR 0.53; p<0.01). In contrast to the other cohorts, female NZWS *clerks* were less likely to be deprived (OR 0.62; p<0.05), whereas *service and sales workers* were more likely to be deprived (OR 1.67; p<0.01). Māori male *trades workers* were less obese (OR 0.65; p<0.05).

Table 4.1. Population characteristics

	NZWS			Māori NZWS		
	N=2970			N=2070		
	Male	Female		Male	Female	
	1407 (47.3%)	1560 (52.5%)	P[c]	927 (44.8%)	1143 (55.2%)	P[c]
	n (%)	n (%)		n (%)	n (%)	
Age at interview			<0.01			0.72
20-34	282 (20.0)	339 (21.7)		159 (17.2)	210 (18.5)	
35-44	336 (23.9)	459 (29.4)		207 (22.4)	255 (22.4)	
45-54	402 (28.6)	471 (30.2)		219 (23.7)	279 (24.5)	
55+	393 (27.0)	291 (18.7)		339 (36.7)	387 (34.0)	
Missing				S	6	
Ethnicity			<0.01			
Pākehā	1125 (80.3)	1233 (79.0)				
Māori	108 (7.7)	165 (10.6)		927 (100.0)	1143 (100.0)	
Pacific peoples	21 (1.5)	33 (2.1)				
Other	150 (10.7)	129 (8.3)				
Missing	S					
Deprivation Index 2006			<0.01			0.10
1-2 (least deprived)	405 (28.8)	381 (24.4)		138 (14.9)	141 (12.3)	
3-4	318 (22.6)	333 (21.3)		147 (15.9)	162 (14.2)	
5-6	288 (20.5)	363 (23.3)		162 (17.5)	204 (17.8)	
7-8	228 (16.2)	291 (18.7)		219 (23.7)	264 (23.1)	
9-10 (most deprived)	171 (12.2)	192 (12.3)		258 (27.9)	372 (32.5)	
Missing		S		S	S	
Smoking						
Ever	714 (50.7)	753 (48.3)	0.23	567 (61.2)	771 (67.5)	<0.01
Current	252 (17.9)	288 (18.5)	0.22	219 (23.6)	348 (30.4)	0.02
Missing	S	6		S	S	
Body Mass Index (kg/m²)			<0.01			<0.01
<18.5	6 (0.4)	30 (2.0)		S	21 (20.1)	
18.5-24.9	477 (34.5)	726 (49.2)		147 (16.4)	306 (30.2)	
25-29.9	633 (45.8)	438 (29.6)		345 (38.6)	270 (26.6)	
30+	270 (19.5)	279 (18.9)		396 (44.3)	417 (41.1)	
Missing	24	84		33	129	
Diabetes Ever						
	153 (10.9)	105 (6.7)	<0.01	180 (19.4)	177 (15.5)	0.03
High blood pressure[a] Ever						
	507 (36.0)	495 (31.7)	<0.01	381 (41.1)	444 (38.8)	0.38
High cholesterol[b] Ever						
	453 (32.2)	288 (18.5)	<0.01	345 (37.2)	312 (27.3)	<0.01
Occupation (NZSCO 1-digit code)			<0.01			<0.01
1. Legislators, admin. & managers	276 (19.7)	171 (11.0)		99 (10.8)	129 (11.4)	
2. Professionals	231 (16.5)	390 (25.0)		111 (12.1)	219 (19.4)	
3. Technicians & assoc. profs.	180 (12.8)	276 (17.7)		105 (11.4)	198 (17.5)	
4. Clerks	66 (4.7)	282 18.1)		42 (4.6)	171 (15.1)	
5. Service & sales workers	87 (6.2)	273 (17.5)		81 (8.8)	225 (19.9)	
6. Agricultural & forestry workers	123 (8.8)	60 (4.3)		75 (8.2)	42 (3.7)	
7. Trades workers	234 (16.7)	18 (1.2)		126 (13.7)	12 (1.1)	
8. Plant & machine operators & assemblers	153 (10.9)	33 (2.1)		213 (23.2)	57 (5.0)	
9. Elementary occupations	60 (4.3)	57 (3.7)		69 (7.5)	72 (6.4)	
Missing	S			9	12	

Following IDI protocols, all frequencies have been rounded to the nearest multiple of three and percentages calculated from the rounded counts. (S = suppressed)

[a]Diagnosed with high blood pressure or received anti-hypertensive medication at any time over the available date range of the data.

[b]Diagnosed with high cholesterol or received cholesterol management medication at any time over the available date range of the data.

[c]Comparing males and females within each cohort.

Table 4.2. Associations between current occupation and conventional cardiovascular risk factors

Occupation (NZSCO 1-digit code)	Deprivation Index 9/10		Obese (>30kg m²)		Ever Smoked		High Blood Pressure[a]		Diabetes		High Cholesterol[b]	
	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)
a) NZWS males	**12.2**	**n=1407**	**19.5**	**n = 1386**	**50.7**	**n = 1404**	**36.0**	**n = 1410**	**10.9**	**n = 1410**	**32.2**	**n = 1410**
1. Legislators, Admin. & Managers (n=276)	6.6	0.50 (0.30-0.82)**	20.9	1.12 (0.81-1.56)	47.8	0.90 (0.69-1.17)	36.7	1.06 (0.79-1.43)	11.0	1.08 (0.70-1.68)	45.1	1.27 (0.94-1.72)
2. Professionals (n=231)	9.0	0.68 (0.42-1.11)	12.0	0.51 (0.33-0.77)**	41.6	0.64 (0.48-0.85)**	34.6	0.87 (0.63-1.20)	7.7	0.64 (0.39-1.08)	31.2	0.87 (0.63-1.21)
3. Technicians & Assoc. Professionals (n=180)	11.7	1.00 (0.62-1.61)	20.0	1.06 (0.71-1.56)	49.2	0.91 (0.67-1.25)	33.3	0.82 (0.57-1.19)	10.0	0.85 (0.49-1.46)	30.0	0.91 (0.63-1.32)
4. Clerks (n=66)	13.6	1.23 (0.61-2.47)	18.2	0.94 (0.49-1.78)	52.2	1.00 (0.61-1.65)	54.5	2.22 (1.26-3.91)**	13.6	1.32 (0.64-2.74)	36.4	1.11 (0.62-1.98)
5. Service & Sales Workers (n=87)	17.2	1.52 (0.85-2.73)	21.4	1.07 (0.62-1.86)	51.7	1.07 (0.69-1.65)	27.6	0.72 (0.43-1.23)	6.9	0.93 (0.43-2.02)	23.3	0.66 (0.38-1.16)
6. Agriculture & Fishery Workers (n=123)	7.3	0.49 (0.23-1.02)	12.5	0.63 (0.37-1.07)	53.7	1.03 (0.71-1.50)	34.1	0.74 (0.48-1.12)	7.3	0.52 (0.25-1.07)	27.5	0.61 (0.39-0.96)*
7. Trades Workers (n=234)	12.8	1.03 (0.67-1.58)	16.0	0.82 (0.56-1.20)	55.1	1.29 (0.97-1.72)	33.3	1.00 (0.73-1.39)	11.4	1.14 (0.71-1.83)	29.5	0.98 (0.70-1.38)
8. Plant & Machine Operators & Assemblers (n=153)	20.0	2.22 (1.44-3.42)**	34.7	2.38 (1.64-3.45)**	60.8	1.58 (1.12-2.23)**	43.1	1.34 (0.92-1.94)	17.3	1.68 (1.03-2.73)*	36.0	1.14 (0.78-1.67)
9. Elementary Occupations (n=60)	25.0	2.33 (1.25-4.36)**	25.0	1.39 (0.74-2.58)	52.6	1.08 (0.64-1.83)	40.0	1.19 (0.66-2.14)	15.0	1.59 (0.74-3.43)	47.6	2.36 (1.30-4.28)**
b) Māori NZWS Males	**27.9**	**n = 921**	**44.3**	**n = 891**	**61.2**	**n = 918**	**41.1**	**n = 918**	**19.4**	**n = 918**	**41.9**	**n = 918**
1. Legislators, Admin. and Managers (n=99)	23.5	0.70 (0.43-1.15)	42.4	0.86 (0.56-1.32)	59.4	0.90 (0.59-1.39)	39.4	0.82 (0.50-1.34)	21.2	0.98 (0.58-1.68)	44.1	1.24 (0.76-2.00)
2. Professionals (n=111)	13.9	0.43 (0.25-0.74)**	40.0	0.84 (0.56-1.28)	43.2	0.45 (0.30-0.68)**	37.8	0.92 (0.57-1.48)	16.2	0.83 (0.48-1.44)	32.4	0.80 (0.49-1.30)
3. Technicians & Assoc. Professionals (n=105)	20.6	0.71 (0.44-1.16)	37.5	0.76 (0.49-1.16)	60.0	0.93 (0.61-1.42)	38.2	0.81 (0.49-1.32)	14.3	0.61 (0.33-1.12)	31.4	0.72 (0.44-1.19)
4. Clerks (n=42)	30.8	1.16 (0.58-2.34)	42.9	1.07 (0.56-2.06)	57.1	0.85 (0.52-1.30)	35.7	1.51 (0.69-3.29)	15.4	0.99 (0.39-2.49)	30.8	1.28 (0.55-2.99)
5. Service & Sales Workers (n=81)	28.6	1.03 (0.62-1.71)	51.9	1.49 (0.93-2.37)	55.6	0.82 (0.52-1.30)	35.7	0.86 (0.49-1.49)	25.0	1.47 (0.83-2.59)	35.7	1.11 (0.64-1.94)
6. Agriculture & Fishery Workers (n=75)	28.0	1.00 (0.59-1.69)	37.5	0.76 (0.46-1.25)	60.0	0.81 (0.50-1.31)	48.0	1.03 (0.59-1.30)	20.0	0.81 (0.43-1.51)	46.2	1.31 (0.75-2.28)
7. Trades Workers (n=126)	28.6	1.09 (0.72-1.65)	35.7	0.65 (0.44-0.97)*	64.3	1.16 (0.78-1.71)	35.7	0.83 (0.53-1.30)	14.0	0.67 (0.39-1.15)	23.4	0.42 (0.26-0.68)**
8. Plant & Machine Operators & Assemblers (n=213)	35.2	1.55 (1.11-2.16)**	52.2	1.41 (1.02-1.94)*	74.6	2.09 (1.48-2.95)**	47.9	1.23 (0.86-1.76)	24.3	1.31 (0.89-1.92)	44.3	1.28 (0.90-1.83)
9. Elementary Occupations (n=69)	39.1	1.69 (1.01-2.83)*	54.5	1.63 (0.98-2.71)	60.9	1.07 (0.64-1.78)	43.5	1.51 (0.84-2.72)	27.3	1.75 (0.96-3.16)	45.8	2.00 (1.09-3.67)*

*P-value < 0.05 ** P-value < 0.01 for the separate logistic regression model for the risk factor represented by each column – models are adjusted for age and compare each occupational group with all other occupational groups.
Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The odds ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)
[a]Diagnosed with high blood pressure or received anti-hypertensive medication at any time over the available date range of the data.
[b]Diagnosed with high cholesterol or received cholesterol management medication at any time over the available date range of the data.
9 Māori NZWS male participants were excluded as they could not be assigned to an occupational group (unemployed, a student, a housewife/husband or did not answer). Less than 6 participants were excluded from the NZWS males (as per IDI rules, the precise number cannot be released).

Occupation (NZSCO 1-digit code)	Deprivation Index 9/10		Obese (>30kg m²)		Ever Smoked		High Blood Pressure[a]		Diabetes		High Cholesterol[b]	
	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)
c) NZWS females	**12.3**	**n = 1557**	**18.9**	**n = 1473**	**48.3**	**n = 1551**	**31.7**	**n = 1557**	**6.7**	**n = 1557**	**18.5**	**n = 1557**
1. Legislators, Admin. & Managers (n=171)	12.1	0.92 (0.56-1.51)	16.7	0.81 (0.52-1.27)	42.1	0.76 (0.55-1.05)	28.1	0.82 (0.56-1.19)	5.3	0.86 (0.44-1.69)	19.0	0.99 (0.64-1.54)
2. Professionals (n=390)	9.2	0.63 (0.43-0.93)*	18.5	0.99 (0.73-1.34)	40.8	0.65 (0.52-0.83)**	27.7	0.75 (0.57-0.98)*	6.2	0.97 (0.61-1.55)	16.9	0.83 (0.60-1.15)
3. Technicians & Assoc. Professionals (n=276)	12.0	0.94 (0.63-1.40)	15.7	0.79 (0.55-1.14)	47.8	0.97 (0.75-1.26)	27.2	0.83 (0.61-1.13)	4.3	0.70 (0.38-1.27)	13.0	0.72 (0.48-1.07)
4. Clerks (n=282)	8.5	0.62 (0.40-0.97)*	21.6	1.25 (0.90-1.74)	52.1	1.19 (0.92-1.54)	37.9	1.47 (1.10-1.95)**	7.4	1.14 (0.69-1.89)	21.3	1.31 (0.93-1.85)
5. Service & Sales Workers (n=273)	17.6	1.67 (1.16-2.38)**	19.0	0.99 (0.70-1.41)	52.8	1.20 (0.92-1.56)	34.1	1.09 (0.81-1.47)	6.6	0.95 (0.56-1.62)	18.7	0.94 (0.66-1.36)
6. Agriculture & Fishery Workers (n=60)	S	S	15.8	0.77 (0.37-1.59)	47.6	1.03 (0.62-1.72)	35.0	1.04 (0.59-1.84)	S	S	23.8	0.99 (0.51-1.94)
7. Trades Workers (n=18)	20.0	1.70 (0.69-4.21)	S	S	S	S	33.3	1.34 (0.47-3.81)	S	S	S	S
8. Plant & Machine Operators & Assemblers (n=33)	18.2	1.70 (0.69-4.21)	22.2	1.24 (0.52-2.94)	60.0	1.85 (0.88-3.93)	40.0	1.47 (0.67-3.24)	S	S	20.0	0.91 (0.34-2.42)
9. Elementary Occupations (n=57)	31.6	3.87 (2.17-6.90)**	33.3	2.01 (1.10-3.65)*	73.7	2.99 (1.64-5.45)**	42.1	1.50 (0.83-2.72)	16.7	2.60 (1.21-5.57)*	36.8	2.78 (1.47-5.26)**
d) Māori NZWS females	**32.5**	**n = 1128**	**41.1**	**n = 1005**	**67.5**	**n = 1128**	**38.8**	**n = 1131**	**15.5**	**n = 1131**	**27.3**	**n = 1131**
1. Legislators, Admin. and Managers (n=129)	21.4	0.53 (0.34-0.83)**	30.0	0.56 (0.37-0.85)*	66.7	0.94 (0.63-1.39)	37.2	0.71 (0.46-1.09)	11.6	0.59 (0.33-1.06)	27.3	0.77 (0.48-1.23)
2. Professionals (n=219)	26.0	0.67 (0.48-0.93)*	38.2	0.90 (0.66-1.24)	50.7	0.42 (0.31-0.57)**	35.1	0.84 (0.59-1.20)	16.2	1.17 (0.77-1.78)	21.9	0.76 (0.51-1.14)
3. Technicians & Assoc. Professionals ((n=198)	29.9	0.88 (0.63-1.23)	39.0	0.91 (0.65-1.27)	65.0	0.89 (0.64-1.23)	33.8	0.75 (0.52-1.08)	10.9	0.81 (0.51-1.28)	27.3	1.15 (0.78-1.71)
4. Clerks (n=171)	32.8	0.96 (0.68-1.36)	43.0	1.12 (0.79-1.59)	64.0	0.84 (0.60-1.18)	41.4	1.32 (0.90-1.95)	13.8	0.84 (0.51-1.36)	24.6	0.88 (0.57-1.36)
5. Service & Sales Workers (n=225)	37.8	1.37 (1.01-1.86)*	47.0	1.33 (0.97-1.83)	75.0	1.56 (1.12-2.18)**	44.0	1.26 (0.89-1.79)	20.0	1.39 (0.94-2.07)	32.0	1.35 (0.93-1.96)
6. Agriculture & Fishery Workers (n=42)	30.8	1.09 (0.56-2.11)	42.0	0.94 (0.47-1.87)	79.0	1.79 (0.84-3.80)	42.6	1.19 (0.57-2.48)	20.0	1.07 (0.45-2.54)	35.7	1.43 (0.66-3.11)
7. Trades Workers (n=12)	50.0	2.14 (0.68-6.71)	S	S	S	S	S	S	S	S	S	S
8. Plant & Machine Operators & Assemblers (n=57)	36.8	1.40 (0.81-2.40)	56.0	1.75 (0.99-3.09)	85.0	3.18 (1.49-6.79)**	47.4	1.35 (0.74-2.46)	20.0	1.18 (0.58-2.36)	30.0	1.08 (0.57-2.07)
9. Elementary Occupations (n=72)	54.2	2.49 (1.54-4.03)**	48.0	1.18 (0.70-1.97)	83.0	2.44 (1.29-4.59)**	45.8	1.08 (0.63-1.88)	20.0	1.20 (0.64-2.25)	28.0	0.96 (0.53-1.74)

*P-value < 0.05 ** P-value < 0.01 for the separate logistic regression model for the risk factor represented by each column – models are adjusted for age and compare each occupational group with all other occupational groups.
Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The odds ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)
[a]Diagnosed with high blood pressure or received anti-hypertensive medication at any time over the available date range of the data.
[b]Diagnosed with high cholesterol or received cholesterol management medication at any time over the available date range of the data.
12 Māori NZWS female participants were excluded as they could not be assigned to an occupational group (unemployed, a student, a housewife/husband or did not answer).

Metabolic risk factors

Similar to the socio-demographic and behavioural risk factors, ORs were also higher for metabolic risk factors in the blue-collar compared to the white-collar occupational groups, although less marked (Table 4.2). *Plant and machine operators and assemblers* in the NZWS males were more likely to have diabetes (OR 1.68, p<0.05), while all *elementary workers*, except Māori females, were more likely to have high cholesterol (ORs ranging from 2.00 to 2.78; p<0.05). Female *elementary workers* in the NZWS also had more diabetes (OR 2.60; p<0.01). NZWS females who worked as *professionals* were less likely to have high blood pressure (OR 0.75; p<0.05), whereas *clerks* were more likely to experience high blood pressure observed in both NZWS males (OR 2.22; p<0.01) and females (OR 1.47; p<0.01). Interestingly, the NZWS male *agricultural workers* and Māori male *trades workers* were less likely to have high cholesterol (OR 0.61; p<0.05 and OR 0.42; p<0.05, respectively

4.4 Discussion

This study, which aimed to characterise the distribution of traditional CVD risk factors across occupational groups, found that *plant and machine operators and assemblers* were more likely than other occupational groups to experience CVD risk factors, including diabetes, high cholesterol, smoking and obesity, whereas, *professionals* were less likely to experience these risk factors. There were some notable ethnic and sex differences, as Māori and males had a greater prevalence of risk factors in comparison to the general population and females, respectively.

In general, our study showed that WCW had fewer CVD risk factors than BCW, which is consistent with the literature, particularly for females.[14] Previous research has shown that BCW are more likely to develop high blood pressure [107, 112] and are also more likely to smoke.[99] Most BCW also have lower SES, which may in part contribute to the higher burden of CVD risk factors. However, adjustment for deprivation (both dichotomous and by NZDep2006 quintiles) only slightly attenuated the results (data not shown), indicating that other factors may be more important. Blue-collar occupations are also often associated with multiple occupational exposures, which may explain, at least in part, the higher prevalence of CVD risk factors in this group. Previous research has linked occupational exposures, such as physical exertion, to an increased burden of CVD risk factors in men [257] e.g. heavy lifting can elevate blood pressure, particularly in those with low fitness levels.[262, 266] Similarly, chemical exposures (e.g. lead) and loud noise have been associated with CVD risk factors such as high blood pressure.[281, 324]

Plant and machine operators and assemblers were most likely to experience CVD risk factors, particularly in males. This is largely driven by specific subgroups, including *drivers and mobile machinery operators* (Supplementary Tables S4.5 – S4.9), which is consistent with previous research that has shown that drivers have an elevated risk of CVD and risk factors such as obesity.[10, 97, 325] Occupational exposures including job stress, irregular schedules, sedentary work and unhealthy dietary patterns may play a role as these are highly prevalent in these subgroups and have all been associated with CVD and CVD risk factors.[20, 326-328]

Within the females, *elementary workers* had the highest risk factor burden. Although there is an overall lack of research in female BCW and CVD, these results support a recent systematic review concluding that female BCW have poorer health than female WCW or blue-collar males.[14] This may at least in part be due to females being exposed to additional occupational hazards e.g. they are more likely to work with equipment designed for males, and they may be subject to gender discrimination, social isolation and sexual harassment. [14] These exposures are prevalent in *elementary workers* and hence may also contribute to the observed greater risk of experiencing CVD risk factors in females in this group.

Male *agricultural and fishery workers* in the general population were less likely to have high cholesterol and several other CVD risk factors, but the same was not found for females or Māori males. The reasons for this are unclear but may be related to the fact that >60% of general males in the agricultural and fishery workers work in the minor group NZSCO code 612 (*market-oriented animal producers*), suggesting that farmers make up a large proportion of this group. In contrast, only 45% of Māori males, 34% of Māori females and 55% of general females work in this minor occupational group. As farmers and agricultural workers have been shown to have healthier and more traditional dietary patterns compared to other occupations, this may explain some of these differences.[100]

Of the white-collar occupations, *professionals* were less likely to experience CVD risk factors. The evidence for white-collar occupations has been conflicting, with some research showing increased exposure to metabolic risk factors in professionals compared to unskilled workers.[90, 107] However, in contrast, female managers have been shown to have a favourable risk factor profile and the prevalence of type 2 diabetes is lowest among professionals.[100, 111] The higher SES of these occupations may also account for a more favourable CVD risk factor profile,[40] however, adjustment of SES only slightly attenuated the results. Our study showed that the pattern is not identical across all white-collar occupational groups, suggesting that conflicting results reported previously may be due to the heterogeneity of occupations within this group of occupations.

In contrast to *professionals*, *clerks* in our study were more likely to have high blood pressure. Office workers have previously been shown to be more likely to experience metabolic risk factors, such as high blood pressure, and this association may be mediated by a lack of physical activity in these occupations.[102, 253, 321] Additionally, job strain (defined as low job control and high demand) [130] and effort-reward imbalance [131] have been associated with cardiovascular risk factors, such as diabetes, high blood pressure and smoking.[153, 154] Clerical work may have a particularly high exposure to these psychosocial factors as suggested by our survey data, which shows that *clerks* were less satisfied with the enjoyment of their work (results not shown) and as has previously been shown, low-status office workers have an elevated risk of CVD risk factors.[13]

Māori are more likely to experience CVD risk factors than European New Zealanders.[39, 80] Our research confirms this as the prevalence of all risk factors was substantially higher in Māori compared to the general population. In addition, we have previously shown that Māori may be more exposed to hazardous occupational exposures, even when compared with non-Māori within the same occupational groups.[26] Combined, this may contribute to the higher overall risk of CVD in Māori.[80, 329] The increased exposure included physical exposures, such as lifting, which may, at least in part, be an explanation for why male Māori trades workers are less likely to have high cholesterol or be obese. This may appear paradoxical, given that physical exertion is associated with an increased risk of experiencing CVD risk factors (see above), however, research suggests the differential effects of physical activity are potentially due to a U-shaped dose-response effect, with both a lack of exercise and too much exercise (exertion) resulting in adverse health effects including lipid dysregulation, weight gain [22] and increased blood pressure.[266] Māori trades workers may have elevated exposures to lifting but this may only be moderate and not equate to exertion.

It is well established that males have a higher incidence of CVD than similar aged pre-menopausal women, however, direct comparisons of the burden of risk factors are limited. Our results are consistent with previous literature showing that males have a higher prevalence of CVD risk factors than females.[28] Sex-specific differences in occupational exposures due to differences in the distribution of tasks and occupations within an occupational group may contribute to these stark differences.[27] Differences in biological susceptibility to similar exposures may be another reason for differences in metabolic risk factors.[14]

Our study had several limitations. Due to the cross-sectional nature of the workforce surveys, lifestyle and occupation information (e.g. smoking status, BMI and deprivation) was only collected at one point in time. Additionally, the measurement of BMI is likely affected by measurement error as it was self-reported. However, there is no direct evidence suggesting that reporting bias differs across occupational groups, and although more misreporting has been associated with lower SES,[330] adjustment for deprivation only slightly attenuated the obesity results.

Our survey sample sizes meant only results for the 9 major occupational groups are presented. Although a limitation, this is comparable to the number of occupational groups used in other studies in this area.[97] To illustrate the heterogeneity within some major groups we also included results for 2-digit occupational groups (Supplementary Tables S4.5 – S4.9). For example, NZSCO group 5 (i.e. *service and sales workers*) includes both waiters and firefighters with very different exposure patterns.

As we assessed 9 occupational groups across males and females of both surveys many comparisons were made, which may result in some false-positive results. In fact, there were 216 statistical tests in total, with 26 significant at <1% and 39 at <5%. Therefore, the expected number of false positives is 2 and 11, respectively. So, even allowing for the multiple testing required due to the exploratory nature of the study, results presented at <1% for a single test are reliable. Those presented as significant at <5% but not <1% should be interpreted with more caution. The principal focus of the paper is consequently on results of the individual tests that are significant at <1%.

The low response rates of the surveys are a potential source of bias. However, in a detailed evaluation of the survey response, no evidence of major non-response bias was found.[26, 301] While some groups were underrepresented in the survey sample, the prevalence of key survey variables (both occupational exposure and health-related variables) were unchanged after standardising to the demographic distribution of the source population, and similar between early and late responses.

In contrast to the cross-sectional nature of the workforce survey, the health information obtained from the IDI was longitudinal, but the available date ranges varied somewhat across various datasets. Also, not all relevant health data is included in the IDI, meaning primary healthcare information could not be included in our definitions of CVD risk factors. In addition, anti-hypertensive and cholesterol management medication can also be used to manage conditions other than those of interest; therefore our results may include an overrepresentation of high blood pressure and high cholesterol, although we do not expect this to differ proportionately between occupational groups.

Another limitation is that the original two surveys were conducted at different time points. As a result, comparisons between both surveys should be interpreted with caution, particularly for risk factors assessed by questionnaire (obesity, smoking and deprivation) as the population-prevalence for these factors may have changed between the time that the initial NZWS and the Māori NZWS were conducted. However, differences in prevalence are likely small as surveys were conducted only 5 years apart. Also, the same limitation does not apply for diabetes, high blood pressure and high cholesterol, as this was assessed using routinely collected data, with the period of assessment for each risk factor being the same for both surveys (and prevalence measured as experiencing any of these events at any point in time throughout this assessment period). The period of assessment of each risk factor (e.g. diabetes and high blood pressure) differed somewhat, but that is not an issue as analyses were presented for each risk factor separately. Nonetheless, it means that comparisons of results across risk factors should be interpreted with caution.

A strength and novel aspect of this study is the linkage between detailed survey data and routinely collected objective CVD risk factor data (as opposed to relying on subjective self-reports). Our sample also consisted of over 50% female and a large proportion (~40%) of Māori, allowing assessment of understudied females, as well as undertaking the first study in Māori on CVD risk factors in different occupational groups.

In conclusion, our results highlight differences in the distribution of conventional CVD risk factors across occupational groups, with notable ethnic and sex differences. In conjunction with previous research evaluating workplace-based health promotion initiatives both in New Zealand [85, 331] and internationally,[37, 332] the data presented may guide effective interventions towards minimising CVD risk factors in high-risk occupations.

4.5 Supplementary material

Table S4.3. IDI datasets

Dataset	Contents	Date range of available data
Mortality data	Mortality	1988- Dec 2015
National Minimum Dataset (NMDS)	Publicly funded hospital events: diagnosis and procedures	1988- Jun 30th 2017
Pharmaceutical data	Pharmaceutical claims	2005- Dec 31st 2017
National Non-Admitted Patient Collection (NNPAC)	Non-admitted secondary care events, such as outpatient visits	2007- June 30th 2017
Laboratory claims collection	Claim and payment information for community laboratory tests	2003- June 30th 2017

Table S4.4. Definitions of health conditions

Condition		ICD Code or Criteria
High blood pressure	Malignant essential hypertension	ICD-9-AM 401.0
	Benign essential hypertension	ICD-9-AM 401.1
	Unspecified essential hypertension	ICD-9-AM 401.9
	Essential (primary) hypertension	ICD-10-AM I10
	Hypertensive heart disease	ICD-9-AM 402, ICD-10-AM I11
	Hypertensive chronic kidney disease	ICD-9-AM 403/4, ICD-10-AM I12/3
	Pharmaceuticals	≥2 dispensings in a 12 month period
High Cholesterol	Pure hypercholesterolemia	ICD-9-AM 272.0, ICD-10-AM E780
	Hyperlipidaemia, unspecified	ICD-9-AM 272.4, ICD-10-AM E785
	Pharmaceuticals	≥2 dispensings in a 12 month period
Diabetes Mellitus	Type 1 diabetes mellitus	ICD-10-AM E10
	Type 2 diabetes mellitus	ICD-10-AM E11
	Other specified diabetes mellitus	ICD-10-AM E13
	Unspecified diabetes mellitus	ICD-10-AM E14
	Pre-existing diabetes mellitus, Type 1 in pregnancy	ICD-10-AM O24.0
	Pre-existing diabetes mellitus, Type 2 in pregnancy	ICD-10-AM O24.1
	Pre-existing diabetes mellitus, other specified type, in pregnancy	ICD-10-AM O24.2
	Pre-existing diabetes mellitus, unspecified, in pregnancy.	ICD-10-AM O24.3
	Pharmaceuticals	≥2 dispensings for all subsidised forms of insulin, oral hypoglycaemics, and glucagon within 2 years. (if only metformin use, must meet other criteria)
	Laboratory claims	≥4 requests for HbA1c and two or more requests for Albumin Creatinine Ratio within 2 years.
	Non-admitted secondary care events, such as outpatient visits	NNPAC events that contain any purchase unit code for Diabetes education and management or Diabetes retinal screening.
	Mortality	Diabetes as the cause of death

Table S4.5. NZSCO two-digit job titles

OE_11	Legislators and Administrators
OE_12	Corporate Managers
OE_21	Physical, Mathematical and Engineering Science Professionals
OE_22	Life Science and Health Professionals
OE_23	Teaching Professionals
OE_24	Other Professionals
OE_31	Physical Science and Engineering Associate Professionals
OE_32	Life Science and Health Associate Professionals
OE_33	Other Associate Professionals
OE_41	Office Clerks
OE_42	Customer Services Clerks
OE_51	Personal and Protective Services Workers
OE_52	Salespersons, Demonstrators and Models
OE_61	Market-Oriented Agricultural and Fishery Workers
OE_71	Building Trades Workers
OE_72	Metal and Machinery Trades Workers
OE_73	Precision Trades Workers
OE_74	Other Craft and Related Trades Workers
OE_81	Industrial Plant Operators
OE_82	Stationary Machine Operators and Assemblers
OE_83	Drivers and Mobile Machinery Operators
OE_84	Building and Related Workers
OE_91	Labourers and Related Elementary Service

Table S4.6. NZWS Males - NZSCO two-digit logistic regression

	N	Deprivation Index 9/10 n=1407		Obese (>30kg m^{2}) n = 1386		Ever Smoked n = 1404		High blood pressure[a] n = 1410		Diabetes n = 1410		High Cholesterol[b] n = 1410	
		%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)
OE_11	21	S	S	S	S	42.9	0.59 (0.25-1.43)	S	S	S	S	S	S
OE_12	252	6.0	0.48 (0.29-0.81)*	21.4	1.21 (0.86-1.69)	48.8	0.94 (0.71-1.24)	37.7	1.12 (0.83-1.53)	10.7	1.14 (0.73-1.79)	36.9	1.37 (1.00-1.87)
OE_21	93	6.7	0.68 (0.32-1.43)	6.5	0.39 (0.19-0.82)*	41.9	0.69 (0.45-1.07)	34.4	0.94 (0.58-1.52)	9.7	0.88 (0.42-1.82)	29.0	0.73 (0.44-1.23)
OE_22	27	S	S	22.2	1.27 (0.50-3.22)	33.3	0.76 (0.34-1.67)	29.6	0.94 (0.38-2.31)	S	S	44.4	1.83 (0.76-4.38)
OE_23	54	11.1	1.04 (0.46-2.34)	11.1	0.60 (0.27-1.35)	44.4	0.70 (0.40-1.20)	44.4	1.21 (0.67-2.21)	S	S	33.3	1.00 (0.54-1.85)
OE_24	60	S	S	10.0	0.45 (0.19-1.06)	35.0	0.59 (0.34-1.01)	28.3	0.61 (0.33-1.13)	10.0	1.02 (0.45-2.36)	30.0	0.75 (0.40-1.39)
OE_31	63	S	S	14.3	0.71 (0.34-1.46)	42.9	0.82 (0.49-1.37)	30.2	0.79 (0.43-1.43)	S	S	28.6	0.96 (0.52-1.77)
OE_32	9	S	S	S	S	S	S	S	S	S	S	S	S
OE_33	114	16.2	1.30 (0.75-2.23)	23.7	1.31 (0.82-2.08)	47.4	0.96 (0.65-1.41)	32.5	0.89 (0.57-1.40)	10.5	1.11 (0.59-2.07)	31.6	0.94 (0.59-1.49)
OE_41	54	16.7	1.18 (0.54-2.54)	11.1	0.62 (0.27-1.39)	55.6	0.93 (0.54-1.61))	53.7	2.24 (1.19-4.21)*	16.7	1.23 (0.55-2.75)	38.9	1.10 (0.58-2.08)
OE_42	12	S	S	S	S	50.0	1.37 (0.76-3.92)	50.0	2.18 (0.61-7.85)	S	S	S	S
OE_51	51	11.8	1.10 (0.49-2.49)	23.5	1.53 (0.82-2.87)	47.1	0.81 (0.46-1.41)	33.3	0.84 (0.44-1.58)	11.8	1.11 (0.45-2.72)	23.5	0.72 (0.37-1.41)
OE_52	33	27.3	2.19 (0.97-4.96)	S	S	63.6	1.65 (0.82-3.35)	21.3	0.58 (0.23-1.46)	S	S	18.2	0.58 (0.22-1.54)
OE_61	123	5.0	0.49 (0.23-1.02)	14.6	0.63 (0.37-1.07)	51.2	1.03 (0.71-1.50)	33.3	0.71 (0.47-1.09)	7.3	0.52 (0.25-1.07)	29.2	0.61 (0.39-0.96)*
OE_71	120	14.6	1.12 (0.64-1.95)	12.5	0.60 (0.34-1.06)	57.5	1.39 (0.95-2.03)	33.3	1.10 (0.71-1.69)	10.0	1.11 (0.58-2.11)	30.0	1.08 (0.69-1.70)
OE_72	75	8.0	0.73 (0.33-1.62)	16.0	0.82 (0.43-1.55)	52.0	1.18 (0.74-1.88)	32.0	0.99 (0.58-1.70)	8.0	1.02 (0.45-2.34)	28.0	0.96 (0.55-1.70)
OE_73	12	S	S	S	S	S	S	S	S	S	S	S	S
OE_74	27	25.0	2.21 (0.87-5.61)	33.3	3.02 (1.36-6.67)**	66.7	1.73 (0.76-3.92)	33.3	0.71 (0.29-1.70)	22.2	2.10 (0.79-5.57)	33.3	1.03 (0.43-2.46)
OE_81	24	S	S	50.0	3.79 (1.59-9.06)**	62.5	2.89 (1.13-7.41)*	29.2	0.73 (0.28-1.88)	S	S	25.0	0.70 (0.26-1.88)
OE_82	51	17.6	1.88 (0.92-3.85)	29.4	1.58 (0.84-2.97)	58.8	1.26 (0.71-2.23)	49.0	1.56 (0.85-2.86)	11.8	0.95 (0.39-2.33)	47.1	1.57 (0.85-2.90)
OE_83	69	22.7	2.37 (1.32-4.26)**	34.8	2.37 (1.41-3.98)**	60.9	1.52 (0.93-2.51)	44.9	1.51 (0.88-2.59)	21.7	2.21 (1.16-4.22)*	30.4	1.01 (0.57-1.77)
OE_84	12	S	S	S	S	S	S	S	S	S	S	S	S
OE_91	60	21.1	2.33 (1.25-4.36)*	25.0	1.39 (0.74-2.58)	50.0	1.08 (0.64-1.83)	38.3	1.21 (0.67-2.18)	15.0	1.59 (0.74-3.43)	50.0	2.36 (1.30-4.28)**

*P-value < 0.05 ** P-value < 0.01 for the separate logistic regression model for the risk factor represented by each column – models are adjusted for age, and analyses compare each occupational group with all other occupational groups.

Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The odds ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)

[a]Diagnosed with high blood pressure or received anti-hypertensive medication at any time over the available date range of the data.

[b]Diagnosed with high cholesterol or received cholesterol management medication at any time over the available date range of the data.

Less than 6 participants were excluded from the NZWS males (as per IDI rules, the precise number cannot be released).

Table S4.7. Māori NZWS Males - NZSCO two-digit logistic regression

	N	Deprivation Index 9/10 n = 921		Obese (>30kg m^2) n = 891		Ever Smoked n = 918		High blood pressure[a] n = 918		Diabetes n = 918		High Cholesterol[b] n = 918	
		%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)
OE_11	9	S	S	S	S	S	S	S	S	S	S	S	S
OE_12	93	20.0	0.65 (0.39-1.11)	41.9	0.87 (0.56-1.36)	61.3	0.98 (0.62-1.54)	38.7	0.80 (0.48-1.34)	19.4	0.89 (0.50-1.58)	41.9	1.16 (0.70-1.91)
OE_21	27	S	S	22.2	0.68 (0.29-1.61)	33.3	0.38 (0.16-0.87)*	37.0	1.52 (0.55-4.19)	S	S	S	S
OE_22	12	S	S	75.0	1.28 (0.43-3.87)	50.0	0.49 (0.16-1.48)	50.0	0.98 (0.29-3.37)	S	S	50.0	1.10 (0.34-3.63)
OE_23	42	21.4	0.69 (0.33-1.47)	42.9	0.95 (0.50-1.80)	50.0	0.57 (0.30-1.09)	31.0	0.50 (0.24-1.07)	14.3	0.92 (0.41-2.07)	28.6	0.53 (0.25-1.12)
OE_24	30	S	S	30.0	0.74 (0.34-1.62)	40.0	0.48 (0.23-1.01)	43.3	1.60 (0.65-3.91)	S	S	30.0	0.74 (0.28-1.95)
OE_31	24	S	S	37.5	1.04 (0.44-2.47)	62.5	0.82 (0.36-1.88)	45.8	1.08 (0.41-2.83)	S	S	50.0	1.10 (0.42-2.89)
OE_32	6	S	S	S	S	S	S	S	S	S	S	S	S
OE_33	75	24.0	0.83 (0.48-1.44)	32.0	0.62 (0.37-1.04)	60.0	0.91 (0.56-1.48)	33.3	0.77 (0.43-1.37)	18.0	0.43 (0.19-0.97)*	28.0	0.62 (0.34-1.12)
OE_41	33	33.3	1.22 (0.57-2.63)	36.4	0.93 (0.45-1.93)	54.5	0.86 (0.42-1.76)	39.4	1.53 (0.65-3.59)	S	S	36.4	1.09 (0.44-2.68)
OE_42	9	S	S	S	S	S	S	S	S	S	S	S	S
OE_51	66	31.8	1.12 (0.64-1.95)	54.5	1.61 (0.95-2.72)	54.5	0.87 (0.52-1.47)	36.4	0.91 (0.49-168)	22.7	1.27 (0.67-2.42)	36.4	1.15 (0.62-2.13)
OE_52	18	S	S	50.0	1.24 (0.47-3.38)	33.3	0.62 (0.24-1.65)	S	S	S	S	S	S
OE_61	78	28.0	1.00 (0.59-1.69)	38.5	0.76 (0.46-1.25)	57.7	0.81 (0.50-1.31)	46.2	1.04 (0.59-1.82)	16.2	0.81 (0.43-1.51)	46.2	1.31 (0.75-2.28)
OE_71	72	26.1	0.93 (0.54-1.61)	33.3	0.69 (0.42-1.16)	58.3	0.89 (0.54-1.45)	34.7	0.72 (0.40-1.30)	16.7	0.84 (0.43-1.63)	25.0	0.44 (0.24-0.82)*
OE_72	39	35.7	1.49 (0.76-2.92)	30.8	0.49 (0.24-1.01)*	61.5	1.34 (0.67-2.66)	28.2	0.49 (0.21-1.11)	S	S	23.1	0.38 (0.16-0.90)*
OE_73	S	S	S	S	S	S	S	S	S	S	S	S	S
OE_74	15	S	S	40.0	1.02 (0.34-3.11)	80.0	7.62 (0.98-59.21)	53.3	3.03 (0.84-10.95)	S	S	S	S
OE_81	27	40.0	1.66 (0.74-3.70)	44.4	1.18 (0.53-2.63)	66.7	2.23 (0.88-5.65)	33.3	0.72 (0.28-1.88)	S	S	33.3	0.91 (0.36-2.312)
OE_82	87	31.0	1.12 (0.69-1.82)	44.8	1.24 (0.78-1.95)	72.4	1.82 (1.10-3.00)*	50.6	1.63 (0.97-2.75)	20.7	1.14 (0.65-1.99)	41.4	1.17 (0.70-1.97)
OE_83	90	37.9	1.59 (1.00-2.52)*	50.0	1.49 (0.94-2.36)	76.7	1.86 (1.12-3.08)*	51.1	1.14 (0.69-1.88)	30.0	1.49 (0.90-2.48)	50.0	1.38 (0.85-2.26)
OE_84	12	S	S	S	S	50.0	1.70 (0.43-6.70)	S	S	S	S	S	S
OE_91	66	39.1	1.69 (1.01-2.83)*	54.5	1.63 (0.98-2.71)	63.6	1.07 (0.64-1.78)	45.5	1.40 (0.78-2.54)	27.3	1.75 (0.96-3.16)	45.5	2.00 (1.09-3.67)*

*P-value < 0.05 ** P-value < 0.01 for the separate logistic regression model for the risk factor represented by each column – models are adjusted for age, and analyses compare each occupational group with all other occupational groups.

Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The odds ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)

[a]Diagnosed with high blood pressure or received anti-hypertensive medication at any time over the available date range of the data.

[b]Diagnosed with high cholesterol or received cholesterol management medication at any time over the available date range of the data.

9 Māori NZWS male participants were excluded as they could not be assigned to an occupational group (unemployed, a student, a housewife/husband or did not answer).

Table S4.8. NZWS Females - NZSCO two-digit logistic regression

	N	Deprivation Index 9/10 n = 1557		Obese (>30kg m^2) n = 1473		Ever Smoked n = 1551		High blood pressure[a] n = 1557		Diabetes n = 1557		High Cholesterol[b] n = 1557	
		%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)
OE_11	9	S	S	S	S	S	S	S	**S**	S	S	S	S
OE_12	162	11.3	1.00 (0.61-1.65)	14.8	0.76 (0.47-1.20)	42.6	0.78 (0.56-1.08)	27.8	0.85 (0.57-1.24)	5.6	0.83 (0.41-1.68)	16.7	0.95 (0.60-1.51)
OE_21	24	S	S	S	S	25.0	0.47 (0.20-1.08)	29.2	1.07 (0.42-2.69)	S	S	S	S
OE_22	93	6.5	0.58 (0.26-1.27)	22.6	1.21 (0.72-2.02)	45.2	0.84 (0.55-1.27)	21.5	0.52 (0.30-0.88)*	S	S	19.4	0.94 (0.53-1.67)
OE_23	165	12.7	1.05 (0.64-1.70)	21.8	1.38 (0.92-2.02)	36.4	0.57 (0.41-0.79)**	33.3	1.05 (0.73-1.52)	7.3	0.95 (0.49-1.83)	14.5	0.68 (0.42-1.10)
OE_24	108	S	S	11.1	0.54 (0.29-0.99)*	47.2	0.92 (0.62-1.37)	24.1	0.67 (0.41-1.08)	5.6	0.99 (0.44-2.20)	19.4	1.05 (0.61-1.81)
OE_31	21	S	S	S	S	57.1	1.32 (0.54-3.22)	28.6	0.93 (0.33-2.58)	S	S	S	S
OE_32	36	S	S	S	S	33.3	0.48 (0.24-0.96)*	33.3	1.29 (0.62-2.72)	S	S	S	S
OE_33	219	12.3	1.03 (0.67-1.59)	16.4	0.87 (0.59-1.29)	50.7	1.07 (0.81-1.43)	25.6	0.78(0.55-1.11)	2.7	0.45 (0.21-0.99)*	13.7	0.70 (0.45-1.09)
OE_41	204	8.8	0.63 (0.37-1.06)	19.1	1.08 (0.74-1.59)	54.4	1.30 (0.97-1.75)	32.8	1.09 (0.78-1.52)	7.4	1.12 (0.63-1.99)	19.1	1.16 (0.78-1.73)
OE_42	81	7.4	0.67 (0.31-1.49)	22.2	1.59 (0.94-2.71)	48.1	0.92 (0.58-1.44)	46.9	2.37 (1.46-3.86)**	7.4	1.16 (0.49-2.75)	22.2	1.58 (0.89-2.82)
OE_51	174	20.3	1.90 (1.27-2.86)**	19.0	1.23 (0.82-1.84)	51.7	1.16 (0.85-1.60)	36.2	1.23 (0.86-1.76)	6.9	1.09 (0.59-2.01)	19.0	0.94 (0.61-1.45)
OE_52	96	12.9	1.10 (0.60-2.01)	12.5	0.67 (0.36-1.22)	53.1	1.21 (0.80-1.83)	30.2	0.90 (0.55-1.46)	S	S	18.8	0.97 (0.55-1.72)
OE_61	63	S	S	14.3	0.77 (0.37-1.59)	47.6	1.03 (0.62-1.72)	33.3	0.97 (0.54-1.73)	S	S	19.0	0.99 (0.51-1.94)
OE_71	S	S	S	S	S	S	S	S	S	S	S	S	S
OE_72	S	S	S	S	S	S	S	S	S	S	S	S	S
OE_73	S	S	S	S	S	S	S	S	S	S	S	S	S
OE_74	6	S	S	S	S	S	S	S	S	S	S	S	S
OE_81	S	S	S	S	S	S	S	S	S	S	S	S	S
OE_82	24	S	S	S	S	62.5	1.91 (0.84-4.35)	37.5	1.02 (0.42-2.49)	S	S	25.0	1.19 (0.43-3.29)
OE_83	S	S	S	S	S	S	S	S	S	S	S	S	S
OE_91	57	33.3	3.87 (2.17-6.90)**	26.3	2.01 (1.10-3.65)*	73.7	2.99 (1.64-5.45)**	42.1	1.53 (0.85-2.77)	15.8	2.60 (1.21-5.57)*	36.8	2.78 (1.47-5.26)**

*P-value < 0.05 ** P-value < 0.01 for the separate logistic regression model for the risk factor represented by each column – models are adjusted for age, and analyses compare each occupational group with all other occupational groups.

Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The odds ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)

[a]Diagnosed with high blood pressure or received anti-hypertensive medication at any time over the available date range of the data.

[b]Diagnosed with high cholesterol or received cholesterol management medication at any time over the available date range of the data.

Table S4.9. Māori NZWS Females - NZSCO two-digit logistic regression

	N	Deprivation Index 9/10 n = 1128		Obese (>30kg m^2) n = 1005		Ever Smoked n = 1128		High blood pressure[a] n = 1131		Diabetes n = 1131		High Cholesterol[b] n = 1131	
		%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)	%	OR (95%CI)
OE_11	9	S	S	S	S	S	S	S	S	S	S	S	S
OE_12	120	20.0	0.55 (0.35-0.86)*	36.0	0.57 (0.38-088)*	67.5	0.95 (0.63-1.42)	39.2	0.77 (0.50-1.20)	12.5	0.64 (0.36-1.14)	30.0	0.86 (0.53-1.38)
OE_21	12	S	S	S	S	S	S	S	S	S	S	S	S
OE_22	54	17.6	0.47 (0.23-0.95)*	38.9	0.79 (0.44-1.41)	55.6	0.64 (0.36-1.12)	37.0	0.86 (0.44-1.65)	22.2	1.44 (0.70-2.94)	22.2	0.67 (0.32-1.39)
OE_23	99	30.3	0.80 (0.50-1.26)	42.4	1.21 (0.78-1.87)	51.5	0.46 (0.30-0.71)**	36.4	0.96 (0.57-1.59)	18.2	1.22 (0.68-2.17)	24.2	0.89 (0.51-1.57)
OE_24	60	30.0	0.92 (0.52-1.62)	25.0	0.61 (0.33-1.13)	50.0	0.46 (0.27-.77)**	31.7	0.83 (0.44-1.59)	15.0	0.95 (0.43-2.10)	25.0	0.89 (0.43-1.83)
OE_31	12	S	S	S	S	75.0	0.90 (0.27-3.04)	S	S	S	S	S	S
OE_32	24	25.0	0.63 (0.25-1.59)	S	S	62.5	0.65 (0.29-1.44)	50.0	2.01 (0.81-4.98)	S	S	37.5	1.43 (0.51-4.02)
OE_33	159	30.2	0.92 (0.64-1.32)	39.6	1.08 (0.76-1.55)	66.0	0.95 (0.67-1.37)	33.3	0.65 (0.44-0.96)	13.2	0.82 (0.50-1.35)	28.3	1.01 (0.66-1.55)
OE_41	123	34.1	1.11 (0.75-1.65)	36.6	1.04 (0.70-1.56)	65.9	0.86 (0.58-1.28)	44.7	1.43 (0.92-2.22)	12.2	0.79 (0.45-1.39)	24.4	0.78 (0.47-1.28)
OE_42	48	29.4	0.68 (0.35-1.30)	43.8	1.29 (0.70-2.37)	62.5	0.83 (0.46-1.51)	35.4	1.01 (0.48-2.13)	12.5	1.01 (0.43-2.38)	25.0	1.28 (0.57-2.88)
OE_51	180	36.7	1.31 (0.94-1.83)	43.3	1.67 (1.18-2.36)**	76.7	1.66 (1.15-2.39)**	44.4	1.22 (0.84-1.78)	23.3	1.78 (1.19-2.68)**	35.0	1.48 (0.99-2.20)
OE_52	42	40.0	1.43 (0.77-2.65)	28.6	0.54 (0.27-1.09)	71.4	1.11 (0.58-2.14)	42.9	1.36 (0.64-2.88)	S	S	28.6	0.84 (0.36-1.93)
OE_61	39	35.7	1.09 (0.56-2.11)	38.5	0.94 (0.47-1.87)	84.6	1.79 (0.84-3.80)	46.2	1.20 (0.58-2.49)	15.4	1.07 (0.45-2.54)	30.8	1.43 (0.66-3.11)
OE_71	S	S	S	S	S	S	S	S	S	S	S	S	S
OE_72	S	S	S	S	S	S	S	S	S	S	S	S	S
OE_73	S	S	S	S	S	S	S	S	S	S	S	S	S
OE_74	9	S	S	S	S	100.0	-	S	S	S	S	S	S
OE_82	42	40.0	1.44 (0.77-2.69)	50.0	1.59 (0.83-3.06)	85.7	2.50 (1.10-5.69)*	47.6	1.50 (0.75-3.02)	14.3	1.28 (0.57-2.88)	28.6	0.79 (0.35-1.76)
OE_83	15	40.0	1.27 (0.46-3.53)	60.0	2.21 (0.72-6.84)	100.0	7.46 (0.98-56.81)	53.3	1.03 (0.34-3.12)	S	S	40.0	2.06 (0.66-6.40)
OE_91	75	50.0	2.49 (1.54-4.03)**	40.0	1.18 (0.70-1.97)	84.0	2.44 (1.29-4.59)**	42.7	1.09 (0.63-1.89)	16.0	1.20 (0.64-2.25)	28.0	0.96 (0.53-1.74)

*P-value < 0.05 ** P-value < 0.01 for the separate logistic regression model for the risk factor represented by each column – models are adjusted for age, and analyses compare each occupational group with all other occupational groups.

Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The odds ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)

[a]Diagnosed with high blood pressure or received anti-hypertensive medication at any time over the available date range of the data.

[b]Diagnosed with high cholesterol or received cholesterol management medication at any time over the available date range of the data.

12 Māori NZWS female participants were excluded as they could not be assigned to an occupational group (unemployed, a student, a housewife/husband or did not answer).

CHAPTER FIVE

A longitudinal linkage study of occupation and ischaemic heart disease in the general and Māori populations of New Zealand

Lucy A Barnes, Amanda Eng, Marine Corbin, Hayley J Denison, Andrea t' Mannetje, Stephen Haslett, Dave McLean, Lis Ellison-Loschmann, Rod Jackson, Jeroen Douwes.

Objectives: Occupation is a poorly characterised risk factor for cardiovascular disease (CVD) with females and indigenous peoples underrepresented in most research. This study assessed associations between occupation and ischaemic heart disease (IHD) in males and females of the general and Māori (indigenous people of NZ) populations of New Zealand.

Methods: Two surveys of the New Zealand adult population (NZ Workforce Survey (NZWS); 2004-2006; n=3003) and of the Māori population (NZWS Māori; 2009-2011; n=2107) with detailed occupational histories were linked with routinely collected health data and followed up until December 2018. Cox regression was used to calculate hazard ratios (HR) for IHD and 'ever worked' in any of the nine major occupational groups or 17 industries. Analyses were controlled for age, deprivation and smoking, and stratified by sex and survey.

Results: *Plant and machine operators and assemblers* and *elementary occupations* were positively associated with IHD in female Māori (HR 2.2 (1.2-4.1) and HR 2.0 (1.1-3.8), respectively) and among NZWS males who had been employed as *plant and machine operators and assemblers* for 10+ years (HR 1.7 (1.2-2.8)). Working in the *manufacturing* industry was also associated with IHD in NZWS females (HR 1.9 (1.1-3.7)), whilst inverse associations were observed for *technicians and associate professionals* (HR 0.5 (0.3-0.8)) in NZWS males. For *clerks*, a positive association was found for NZWS males (HR 1.8 (1.2-2.7)), whilst an inverse association was observed for Māori females (HR 0.4 (0.2-0.8)).

Conclusion: Associations with IHD differed significantly across occupational groups and were not consistent across males and females or for Māori and the general population, even within the same occupational groups, suggesting that current knowledge regarding the association between occupation and IHD may not be generalisable across different population groups.

5.1 Introduction

Cardiovascular disease (CVD) is a leading cause of death, with ischaemic heart disease (IHD) making up the largest proportion.[30] Risk factors include smoking, lack of physical activity, overweight, high blood pressure, high cholesterol and diabetes, as well as non-modifiable characteristics such as age, sex, ethnicity and family history.[320]

Occupation is another modifiable risk factor, but the evidence is limited.[10] In New Zealand, 19% of IHD mortality may be attributable to occupational risk factors for males and 9% for females.[11] The first New Zealand study to examine specific occupations and IHD mortality identified elevated risks in spinners, weavers and dyers, and shoemakers and leather good workers, but the study was conducted more than three decades ago and only included males.[16] A later update for the period 2001-2005 identified *plant and machine operators and assemblers* as having high rates of IHD mortality for New Zealand males.[17] International research has highlighted drivers, police officers, factory workers and blue-collar occupations more generally, to be among high-risk occupations.[15, 123]

Previous research on occupational risk factors had some limitations; many cohort studies were based on death records, which did not include full occupational histories and were not controlled for important confounders (e.g. smoking), while cross-sectional studies relied on self-reported IHD. Furthermore, despite sex-specific differences in CVD risk [68] and occupational exposure profiles,[27] previous research has predominantly focused on male-dominated occupations or only included men. Similarly, little research has been conducted in ethnic minority groups, despite evidence suggesting differences in cardiovascular risk between ethnicities.[333] In New Zealand, Māori (the indigenous people of New Zealand) have a considerably greater CVD mortality compared to New Zealand Europeans,[9] and occupational exposure patterns are also significantly different,[26] hence associations between occupation and IHD may be different for Māori and non-Māori. Therefore, effective workplace interventions may need to take into account sex and ethnic differences.

In this study, we used detailed occupational history and lifestyle information previously collected as part of two New Zealand workforce surveys: one in Māori[26] who make up ~16.5% of the population[302] and one in the general population[300] comprising 70.2% European origin, 8.1% Pacific and 15.1% Asian peoples. Data was linked to routinely recorded health information to identify incident IHD and assess association with occupation for males and females of the Māori and general populations.

5.2 Methods

This is a longitudinal study using occupational history and lifestyle information from two previous occupational surveys. Participants were followed up for 7-14 years from the date of interview for incident IHD using linked administrative health data.

Workforce surveys

The methods for the New Zealand Workforce Surveys (NZWS[300] and Māori NZWS[26]) have been described in detail previously. Briefly, a random proportionally stratified, systematic and self-weighted sample of people aged 20-64 years was selected from the Māori and general electoral rolls. For the NZWS, participants (including New Zealand European, Māori, Pasifika and other ethnic groups) were interviewed by phone from 2004-2006. In total, 3003 participants (37%) completed the survey.[300] The Māori NZWS was conducted from 2009-2011 using the same methodology and resulted in 2107 participants (29%) completing the survey.[26] Two participants were included in both surveys and their most recent interview was therefore excluded (i.e. the Māori NZWS).

The questionnaire included questions about lifetime work history, current workplace exposures, and demographic and lifestyle factors. Ethics approval was granted by the Massey University Human Ethics Committee (NZWS –WGTN 03/133, Māori NZWS – MUHEC 08/28) and the current linkage study was granted ethics approval by the New Zealand Health and Disability Ethics Committees (16/NTB/173).

As previously described, potential response bias from low survey response was small.[301] While some groups were underrepresented including, younger ages, higher deprivation and Māori (in the NZWS), the prevalence of key survey variables (both occupational exposure and health-related variables) were unchanged after standardising to the demographic distribution of the source population, and similar between early and late responders.

CVD risk factors assessed via questionnaire

Start date, end date and duration for each job were recorded, and jobs of ≥6 months were coded using the NZ Standard Classification of Occupations (NZSCO) 1999, which is a hierarchical skills-based classification with nine major occupational groups.[303] Work histories also included information about the main activity of the organisation for each job, which was coded for industry using the Australian and New Zealand Standard Industrial Classification (ANZSIC) 1996.[304] Age at interview was categorised: 20-34; 35-44; 45-54; and 55+ years. Socioeconomic status (SES) was assessed using the New Zealand Deprivation Index 2006 (NZDep2006), a census-based index with a relative deprivation score ranging from 1-10, based on place of residence. The distribution of deprivation is presented in quintiles, but for subsequent analyses it was dichotomised, combining scores 9-10 (people living in neighbourhoods of high deprivation) and scores 1-8 (people living in neighbourhoods of lower deprivation).

Smoking was analysed as never/ever and as pack-years, calculated from the number of cigarettes smoked per day divided by 20, multiplied by the number of years smoked. Body Mass Index (BMI) was calculated using self-reported height and weight grouped into four categories (i.e. <18.5; 18.5-24.9; 25-29.9; 30+).[319]

IHD events identified from linked health data

IHD events were ascertained by linking de-identified survey information to the Integrated Data Infrastructure (IDI), a longitudinal meta-dataset linked at the individual level, consisting of data from government agencies.[307] Before linkage, probabilistic matching was conducted to determine participants' National Health Index (NHI) number, which resulted in 98% of respondents being successfully matched and linkable.[319] Ministry of Health records including mortality, public hospital diagnoses and pharmaceutical dispensing were accessed for all participants.

IHD was identified using a previously developed definition:[312] it included IHD deaths, hospital diagnoses and procedures, using International Classification of Disease (ICD) codes, and dispensings of anti-anginal drugs recorded in the pharmaceutical claims dataset (Table 5.1). Primary healthcare information was not used as it is not available in the IDI.

Follow-up of IHD events

Participants were followed from the date of interview: 2004-2006 for the NZWS and 2009-2011 for the Māori NZWS. Participants with prior IHD were excluded. Participants who had moved overseas or died were identified through immigration and mortality data, respectively, and were censored from that time point. The end of follow-up was 31 December 2018; this is the date last observed for those that were not lost to follow-up, not deceased, or did not have an IHD event.

Table 5.1. IHD definition

IDI Data Source	ICD Code or Criteria	Details
Mortality data (1988-31 Dec 2016)	ICD-9-CMA 410, ICD-10-AM I21	Acute myocardial infarction
	ICD-10-AM I22	Subsequent myocardial infarction
National Minimum Dataset (NMDS): Publicly funded hospital discharges – diagnosis/procedure information (1988- 31 Dec 2018)	ICD-9-CMA 411, ICD-10-AM I24	Other acute and subacute forms of ischaemic heart disease
	ICD-9-CMA I23	Certain current complications following myocardial infarction (within the 28-day period)
	ICD-9-CMA 413, ICD-10-AM I20	Angina pectoris
	ICD-9-CMA 412, ICD-9-CMA 414, ICD-10-AM I25*	Chronic ischaemic heart disease
	ICD-9-CMA V45.81	Aortocoronary bypass status
	ICD-9-CMA V45.82	Percutaneous transluminal coronary angioplasty status
	ICD-10-AM Z95.1	Presence of aortocoronary bypass graft
	ICD-10-AM Z95.5	Presence of coronary angioplasty implant and graft
	ICD-9- CMA 36.0x, 36.1x ICD-10-AM 3530400, 3530401, 3530500, 3530501, 3531000, 3531001, 3531002, 3531003, 3531004, 3531005, 3849700, 3849701, 3849702, 3849703, 3849704, 03849705, 3849706, 3849707, 3850000, 3850001, 3850002, 3850003, 3850004, 3850300, 3850301, 3850302, 3850303, 3850304, 9020100, 9020101, 9020102, 9020103, 3863700, 3845619, 3865308, 3850500	Procedures
Pharmaceutical Data (2005-31 Dec 2018)	1577 Glyceryl trinitrate 2377 Isosorbide dinitrate 2836 Isosorbide mononitrate 1292 Nicorandil 1949 Perhexiline maleate	≥2 dispensings of any of these medications within 12 months

*ICD-10-AM I25.2 was excluded as it refers to old myocardial infarction and therefore, not an incident IHD event

Statistical Analyses

Cox proportional hazards regression was used to estimate hazard ratios (HR) of first IHD event for “ever worked” in a particular occupational group compared to “never worked” in that group, for each of the nine major NZSCO occupational groups and 17 ANZSIC industry groups. HRs were also estimated for the 23 two-digit NZSCO codes.

Work duration in each occupational group was categorised as never or <0.5; 0.5-2; >2-10; and >10 years. Duration was missing for 116 job records and these were excluded from duration analyses. Duration was fitted as a categorical variable using the ‘never’ category as the reference group (only HRs for 10+ years are presented).

Analyses are presented as age-adjusted (with no further adjustments) and age group (4 categories), SES (highest deprivation quintile vs all other) and smoking status (ever/never)-adjusted HRs. Additional analyses adjusting for pack-years and BMI were also conducted (Supplementary Table S5.6).

In compliance with the IDI confidentiality requirements, all frequencies were rounded to the nearest multiple of three and percentages were calculated from the rounded counts (hence, the number of total participants in each table varying slightly and percentages not adding up to exactly 100%). All statistical tests were performed on unrounded counts. All counts under six and the HRs derived from these are suppressed according to IDI confidentiality requirements (marked as ‘S’ in the tables).

5.3 Results

The average follow-up for the NZWS and Māori NZWS was 12.1 and 7.5 years, respectively. For 85 participants (45 NZWS and 40 Māori NZWS) we were not able to link survey and health data, and 213 participants had had an IHD event prior to interview (83 NZWS and 130 Māori NZWS), leaving a total of 2875 participants in the NZWS survey and 1935 in the Māori NZWS survey for analyses.

Incident IHD

The percentage of incident IHD cases identified through hospital discharges ranged from 73-80% across males and females of both surveys, while 19-25% of cases were identified through pharmaceutical dispensings only; mortality records identified the remainder. In the NZWS survey, 99 males and 36 females had a first IHD event during follow-up; in the Māori survey, 51 males and 42 females had a first IHD event. Males had proportionally more incident IHD in both surveys (Table 5.2). Incident IHD cases were more likely to be aged 55+ years, to have ever smoked, be deprived, and be obese (Table 5.2). Obesity, smoking and deprivation were more common in the Māori than in the general NZWS survey. These risk factors were also most prevalent in males, but differences were less pronounced in the Māori NZWS.

Occupation and industry and IHD

Among NZWS males, an inverse association with IHD was observed for *technicians and associate professionals* (Table 5.3; HR 0.5 (0.3-0.8)), whereas *clerks* had a greater risk (HR 1.8 (1.2-2.7)). Associations were less pronounced for those who worked 10+ years in these occupations (HR 0.8, (0.8-1.0) and 1.6 (0.8-3.0), respectively; Table 5.3). Employment for 10+ years as a *plant and machine operator and assembler* was also positively associated with IHD (HR 1.7 (1.2-2.8)).

For NZWS females, none of the main occupational groups showed statistically significant associations (Table 5.3). When considering more detailed 2-digit occupational groups (Supplementary Table S5.7), the 52- *Salespersons, Demonstrators and Models* occupational group was associated with an increased risk (HR 2.2 (1.2-4.3)). An increased risk was also observed for ever employed in the *manufacturing* industry (HR 1.9 (1.1-3.7); Table 5.4).

For Māori males, none of the main occupational or industry groups showed statistically significant associations after adjustment for age, high deprivation and smoking (Tables 5.3 and 5.5).

Among Māori females, *clerks* were less likely to have new-onset IHD (HR 0.4 (0.2-0.8); Table 5.3), in contrast with what was observed for NZWS males. Employment as *plant and machine operators and assemblers* or in *elementary occupations* was associated with an increased risk (HR 2.2 (1.2-4.1) and HR 2.0 (1.1-3.8), respectively) and 10+ years of employment in *elementary occupations* was associated with a three-times greater risk (HR 3.2 (1.3-7.9)). Employment in the *agriculture, forestry and fishing* industry for 10+ years was also associated with an increased risk (HR 2.5 (1.0-6.1); Table 5.5)

Table 5.2. Population characteristics

	NZWS Survey				Māori NZWS Survey			
	Total n = 2874		IHD cases n = 135		Total n = 1935		IHD cases n = 93	
	Males	Females	Males	Females	Males	Females	Males	Females
	1350 (47.0%) n (%)	1524 (53.0%) n (%)	99 (73.3%) n (%)	36 (26.6%) n (%)	852 (44.0%) n	1083 (56.0%) n (%)	51 (54.8%) n (%)	42 (45.2%) n (%)
Age at interview								
20-34	279 (20.7)	333 (21.9)	S	S	159 (18.7)	207 (19.1)	S	S
35-44	324 (24.0)	459 (30.1)	15 (15.2)	6 (16.7)	201 (23.6)	255 (23.5)	S	S
45-54	390 (28.9)	462 (30.3)	30 (30.3)	15 (41.7)	210 (24.6)	273 (25.2)	9 (17.6)	12 (28.6)
55+	354 (26.2)	273 (17.9)	54 (54.5)	15 (41.7)	288 (33.8)	348 (32.1)	39 (76.5)	27 (64.3)
Interview year								
2004	147 (10.9)	198 (13.0)	15 (15.2)	S	N/A	N/A	N/A	N/A
2005	678 (50.2)	738 (48.4)	57 (57.6)	24 (66.7)	N/A	N/A	N/A	N/A
2006	528 (39.1)	588 (38.6)	30 (30.3)	9 (25.0)	N/A	N/A	N/A	N/A
2007	S	S			N/A	N/A	N/A	N/A
2009	N/A	N/A	N/A	N/A	156 (18.3)	207 (19.1)	12 (23.5)	12 (28.6)
2010	N/A	N/A	N/A	N/A	456 (53.5)	585 (54.0)	30 (58.8)	21 (50.0)
2011	N/A	N/A	N/A	N/A	240 (28.2)	288 (26.6)	12 (23.5)	9 (21.4)
Ethnicity								
Pākehā	1080 (80.4)	1206 (79.1)	78 (78.8)	33 (91.7)				
Māori	99 (7.4)	156 (10.2)	12 (12.1)	S	852 (100.0)	1083 (100.0)	51 (100.0)	42 (100.0)
Pacific peoples	21 (1.6)	33 (2.1)	S	S				
Other	144 (10.7)	129 (8.5)	9 (9.1)	S				
Missing	S							
Smoking								
Ever	681 (50.6)	738 (48.4)	60 (60.6)	21 (58.3)	513 (60.4)	717 (66.4)	30 (58.8)	30 (71.4)
Current	246 (18.2)	282 (18.5)	27 (27.3)	9 (25.0)	210 (24.6)	327 (30.2)	6 (11.8)	12 (28.6)
Missing	S	S			S	S		
BMI								
<18.5	S	27 (1.9)	S	S	S	18 (1.9)	S	S
18.5-24.9	465 (35.1)	717 (49.7)	2 (28.1)	12 (36.4)	135 (16.4)	297 (30.8)	S	9 (23.1)
25-29.9	606 (45.7)	429 (29.7)	39 (40.6)	12 (36.4)	318 (38.7)	255 (26.5)	12 (24.5)	12 (30.8)
30+	249 (18.8)	267 (18.5)	30 (31.3)	9 (27.3)	366 (44.5)	387 (40.2)	30 (61.2)	18 (46.2)
Missing	21	84	S	S	30	120	S	S
NZDep 2006								
1-2 (least)	390 (28.9)	378 (24.8)	24 (24.2)	9 (25.0)	129 (15.2)	138 (12.7)	S	S
3-4	306 (22.7)	324 (21.3)	21 (21.2)	6 (16.7)	135 (15.9)	156 (14.4)	6 (11.8)	S
5-6	273 (20.2)	354 (23.2)	18 (18.2)	6 (16.7)	147 (17.3)	195 (18.0)	9 (17.6)	S
7-8	222 (16.4)	279 (18.3)	12 (12.1)	9 (25.0)	201 (23.7)	246 (22.7)	12 (23.5)	18 (42.9)
9-10 (most)	162 (12.0)	186 (12.2)	18 (18.2)	6 (16.7)	234 (27.6)	351 (32.4)	21 (41.2)	15 (35.7)
Missing		S			S	S		

Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts.
(S = suppressed)
BMI: Body Mass Index; NZDep: New Zealand Deprivation Index 2006

Table 5.3. Associations between occupational groups and IHD

Occupational Group	Total (n)	IHD cases (n)	HR (95%CI)[a]	HR (95%CI)[b]	Total (n)	IHD cases (n)	HR (95%CI)[a]	HR (95%CI)[b]
NZWS			**Males**				**Females**	
1. Legislators, Admin. & Managers (ever)	435	33	0.9 (0.6-1.4)	0.9 (0.6-1.4)	333	9	1.0 (0.5-2.2)	1.0 (0.5-2.2)
(employed 10+ years)	198	12	0.6 (0.4-1.2)	0.7 (0.4-1.3)	69	S	S	S
2. Professionals (ever)	399	24	0.7 (0.4-1.0)	0.7 (0.4-1.1)	591	18	1.0 (0.5-2.0)	1.1 (0.6-2.1)
(employed 10+ years)	180	12	0.6 (0.3-1.1)	0.9 (0.7-1.1)	249	6	0.7 (0.3-1.8)	0.8 (0.3-1.9)
3. Technicians & Assoc. Professionals (ever)	447	21	0.5 (0.3-0.8)**	0.5 (0.3-0.8)**	633	12	0.7 (0.4-1.4)	0.7 (0.4-1.5)
(employed 10+ years)	147	12	0.7 (0.4-1.3)	0.8 (0.7-1.0)*	159	S	S	S
4. Clerks (ever)	285	33	1.7 (1.1-2.6)**	1.8 (1.2-2.7)**	822	21	1.0 (0.5-2.0)	1.0 (0.5-1.9)
(employed 10+ years)	72	12	1.5 (0.8-3.0)	1.6 (0.8-3.0)	255	9	1.1 (0.5-2.6)	1.1 (0.5-2.6)
5. Service & Sales Workers (ever)	369	18	0.6 (0.4-1.1)	0.6 (0.4-1.0)	750	21	1.6 (0.8-3.2)	1.5 (0.8-3.0)
(employed 10+ years)	81	6	0.8 (0.3-1.8)	0.8 (0.3-1.8)	165	9	2.0 (0.8-4.9)	1.9 (0.8-4.6)
6. Agriculture & Fishery Workers (ever)	315	24	1.0 (0.6-1.5)	0.9 (0.6-1.5)	183	6	1.2 (0.5-2.9)	1.2 (0.5-2.8)
(employed 10+ years)	120	9	0.8 (0.4-1.6)	0.8 (0.4-1.6)	42	S	S	S
7. Trades Workers (ever)	459	36	1.1 (0.8-1.7)	1.1 (0.7-1.6)	60	S	S	S
(employed 10+ years)	246	24	1.2 (0.7-1.9)	1.1 (0.7-1.8)	18	S	S	S
8. Plant & Machine Operators & Assemblers (ever)	393	36	1.3 (0.9-2.0)	1.2 (0.8-1.8)	198	6	1.3 (0.6-3.1)	1.2 (0.5-2.8)
(employed 10+ years)	141	21	1.9 (1.2-3.2)**	1.7 (1.2-2.8)*	33	S	S	S
9. Elementary Occupations (ever)	327	27	1.3 (0.8-2.1)	1.2 (0.8-1.9)	237	6	1.2 (0.5-2.6)	1.1 (0.5-2.5)
(employed 10+ years)	33	S	s	S	33	S	S	S
Māori NZWS			**Males**				**Females**	
1. Legislators, Admin. & Managers (ever)	201	18	1.2 (0.7-2.2)	1.3 (0.7-2.3)	264	S	S	S
(employed 10+ years)	81	9	1.0 (0.5-2.2)	1.1 (0.5-2.4)	66	S	S	S
2. Professionals (ever)	180	9	0.8 (0.4-1.6)	0.8 (0.4-1.6)	363	9	0.7 (0.3-1.4)	0.7 (0.3-1.4)
(employed 10+ years)	69	6	1.1 (0.5-2.4)	1.1 (0.5-2.6)	120	6	0.8 (0.3-1.9)	0.8 (0.3-1.9)
3. Technicians & Assoc. Professionals (ever)	234	12	0.7 (0.4-1.4)	0.7 (0.4-1.4)	402	9	0.5 (0.3-1.1)	0.5 (0.3-1.1)
(employed 10+ years)	60	S	S	S	96	S	S	S
4. Clerks (ever)	174	9	0.8 (0.4-1.4)	0.8 (0.4-1.7)	543	12	0.4 (0.2-0.8)**	0.4 (0.2-0.8)*
(employed 10+ years)	33	S	S	S	144	S	S	S
5. Service & Sales Workers (ever)	270	15	1.0 (0.6-1.9)	1.0 (0.6-1.9)	651	27	1.5 (0.7-2.9)	1.4 (0.7-2.8)
(employed 10+ years)	63	6	1.2 (0.5-2.9)	1.3 (0.5-3.1)	153	S	S	S
6. Agriculture & Fishery Workers (ever)	297	24	1.3 (0.7-2.2)	1.2 (0.7-2.2)	210	12	1.5 (0.8-3.0)	1.5 (0.8-3.0)
(employed 10+ years)	81	9	1.2 (0.5-2.5	1.1 (0.5-2.4)	48	S	S	S
7. Trades Workers (ever)	336	21	0.8 (0.4-1.4)	0.7 (0.4-1.3)	72	6	2.1 (0.9-4.9)	2.0 (0.8-4.8)
(employed 10+ years)	132	9	0.8 (0.4-1.6)	0.7 (0.3-1.5)	S	S	S	S
8. Plant & Machine Operators & Assemblers (ever)	435	33	1.4 (0.8-2.4)	1.3 (0.7-2.3)	312	24	2.2 (1.2-4.1)**	2.2 (1.2-4.1)*
(employed 10+ years)	204	21	1.5 (0.8-2.9)	1.5 (0.8-2.8)	54	S	S	S
9. Elementary Occupations (ever)	369	21	1.5 (0.8-2.6)	1.4 (0.8-2.4)	294	18	2.0 (1.1-3.8)**	2.0 (1.1-3.8)*
(employed 10+ years)	42	S	S	S	45	6	3.2 (1.37.9)**	3.2 (1.3-7.9)*

*P value <0.05, **P value <0.01.

Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The hazard ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)

[a]Adjusted for age group

[b]Adjusted for age group, high deprivation and smoking status

Table 5.4. Associations between industry group and IHD - NZWS

Industry		Total (n)	IHD cases (n)	HR (95%CI)[a]	HR (95%CI)[b]	Total (n)	IHD cases (n)	HR (95%CI)[a]	HR (95%CI)[b]
NZWS				**Males**				**Females**	
A. Agriculture, Forestry & Fishing	(ever)	309	27	1.0 (0.6-1.6)	0.9 (0.6-1.5)	201	S	S	S
	(employed 10+ years)	129	12	0.8 (0.4-1.6)	0.8 (0.4-1.6)	51	S	S	S
C. Manufacturing	(ever)	609	54	1.2 (0.8-1.8)	1.2 (0.8-1.7)	468	18	2.0 (1.1-3.9)*	1.9 (1.1-3.7)*
	(employed 10+ years)	252	30	1.6 (1.0-2.5)	1.5 (0.9-2.3)	102	S	S	S
E. Construction	(ever)	381	27	1.0 (0.6-1.5)	0.9 (0.6-1.4)	93	S	S	S
	(employed 10+ years)	159	12	0.8 (0.4-2.3)	0.7 (0.4-1.4)	9	S	S	S
F. Wholesale Trade	(ever)	213	12	0.8 (0.4-1.4)	0.8 (0.5-1.5)	165	S	S	S
	(employed 10+ years)	51	S	S	S	9	S	S	S
G. Retail Trade	(ever)	396	21	0.7 (0.4-1.2)	0.7 (0.4-1.1)	609	15	1.1 (0.8-2.1)	1.0 (0.5-2.0)
	(employed 10+ years)	111	9	0.8 (0.4-1.7)	0.9 (0.4-1.7)	99	S	S	S
H. Accommodation, Cafes & Restaurants	(ever)	132	6	0.9 (0.4-1.9)	0.8 (0.4-1.8)	333	9	1.2 (0.6-2.5)	1.2 (0.5-2.5)
	(employed 10+ years)	18	S	S	S	30	S	S	S
I. Transport & Storage	(ever)	204	18	1.1 (0.7-1.9)	1.1 (0.7-1.8)	126	6	2.2 (0.9-5.3)	2.1 (0.9-5.1)
	(employed 10+ years)	69	12	1.9 (1.0-3.5)	1.8 (1.0-3.4)	18	S	S	S
J. Communication Services	(ever)	102	9	1.4 (0.7-2.8)	1.5 (0.7-2.9)	144	S	S	S
	(employed 10+ years)	24	S	S	S	27	S	S	S
L. Property & Business Services	(ever)	324	21	0.8 (0.5-1.3)	0.9 (0.5-1.4)	474	12	0.8 (0.4-1.6)	0.8 (0.4-1.7)
	(employed 10+ years)	93	6	0.6 (0.2-1.3)	0.6 (0.3-1.4)	75	S	S	S
M. Government Admin. & Defence	(ever)	267	27	1.3 (0.8-2.0)	1.3 (0.8-0.2)	285	12	1.7 (0.8-3.4)	1.7 (0.9-3.6)
	(employed 10+ years)	90	9	1.3 (0.7-2.5)	1.4 (0.7-2.7)	54	S	S	S
N. Education	(ever)	150	9	0.9 (0.5-1.7)	0.9 (0.5-1.7)	447	12	0.7 (0.3-1.5)	0.7 (0.4-1.6)
	(employed 10+ years)	54	6	0.9 (0.4-2.0)	0.9 (0.4-2.1)	141	S	S	S
O- Health Communication Services	(ever)	78	S	S	S	561	15	1.0 (0.5-1.9)	0.9 (0.5-1.7)
	(employed 10+ years)	30	S	S	S	198	S	S	S
P. Cultural & Recreational Services	(ever)	108	6	0.8 (0.4-1.9)	0.8 (0.4-1.9)	180	S	S	S
	(employed 10+ years)	12	S	S	S	24	S	S	S
Q. Personal & Other Services	(ever)	129	12	1.3 (0.7-2.4)	1.3 (0.7-2.4)	228	9	1.3 (0.6-2.9)	1.3 (0.6-2.8)
	(employed 10+ years)	33	S	S	S	30	S	S	S

*P value <0.05, **P value <0.01.
Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The hazard ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)
[a]Adjusted for age group
[b]Adjusted for age group, high deprivation and smoking status
Industries with no participants from either sex have been excluded - B (mining), D, and K

Table 5.5. Associations between industry group and IHD – Māori NZWS

Industry		Total (n)	IHD cases (n)	HR (95%CI)[a]	HR (95%CI)[b]	Total (n)	IHD cases (n)	HR (95%CI)[a]	HR (95%CI)[b]
Māori NZWS				**Males**				**Females**	
A. Agriculture, Forestry & Fishing	(ever)	288	24	1.3 (0.7-2.3)	1.2 (0.7-2.2)	228	12	1.4 (0.7-2.8)	1.4 (0.7-2.8)
	(employed 10+ years)	84	9	1.4 (0.7-3.0)	1.4 (0.6-2.9)	54	6	2.6 (1.1-6.2)**	2.5 (1.0-6.1)*
C. Manufacturing	(ever)	435	30	1.1 (0.6-1.9)	1.0 (0.6-1.7)	456	24	1.6 (0.8-3.1)	1.6 (0.8-3.0)
	(employed 10+ years)	189	15	1.0 (0.5-2.0)	1.0 (0.5-1.9)	90	6	1.7 (0.6-4.3)	1.6 (0.6-4.2)
E. Construction	(ever)	294	18	0.9 (0.5-1.6)	0.9 (0.5-1.5)	57	S	S	S
	(employed 10+ years)	114	6	0.8 (0.4-1.8)	0.8 (0.4-1.8)	6	S	S	S
G. Retail Trade	(ever)	261	15	0.8 (0.4-1.6)	0.8 (0.4-1.6)	468	12	0.7 (0.4-1.4)	0.7 (0.4-1.4)
	(employed 10+ years)	60	S	S	S	63	S	S	S
H. Accommodation, Cafes & Restaurants	(ever)	81	S	S	S	306	12	0.7 (0.4-1.5)	0.7 (0.4-1.5)
	(employed 10+ years)	9	S	S	S	45	S	S	S
I. Transport & Storage	(ever)	162	12	0.8 (0.4-1.6)	0.8 (0.4-1.6)	111	S	S	S
	(employed 10+ years)	57	S	S	S	18	S	S	S
J. Communication Services	(ever)	66	S	S	S	141	6	1.0 (0.4-2.4)	1.0 (0.4-2.4)
	(employed 10+ years)	15	S	S	S	21	S	S	S
L. Property & Business Services	(ever)	144	9	0.7 (0.3-1.6)	0.8 (0.3-1.7)	270	15	1.8 (1.0-3.5)	1.9 (1.0-3.5)
	(employed 10+ years)	30	S	S	S	39	S	S	S
M. Government Admin. & Defence	(ever)	210	18	1.4 (0.8-2.4)	1.4 (0.8-2.5)	231	6	0.6 (0.3-1.4)	0.6 (0.3-1.4)
	(employed 10+ years)	69	6	1.1 (0.5-2.7)	1.2 (0.5-3.0)	45	S	S	S
N. Education	(ever)	111	9	1.3 (0.7-2.8)	1.3 (0.6-2.7)	336	15	1.1 (0.6-2.2)	1.1 (0.6-2.2)
	(employed 10+ years)	39	S	S	S	108	9	1.2 (0.5-2.7)	1.2 (0.5-2.7)
O. Health Communication Services	(ever)	75	6	1.8 (0.9-3.9)	1.9 (0.9-4.0)	426	24	1.4 (0.8-2.7)	1.5 (0.8-2.7)
	(employed 10+ years)	39	S	S	S	108	9	1.7 (0.8-3.7)	1.2 (0.5-2.7)
Q. Personal and Other Services	(ever)	120	9	0.8 (0.4-1.8)	0.8 (0.4-1.8)	189	9	0.9 (0.4-2.1)	0.9 (0.4-2.1)
	(employed 10+ years)	27	S	S	S	30	S	S	S

*P value <0.05, **P value <0.01.

Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The odds ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)

[a]Adjusted for age group

[b]Adjusted for age group, high deprivation and smoking status

Industries with no participants from either sex have been excluded - B (mining), D, F, K and P

Additional adjustments for potential confounders

Results reported above were adjusted for “ever/never having smoked” as “pack-years” had more missing information. Also, as 5% of participants had missing information on BMI we did not adjust the analyses for this. HRs adjusted for pack-years and BMI are presented in Supplementary Table S5.6 and compared with the age-adjusted HRs, whilst restricting the sample to participants for whom we had complete data on pack-years and BMI to ensure comparisons were done on the same groups. These additional analyses did not change the results.

5.4 Discussion

This study, which linked detailed occupational histories to IHD using routinely collected health data, showed increased IHD risks for several occupational groups, with some marked sex and ethnic differences.

Incident IHD was more common in several blue-collar occupational groups such as *plant and machine operators and assemblers* and *elementary workers,* consistent with previous research showing increased IHD risk in blue-collar workers and New Zealand-specific data showing higher IHD mortality in *plant and machine operators and assemblers*.[14, 17, 123] Smoking and BMI are unlikely to explain this as analyses were adjusted for these factors. Blue-collar workers are often exposed to multiple hazards, including carbon monoxide, irregular schedules, noise and vigorous physical activity, which have previously been associated (inconsistently) with CVD.[92] The increased risk for blue-collar occupations was most pronounced for Māori females, with employment in *plant and machine operators and assemblers* and *elementary workers* doubling the risk of IHD. It is possible that within these broad occupational groups Māori women are more frequently involved in specific high-risk jobs, but this could not be confirmed. Alternatively, increased risk in women may be due to differential physiological responses to the same occupational exposures. Furthermore, as these occupations traditionally employ more males, it is possible that females are exposed to additional risk factors including working with equipment not designed for females, social isolation, gender discrimination and sexual harassment.[14] In addition, as we have shown previously, Māori are more exposed to physical factors[26] and may also suffer racial discrimination within the workplace, potentially compounding the risk for female Māori.[334]

A strong positive association with IHD was shown for *clerks* in male NZWS participants. An increased CVD risk has previously been reported for clerical workers, although this has primarily been found in females [117] and may be due to obesity and other CVD risk factors.[102] However, as we reported earlier,[319] no differences in obesity, smoking, diabetes or high cholesterol were observed between male NZWS *clerks* and other occupational groups; nonetheless, male *clerks* in the NZWS survey had twice the risk of high blood pressure (females had 1.5 times the risk). Although evidence is inconsistent, some studies have linked sedentary behaviour, common among clerical workers, to increased blood pressure, which may explain the increase in CVD risk.[253] In addition, job strain among clerical workers, which is associated with CVD,[146] may also play a role.

Our finding of a reduced risk among female Māori *clerks* is opposite to what we found for men and inconsistent with previous research (see above). When comparing specific job titles within the group of *clerks*, there were notable differences, with substantially more females working as *secretaries and keyboard operating clerks*, while a greater proportion of males worked as *material recording and transport clerks*. The occupational exposures associated with these sub-groups may be different and may explain the differential findings for male and female *clerks*. In addition, as we have shown previously, occupational exposures between Māori and non-Māori may differ, even within the same job title, with Māori involved in more physically demanding tasks.[26]

Opposing results were also found for male and female *service and sales workers* in the general survey: NZWS females who worked as 52-*Salespersons, Demonstrators and Models* had an increased IHD risk, while NZWS males in the same occupational group had a reduced risk. Specific occupations, tasks and related exposures may differ between males and females working in this occupational group, such as sedentary work and standing at work, both of which have been (inconsistently) linked to IHD.[239, 244]

Different results were observed for employment in agriculture, which was a risk factor for Māori females but not NZWS males. Previous research of agricultural workers has been inconsistent with some studies showing a high prevalence of multiple risk factors, such as high blood pressure and high cholesterol,[335] while other studies have demonstrated favourable CVD risk factor profiles in this industry, possibly due to better access to nutritious foods and higher levels of physical activity.[100] Our study suggests that Māori females working in agriculture may not equally benefit from this, but the reasons for this are unclear.

Technicians and associate professionals were less likely to experience IHD and this was shown across all subgroups, although it only reached statistical significance in NZWS males. In general, white-collar occupations are less exposed to hazardous occupational exposures, and this may in part explain the protective effect. However, similar trends were not found for other non-manual workers (*clerks* and *legislators, administrators and managers*).

This study has several limitations. The number of IHD cases was relatively small, with insufficient power to focus on more detailed occupational groups. Also, as we studied 9 occupational groups and 17 industries across both sexes in two surveys, involving many comparisons, some of our results may be due to chance. In fact, 222 statistical tests were conducted, equating to an expected number of false positives of 2 and 11 at the $p<0.01$ levels $p<0.05$ level, respectively. A similar rider applies to the supplementary tables, with 195 further tests, 17 and 11 significant at $p<0.05$ and $p<0.01$, and expected false positives of 10 and 2, respectively. Therefore, results, particularly those not previously reported in the literature, should be interpreted with caution.

There are other limitations including the variable date ranges of available health data (Table 5.1), which meant that the IHD definition varied somewhat across time. In particular, pharmaceutical data were only available from 2005, and mortality data with confirmed ICD codes only up to December 2016 (although non-coded death data was available until the end of December 2018). However, the impact of this is likely small as the majority of case ascertainment was based on hospital discharges, which covered the whole follow-up period. In addition, the definition of IHD was based on healthcare use, which may be different between genders and ethnicities.[29, 82] As primary healthcare data and private hospitalisation information are not available in the IDI, IHD events that did not result in a public hospitalisation, specific pharmaceutical prescription or death, were not included, potentially resulting in a small undercount of IHD cases.

Residual confounding is another limitation of this study. We did not adjust the analyses for high blood pressure, high cholesterol or diabetes as it is likely that these conditions are on the causal pathway between exposure and IHD, in which case it would be inappropriate to adjust for these factors. However, there is potential for other unmeasured confounding (e.g. diet, physical activity and alcohol consumption) that we were not able to account for.

A strength of the study is its longitudinal design, which did not rely on self-reports of outcomes, therefore reducing the risk of bias. The occupational histories also allowed for assessment of duration of employment. A unique feature of this study is that it included >50%

females and a large proportion of Māori, a population that has not previously been studied with respect to occupation and IHD.

In conclusion, the association between occupation and IHD differed across occupational groups, even after adjustment for known risk factors. Occupational risk patterns also differed between males and females, and between the Māori and general populations, suggesting that current knowledge regarding occupation and IHD may not be generalisable across all population groups.

5.5 Supplementary material

Table S5.6. Associations between occupational groups and IHD, excluding participants with missing BMI or pack-years information[a]

Occupational Group		Total (n)	IHD cases (n)	HR (95%CI)[a]	HR (95%CI)[c]	HR (95%CI)[d]	Total (n)	IHD cases (n)	HR (95%CI)[a]	HR (95%CI)[c]	HR (95%CI)[d]
NZWS				**Males**					**Females**		
1. Legislators, Admin. & Managers	(ever)	432	33	0.9 (0.6-1.4)	0.9 (0.6-1.4)	0.9 (0.6-1.4)	318	6	1.0 (0.4-2.1)	1.0 (0.4-2.2)	1.0 (0.4-2.1)
2. Professionals	(ever)	390	21	0.7 (0.4-1.1)	0.7 (0.4-1.1)	0.7 (0.4-1.1)	564	15	1.0 (0.5-2.0)	1.0 (0.5-2.1)	1.0 (0.5-2.0)
3. Technicians & Assoc. Professionals	(ever)	444	21	0.5 (0.3-0.9)*	0.5 (0.3-0.9)**	0.5 (0.3-0.9)*	603	12	0.7 (0.4-1.5)	0.7 (0.4-1.5)	0.7 (0.4-1.5)
4. Clerks	(ever)	282	30	1.8 (1.2-2.8)**	1.9 (1.2-2.9)**	1.9 (1.2-2.9)**	777	18	1.0 (0.5-1.9)	1.0 (0.5-2.0)	0.9 (0.5-1.9)
5. Service & Sales Workers	(ever)	366	18	0.6 (0.4-1.0)	0.6 (0.4-1.0)	0.6 (0.3-1.0)	708	21	1.4 (0.7-2.9)	1.5 (0.7-2.9)	1.5 (0.7-3.0)
6. Agriculture & Fishery Workers	(ever)	309	27	1.0 (0.6-1.5)	0.9 (0.6-1.4)	0.9 (0.6-1.5)	174	S	S	S	S
7. Trades Workers	(ever)	453	36	1.1 (0.7-1.6)	1.1 (0.8-1.8)	1.1 (0.7-1.6)	60	S	S	S	S
8. Plant & Machine Operators & Assembler	(ever)	384	36	1.2 (0.8-1.8)	1.2 (0.8-1.8)	1.1 (0.7-1.7)	192	6	1.1 (0.5-2.7)	1.1 (0.4-2.7)	1.1 (0.5-2.8)
9. Elementary Occupations	(ever)	321	27	1.2 (0.8-1.9)	1.2 (0.8-1.9)	1.2 (0.8-1.9)	219	6	1.0 (0.4-2.4)	1.0 (0.4-2.4)	1.0 (0.4-2.4)
Māori NZWS				**Males**					**Females**		
1. Legislators, Admin. & Managers	(ever)	198	15	1.3 (0.7-2.3)	1.4 (0.8-2.6)	1.3 (0.7-2.5)	252	S	S	S	S
2. Professionals	(ever)	177	9	0.7 (0.3-1.5)	0.7 (0.4-1.5)	0.8 (0.4-1.6)	333	12	0.7 (0.4-1.5)	0.7 (0.4-1.5)	0.7 (0.4-1.5)
3. Technicians & Assoc. Professionals	(ever)	228	12	0.8 (0.4—1.5)	0.8 (0.4-1.5)	0.8 (0.4-1.7)	363	12	0.5 (0.3-1.1)	0.5 (0.3-1.1)	0.5 (0.3-1.1)
4. Clerks	(ever)	174	9	0.9 (0.4-1.8)	1.0 (0.5-2.0)	1.0 (0.5-2.1)	480	12	0.5 (0.2-0.9)*	0.5 (0.2-0.9)*	0.5 (0.2-0.9)*
5. Service & Sales Workers	(ever)	264	15	1.1 (0.6-2.0)	1.2 (0.6-2.1)	1.0 (0.6-1.9)	579	27	1.3 (0.7-2.7)	1.3 (0.7-2.6)	1.3 (0.7-2.9)
6. Agriculture & Fishery Workers	(ever)	279	21	1.2 (0.7-2.1)	1.1 (0.6-1.9)	1.1 (0.6-2.0)	186	12	1.4 (0.7-2.9)	1.4 (0.7-2.9)	1.4 (0.7-2.9)
7. Trades Workers	(ever)	324	18	0.8 (0.4-1.4)	0.8 (0.4-1.4)	0.8 (0.4-1.4)	63	6	2.1 (1.1-3.9)	2.2 (0.9-5.4)	2.2 (1.1-5.3)
8. Plant & Machine Operators & Assemblers	(ever)	420	30	1.2 (0.7-2.2)	1.2 (0.6-2.2)	1.2 (0.6-2.1)	273	21	2.0 (1.1-3.9)*	2.0 (1.0-3.9)*	2.1 (1.1-4.0)*
9. Elementary Occupations	(ever)	261	18	1.2 (0.7-2.2)	1.0 (0.6-1.9)	1.2 (0.6-2.1)	261	18	2.1 (1.1-4.0)*	2.0 (1.1-3.9)*	2.1 (1.1-4.0)*

*P value <0.05, **P value <0.01.

Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The hazard ratios and associated 95% confidence intervals are presented in Their raw form and were calculated using the unrounded counts. (S = suppressed)

[a]Participants with missing BMI or pack years information excluded: 82 female and 22 male NZWS survey participants and 122 female and 31 Māori NZWS survey.

[b]Adjusted for age group, high deprivation, and smoking status.

[c]Adjusted for age group, high deprivation and pack years.

[d]Adjusted for age group, high deprivation smoking status, and BMI.

Table S5.7. Associations between NZSCO two-digit occupational groups and IHD - NZWS

	Total (n)	IHD cases (n)	HR (95%CI)[a]	HR (95%CI)[b]	Total (n)	IHD cases (n)	HR (95%CI)[a]	HR (95%CI)[b]
NZWS			**Males**				**Females**	
11. Legislators & Administrators	30	S	S	S	21	S	S	S
12. Corporate Managers	426	33	0.9 (0.6-1.4)	1.0 (0.6-1.5)	324	9	1.1 (0.5-2.3)	1.1 (0.5-2.3)
21. Physical, Mathematical & Engineering Science Professionals	171	9	0.8 (0.4-1.4)	0.8 (0.4-1.5)	45	S	S	S
22. Life Science & Health Professionals	48	S	S	S	159	S	S	S
23. Teaching Professionals	96	6	0.9 (0.5-1.9)	0.9 (0.5-2.0)	270	9	1.0 (0.5-2.2)	1.0 (0.5-2.3)
24. Other Professionals	132	S	S	S	195	S	S	S
31. Physical Science & Engineering Assoc. Professionals	168	12	0.8 (0.4-1.5)	0.8 (0.4-1.5)	102	S	S	S
32. Life Science & Health Assoc. Professionals	21	S	S	S	144	S	S	S
33. Other Assoc. Professionals	312	12	0.4 (0.2-0.8)**	0.4 (0.2-0.8)**	468	9	0.7 (0.3-1.5)	0.7 (0.3-1.5)
41. Office Clerks	240	24	1.5 (0.9-2.3)	1.6 (1.0-2.5)	663	18	0.8 (0.4-1.6)	0.8 (0.4-1.6)
42. Customer Services Clerks	63	6	1.7 (0.8-3.6)	1.6 (0.8-3.5)	366	9	1.0 (0.5-2.1)	1.0 (0.5-2.1)
51. Personal & Protective Services Workers	201	9	0.8 (0.4-1.5)	0.7 (0.4-1.4)	540	12	0.9 (0.5-1.9)	0.9 (0.4-1.7)
52. Salespersons, Demonstrators & Models	222	9	0.5 (0.2-1.1)	0.5 (0.2-1.0)*	411	18	2.3 (1.2-4.5)**	2.2 (1.2-4.3)*
61. Market-Oriented Agricultural & Fishery Workers	315	24	1.0 (0.5-1.5)	0.9 (0.6-1.5)	183	6	1.2 (0.5-2.9)	1.2 (0.5-2.8)
71. Building Trades Workers	231	18	1.2 (0.8-2.3)	1.2 (0.5-2.8)	12	S	S	S
72. Metal & Machinery Trades Workers	195	18	1.4 (0.8-2.3)	1.3 (0.8-2.1)	6	S	S	S
73. Precision Trades Workers	27	S	S	S	18	S	S	S
74. Other Craft & Related Trades Workers	66	6	1.2 (0.5-2.8)	1.1 (0.5-2.5)	27	S	S	S
81. Industrial Plant Operators	78	S	S	S	12	S	S	S
82. Stationary Machine Operators & Assemblers	210	21	1.5 (0.9-2.4)	1.3 (0.8-2.1)	177	6	1.5 (0.7-3.4)	1.4 (0.6-3.2)
83. Drivers & Mobile Machinery Operators	171	15	1.0 (0.6-1.7)	0.9 (0.5-1.6)	15	S	S	S
84. Building & Related Workers	33	S	S	S	S	S	S	S
91. Labourers & Related Elementary Service	327	27	1.3 (0.8-2.1)	1.2 (0.8-1.9)	237	6	1.2 (0.5-2.6)	1.1 (0.5-2.5)

*P value <0.05, **P value <0.01.
Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The hazard ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)
[a]Adjusted for age group.
[b]Adjusted for age group, high deprivation and smoking status.

Table S5.8. Associations between NZSCO two-digit occupational groups and IHD - Māori NZWS

	Total (n)	IHD cases (n)	HR (95%CI)[a]	HR (95%CI)[b]	Total (n)	IHD cases (n)	HR (95%CI)[a]	HR (95%CI)[b]
Māori NZWS			**Males**				**Females**	
11. Legislators & Administrators	18	S	S	S	18	S	S	S
12. Corporate Managers	192	15	1.3 (0.7-2.3)	1.4 (0.7-2.5)	255	S	S	S
21. Physical, Mathematical & Engineering Science Professionals	45	S	S	S	27	S	S	S
22. Life Science & Health Professionals	21	S	S	S	96	S	S	S
23. Teaching Professionals	66	6	1.3 (0.5-3.0)	1.2 (0.5-2.9)	186	9	1.4 (0.6-2.9)	1.4 (0.7-2.9)
24. Other Professionals	69	S	S	S	111	S	S	S
31. Physical Science & Engineering Assoc. Professionals	63	S	S	S	42	S	S	S
32. Life Science & Health Assoc. Professionals	18	S	S	S	66	S	S	S
33. Other Assoc. Professionals	177	9	0.9 (0.5-1.9)	0.9 (0.5-1.9)	333	9	0.4 (0.2-1.0)*	0.4 (0.2-1.0)*
41. Office Clerks	150	9	1.0 (0.5-2.0)	1.0 (0.5-2.1)	417	9	0.3 (0.2-0.7)**	0.3 (0.2-0.7)**
42. Customer Services Clerks	39	S	S	S	249	6	0.6 (0.2-1.4)	0.6 (0.2-1.3)
51. Personal & Protective Services Workers	195	12	1.3 (0.7-2.5)	1.3 (0.7-2.4)	528	24	1.4 (0.7-2.6)	1.4 (0.7-2.6)
52. Salespersons, Demonstrators & Models	123	S	S	S	270	12	1.1 (0.5-2.2)	1.1 (0.5-2.2)
61. Market-Oriented Agricultural & Fishery Workers	297	24	1.3 (0.7-2.2)	1.2 (0.7-2.2)	210	12	1.5 (0.8-3.0)	1.5 (0.8-3.0)
71. Building Trades Workers	174	9	0.6 (0.3-1.3)	0.6 (0.3-1.2)	18	S	S	S
72. Metal & Machinery Trades Workers	114	6	0.9 (0.4-1.9)	0.9 (0.4-2.0)	9	S	S	S
73. Precision Trades Workers	15	S	S	S	18	S	S	S
74. Other Craft & Related Trades Workers	69	6	1.9 (0.9-4.3)	1.8 (0.8-4.1)	24	S	S	S
81. Industrial Plant Operators	108	9	1.0 (0.5-2.2)	0.9 (0.4-2.1)	24	S	S	S
82. Stationary Machine Operators & Assemblers	285	24	1.5 (0.9-2.6)	1.4 (0.8-2.5)	282	21	2.4 (1.3-4.4)**	2.4 (1.3-4.4)**
83. Drivers & Mobile Machinery Operators	177	12	0.9 (0.5-1.8)	0.9 (0.5-1.8)	36	S	S	S
84. Building & Related Workers	36	S	S	S	S	S	S	S
91. Labourers & Related Elementary Service	369	21	1.5 (0.8-2.6)	1.4 (0.8-2.4)	294	18	2.0 (1.1-3.8)*	2.0 (1.1-3.8)*

*P value <0.05, **P value <0.01.

Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The hazard ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)

[a]Adjusted for age group.

[b]Adjusted for age group, high deprivation and smoking status.

CHAPTER SIX

Ischaemic heart disease and occupational exposures – a longitudinal linkage study in the general and Māori populations of New Zealand

Lucy A Barnes, Amanda Eng, Marine Corbin, Hayley J Denison, Andrea t' Mannetje, Stephen Haslett, Dave McLean, Lis Ellison-Loschmann, Rod Jackson, Jeroen Douwes.

Objectives: This study assessed associations between occupational exposures and ischaemic heart disease (IHD) for males and females in the general and Māori (indigenous peoples) populations of New Zealand.

Methods: Two surveys of the general adult (New Zealand Workforce Survey (NZWS); 2004-2006; n=3003) and Māori population (NZWS Māori; 2009-2011; n=2107), with information on occupational exposures, were linked with administrative health data and followed up until December 2018. Cox proportional hazards regression (adjusted for age, deprivation and smoking) was used to assess associations between organisational factors, dust and chemicals, stress and physical exposures, and IHD.

Results: Dust (HR 1.6 (1.1-2.4)), smoke or fumes (HR 1.5 (1.0-2.3)), and oils and solvents (HR 1.5 (1.0-2.3)) were associated with IHD in NZWS males. High frequency exposure to awkward or tiring hand positions was associated with IHD in both males and females of the NZWS (HRs 1.8 (1.1-2.8) and 2.4 (1.1-5.0) respectively). Repetitive tasks and working at very high speeds were associated with IHD among NZWS females (HRs 3.4 (1.1-10.4) and 2.6 (1.2-5.5), respectively). Māori females working with vibrating tools and those exposed to a high frequency of loud noise were more likely to experience IHD (HRs 2.3 (1.1-4.8) and 2.1 (1.0-4.4), respectively). Exposure to multiple dust and chemical factors was associated with IHD in the NZWS males, as was exposure to multiple physical factors in males and females of the NZWS.

Conclusions: Several occupational exposures were associated with IHD, but results differed for males and females and Māori and the general population.

6.1 Introduction

Growing evidence suggests a role for psychosocial, organisational and environmental workplace factors in CVD.[20] In particular, work-related psychosocial factors, such as stress, have been linked to CVD with relative risk estimates ranging from 1.5– 1.9,[21] and there is suggestive evidence for modest associations with loud noise and shift work. A meta-analysis of a small number of prospective studies on noise estimated an overall relative risk for CVD of 1.34 (95% CI 1.15-1.56).[213] Organisational factors, such as shift work, have also been studied, with a 2018 meta-analysis suggesting an almost 20% higher risk of CVD for those involved in shift work,[182] and another recent meta-analysis demonstrating a dose-response relationship.[183] Physical exertion, sedentary behaviour, long working hours and chemicals (such as pesticides), are among other occupational exposures associated with CVD. However, the extent of research into occupational risk factors for CVD and the consistency of findings has been variable.[18-20]

Most research has been undertaken in males or male-dominated occupations, with effects on female cardiovascular health largely unexplored. Ethnic minorities have also rarely been studied, despite considerable differences in CVD burden and occupational exposure profiles between ethnic groups and males and females.[26, 27, 67] In New Zealand, Māori (the indigenous population) have a considerably greater CVD burden compared to New Zealand Europeans[67] and they are overrepresented in blue-collar occupations.[307] The same is true for other indigenous populations[336, 337] but, to our knowledge, no previous occupational CVD studies have focused on indigenous workers.

In this study we used information from two New Zealand Workforce Surveys (NZWS), one conducted among Māori and one in the general population, to assess associations between occupational exposures and ischaemic heart disease (IHD), which makes up the greatest proportion of CVD cases, by linkage to routinely collected health records. To allow ethnic- and sex-specific associations to be studied, analyses were stratified by survey and sex.

6.2 Methods

This is a longitudinal study using occupational history and lifestyle information from two previously conducted occupational surveys in the general and Māori populations (see below). Incident IHD events were identified using routinely collected health data for a 7-14 year period from the date of interview until 31 December 2018.

Workforce surveys

The methods for the two New Zealand workforce Surveys (general population NZWS[300] and Māori NZWS[26]) have been described in detail previously. Briefly, for each survey, a random proportionally stratified, systematic and self-weighted sample of people aged 20-64 years were selected from the Māori and general electoral rolls. The general population NZWS was conducted from 2004-2006. Invitations to participate in a telephone interview were mailed up to three times and non-responders were contacted by phone (if available), with 3003 participants (37%) completing the survey. The Māori NZWS was conducted from 2009-2011 using the same methodology and resulted in 2107 participants (29%) completing the survey.[26] Two participants were included in both surveys and their most recent interview was therefore excluded (i.e. the Māori NZWS). Potential for participation bias from low survey response was evaluated and considered to be small,[301] i.e. while some groups were underrepresented, the prevalence of key survey variables (both occupational exposure and health-related variables) were unchanged after standardising to the demographic distribution of the source population, and similar between early and late responders.

The questionnaire included questions about lifetime work history, current workplace exposures, and demographic and lifestyle factors. Ethics approval was granted by the Massey University Human Ethics Committee (NZWS –WGTN 03/133, Māori NZWS – MUHEC 08/28) and the current linkage study was granted ethics approval by the New Zealand Health and Disability Ethics Committees (16/NTB/173).

Self-reported occupational exposures

Participants were asked whether the following exposures were present (yes/no) in their current or most recent workplace environment: dust; smoke or fumes; gas; oil and solvents; acids or alkalis; and pesticides. They were also asked about organisational factors, including working irregular hours (outside 7:00 to 20:30) and night shift (for at least 3 hours between 00:00-5:00) in the previous month, as well as the number of hours worked per week (<35; 35-45; 46-54; ≥55 hours). Participants were asked how often their job involved exposure to the following physical factors: awkward and tiring positions; awkward grip or hand movements; lifting; standing; sitting; using tools that vibrate; loud noise; repetitive tasks; working at very high speeds; and working to tight deadlines. This was measured on a scale from never to always (provided as a percentage of time or a point on the scale from never, ¼, ½, ¾, or all the time) and the median frequency of exposure (averaged across the cohorts), was calculated to determine a cut-point for ‘low exposure’ (i.e. <=median) and ‘high exposure’ (i.e. >median). Lastly, participants rated how stressful they found their current job (not at all, mild, moderate, very and extremely stressful), and this was categorised into: not at all/mild; moderate; and very/extremely.

The occupational exposures included in the analyses were selected based on previously reported associations with CVD.[20] We also asked about other exposures (e.g. boring work and working outside), but these were not included as they have not previously been shown to be associated with CVD.

Other CVD risk factors assessed via questionnaire

Participants provided current or most recent job title with a description of job tasks, and each job was coded using the New Zealand Standard Classification of Occupations (NZSCO) 1999, which is a hierarchical skills-based classification with nine major occupational groups.[303] Age at interview was categorised as follows: 20-34; 35-44; 45-54; ≥55 years. Socioeconomic status (SES) was assessed using the New Zealand Deprivation Index 2006, a census-based index with a relative deprivation score from 1-10, based on place of residence. The distribution of deprivation is presented in quintiles, but for subsequent analyses, it was dichotomised combining scores 9-10 (people living in neighbourhoods of high deprivation) and 1-8 (people living in neighbourhoods of lower deprivation).

Smoking status at the time of interview was analysed as never/ever and as pack-years, which was calculated from the number of cigarettes smoked per day divided by 20 and multiplied by the number of years smoked. Results for both measures were very similar (not shown); therefore, only results for ever/never are presented. Body Mass Index (BMI) at the time of interview was calculated using self-reported height and weight grouped into four categories (<18.5; 18.5-24.9; 25-29.9; ≥30) based on WHO guidelines.[322]

IHD identified from linked health data

To identify incident IHD, survey information from participants was linked to the Integrated Data Infrastructure (IDI), a longitudinal meta-dataset of de-identified data from government agencies, at the individual level.[307] Before linkage, probabilistic matching was conducted based on date of birth, sex, family name and first two given names, to identify National Health Index numbers to enable linkage to Ministry of Health (MoH) datasets. From the surveys, 98% of respondents were successfully matched and could be linked to mortality, public hospital diagnoses and pharmaceutical dispensing records.

The IHD definition included IHD deaths, hospital discharges and procedures using International Classification of Disease (ICD) codes, and ≥2 dispensings of anti-anginal drugs from the pharmaceutical claims dataset (Table 6.1) and was based on a previously developed definition.[312] Primary healthcare information was not used as it is not available in the IDI.

Follow-up of IHD events

Participants with prior IHD were excluded. For incident IHD, participants were followed from the date of interview: 2004-2006 for the general population and 2009-2011 for Māori. Participants that moved overseas or died from other causes were identified through immigration and mortality data in the IDI, respectively, and were censored from that time point. The date last observed for participants that were not lost to follow-up, not deceased, or did not have an IHD event, was 31 December 2018.

Table 6.1. IHD Definition

Data Source	ICD Code or Criteria	Details
Mortality data (1988-31 Dec 2016)	ICD-9-CMA 410, ICD-10-AM I21	Acute myocardial infarction
	ICD-10-AM I22	Subsequent myocardial infarction
National Minimum Dataset (NMDS): Publicly funded hospital discharges – diagnosis/procedure information (1988- 31 Dec 2018)	ICD-9-CMA 411, ICD-10-AM I24	Other acute and subacute forms of ischaemic heart disease
	ICD-9-CMA I23	Certain current complications following myocardial infarction (within the 28-day period)
	ICD-9-CMA 413, ICD-10-AM I20	Angina pectoris
	ICD-9-CMA 412, ICD-9-CMA 414, ICD-10-AM I25*	Chronic ischaemic heart disease
	ICD-9-CMA V45.81	Aortocoronary bypass status
	ICD-9-CMA V45.82	Percutaneous transluminal coronary angioplasty status
	ICD-10-AM Z95.1	Presence of aortocoronary bypass graft
	ICD-10-AM Z95.5	Presences of coronary angioplasty implant and graft
	ICD-9- CMA 36.0x, 36.1x ICD-10-AM 3530400, 3530401, 3530500, 3530501, 3531000, 3531001, 3531002, 3531003, 3531004, 3531005, 3849700, 3849701, 3849702, 3849703, 3849704, 03849705, 3849706, 3849707, 3850000, 3850001, 3850002, 3850003, 3850004, 3850300, 3850301, 3850302, 3850303, 3850304, 9020100, 9020101, 9020102, 9020103, 3863700, 3845619, 3865308, 3850500	Procedures
Pharmaceutical Data (2005-31 Dec 2018)	1577 Glyceryl trinitrate 2377 Isosorbide dinitrate 2836 Isosorbide mononitrate 1292 Nicorandil 1949 Perhexiline maleate	≥2 dispensings within 12 months

*ICD-10-AM I25.2 was excluded as it refers to old myocardial infarction and therefore, not an incident IHD

Statistical analyses

Analyses were stratified by survey and sex. Cox proportional hazards regression was used to calculate hazard ratios (HR) for associations between incident IHD and occupational exposures in participants' current or most recent job. Models were adjusted for age groups, deprivation and smoking status. For physical exposures, the 3-level variable (none/low/high frequency exposure) was used. For analyses where the number of IHD cases were <6 and had to be suppressed due to IDI requirements (see below), both low- and high-level exposures were combined to create a dichotomous variable of exposed versus not exposed.

Associations for combined workplace exposures were assessed by combining dust and chemical exposure variables if they were significantly ($p<0.05$) and positively associated with IHD in at least one cohort, summing the number of exposures for each participant (0 (reference), 1, 2 or 3). We used the same approach for physical exposures resulting in a summed exposure variable ranging from 0-4. The focus for statistical analysis of ordinal exposure variables was on whether exposure indicated increased IHD risk.

In compliance with the IDI confidentiality requirements, all frequencies were rounded to the nearest multiple of three and percentages were calculated from the rounded counts (hence total numbers of participants in each table vary slightly and do not add to exactly 100%). All statistical tests used the unrounded counts. All counts under six and the HRs from these are suppressed (marked 'S' in the tables).

6.3 Results

A total of 70 participants could not be linked to health data and for 15 a date last observed could not be determined. A further 213 participants (83 NZWS and 130 Māori NZWS) with an IHD event before interview were excluded, resulting in 2875 participants of the NZWS and 1935 participants of the Māori NZWS survey for analyses. Mean follow-up for the NZWS and Māori NZWS were 12.1 and 7.5 years, respectively.

Incident IHD

There were 135 incident IHD cases in the NZWS and 93 in the Māori NZWS. As expected, IHD cases were overrepresented in males, the oldest age group, ever smokers, overweight/obese BMI categories and high deprivation group (Table 6.2). IHD cases were also overrepresented in *plant and machine operators and assemblers* and *elementary occupations*.

Organisational factors, dust and chemicals, and stress

Dust, smoke or fumes, and oils and solvents (Table 6.3) were associated with IHD after adjusting for age, high deprivation and smoking in males of the NZWS (HRs 1.6 (1.1-2.4); 1.5 (1.0-2.3); and 1.5 (1.0-2.3), respectively). This was not found in the Māori NZWS. There were no associations observed for other chemicals, organisational factors or stress in either survey. The proportion of participants exposed to all three chemical exposures (dust, smoke or fumes, and oils and solvents) was similar between surveys but considerably greater in males compared to females (NZWS males 12.2%, Māori males 12.7%, NZWS females 2.6%, Māori females 2.5%). In NZWS males, exposure to two dust and chemical factors was significantly associated with IHD (HR 2.3 (1.4-3.8)); this was not found in the other groups (Table 6.3).

Physical exposures

High exposure to awkward grip or hand movements was associated with IHD in both males and females of the NZWS (Table 6.4; males HR 1.8 (1.1-2.8); females HR 2.4 (1.1-5.0)), but this was not found in Māori NZWS. Females in the NZWS (but not Māori NZWS females) also had an increased risk of IHD associated with high frequency exposure to repetitive tasks (HR 3.4 (1.1-10.4)) and working at very high speeds (HR 2.6 (1.2-5.5)).

An inverse association with IHD was found for exposure to awkward or tiring positions and loud noise in Māori males, statistically significant only for the low exposure categories (HRs 0.5 (0.2-1.0) and 0.4 (0.2-0.9), respectively). Among Māori females, there was a positive association with high frequency exposure to loud noise (HR 2.3 (1.1-4.8)); exposure to tools that vibrate was also positively associated (HR 2.1 (1.0-4.4)). Sitting was inversely associated with IHD among female Māori (but not NZWS females), significant for the high exposure category (HR 0.3 (0.1-0.8)).

The proportion of participants exposed to all four physical exposures that were positively associated with IHD in at least one cohort (high frequency of awkward grip or hand movements, repetitive tasks, working at very high speeds, loud noise) was higher among Māori NZWS compared to the NZWS (NZWS males 3.1%, Māori males 9.2%, NZWS females 2.4%, Māori females 6.1%). In NZWS males, exposure to four of these physical factors was associated with an almost four times greater IHD risk and in NZWS females, exposure to two physical factors was associated with a 4.6 times greater risk (Table 6.4). No associations were found for Māori.

Physical exposure analyses were repeated using a dichotomous cut-off representing exposure occurring ≥25% of the time (rather than using a median cut-off), which showed very similar results (Supplementary Table S6.5).

Table 6.2. Population characteristics

	NZWS Survey				Māori NZWS Survey			
	Total n = 2874		IHD cases n = 135		Total n = 1935		IHD cases n = 93	
	Males	Females	Males	Females	Males	Females	Males	Females
	1350 (47.0%) n (%)	1524 (53.0%) n (%)	99 (73.3%) n (%)	36 (26.6%) n (%)	852 (44.0%) n	1083 (56.0%) n (%)	51 (54.8%) n (%)	42 (45.2%) n (%)
Age at interview								
20-34	279 (20.7)	333 (21.9)	S	S	159 (18.7)	207 (19.1)	S	S
35-44	324 (24.0)	459 (30.1)	15 (15.2)	6 (16.7)	201 (23.6)	255 (23.5)	S	S
45-54	390 (28.9)	462 (30.3)	30 (30.3)	15 (41.7)	210 (24.6)	273 (25.2)	9 (17.6)	12 (28.6)
55+	354 (26.2)	273 (17.9)	54 (54.5)	15 (41.7)	288 (33.8)	348 (32.1)	39 (76.5)	27 (64.3)
Interview year								
2004	147 (10.9)	198 (13.0)	15 (15.2)	S	N/A	N/A	N/A	N/A
2005	678 (50.2)	738 (48.4)	57 (57.6)	24 (66.7)	N/A	N/A	N/A	N/A
2006	528 (39.1)	588 (38.6)	30 (30.3)	9 (25.0)	N/A	N/A	N/A	N/A
2007	S	S			N/A	N/A	N/A	N/A
2009	N/A	N/A	N/A	N/A	156 (18.3)	207 (19.1)	12 (23.5)	12 (28.6)
2010	N/A	N/A	N/A	N/A	456 (53.5)	585 (54.0)	30 (58.8)	21 (50.0)
2011	N/A	N/A	N/A	N/A	240 (28.2)	288 (26.6)	12 (23.5)	9 (21.4)
Ethnicity								
Pākehā	1080 (80.4)	1206 (79.1)	78 (78.8)	33 (91.7)				
Māori	99 (7.4)	156 (10.2)	12 (12.1)	S	852 (100.0)	1083 (100.0)	51 (100.0)	42 (100.0)
Pacific peoples	21 (1.6)	33 (2.1)	S	S				
Other	144 (10.7)	129 (8.5)	9 (9.1)	S				
Missing	S							
Smoking								
Ever	681 (50.6)	738 (48.4)	60 (60.6)	21 (58.3)	513 (60.4)	717 (66.4)	30 (58.8)	30 (71.4)
Current	246 (18.2)	282 (18.5)	27 (27.3)	9 (25.0)	210 (24.6)	327 (30.2)	6 (11.8)	12 (28.6)
Missing	S	S			S	S		
BMI								
<18.5	S	27 (1.9)	S	S	S	18 (1.9)	S	S
18.5-24.9	465 (35.1)	717 (49.7)	2 (28.1)	12 (36.4)	135 (16.4)	297 (30.8)	S	9 (23.1)
25-29.9	606 (45.7)	429 (29.7)	39 (40.6)	12 (36.4)	318 (38.7)	255 (26.5)	12 (24.5)	12 (30.8)
30+	249 (18.8)	267 (18.5)	30 (31.3)	9 (27.3)	366 (44.5)	387 (40.2)	30 (61.2)	18 (46.2)
Missing	21	84	S	S	30	120	S	S
NZDep2006								
1-2 (least deprived)	390 (28.9)	378 (24.8)	24 (24.2)	9 (25.0)	129 (15.2)	138 (12.7)	S	S
3-4	306 (22.7)	324 (21.3)	21 (21.2)	6 (16.7)	135 (15.9)	156 (14.4)	6 (11.8)	S
5-6	273 (20.2)	354 (23.2)	18 (18.2)	6 (16.7)	147 (17.3)	195 (18.0)	9 (17.6)	S
7-8	222 (16.4)	279 (18.3)	12 (12.1)	9 (25.0)	201 (23.7)	246 (22.7)	12 (23.5)	18 (42.9)
9-10 (most deprived)	162 (12.0)	186 (12.2)	18 (18.2)	6 (16.7)	234 (27.6)	351 (32.4)	21 (41.2)	15 (35.7)
Missing		S			S	S		
1-digit NZSCO occupational group								
1. Legislators, admin. & managers	261 (19.3)	165 (10.8)	15 (15.2)	S	93 (11.0)	123 (11.5)	9 (17.6)	S
2. Professionals	228 (16.9)	387 (25.4)	12 (12.1)	S	102 (12.1)	213 (19.9)	6 (11.8)	6 (14.3)
3. Technicians & assoc. prof.	168 (12.4)	273 (17.9)	12 (12.1)	9 (25.0)	96 (11.3)	189 (17.6)	S	S
4. Clerks	60 (4.4)	273 (17.9)	9 (9.1)	9 (25.0)	42 (5.0)	156 (14.6)	S	S
5. Service & sales workers	81 (6.0)	267 (17.5)	S	S	75 (8.9)	216 (20.2)	S	12 (28.6)
6. Agriculture & fishery workers	120 (8.9)	60 (3.9)	9 (9.1)	S	69 (8.2)	39 (3.6)	S	S
7. Trade workers	225 (16.7)	18 (1.2)	18 (18.2)	S	120 (14.2)	12 (1.1)	S	S
8. Plant & machine operators & assemblers	144 (10.7)	30 (2.0)	15 (15.2)	9 (25.0)	192 (22.7)	57 (5.3)	9 (17.6)	S
9. Elementary occupations	57 (4.2)	54 (3.5)	9 (9.1)	6 (16.7)	63 (7.4)	69 (6.4)	17 (23.5)	6 (14.3)
Missing	S				6	12		S

Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. (S = suppressed)
BMI: body mass index; NZDep2006: New Zealand deprivation index 2006

Table 6.3. Associations between IHD and organisational factors, stress, and dust and chemical exposures

	NZWS						Māori NZWS					
	Males			Females			Males			Females		
	Total	IHD	HR (95% CI)[a]	Total	IHD	HR (95% CI)[a]	Total	IHD	HR (95% CI)[a]	Total	IHD	HR (95% CI)[a]
	1350	99		1524	36		852	51		1083	42	
Organisational factors												
Hours worked												
<35	147	12	0.9 (0.5-1.7)	654	12	0.7 (0.3-1.3)	114	12	1.1 (0.5-2.3)	399	18	2.0 (1.0-4.2)
35-45	642	48	Ref 1.00	660	18	Ref 1.00	375	24	Ref 1.00	498	12	Ref 1.00
46-54	291	18	0.9 (0.5-1.5)	111	S	S	201	9	0.8 (0.4-1.6)	84	S	S
55+	267	21	0.9 (0.5-1.5)	99	S	S	144	S	S	72	S	S
Irregular hours	381	33	1.4 (0.9-2.1)	264	6	0.9 (0.4-2.2)	279	15	0.8 (0.4-1.4)	246	9	1.1 (0.5-2.2)
Night shift	129	12	1.7 (0.9-3.0)	63	S	S	117	S	S	87	S	S
Stress												
none/mild	486	42	Ref 1.00	642	15	Ref 1.00	405	30	Ref 1.00	489	21	Ref 1.00
moderate	663	42	0.7 (0.5-1.1)	645	15	1.3 (0.6-2.7)	339	15	0.7 (0.4-1.3)	414	12	0.6 (0.3-1.3)
very/extremely	198	12	0.6 (0.3-1.1)	234	6	1.7 (0.7-4.4)	105	S	S	165	6	0.9 (0.4-2.2)
Dust and chemical exposures												
Dust	546	48	1.6 (1.1-2.4)*	297	S	S	387	24	1.3 (0.8-2.3)	279	12	1.3 (0.7-2.5)
Smoke or Fumes	351	33	1.5 (1.0-2.3)*	171	S	S	261	12	0.6 (0.3-1.1)	135	6	2.0 (0.9-4.4)
Gas	150	12	1.3 (0.7-2.3)	78	S	S	99	6	1.1 (0.5-2.6)	81	S	S
Oil and Solvents	405	36	1.5 (1.0-2.3)*	198	S	S	246	12	1.0 (0.5-1.9)	114	6	1.8 (0.7-4.2)
Acids or Alkalis	186	15	1.2 (0.7-2.0)	90	S	S	126	S	S	57	S	S
Pesticides	195	9	0.6 (0.3-1.1)	78	S	S	114	12	1.0 (0.6-1.8)	69	S	S
Combined exposures: dust, smoke or fumes, and oils and solvents												
0	630	36	Ref 1.00	1053	30	Ref 1.00	348	21	Ref 1.00	699	24	Ref 1.00
1	306	18	1.0 (0.6-1.8)	321	S	S	219	15	1.2 (0.6-2.3)	252	9	1.3 (0.6-2.7)
2	252	30	2.3 (1.4-3.8)**	114	S	S	177	9	0.9 (0.4-2.1)	99	S	S
3	165	12	1.6 (0.8-3.0)	39	S	S	108	S	S	27	S	S

*P value <0.05, **P value <0.01.

Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The hazard ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)

[a]Adjusted for age group, high deprivation and smoking status.

Table 6.4. Associations between IHD and physical exposures

	NZWS						Māori NZWS					
	Males			Females			Males			Females		
	Total	IHD	HR (95% CI)[a]	Total	IHD	HR (95% CI)[a]	Total	IHD	HR (95% CI)[a]	Total	IHD	HR (95% CI)[a]
	1350	99		1524	36		852	51		1083	42	
Awkward or tiring positions												
No exposure	345	24	Ref 1.00	411	9	Ref 1.00	159	18	Ref 1.00	291	12	Ref 1.00
Low	582	39	1.1 (0.7-1.8)	606	12	0.9 (0.4-2.2)	345	18	0.5 (0.2-1.0)*	393	12	1.2 (0.5-2.7)
High (≥37.5% of the time)	417	36	1.5 (0.9-2.5)	498	18	1.9 (0.9-4.3)	342	18	0.5 (0.3-1.0)	387	18	1.5 (0.7-3.3)
Awkward grip/hand movements												
No exposure	543	39	Ref 1.00	759	12	Ref 1.00	258	21	Ref 1.00	474	18	Ref 1.00
Low	462	27	1.0 (0.6-1.7)	420	9	1.3 (0.6-3.1)	279	12	0.5 (0.3-1.0)	297	12	1.1 (0.5-2.2)
High (≥12.5% of the time)	339	33	1.8 (1.1-2.8)*	339	15	2.4 (1.1-5.0)*	309	15	0.6 (0.3-1.2)	297	9	1.0 (0.5-2.1)
Repetitive tasks												
No exposure	306	24	Ref 1.00	300	5	Ref 1.00	123	6	Ref 1.00	153	6	Ref 1.00
Low	735	45	0.9 (0.6-1.6)	831	18	2.1 (0.7-6.3)	426	27	1.5 (0.6-3.7)	504	18	0.9 (0.4-2.2)
High (≥75% of the time)	300	30	1.5 (0.9-2.7)	384	12	3.4 (1.1-10.4)*	297	18	1.7 (0.7-4.4)	408	15	1.0 (0.4-2.5)
Working at very high speeds												
No exposure	552	45	Ref 1.00	549	12	Ref 1.00	264	21	Ref 1.00	369	18	Ref 1.00
Low	495	30	0.9 (0.6-1.5)	549	6	0.7 (0.3-1.9)	315	18	0.8 (0.4-1.6)	348	9	0.8 (0.4-1.7)
High (≥50% of the time)	294	24	1.2 (0.7-2.0)	417	18	2.6 (1.2-5.5)*	270	12	0.7 (0.4-1.5)	357	9	0.7 (0.3-1.6)
Working to tight deadlines												
No exposure	201	21	Ref 1.00	303	9	Ref 1.00	111	15	Ref 1.00	180	12	Ref 1.00
Low	753	51	0.8 (0.5-1.4)	813	21	0.9 (0.4-2.0)	417	21	0.6 (0.3-1.1)	537	15	0.6 (0.3-1.3)
High (≥75% of the time)	387	27	1.0 (0.6-1.8)	405	9	0.8 (0.3-2.2)	324	15	0.5 (0.3-1.0)	354	12	0.6 (0.3-1.5)
Standing												
No exposure	705	51	Ref 1.00	891	24	Ref 1.00	417	30	Ref 1.00	615	24	Ref 1.00
Low	390	27	0.9 (0.6-1.4)	630	12	0.6 (0.3-1.2)	276	12	0.7 (0.4-1.4)	258	9	0.7 (0.3-1.7)
High (12.5% of the time)	249	18	1.0 (0.6-1.6)				153	9	0.9 (0.4-2.0)	198	9	1.3 (0.6-2.8)
Sitting												
No exposure	288	27	Ref 1.00	354	9	Ref 1.00	225	18	Ref 1.00	285	18	Ref 1.00
Low	645	48	0.8 (0.5-1.3)	600	12	1.0 (0.4-2.5)	399	21	0.7 (0.4-1.4)	447	15	0.5 (0.3-1.0)
High (≥50% of the time)	414	24	0.7 (0.4-1.3)	567	18	2.0 (0.9-4.7)	222	15	0.9 (0.4-1.9)	339	6	0.3 (0.1-0.8)*
Lifting												
No exposure	444	33	Ref 1.00	639	15	Ref 1.00	198	15	Ref 1.00	396	15	Ref 1.00
Low	582	39	1.0 (0.6-1.6)	618	12	0.7 (0.3-1.6)	312	18	0.8 (0.4-1.6)	363	12	1.0 (0.5-2.1)
High (≥37.5% of the time)	321	27	1.3 (0.8-2.2)	267	9	1.5 (0.7-3.4)	339	18	0.8 (0.4-1.5)	312	12	1.3 (0.6-2.7)
Tools that vibrate												
No exposure	915	63	Ref 1.00	1359	33	Ref 1.00	438	33	Ref 1.00	894	30	Ref 1.00
Low	279	27	1.4 (0.9-2.2)	159	S	S	225	9	0.6 (0.3-1.2)	177	9	2.1 (1.0-4.4)*
High (≥12.5% of the time)	147	9	1.2 (0.6-2.4)				183	12	1.0 (0.5-2.0)			

	NZWS						Māori NZWS					
	Males			Females			Males			Females		
	Total	**IHD**	**HR (95% CI)[a]**	**Total**	**IHD**	**HR (95% CI)[a]**	**Total**	**IHD**	**HR (95% CI)[a]**	**Total**	**IHD**	**HR (95% CI)[a]**
	1350	99		1524	36		852	51		1083	42	
Loud noise												
No exposure	585	42	Ref 1.00	1077	27	Ref 1.00	264	24	Ref 1.00	606	18	Ref 1.00
Low	378	30	1.1 (0.7-1.8)	441	12	1.4 (0.7-2.7)	234	9	0.4 (0.2-0.9)*	240	9	1.6 (0.8-3.6)
High (≥37.5% of the time)	387	27	1.2 (0.7-1.9)				345	18	0.7 (0.4-1.3)	225	12	2.3 (1.1-4.8)*
Combined exposures: high frequency exposure to awkward grip or hand movements, repetitive tasks, working high speeds and loud noise.												
0	612	42	Ref 1.00	714	9	Ref 1.00	258	21	Ref 1.00	408	18	Ref 1.00
1	360	24	1.1 (0.7-1.8)	465	12	2.2 (0.9-5.2)	231	12	0.7 (0.3-1.5)	297	9	0.7 (0.3-1.6)
2	219	15	1.3 (0.7-2.3)	213	9	4.6 (1.9-11.1)**	165	6	0.6 (0.2-1.5)	189	6	0.8 (0.3-1.9)
3	117	6	1.1 (0.5-2.4)	96	S	S	117	12	1.8 (0.9-3.9)	114	S	S
4	42	9	3.7 (1.8-7.7)**	36	S	S	78	S	S	66	S	S

*P value <0.05, **P value <0.01.

Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The hazard ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)

[a]Adjusted for age group, high deprivation and smoking status.

6.4 Discussion

Several occupational exposures were associated with incident IHD. Oils and solvents, dust, and smoke or fumes were associated with an increased risk in general population males, and high frequency exposures of awkward grip or hand movements was associated with an increased risk in both males and females of the general population. High frequency exposure to repetitive tasks and working at very high speeds were positively associated with IHD among general population females. Among Māori females, working with tools that vibrate and exposure to loud noise were associated with an increased risk of IHD. No associations for physical factors were observed in Māori males or females, and none of the occupational exposures were associated with IHD in Māori males.

There are several explanations why associations differed for Māori and the general population. Firstly, statistical power was lower in the Māori cohort due to fewer participants and shorter follow-up, resulting in fewer incident IHD events, even though exposure prevalence of physical factors was higher for Māori males.

Secondly, results are based on self-reported exposures and differences in perception of exposure can therefore not be excluded. Also, the nature, as well as circumstance of exposure, may differ between Māori and the general population. This may, for example, have contributed to the effect observed for sitting, which was inversely associated with IHD only in Māori females.

Thirdly, the distribution of occupations in Māori differs from that of the general population,[87, 338] which impacts exposure prevalence, the occupational composition of the reference group, and the interpretation of HR relative to each survey's reference group. Adjustment for the 1-digit occupational group did not affect results (data not shown), although this adjustment alone may not be sufficient to address this issue, particularly in stratified analyses as reported here.

Fourthly, this study included only a limited number of exposures that have previously been associated with IHD, predominantly in populations of European descent. Other exposures that may be of greater relevance to ethnic minorities may therefore not have been included as they were not collect in the original workforce surveys. Job insecurity, for example, was not assessed, but has been associated with IHD[339] and is higher for ethnic minorities.[340] There may also be additional exposures such as discrimination, which could influence the effect of occupational exposures on cardiovascular health.[88]

Similar issues may also explain the differences in associations observed between males and females. In particular, reduced statistical power due to lower exposure prevalence and fewer IHD cases may have contributed to fewer significant associations observed for women. Differences in the nature of exposure between males and females also likely play a role. For example, 'tools that vibrate' used by women can be different from those used by men, which may explain the difference in IHD risk observed between men and women reporting this exposure. In addition, sex-specific differences in susceptibility and pathophysiology of CVD may play a role.[341]

We observed an association with dust among general population males, which is consistent with previous studies on occupational particulate matter and CVD.[287] Similarly, our finding that smoke or fumes were associated with IHD, is consistent with previous observations that smoke and fumes, such as carbon monoxide and combustion products, were associated with CVD.[19, 272] The evidence is less clear for solvent exposure; earlier work suggested organic solvents may be linked to CVD,[342] while a more recent study found no link between solvent exposure and CVD or IHD.[269]

In contrast to leisure time physical activity, occupational physical exertion and heavy lifting have been linked to increased IHD risk.[258, 266, 343] In our study, lifting was not significantly associated with IHD; high frequency of awkward grip or hand movements, on the other hand, was associated with IHD for both sexes, and repetitive tasks and working at very high speeds was also associated with IHD for NZWS females. These specific exposures have not previously been studied in the context of IHD and may explain the increased IHD risk observed for occupational groups such as *plant and machine operators and assemblers*.[17]

Sedentary behaviour has been associated with CVD,[253] but there is limited evidence for occupational sedentary behaviour.[239] In this study, sitting >50% of working time was associated with a reduced risk in female Māori only. The reasons are unclear, but it is possible that sitting times at work may not adequately represent sedentariness or that it is associated with other work-related risk/protective factors.

Noise has repeatedly been associated with CVD.[213] In this study, frequent exposure to noise was associated with >2 times the risk of IHD in Māori females, whereas noise exposure in Māori males was inversely associated. This supports previous literature indicating women may be more adversely affected by noise exposure.[212] In Māori females, we also found an association with tools that vibrate, and whilst modest evidence of a relationship between vibration and CVD exists, there are limited studies among female workers.[290]

Chronic stress, including occupational stress, has been associated with CVD,[21] but this was not observed in the current study. It is possible that our measure of stress was insufficiently nuanced, with previous studies using more refined measures, such as job strain and effort-reward imbalance.[21] Stress may also play a mediating role in the association between other occupational exposures and IHD, however, this was not assessed in the current analyses.

Both shift work and long working hours have previously been linked to IHD, however, the overall evidence is inconsistent.[162] In this study, we did not observe significant associations with IHD for the number of hours worked, for working irregular hours or for working night shifts, but the prevalence of these exposures was low.

Few studies have considered the combined effect of occupational exposures, but some analysed exposure interactions (for e.g. physical activity, noise, job strain and shift work), and reported additive effects.[215, 292] In this study, exposure to multiple occupational factors was associated with greater IHD risk, which may explain the elevated prevalence of IHD we have previously observed for some occupational groups (e.g. *plant and machine operators and assemblers* and *elementary occupations*[319]) where exposure to multiple risk factors is common. However, it must be acknowledged that combining exposures as was done in this analysis implies an additive effect of occupational exposures; this assumption may not be entirely valid but could not be tested as the exposure metric used was relatively crude (yes/no).

Strengths of this study include the large proportion of females (>50%) and Māori (~40%), the inclusion of a range of exposures, which were collected before IHD diagnosis, limiting recall bias, as well as the measure of IHD incidence not relying on self-reports. It also has limitations, which include limited study power especially after adjustment for multiple testing as shown in Supplementary Table S6.6 and discussed further below. A lack of power was also an issue when comparing different levels of exposure, with higher levels of exposure often having small counts, which may be why sometimes a lower level of exposure was statistically significant but a higher level was not. We also used relatively crude measures of (self-reported) exposures, resulting in possible exposure misclassification. For the identification of IHD, we did not have access to private hospital or primary healthcare information, which may have resulted in an underestimation. Although we did have access to community dispensings of anti-angina medications, which likely captured most IHD cases that did not result in public hospitalisation.

Community dispensings data had limited date range availability (2005 onwards); however, most incident IHD events were identified through hospital admissions (73-80% across males and females of both surveys), which were available from 1988 onwards. Furthermore, occupational exposures were only available for current or most recent job. The average number of jobs in males and females was similar (NZWS males 4.4; NZWS females 4.9; Māori NZWS males 4.5; Māori NZWS females 4.9), but the average duration of employment of the last job was longer for males (NZWS males 9.5. years; NZWS females 6.0 years; Māori NZWS males 9.2 years; Māori NZWS females 6.9 years). Last occupation may therefore not be equally representative for males and females.

There may be confounders that were not considered, such as diet, leisure-time physical activity and alcohol consumption. However, analyses adjusting for BMI did not significantly alter results other than slightly change p-values (data not shown). We did not adjust analyses for high blood pressure, diabetes or elevated cholesterol as these may be on the causal pathway between (some) occupational exposures and IHD.

Finally, assessing multiple exposures stratified by sex and survey resulted in a large number of comparisons so some statistically significant results may be due to chance. Findings should therefore be interpreted with caution, particularly for those not previously reported in the literature and for subgroup analyses with a small number of significant findings (Supplementary Table S6.6).

In conclusion, associations with exposure to dust, smoke or fumes, oils and solvents, awkward grip or hand movements, carrying out repetitive tasks, working at very high speeds, loud noise and working with tools that vibrate and IHD were found, but results were often not consistent for males and females and between the general and Māori populations. These findings suggest that occupational risk factors for IHD may differ across populations.

6.5 Supplementary Material

Table S6.5. Association between physical exposures (25% cut-off point) and IHD

	NZWS						Māori NZWS					
	Males			Females			Males			Females		
	Total	IHD	HR (95%CI)[a]	Total	IHD	HR (95%CI)[a]	Total	IHD	HR (95%CI)[a]	Total	IHD	HR (95%CI)[a]
	1350	99		1524	36		852	51		1083	42	
Physical exposures												
Awkward or tiring positions	729	57	1.4 (0.9-2.0)	882	24	1.6 (0.8-3.1)	597	30	0.7 (0.4-1.2)	663	27	1.4 (0.7-2.7)
Awkward grip/hand movements	546	48	1.6 (1.09-2.4)*	555	18	2.0 (1.0-3.8)*	477	24	0.7 (0.4-1.2)	465	18	0.9 (0.5-1.7)
Repetitive tasks	870	66	1.4 (0.9-2.2)	1089	33	2.5 (1.0-6.0)*	681	45	1.7 (0.7-3.7)	846	27	0.8 (0.4-1.6)
Working at very high speeds	639	45	1.2 (0.83-1.9)	834	24	1.7 (0.8-3.3)	507	27	0.9 (0.5-1.6)	639	18	0.7 (0.3-1.3)
Working to tight deadlines	1005	66	1.0 (0.6-1.5)	1092	24	1.1 (0.5-2.2)	690	36	0.5 (0.3-1.0)*	846	24	0.7 (0.4-1.5)
Standing	363	30	1.1 (0.7-1.7)	435	9	0.9 (0.4-1.8)	285	15	0.7 (0.4-1.4)	318	12	1.1 (0.5-2.2)
Sitting	861	57	0.8 (0.5-1.2)	1002	30	2.1 (1.0-4.7)	510	30	0.9 (0.5-.7)	693	18	0.5 (0.3-0.9)*
Lifting	582	42	1.1 (0.7-1.7)	546	15	1.3 (0.7-2.4)	531	33	0.9 (0.5-1.6)	516	18	1.1 (0.8-2.0)
Tools that vibrate	231	18	1.2 (0.7-2.1)	87	S	S	291	12	0.7 (0.4-1.4)	117	9	3.0 (1.4-6.3)**
Loud noise	543	39	1.1 (0.7-.1.6)	309	9	1.4 (0.7-3.0)	492	27	0.8 (0.5-1.4)	363	18	1.9 (1.0-3.6)*

*P value <0.05, **P value <0.01.

Following IDI protocols, frequencies have been rounded to the nearest multiple of three and percentages calculated from those rounded counts. The hazard ratios and associated 95% confidence intervals are presented in their raw form and were calculated using the unrounded counts. (S = suppressed)

[a]Adjusted for age, high deprivation and smoking status

Table S6.6. Multiple comparisons

	Total number of tests	Expected number significant by chance at p<0.05	Number significant at p<0.05	Multiple test adjustment p-value[a]
Main tables				
NZWS males	40	2.0	6	0.014*
NZWS females	22	1.1	4	0.023*
Māori NZWS males	34	1.7	2	0.512
Māori NZWS females	29	1.5	3	0.175
Supplementary tables				
NZWS males	10	0.5	1	0.401
NZWS females	9	0.5	2	0.071
Māori NZWS males	10	0.5	1	0.401
Māori NZWS females	10	0.5	3	0.012*

[a]The probability tabulated is for the actual number of successes (i.e. significant findings) from a sequence of Bernoulli trials when the probability of success for each trial or test is p=0.05

CHAPTER SEVEN

Discussion and conclusions

7.1 Introduction

The aim of this thesis was to improve the understanding of occupational risk factors for IHD. The two previous studies conducted in New Zealand about occupation and CVD were mortality studies which only included males[16, 17] and did not present results by ethnicity, despite nearly 48% of the working population being female[344] and 12.7%, Māori[338] (2017 statistics). International research in this area has also predominantly been conducted in male populations and seldom involves ethnic minorities. In this thesis, results are presented separately for males and females and for the general and Māori populations, making it possible to ascertain whether occupational risk factors for IHD differed across these populations. In this final chapter, the main findings are discussed, methodological strengths and limitations are explored, and the implications for practice and future research are considered.

7.2 Discussion of main findings

7.2.1 Occupational risk factors for IHD

The work described in this thesis has identified several occupational groups (Chapter 5), industries (Chapter 5) and specific occupational exposures (Chapter 6) that were associated with an increased risk of IHD. The associations between these occupational factors and IHD are summarised in the tables below (Tables 7.1-7.3).

Table 7.1. Associations between IHD and occupational groups

	General population		Māori population	
NZSCO Occupational group	*Male*	*Female*	*Male*	*Female*
1. Legislators, administrators, and managers				
2. Professionals				
3. Technicians and associate professionals	-			
4. Clerks	+			-
5. Service and sales workers	(-)	(+)		
6. Agriculture and fishery workers				(+)
7. Trade workers				(+)
8. Plant and machine operators and assemblers	+			+
9. Elementary occupations				+

-	Statistically significant inverse association (reduced risk of IHD)
+	Statistically significant positive association (increased risk of IHD)
(-)	Borderline significant inverse association
(+)	Borderline significant positive association

Table 7.2. Associations between IHD and industries

	General population		Māori population	
ANZSIC Industry group	*Male*	*Female*	*Male*	*Female*
A. Agriculture, Forestry and Fishing				+
B. Mining				
C. Manufacturing	(+)	+		(+)
D. Electricity, Gas and Water Supply				
E. Construction				
F. Wholesale Trade				
G. Retail Trade				
H. Accommodation, Cafes, and Restaurants				
I. Transport and Storage	(+)	(+)		
J. Communication Services				
K. Finance and Insurance				
L. Property and Business Services				(+)
M. Government Administration and Defence		(+)		
N. Education				
O. Health and Community Services			(+)	(+)
P. Cultural and Recreational Services				
Q. Personal and Other Services				

-	Statistically significant inverse association (reduced risk of IHD)
+	Statistically significant positive association (increased risk of IHD)
(-)	Borderline significant inverse association
(+)	Borderline significant positive association

Table 7.3. Associations between IHD and specific occupational exposures

	General population		Māori population	
Occupational exposures	*Male*	*Female*	*Male*	*Female*
Working ≥55 hours per week				
Irregular hours				
Nightshift	(+)			
Dust	+			
Smoke or fumes	+			(+)
Gas				
Oils and solvents	+			(+)
Acids and alkalis				
Pesticides				
Very/extremely stressed	(-)	(+)		
Awkward or tiring position *(high freq.)*	(+)	(+)	(-)	(+)
Awkward grip or hand movement *(high freq.)*	+	+	(-)	
Repetitive tasks *(high freq.)*	(+)	+	(+)	
Working at very high speeds *(high freq.)*		+		
Working to tight deadlines *(high freq.)*			(-)	
Standing *(high freq.)*				
Sitting *(high freq.)*		(+)		-
Lifting *(high freq.)*				
Tools that vibrate				+
Loud noise *(high freq.)*				+

-	Statistically significant inverse association (reduced risk of IHD)
+	Statistically significant positive association (increased risk of IHD)
(-)	Borderline significant inverse association
(+)	Borderline significant positive association

This research found that blue-collar occupations and industries were most consistently associated with increased risks of IHD, in particular the occupational groups *plant and machine operators and assemblers* and *elementary occupations*. Similar occupational groups, such as factory workers and labourers, have previously been linked to increased CVD risk in the previous New Zealand mortality studies of men[16, 17] and internationally.[15] HRs increased with longer duration of employment (Chapter 5, Table 5.3); Māori females with more than 10 years employment in *elementary occupations* showed a three times higher IHD risk (HR 3.2 (1.3-7.9)) compared to those who had not worked in that occupational group. As demonstrated in Chapter 4, people working as *plant and machine operators and assemblers* and *elementary workers* were often more likely to live in deprived areas, be obese or have ever smoked, consistent with previous evidence.[99, 107] However, adjustment for these cardiovascular risk factors only slightly attenuated the results for these occupational groups, indicating that the observed associations cannot be explained by these risk factors. *Plant and machine operators and assemblers* and *elementary workers* were also more likely to ever be diagnosed or treated for high blood pressure, diabetes and high cholesterol (Chapter 4), indicating plausible physiological pathways linking these occupational groups to IHD.

Similarly, employment in the blue-collar the *manufacturing* industry was associated with IHD, particularly for general population females (HR 1.9 (1.1-3.7); Chapter 5, Table 5.4). Employment in the *transport and storage* industry was also associated with an elevated risk of IHD for the general population (although not statistically significant; Chapter 5, Table 5.4). This is consistent with previous work that has shown that transport-based occupational groups are associated with a higher prevalence of cardiovascular risk factors and CVD risk, with findings for drivers being most consistent.[84, 118, 125] Employment of 10 or more years in the *agriculture, forestry and fishery* industry was also associated with IHD among Māori females (HR 2.5 (1.0-6.1); Chapter 5, Table 5.4). As demonstrated in Chapter 4, Māori workers in the *agricultural and fishery workers* group were more likely to have high cholesterol, potentially indicating a key physiological pathway. However, this increased risk of IHD was not observed for general population males, who were also less likely to have high cholesterol (Chapter 4 and 5). Previous literature has also been inconsistent for agricultural work, although studies have demonstrated a lower prevalence of risk factors, such as metabolic syndrome, diet and smoking, among agricultural workers.[100, 104]

Chapter 6 found several exposures to be associated with IHD, particularly exposures that are more prevalent in blue-collar occupations,[300] such as dust, smoke or fumes, and oils and solvents (Chapter 6, Table 6.3), which may, at least in part, explain the increased risk of IHD observed in blue-collar occupational groups. Other studies have found similar results, with particulate matter linked to CVD[287] and smoke or fumes products, such as carbon monoxide and combustion products, also associated with CVD.[19, 272] Solvent-use has been linked to CVD,[342] although recent work has shown conflicting evidence with some studies showing no association.[269] Inflammation and endothelial dysfunction are some of the hypothesised mechanisms linking dust and chemical exposure to CVD.[273, 280, 287] Pesticide use is also prevalent in blue-collar work, particularly in the agriculture sector, and several previous studies have found associations between pesticide exposure and CVD.[269, 273] However, there was no evidence of an association with pesticide exposure in the findings described in Chapter 6. The results of this thesis, therefore, suggest that the observed increased IHD risk observed for Māori female agricultural workers may not be due to pesticide exposure.

A range of physical factors were associated with IHD in this research and may also explain some of the association between IHD and blue-collar occupations; these factors include loud noise, awkward grip or hand movements, carrying out repetitive tasks and working at very high speeds (Chapter 6, Table 6.4). Previous literature has identified a link between loud noise exposure and CVD,[213] similar to the findings of an association between loud noise exposure and IHD among Māori women as described in this thesis. As noise exposure likely acts as a stressor, it may, among other physiological responses to stress, contribute to elevated cholesterol and blood pressure.[23] Occupational physical exertion has also been linked to CVD risk in other studies, with prolonged exposure, little rest time, elevation of heart rate and promotion of inflammation differentiating exertion from recreational physical activity.[266] However, there was no specific measure of physical exertion in the surveys and a range of physical factors were analysed instead. The associations with IHD and specific physical factors such as awkward grip or hand movements, carrying out repetitive tasks and working at very high speeds are novel findings; however, these may represent other exposures that have already been linked to CVD, such as physical exertion,[258] particularly if the task being repeated or carried out at high speeds is physically demanding. Although the causal pathways between these physical factors and IHD are not clear, the work described in this thesis suggests the potential relevance of a range of physical aspects with regard to IHD risk.

Shift work (including nightshift) and working long hours are also common in blue-collar occupations and have been associated with CVD in previous literature.[194, 207] However, in this thesis (Chapter 6), results were not statistically significant for shift work and long hours, which may be due to limitations of the assessment of these exposures in the surveys; the questionnaire asked participants if they had worked nightshift over the last 4 weeks (defined as 3 hours between midnight and 5 am) and irregular hours was arbitrarily defined as outside 7:00 to 20:30. These measurements may not accurately represent overall exposure and differ from how these exposures were assessed in previous studies. Job stress is another occupational exposure potentially associated with IHD in blue-collar workers, but this was not statistically significant in the findings of this thesis. Stress is a complex exposure and the crude measurement of stress used in the studies described in this thesis may not be sufficiently sensitive. Furthermore, stress may act as a mediating factor in the relationship between other occupational exposures and IHD, including noise or bullying, suggesting that further work is likely required to fully elucidate the role of stress in occupation and cardiovascular health.

While the work described in this thesis suggests that occupational risk factors are mainly concentrated in blue-collar occupations and industries, there were some exceptions. An increased risk of IHD was observed among male *clerks* in the general population (HR 1.8 (1.2-2.7)); Chapter 5, Table 5.3). The literature on clerical or office workers has been reasonably consistent, demonstrating an increased risk of CVD, however, the majority of work has been done in females.[93, 117] A key physiological pathway linking clerical work and IHD risk may be elevated blood pressure as a strong association was observed between clerks and high blood pressure in Chapter 4, with general population males having double the risk of high blood pressure compared to those working in other occupations (OR 2.22 (1.26-3.91); Chapter 4, Table 2a). In contrast to male clerks in the general population, reduced risk of IHD was observed for female clerks in the Māori cohort. The potential reasons for these and other differences between males and females and Māori and the general population are discussed in Sections 7.2.2 and 7.2.3.

Service and sales work is another white-collar occupational group and industry that has previously been linked to CVD.[13, 123] This is consistent with findings described in this thesis, which showed that although no results were statistically significant at the 1-digit occupational group, there was some evidence of a positive association between *service and sales* work and IHD for females. The 2-digit occupational group *NZSCO 52 – Salespersons, Demonstrators and Models* was associated with IHD for general population females (HR 2.2 (1.2-4.3); Chapter 5, Table S2); in contrast, an inverse association was shown in general population males (HR 0.5 (0.2-1.0)).

Relevant occupational exposures that may contribute to the increased risk observed among *clerks* and *service and sales workers* include prolonged sitting, stress or certain physical factors. In general, the literature linking occupational sedentary behaviour to CVD is mixed,[239] although some evidence suggests that it has direct effects on metabolic and inflammatory processes.[253]

However, as presented in Chapter 6, sitting more than 50% of the time was not associated with an increased risk of IHD, with a reduced risk observed for Māori females (Chapter 6, Table 6.4). The contrasting results may be due to the relevance of the context of sitting; for example, as Māori are often exposed to more physical occupational factors than non-Māori,[26] increased sitting may therefore represent a lack of these other occupational exposures in Māori females, thus showing a protective effect. Stress has more consistently been linked to increased blood pressure and CVD in other studies.[21, 156] However, stress was not significantly associated with IHD in our study (Chapter 6), which may be due to the relative crudeness of the measure of stress used in this thesis. In addition, the physical exposures associated with IHD, such as carrying out repetitive tasks, awkward grip or hand movements and working at very high speeds, may also be prevalent *clerical* and *service and sales workers,* although the nature of the tasks likely differs from those experienced by BCW; for example, repetitive tasks in an office likely differ from repetitive tasks in a factory.

In contrast to clerical or office white-collar work, the literature suggests that white-collar occupational groups with greater responsibility, such as professionals, have a reduced risk of CVD,[120] and similar findings were observed in this thesis for *professionals* and *technicians and associate professionals*. As demonstrated in Chapter 4, *professionals* were less likely to experience conventional cardiovascular risk factors. Working in the occupational group *technician and associate professionals* was also associated with a decreased risk of IHD (Chapter 5, Table 5.3), and risk factors such as deprivation, age, smoking and BMI could not explain these findings. This inverse association may be due to a lack of specific high-risk physical exposures (e.g. repetitive tasks or working at very high speeds) or dust and chemical factors in these occupational groups, although confounding factors that have not been accounted for in these analyses, such as diet, may also play a role.

7.2.2 Occupational risk factors for IHD in males and females

One aim of this thesis was to assess whether associations between occupations/occupational exposures and IHD were consistent for males and females. However, direct comparisons between males and females require caution as there are differences in statistical power due to lower IHD incidence (7.3% in NZWS males; 2.4% in NZWS females; 6.0% Māori NZWS males; 3.9% Māori NZWS females) and less exposure to some occupational risk factors among females. As females are less likely to work in blue-collar occupations (Figure 7.1), the prevalence of exposure to physical and dust or chemical factors is significantly lower among females (Chapter 6, Table 6.3 and 6.4), reducing the power to detect associations for these occupational exposures. Differences in occupational distributions also affect the composition of the reference groups to which all comparisons are made, with the male reference groups comprising more blue-collar workers.

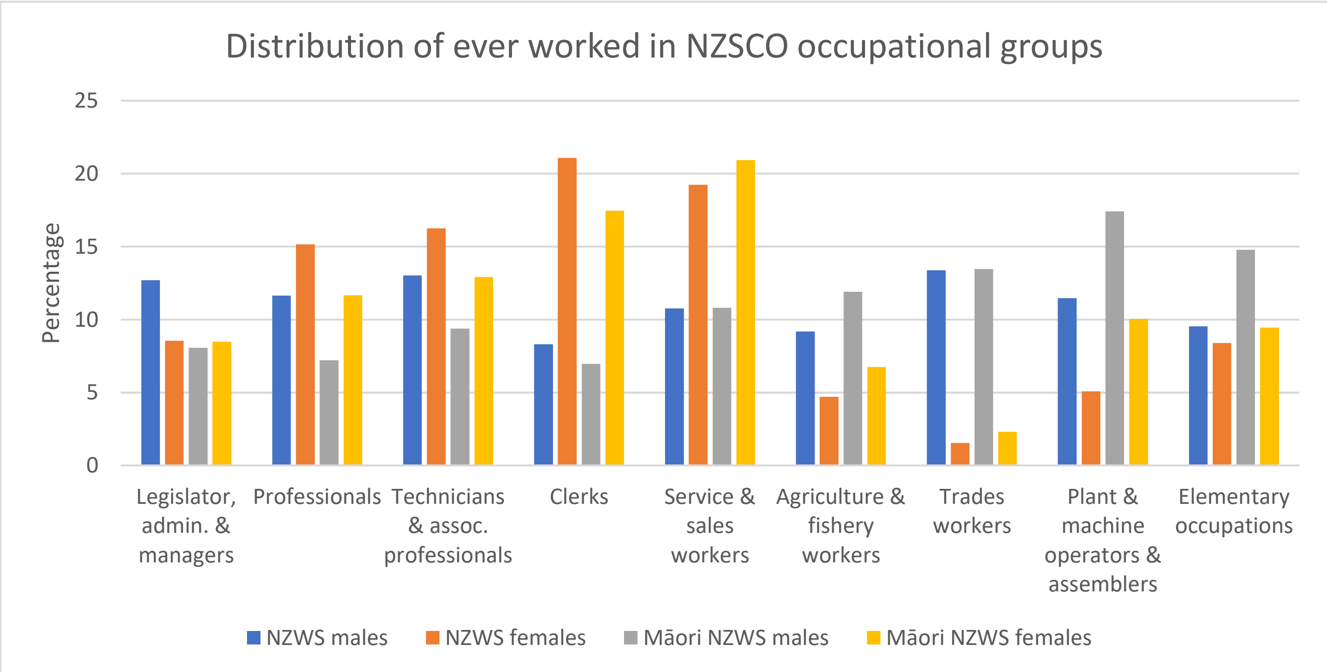

Figure 7.1. Distribution of ever worked in NZSCO occupational groups for the NZWS and Māori NZWS

Despite these differences in statistical power, there were notable differences in the relationships between some occupational risk factors and IHD for males and females that deserve discussion. Contrasting associations were observed for the occupational group *clerks* i.e. Chapter 5 showed an increased risk among general population males, but a decreased risk was found in Māori females. Another difference was observed in *service and sales* workers, with an increased risk of IHD observed in female workers of the general population, while a decreased risk was found for males. These differences may be due to differences in job title distribution between genders within broad occupational groups, and hence, different occupational exposure profiles. For example, even within the smaller occupational classification of NZSCO 52 -*Salespersons, Demonstrators and Models*, occupations range from salespersons and demonstrators to street vendors and fashion models, each with a different proportion of male/female workers and different levels and types of exposures.

There may also be other factors that contribute to inconsistent findings for males and females within the same job. Firstly, previous research has demonstrated that males and females in the same job often have different occupational exposure patterns.[27] Secondly, women may face additional exposures, such as sexism, discrimination and social isolation,[14] which are potentially more prevalent in traditionally male-dominated blue-collar occupations. This has been shown to be particularly detrimental for females from ethnic minorities.[88, 334] These additional exposures may contribute to elevated stress levels and hence, IHD risk.[21, 88] Furthermore, females often have to use equipment and personal protective equipment (PPE) designed for males, which means females may not be equally protected against hazardous exposures. Due to physiological differences, there are also likely differences in the physical capacities of men and women, therefore, physically demanding tasks may have more detrimental effects at a lower exposure for women. Finally, women also traditionally have more responsibilities outside of paid work and these multiple roles have been associated with poor health outcomes.[345] These factors may contribute to the strong associations between blue-collar work and IHD observed among Māori females (Chapter 5, Table 5.3) and between occupational exposure to physical factors and IHD among the general population females (Chapter 6, Table 6.4).

It is plausible that the significant genetic, hormonal and physiological differences between males and females also play a role in occupational risk factors as evidence already demonstrates differences in the effect of conventional cardiovascular risk factors on CVD risk between sexes.[341] For example, both smoking and diabetes have been associated with a greater risk of IHD among women compared to men due to sexually dimorphic physiological responses i.e. greater absorption of toxic chemicals from smoking and more detrimental effects of glucose impairment in diabetes.[73] Additionally, there are CVD risk factors unique to women, such as menopausal status, hypertensive disorders of pregnancy, pre-eclampsia, preterm delivery and polycystic ovarian syndrome,[71, 72, 346] demonstrating that cardiovascular risk factors are not the same across males and females. Therefore, the physiological response to occupational risk factors may be sexually dimorphic, with differences in both the susceptibility and high-risk thresholds at which exposures cause adverse outcomes.

Overall, these results indicate that occupational risk factors for IHD may be different between males and females and suggest that physical exposures and blue-collar occupations are particularly important in the context of female cardiovascular risk.

7.2.3 Occupational risk factors for IHD in the general and Māori populations

Another aim of this thesis was to assess whether associations between occupations/occupational exposures and IHD were consistent for the Māori and general populations. However, there are several reasons why directly comparing results for the general and Māori populations requires caution.

Firstly, statistical power was not the same for the general and Māori surveys, and this difference was particularly marked for males. Because the Māori survey was conducted later than the general population survey, the follow-up period was shorter for Māori (51 incident IHD cases were identified in the male Māori cohort and 99 in the male general population cohort). Secondly, the occupational profile of the Māori population is markedly different from that of the general population, with Māori less likely to work in white-collar occupations (Figure 7.1). This means that the reference groups used for comparing occupational groups/exposures are made up of different workers; for example, the reference groups in the Māori comparisons comprise more blue-collar workers than in the general population reference groups. Thirdly, as occupational exposures were measured by self-report, there may be differences in exposure perception, potentially resulting in different degrees of exposure misclassification between groups. Finally, as the measure of IHD in this thesis relied on interactions with healthcare providers in New Zealand, IHD may be underestimated for Māori as there are significant disparities in access to healthcare between Māori and non-Māori.[82]

While being mindful of these differences between populations, there are notable differences in associations for Māori males that deserve further discussion. In particular, several occupational groups and exposures that were found to be associated with an increased IHD risk in general population males were not replicated for Māori males. For example, working in the *manufacturing* industry was associated with elevated IHD for all populations, except Māori males (Chapter 5, Table 5.4). Also, among the occupational exposures significantly associated with IHD, exposure to dust, smoke or fumes, awkward grip or tiring hand movements, using tools that vibrate, loud noise, awkward or tiring positions, working to tight deadlines and working at very high speeds, were either not or inversely associated with IHD in Māori males (Chapter 6, Table 6.4).

Another marked difference between the general and Māori populations was observed in agricultural occupations, where general population males had a low prevalence of cardiovascular risk factors and did not have an elevated risk of IHD, in contrast to Māori workers. Among Māori females, an elevated (although not statistically significant) risk of IHD was observed for *agriculture and fishery workers* and IHD was significantly associated with working in the *agriculture, forestry and fishing* industry for at least 10 years (HR 2.5 (1.0-6.1); Chapter 5, Table 5.4).

These marked differences in results for Māori and the general population may be due to a range of reasons. Firstly, the specific jobs distribution differs between the general and Māori population with an overrepresentation of Māori in lower skilled work. For example, within the broad agricultural classification, the proportion of Māori males working in *NZSC0 611 – Market Farmers and Crop growers* and *NZSCO 613 – Forestry and Related Workers* was higher than that of the general population males. Secondly, there may be differential delegation of tasks for Māori workers, potentially resulting in differential exposure and task patterns within the same occupation.[26, 347] The specific nature and context of the tasks being completed during exposure may be relevant and differ between Māori and the general population; for example, two workers in the same occupation reporting exposure to an occupational factor are not necessarily doing the same task and each potentially has different effects on the body. Since this research and previous work has demonstrated a compounding effect of multiple exposures, this may further contribute to differences observed between Māori and general populations for associations between occupational groups and IHD as Māori experience greater exposure to occupational hazards.[26]

Systemic racism may be a root cause underlying some of these differences. For example, it contributes to the overrepresentation of Māori in lower-skilled occupations due to differential access to education and job opportunities,[89] as well as potentially contributing to the differences in task delegation and hence, occupational exposures experienced by Māori. This may also in part explain why the healthy worker effect may be more pronounced for Māori males; a selection effect where ‘healthy’ workers are more likely to be employed in more physical or hazardous jobs (discussed in more detail in Section 7.3.2). As previous work has shown that compared to non-Māori workers, Māori workers report higher exposure to physical factors even within the same job,[26] a higher overall level of health may be required for Māori employed in blue-collar and physical occupations. Therefore, Māori male workers in blue-collar jobs may consequently be ‘healthier’ than those employed in other jobs, potentially masking associations. Furthermore, racial discrimination or bullying in the workplace, whether it be between workers or from management, is associated with psychological and physiological responses that may exacerbate the effect of conventional cardiovascular risk factors.[88]

Overall, these results indicate that occupational risk factors for IHD identified in the general population do not necessarily apply to Māori (and potentially other ethnic minorities) and that certain risk factors relevant for Māori may be missed in general population studies and current occupational health and safety strategies. Identification of and targeting ethnic-specific occupational risk factors may therefore help to reduce health inequities experienced by Māori (and potentially other ethnic minorities).

7.3 Methodological strengths and limitations

7.3.1 Strengths

Diverse survey populations

The survey populations used in the analyses of this thesis comprised >50% females and nearly 40% Māori, representing populations that have not been thoroughly studied in the context of occupation and IHD. As demonstrated in Chapter 2, most studies have focused on males or adjusted for sex rather than report sex-specific results. Furthermore, the majority of studies have been undertaken in dominant ethnic groups with limited studies adjusting for ethnicity and even fewer reporting associations by ethnic group. As the findings of this thesis demonstrate that occupational risk factors may not be consistent between different populations, these findings emphasise the importance of considering sex and ethnicity in future investigations of occupation and IHD.

Detailed occupation and occupational exposures

The original workforce surveys collected occupational history information, which allowed a comprehensive evaluation of work history by analysing 'ever working' in an occupational group and duration of employment. This is an important advantage over many other studies on this topic in which generally only the last occupation was available for analysis. Furthermore, the occupational exposure information collected covered a wide range of occupational exposures, allowing assessment of varying levels of exposure for physical exposures and working hours, and investigations into the effects of multiple exposures.

Study design

Occupational information and lifestyle factors were collected before the incident IHD occurred (i.e. at baseline) and IHD was not an original outcome of the investigation, limiting the possibility of recall bias that can occur in other study designs, such as case-control studies. Furthermore, the analyses in Chapters 5 and 6 were longitudinal, capturing the development of incident IHD over a 7-14 year period.

Routinely collected health information

A major strength of this research was that it used routinely collected health records in the IDI to obtain health information because it eliminated reliance on the participants' self-report of their health status and provided access to detailed and clinically coded information on IHD related events and pharmaceutical use. All health events, including death, diagnosis and pharmaceutical use, had a specific date allowing more accurate and reliable determination of incident IHD than self-reports of IHD, and captured a wider range of IHD than death records used in previous studies.

Loss to follow-up

By using additional linked data sources available within the IDI, including migration and death data, complete follow-up and loss to follow-up could be determined with high accuracy in this study, which may have been unobtainable through manual follow-up.

7.3.2 Limitations

Small sample sizes

The NZWS and Māori NZWS are currently the only available occupational surveys in New Zealand, but for the purpose of studying occupational risk factors of IHD, they are relatively small. This is compounded by the fact that these samples contained a range of ages at baseline (20 upwards), there was a relatively short follow-up period (on average, 12.1 years for NZWS and 7.5 years for the Māori NZWS), and IHD events usually manifest in older adults (i.e. >45 years old in general population males; >55 years old in general population females; >30 years old in Māori males; >40 years old in Māori females[309]). As a consequence, the incidence of IHD was relatively low (4.7% NZWS and 4.8% Māori NZWS).

Therefore, statistical power was limited and analyses had to be restricted to broad occupational groups and industries. Furthermore, sensitivity analyses evaluating the associations within different age groups was not possible due to the small sample sizes. Due to the limited study power, it was not possible to formally test the differences between males and females or use interaction terms to assess the relationship between sex and occupation as the large confidence intervals associated with the point estimates of small samples may lead to false negatives. Additionally, the surveys could not be combined to increase study power and formally test differences between the general and Māori populations as the surveys were undertaken at different time points, with different response rates and follow-up periods. In particular, combining the surveys and restricting to a fixed-time period for both surveys would result in censoring approximately 25% of the incident IHD cases in the NZWS, thus reducing overall study power. Therefore, qualitative assessment of differences between population groups was the most appropriate method for the studies described in this thesis and findings suggest that it is likely there are differences between population groups. Concurrent work examining the risk of IHD and occupational information (albeit limited) from the 2013 Census for the entire employed population of NZ, which does not have the same study power limitations as the research presented in this thesis and aims to address some of these additional questions, is discussed in Section 7.4.1.

Low survey response rates

The survey response rates were low at 37% and 29% for the NZWS and Māori NZWS, respectively. Therefore, there is the possibility that participants who responded to the surveys are not representative of the Māori and general populations of New Zealand. However, previous work has evaluated whether the participants were representative of the source populations and found that potential response bias from relatively low survey response was small for both surveys.[26, 301] While some groups were underrepresented, the prevalence of key survey variables (both occupational exposure and health-related variables) were unchanged after standardising to the demographic distribution of the source population (using data from the Electoral Rolls) and similar between early and late responders. These comparisons between non-responders and early and late-responders were not possible for IDI health outcomes due to the confidentiality requirements of the IDI; however, non-response bias concerning IHD is unlikely as the outcome was incident IHD (i.e. measured after interview) and collected from health records rather than self-report.

Self-reported lifestyle and occupational information

The occupational and lifestyle information collected from the two workforce surveys were self-reported and therefore subjective, particularly for some of the physical exposures; for example 'carrying out repetitive tasks' could be interpreted as assembly line work in a factory and/or typing on a computer in an office. Therefore, the effect on the results of gender and ethnic differences in perception and reporting of exposures cannot be ruled out. However, the prevalences for certain occupational exposure reported in the NZWS were similar to self-reported estimates from overseas workforce surveys.[300] Also, the workforce surveys were carried out long before the conception of the current study with IHD as an outcome and thus any exposure misclassification is likely to be non-differential.

The occupational exposure information examined in Chapter 6 (e.g. physical, chemical, organisational and dust exposure in the workplace) was only available for the current or most recent occupation. As a result, the likely contribution of occupational exposures that occurred earlier in the participants' work history was not assessed. However, the availability of the full occupational history for all study participants provides future opportunities to apply information tools such as job-exposure-matrices (JEMs), which assign exposure based on occupation, and may therefore circumvent this limitation; this is discussed in Section 7.4.1.

Additionally, the lifestyle information collected at interview was limited and did not include information on other possible confounders, effect modifiers or mediators, as the surveys were not originally designed to assess CVD risk. These factors may include diet, alcohol intake, leisure-time physical activity and sleep duration, and their inclusion in the analysis may have allowed more valid modelling of the relationship between occupation or occupational exposures and IHD. However, several results are consistent with findings that have been previously reported in studies adjusting for these factors.

Linkage to IDI datasets

Linkage restrictions also contributed to the smaller sample size; 70 participants were excluded as NHI numbers could not be obtained and an additional 15 were excluded as they could not be linked to other datasets to determine loss to follow-up. Furthermore, linkage across IDI datasets introduces the potential of linkage errors. Statistics New Zealand establishes linkage through common unique identifiers, where available, or otherwise through probabilistic linkage based on overlapping personal and demographic variables.[308] As of the September 2019 refresh, the linkage rate of individuals between the spine (dataset to which all other datasets are linked) and the MoH dataset was 85.2% with a false positive rate of 1.1%.[348] The consequence of linkage error is the possibility of incorrect health records being matched to survey participants, however, due to the detailed identifying information collected in the original surveys, the chance of this is low. The greater issue is a mismatch in the linkage to the spine to identify loss to follow-up; if these records were not correctly matched and participants were censored incorrectly, the accuracy of IHD follow-up may have been affected, however, again, the likelihood of this is low due to the high linkage rate.

Coverage of health information

As previously mentioned, the identification of IHD relied on participant interactions with healthcare providers in New Zealand. Although public hospital records capture the majority (>95%) of CVD hospitalisations,[312] primary healthcare information is not currently available in the IDI and private hospitalisation information is incomplete and was therefore not used in the current research. The use of community pharmaceuticals dispensings in the IHD definition largely mitigates this issue as patients are often prescribed medication in the management of CVD regardless of where the initial diagnosis takes place. However, only pharmaceuticals that are prescribed exclusively, or close to exclusively, for CVD could be used in the IHD definition to maintain its specificity. The pharmaceuticals used in the IHD definition therefore only included anti-angina medication as these are solely used to treat the symptoms of IHD and did not include identification of patients on triple therapy (a common combination of antiplatelet/anticoagulant, blood pressure lowering and statin medication[311]) as these medications are also used to treat other conditions. Thus, IHD events may have been underestimated, potentially affecting the validity of associations observed. However, as anti-angina medications are routinely prescribed with IHD diagnosis,[310] the number of people solely on triple therapy is likely small, and therefore, the underestimation of IHD is probably minimal.

The start date of available data varies across different datasets (e.g. pharmaceuticals from 2005; hospital diagnoses from 1988) and the availability end-date changed with each refresh. Consequently, this means the health definitions are composed of multiple datasets that cover variable time ranges. Restricting analyses to a period where all datasets included in the definition had available data was not a viable option as it would substantially reduce the number of IHD cases and further reduced the already limited statistical power. However, the majority of IHD cases have been primarily identified through hospital admissions (73-80% across males and females of both surveys), which is the dataset with the longest range of available data, and only a small number of participants that were interviewed in the year 2004 (the start year of the general population survey) are affected by the limited pharmaceutical date range.

High blood pressure and high cholesterol in the IDI

For the conventional cardiovascular risk factor analyses in Chapter 4, participants were identified as having high cholesterol or high blood pressure if they had been prescribed statins or anti-hypertensives, respectively. However, these pharmaceuticals are also used in the management of other conditions. Therefore, this approach may have introduced a lack of specificity and the prevalence of high blood pressure and high cholesterol may be overestimated.

These medications are also often prescribed if a patient is classified as high cardiovascular risk, regardless of the presence of specific risk factors. Consequently, high blood pressure and high cholesterol identified using pharmaceutical dispensing would be highly correlated with the outcome in analyses assessing associations with IHD. However, this was not an issue as analyses (Chapter 5 and 6) were not controlled for these factors as they are likely on the causal pathway between occupation and IHD [21-23] and adjusting for metabolic factors and BMI may be an overadjustment.

Healthy worker effect

The healthy worker effect is common in occupational cohort studies.[349] In general, people who are ‘not healthy’ are less likely to be employed or remain in certain occupations (for example occupations with high physical demands), and workers with IHD symptoms may change their employment, leave work altogether or reduce tasks/exposures within the workplace. Therefore, reduced IHD risk for some occupations or exposures may be due to ‘unhealthy’ participants having never entered or moved out of these occupations, even prior to their first IHD event. The analysis of duration of employment for at least 10 years was one method to minimise the possibility for the healthy worker effect to impact the study results, as it is unlikely that symptoms that developed 10 years prior to a first IHD event would be severe enough to result in a job change. However, this strategy could not be used for occupational exposures as the survey did not collect the duration of exposure. Furthermore, the healthy worker effect is often not uniform across gender, ethnicities or age groups[349] and the different populations assessed in this thesis may therefore be affected to varying extents, potentially contributing to some of the differences observed between populations (as discussed in Section 7.2.3).

7.4 Implications

This is only the third study in New Zealand to examine the association between occupation and IHD risk (the first two being male mortality studies), and although more research is needed to further clarify the findings of this thesis, a number of implications follow from this body of work.

Firstly, the work in this thesis shows that people's occupation plays a role in their IHD risk, confirming that occupation and IHD should continue to be a focus of future research; occupation-based interventions may contribute to a reduced prevalence of cardiovascular risk factors and resultant IHD incidence in New Zealand.

Secondly, the findings of this thesis show a difference in occupational risk factors for IHD between both males and females, and for Māori, thus emphasising the relevance of considering sex and ethnicity in future work and the design of potential interventions.

Finally, this thesis has consistently highlighted blue-collar work as positively associated with IHD and that exposures such as dust, chemicals and physical factors, are also associated with an increased risk of IHD. Therefore, blue-collar work should be a particular focus in future work, especially as the presence of multiple occupational exposures appears to increase IHD risk.

The following sections will discuss the implications of the work presented in this thesis in the context of recommendations for future research and development of occupational exposure and public health interventions

7.4.1 Recommendations for future research

Although this research has confirmed that occupation plays a role in IHD risk in New Zealand, the limitations of this work and the current body of evidence mean that additional research is still needed. Based on the findings of this thesis, it is recommended that all future work analyse results separately for different ethnic groups and for males and females, allowing specific occupational risk factors for IHD to be identified for a range of populations.

Updates to the current research using both NZWS

Future IDI refreshes, which include additions of recent MoH data, will allow the identification of incident IHD cases for the NZWS cohorts over a longer follow-up period. This would be particularly useful for the Māori NZWS as it was conducted most recently and the shorter follow-up period contributed to reduced study power.

Additionally, JEMs could be applied to the work histories of participants of both NZWS to assess the relationship between IHD and ever being exposed to and/or the duration of exposure to occupational hazards. This could also include occupational exposures for which information was not collected as part of the surveys (i.e. using other available JEMs), as well as analysing sub-categories of exposure (e.g. type of dust or pesticide). The use of extensive MoH data also presents the possibility of analysing other health conditions, such as cancers or diabetes as an outcome, for the NZWS cohorts.

Analysis of census-based occupational information and IHD in the IDI

One major limitation of this thesis was the small sample size and hence, low IHD incidence. Therefore, assessing a much larger sample of working adults in future work would allow more detailed analyses and significantly extend the knowledge base. Although occupational information is limited in the IDI, current occupation is recorded in the Census, providing occupational information for all employed adults at one time point that can be linked to MoH health data. This approach is currently being used CPHR and it involves a cohort of all employed persons (n=2,166,444) identified from the 2013 Census (aged 20-64 years). The definition of IHD events is the same as used in this thesis i.e. hospitalisations, prescriptions and deaths (from 2013 to 2018). The large sample size enables the examination of the associations between IHD and NZSCO occupational groups up to the most specific 5-digit level. Preliminary results from this work appear to confirm the increased risk of IHD for *elementary workers* and *plant and machine assemblers and operators* observed in this thesis and the absence of associations between IHD and blue-collar occupations for Māori males.

Furthermore, the Census contains information about working hours and sedentary work, loud noise, and night shift are being studied using New Zealand-specific JEMs uploaded to the IDI; this involved 2-3 New Zealand experts using a variety of data sources to assess exposure levels for these three exposures for each 5-digit occupation in New Zealand. Preliminary results suggest an association between loud noise exposure and IHD, as was found in this thesis, and have additionally highlighted a relationship between night shift and IHD.

Using the Census in the IDI to provide occupational information provides greater statistical power as the entire employed population is included. A limitation of this approach is that it results in a significantly shorter follow-up time (2013-2018) and does not provide access to full occupational histories (only 2013 occupation is recorded in the Census). However, a Census approach would enable separate analyses for other ethnic groups, such as Pacific peoples and

Asians, and provide the statistical power required to formally tests population differences and the significance of interaction terms.

Mediation analyses

This thesis has presented evidence of associations but only presented hypothesised causal factors and intermediates in the relationship. Clarifying the role of some intermediary factors (e.g. high blood pressure, diabetes, diet and occupational exposures) with formal testing may provide useful insight into mechanisms to aid the identification of specific causal exposures and the development of effective interventions. Previous work has already identified some mediating factors; for example, psychological distress is a mediating factor in the relationship between job insecurity and IHD.[350] Another study, based on the NZWS data and currently underway at CPHR, is using mediation analyses to assess which occupational exposures may explain the associations observed between occupational groups and IHD, as well as the intermediate role of BMI. Preliminary findings support some of the hypothesises of this discussion, including a mediating role of repetitive tasks and awkward grip or hand movements in the IHD risk observed for *plant and machine operators and assemblers* and in *elementary occupations*. BMI also appears to play a mediating role in some of these associations. Similar studies on larger samples with information about physiological intermediates (e.g. high blood pressure, high cholesterol or diabetes) will further help to elucidate these causal pathways.

Multiple exposures

The findings of this thesis showed that exposure to multiple occupational factors had a more significant impact on IHD risk than being exposed to only one factor, which is of particular relevance for blue-collar occupations. To date, the literature examining interactions between occupational risk factors is limited and a greater focus on this in future research would be useful; principal component analysis is one statistical method that could be employed to investigate clustering of occupational exposures and the effect of this on IHD risk.

Occupational exposure measurements in the workplace

More information is required regarding the high-risk thresholds and specific aspects of the associations between occupational exposures and IHD. Therefore, conducting workplace exposure measurements may be one approach to provide a more quantitative assessment of relevant exposures in relation to IHD risk.

For occupational exposures such as dust, chemicals or loud noise, quantitative assessment may enable the identification of high-risk thresholds associated with increased IHD risk. Monitoring tasks being completed during peak exposure, parts of the body exposed and the PPE used to minimise exposure to these occupational risk factors will provide useful information to aid the development of interventions. Furthermore, occupational exposure measurements in the workplace may reduce exposure misclassification and help clarify some of the results found for ambiguous occupational exposures, such as repetitive tasks and awkward grip or hand movements.

Studies in specific workplaces would provide an opportunity for a closer look at occupations for which results were inconsistent across populations or for which the causal factors are unclear, such as *clerks* or *agriculture and fishery workers.* For example, for the latter, increased risks were observed for the agricultural group and industry among Māori females; however, no association with pesticides was found, a common exposure in agriculture work and previously linked to CVD.[273, 277] Observing and measuring all occupational exposures present in some of the high-risk occupations may identify differences in occupational exposures between demographic groups within the same occupation and highlight other exposures that have not previously been recognised as associated with CVD. This may be particularly important for Māori or females who may experience additional occupational exposures that were not assessed in the original surveys. For example, racism, bullying or discrimination in the workplace could be assessed using questionnaires or qualitative methods, such as focus groups or interviews, to determine their role in the association between occupation and IHD.

7.4.2 Implications for occupational exposure interventions

This thesis has indicated that occupation plays a role in IHD risk, supporting previous suggestions that IHD burden could be reduced with widespread changes within the workplace.[351] Current preventive cardiology is aimed at the individual, meaning improvements in cardiovascular health are dependent on individuals implementing healthy lifestyle choices; however, widening this individual-level approach to population-level interventions, such as occupation-based interventions, may be more effective. The benefits of using the workplace for CVD interventions are that a large proportion of the adult working population can be targeted before the disease develops, and the workplace is an environment that can be significantly influenced (e.g. reducing casual exposures in a workplace or changing workplace behaviours to minimise exposure to hazardous factors) and already contains a support network for the workers.[85] Thus, changes to workplace practices to reduce exposure to occupational risk factors presents a relatively novel opportunity for reducing IHD risk and the prevalence of cardiovascular risk factors.

In general, white-collar occupations have been the focus of the majority of CVD occupational interventions and minimising prolonged sitting has been a common focus.[352] However, this thesis and previous literature have demonstrated that BCW have the greatest risk of CVD and would benefit most from occupational interventions. Furthermore, low SES groups and ethnic minorities are often overrepresented in blue-collar occupations,[338, 353] therefore, targeting blue-collar worksites may in turn help to reduce health inequities experienced by these groups. The research in this thesis has highlighted a few occupational exposures to prioritise for the focus of future interventions, as described below. Although more research is required, the findings suggest that targeting those in blue-collar worksites specifically would be of the most benefit.

Occupational exposures that were associated with IHD in both the findings of this research and previous work include dust, loud noise, and smoke or fumes. Although further research is required for these risk factors, likely interventions to encourage a reduction of exposure at the source include stricter enforcement of, and in some cases a review of, workplace exposure standards, highlighting the health risks associated with these exposures to employers, through, for example, health and safety guides and other education material, as well as industry-led initiatives for priority industries. Encouraging workplaces to eliminate or substitute these hazardous exposures would be preferable, however, this is often not practical. Alternative options to minimise exposure may include physical barriers to protect workers from hazards, restricting exposure time and/or using PPE. As most PPE is not specifically designed for females, it may not be as effective for women, and should therefore be re-evaluated to ensure adequate protection for women.

Occupational physical exposures were also associated with IHD, including working at very high speeds, awkward or tiring positions, and carrying out repetitive tasks. However, these are novel findings and due to the limited level of detail in the questionnaire, it is not clear which specific tasks are associated with IHD risk. Fieldwork and specific analyses of high-risk occupations will help to elucidate this but at a relatively crude level, workplace changes may include regular rest periods for physically exerting jobs or switching tasks throughout the day to avoid prolonged repetition of tasks. Using health and safety guidelines and workplace education to highlight the risks associated with physical factors may help to change workplace behaviour and perception of physical activity.

The findings of this thesis suggest that occupational risk factors are not the same for all populations and this needs to be considered in the development and implementation of interventions to ensure health inequities are reduced, rather than increased. Māori and females are both populations that have not experienced the same extent of CVD mortality reduction as non-Māori males in response to current public health initiatives.[8, 9] Therefore, when designing and implementing workplace interventions, the occupational risk factors relevant to these populations must be considered. The following paragraphs discuss the relevant findings and intervention implications for these specific populations.

Females

While minimising prolonged sitting has been a common focus in occupational intervention studies for CVD,[352] the results presented in this thesis suggest that sitting at work may not be an important risk factor for IHD among women and interventions focusing on occupational sitting may therefore not benefit women. Notably, sitting was found to be associated with a reduced IHD risk for Māori women. For women, occupational exposure to physical factors (i.e. awkward grip or hand movements, carrying out repetitive tasks, and working at high speeds) were most consistently associated with IHD, yet currently, there are limited interventions and health and safety guidelines targeted at these physical risk factors. Knowledge of female-specific occupational risk factors from this thesis and future work will guide health and safety practices moving forward to re-evaluate current practices and develop future interventions in a gender-sensitive manner.

Māori

The lack of associations between occupation and occupational exposure and IHD for Māori males observed in this research suggests that Māori men may not benefit equally from occupation-based interventions. However, among Māori females working as a *plant and machine operator and assembler* and in *elementary occupations* was associated with an increased risk of IHD. Therefore, interventions based in blue-collar occupations may help reduce IHD among Māori women. Future research with larger groups of Māori workers may provide more information on occupational risk factors relevant to Māori males and the specific tasks that contribute to risk among Māori females, facilitating the development of occupational interventions that may be more specific for Māori.

7.4.3 Implications for general public health interventions

Although targeting the specific occupational exposures in the workplace is probably the most effective strategy, understanding there is a difference in cardiovascular risk factor distribution and IHD risk across occupations may help to more effectively direct general public health interventions. Public health services targeting CVD include education/media and policy/legislation changes, which are either targeted at whole communities or smaller groups, such as schools, religious organisations, worksites or healthcare facilities.[354] These initiatives have aimed to promote healthy lifestyle behaviours and have resulted in an overall reduction in smoking, high cholesterol and high blood pressure in the population; these reductions in cardiovascular risk factors account for about half the decline of CVD mortality observed in New Zealand and internationally.[6, 7] Using the workplace to facilitate these initiatives may be an efficient approach for targeting high-risk groups.

In New Zealand and internationally, improvements in health behaviours have been observed in worksites where health literacy and education programmes have been implemented.[85, 331, 355] Such interventions have included medical screening and cardiovascular risk assessments on worksites, education on diet and exercise, introductions of behaviour change incentives, and personal counselling.[356] Further development of health literacy and education programmes require integration into the structure of the workplace, environmental modifications to promote healthy behaviours, and must meet the relevant needs of the employees, be culturally sensitive and potentially provide incentives.[332] This thesis is the first step in identifying specific occupations within workplaces to target (e.g. *elementary workers*), the populations that may be of greatest risk in these occupations (e.g. Māori females), and cardiovascular risk factors that may be particularly relevant for those workers (e.g. smoking and diabetes).

In general, the findings of this thesis (and previous studies) have consistently shown that blue-collar workers have the highest prevalence of cardiovascular risk factors and are at the greatest risk of IHD, indicating that these occupations would benefit most from workplace interventions. Education and support programmes to encourage smoking cessation and healthy lifestyle behaviours (including improved diet, reduced alcohol intake, sufficient sleep and physical activity), in occupational groups such as *plant and machine operators and assemblers* or *elementary occupations,* could help to reduce the elevated cardiovascular risk factor prevalence in these occupations. Furthermore, Māori are often overrepresented in these occupations and in general have reduced access/interaction with general public health initiatives,[82, 357] therefore, Māori may particularly benefit from and should have input into, occupation-directed public health interventions.

7.5 Conclusions

The results described in this thesis strongly suggest that occupation plays a role in IHD risk and provide support for the following conclusions, which directly relate to the specific aims of this thesis (summarised in Chapter 1):

- There were differences in the distribution of conventional cardiovascular risk factors (high deprivation, smoking, obesity, high blood pressure, high cholesterol and diabetes) across occupational groups; BCW were more likely to experience these cardiovascular risk factors. (Aim 1);
- Differences in associations between occupational groups and IHD were observed, with positive associations most consistently demonstrated in blue-collar occupations such as *plant and machine operators and assemblers* and *elementary occupations.* These differences were not explained by high deprivation, smoking or age. (Aim 2);
- Occupational exposure to dust, chemicals and physical factors was associated with IHD; differences were not explained by high deprivation, smoking or age. Additionally, exposure to multiple dust and chemical factors was associated with increased IHD risk, as was exposure to multiple physical factors. (Aim 3);
- Associations between occupation and IHD were not consistent between males and females, with different results for occupational groups such as *clerks* and *service and sales workers*. (Aim 4);

- Associations between occupation and IHD were not consistent for the Māori and general populations, with particularly great differences for agricultural work and a general lack of associations observed in Māori males. (Aim 5).

In summary, these findings present potential high-risk occupational groups, occupational exposures and physiological pathways that could be targeted for occupation-based interventions in the future. Future work elucidating the mechanisms and clarifying salient occupational factors for a range of populations would be of considerable benefit to ensure effective application of knowledge. Although further investigations are required, current results indicate that physical factors and manual occupations may present the highest risk of IHD. As associations between occupation and IHD appear to differ between males and females and between the Māori and general populations, future research should assess the underlying reasons for these differences; an improved understanding of the role of co-exposures exposures such as racism and discrimination may be required to ensure health inequities reduce rather than increase with future occupational interventions.

References

1. Lozano R, Naghavi M, Foreman K, Lim S, Shibuya K, Aboyans V, et al. Global and regional mortality from 235 causes of death for 20 age groups in 1990 and 2010: a systematic analysis for the Global Burden of Disease Study 2010. Lancet 2012;380(9859):2095-128.
2. Ministry of Health. Mortality and demographic data 2013 (provisional). Wellington: Ministry of Health; 2015.
3. Gistera A, Hansson GK. The immunology of atherosclerosis. Nat Rev Nephrol 2017;13(6):368-80.
4. Libby P, Buring JE, Badimon L, Hansson GK, Deanfield J, Bittencourt MS, et al. Atherosclerosis. Nat Rev Dis Primers 2019;5(1):56.
5. D'Agostino RB, Sr., Pencina MJ, Massaro JM, Coady S. Cardiovascular disease risk assessment: insights from Framingham. Glob Heart 2013;8(1):11-23.
6. Ford ES, Ajani UA, Croft JB, Critchley JA, Labarthe DR, Kottke TE, et al. Explaining the decrease in U.S. deaths from coronary disease, 1980-2000. N Engl J Med 2007;356(23):2388-98.
7. Capewell S, Beaglehole R, Seddon M, McMurray J. Explanation for the decline in coronary heart disease mortality rates in Auckland, New Zealand, between 1982 and 1993. Circulation 2000;102(13):1511-6.
8. Bots SH, Peters SAE, Woodward M. Sex differences in coronary heart disease and stroke mortality: a global assessment of the effect of ageing between 1980 and 2010. BMJ Glob Health 2017;2(2):e000298-e.
9. Grey C, Jackson R, Wells S, Wu B, Poppe K, Harwood M, et al. Trends in ischaemic heart disease: patterns of hospitalisation and mortality rates differ by ethnicity (ANZACS-QI 21). N Z Med J 2018;131(1478):21-31.
10. Driscoll T. Work-related cardiovascular disease Australia. Australian Safety and Compensation Council 2006.
11. 't Mannetje A, Pearce N. Quantitative estimates of work-related death, disease and injury in New Zealand. Scand J Work Environ Health 2005;31(4):266-76.
12. Morris JN, Heady JA, Raffle PA, Roberts CG, Parks JW. Coronary heart-disease and physical activity of work. Lancet 1953;265(6796):1111-20; concl.
13. MacDonald LA, Bertke S, Hein MJ, Judd S, Baron S, Merritt R, et al. Prevalence of cardiovascular health by occupation: a cross-sectional analysis among U.S. workers aged >/=45 Years. Am J Prev Med 2017;53(2):152-61.
14. Elser H, Falconi AM, Bass M, Cullen MR. Blue-collar work and women's health: A systematic review of the evidence from 1990 to 2015. SSM Popul Health 2018;6:195-244.
15. Steenland K, Fine L, Belkic K, Landsbergis P, Schnall P, Baker D, et al. Research findings linking workplace factors to CVD outcomes. Occup Med 2000;15(1):7-68.
16. Pearce NE, Howard JK. Occupational mortality in New Zealand males 1974-78. Community Health Stud 1985;9(3):212-9.
17. Holmes E, Davies A, Wright C, Pearce N, Borman B. Mortality rates according to occupation in New Zealand males: 2001-2005. N Z Med J 2011;124(1328):16-28.

18. Kristensen TS. Cardiovascular diseases and the work environment. A critical review of the epidemiologic literature on nonchemical factors. Scand J Work Environ Health 1989;15(3):165-79.
19. Kristensen TS. Cardiovascular diseases and the work environment. A critical review of the epidemiologic literature on chemical factors. Scand J Work Environ Health 1989;15(4):245-64.
20. Hwang WJ, Hong O. Work-related cardiovascular disease risk factors using a socioecological approach: implications for practice and research. Eur J Cardiovasc Nurs 2012;11(1):114-26.
21. Sara JD, Prasad M, Eleid MF, Zhang M, Widmer RJ, Lerman A. Association between work-related stress and coronary heart disease: a review of prospective studies through the job strain, effort-reward balance, and organizational justice models. J Am Heart Assoc 2018;7(9).
22. Booth FW, Roberts CK, Laye MJ. Lack of exercise is a major cause of chronic diseases. Compr Physiol 2012;2(2):1143-211.
23. Münzel T, Schmidt FP, Steven S, Herzog J, Daiber A, Sørensen M. Environmental noise and the cardiovascular system. J Am Coll Cardiol 2018;71(6):688-97.
24. Ministry of Health. Maori health status indicators: cardiovascular disease. Wellington: Ministry of Health; 2015.
25. Ministry of Health. Annual update of key results 2018/19: New Zealand Health Survey 2019. Available at https://www.health.govt.nz/publication/annual-update-key-results-2018-19-new-zealand-health-survey Accessed 28 April 2020.
26. Denison HJ, Eng A, Barnes LA, Cheng S, 't Mannetje A, Haddock K, et al. Inequities in exposure to occupational risk factors between Maori and non-Maori workers in Aotearoa New Zealand. J Epidemiol Community Health 2018;72(9):809-16.
27. Eng A, 't Mannetje A, McLean D, Ellison-Loschmann L, Cheng S, Pearce N. Gender differences in occupational exposure patterns. Occup Environ Med 2011;68(12):888-94.
28. Simon M, Boutouyrie P, Narayanan K, Gaye B, Tafflet M, Thomas F, et al. Sex disparities in ideal cardiovascular health. Heart 2017;103(20):1595-601.
29. Park SM, Merz CN. Women and ischemic heart disease: recognition, diagnosis and management. Korean Circ J 2016;46(4):433-42.
30. World Health Organization. Global health estimates 2016: disease burden by cause, age, sex, by country and by region, 2000-2016. Geneva: World Health Organization; 2018.
31. World Health Organization. Cardiovascular diseases. Geneva: World Health Organization; 2017.
32. Ministry of Health. New Zealand Health Survey: Ischaemic heart disease 2019. Available at https://minhealthnz.shinyapps.io/nz-health-survey-2018-19-annual-data-explorer/_w_07361577/_w_adefcc8b/#!/home Accessed 22 March 2020.
33. Yusuf S, Hawken S, Ounpuu S, Dans T, Avezum A, Lanas F, et al. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. Lancet 2004;364(9438):937-52.
34. Khot UN, Khot MB, Bajzer CT, Sapp SK, Ohman EM, Brener SJ, et al. Prevalence of conventional risk factors in patients with coronary heart disease. JAMA 2003;290(7):898-904.
35. Tzoulaki I, Elliott P, Kontis V, Ezzati M. Worldwide exposures to cardiovascular risk factors and associated health effects: current knowledge and data gaps. Circulation 2016;133(23):2314-33.
36. Kane AE, Howlett SE. Differences in cardiovascular aging in men and women. Adv Exp Med Biol 2018;1065:389-411.

37. Cipriano G, Jr., Neves LM, Cipriano GF, Chiappa GR, Borghi-Silva A. Cardiovascular disease prevention and implications for worksite health promotion programs in Brazil. Prog Cardiovasc Dis 2014;56(5):493-500.
38. Rankinen T, Sarzynski MA, Ghosh S, Bouchard C. Are there genetic paths common to obesity, cardiovascular disease outcomes, and cardiovascular risk factors? Circ Res 2015;116(5):909-22.
39. Chan WC, Wright C, Riddell T, Wells S, Kerr AJ, Gala G, et al. Ethnic and socioeconomic disparities in the prevalence of cardiovascular disease in New Zealand. N Z Med J 2008;121(1285):11-20.
40. Kozakiewicz K, Podolecka E, Kwasniewska M, Drygas W, Pajak A, Tendera M. Association between socioeconomic status and cardiovascular risk. Kardiol Pol 2016;74(2):179-84.
41. Pickering T. Cardiovascular pathways: socioeconomic status and stress effects on hypertension and cardiovascular function. Ann N Y Acad Sci 1999;896:262-77.
42. Hiscock R, Bauld L, Amos A, Fidler JA, Munafo M. Socioeconomic status and smoking: a review. Ann N Y Acad Sci 2012;1248:107-23.
43. Bhatnagar A. Environmental determinants of cardiovascular disease. Circ Res 2017;121(2):162-80.
44. Messner B, Bernhard D. Smoking and cardiovascular disease: mechanisms of endothelial dysfunction and early atherogenesis. Arterioscler Thromb Vasc Biol 2014;34(3):509-15.
45. Torres N, Guevara-Cruz M, Velázquez-Villegas LA, Tovar AR. Nutrition and atherosclerosis. Arch Med Res 2015;46(5):408-26.
46. Leong DP, Smyth A, Teo KK, McKee M, Rangarajan S, Pais P, et al. Patterns of alcohol consumption and myocardial infarction risk: observations from 52 countries in the INTERHEART case-control study. Circulation 2014;130(5):390-8.
47. Klatsky AL. Alcohol and cardiovascular diseases: where do we stand today? J Intern Med 2015;278(3):238-50.
48. Toma A, Paré G, Leong DP. Alcohol and cardiovascular disease: how much is too much? Curr Atheroscler Rep 2017;19(3):13.
49. Piano MR. Alcohol's effects on the cardiovascular system. Alcohol Res 2017;38(2):219-41.
50. Lavie CJ, Ozemek C, Carbone S, Katzmarzyk PT, Blair SN. Sedentary behavior, exercise, and cardiovascular health. Circ Res 2019;124(5):799-815.
51. Huang PL. A comprehensive definition for metabolic syndrome. Dis Model Mech 2009;2(5-6):231-7.
52. Wu CY, Hu HY, Chou YJ, Huang N, Chou YC, Li CP. High blood pressure and all-cause and cardiovascular disease mortalities in community-dwelling older adults. Medicine 2015;94(47):e2160-e.
53. Foëx P, Sear JW. Hypertension: pathophysiology and treatment. Continuing Education in Anaesthesia Critical Care & Pain 2004;4(3):71-5.
54. Gao S, Liu J. Association between circulating oxidized low-density lipoprotein and atherosclerotic cardiovascular disease. Chronic Dis Transl Med 2017;3(2):89-94.
55. Sandesara PB, Virani SS, Fazio S, Shapiro MD. The forgotten lipids: triglycerides, remnant cholesterol, and atherosclerotic cardiovascular disease risk. Endocr Rev 2019;40(2):537-57.
56. Kosmas CE, Martinez I, Sourlas A, Bouza KV, Campos FN, Torres V, et al. High-density lipoprotein (HDL) functionality and its relevance to atherosclerotic cardiovascular disease. Drugs Context 2018;7:212525-.
57. Rodriguez-Araujo G, Nakagami H. Pathophysiology of cardiovascular disease in diabetes mellitus. Cardiovasc Endocrinol Metab 2018;7(1):4-9.

58. Dokken BB. The pathophysiology of cardiovascular disease and diabetes: beyond blood pressure and lipids. Diabetes Spectr 2008;21(3):160.
59. Zhu Y, Xian X, Wang Z, Bi Y, Chen Q, Han X, et al. Research progress on the relationship between atherosclerosis and inflammation. Biomolecules 2018;8(3):80.
60. Deeks SG, Overbaugh J, Phillips A, Buchbinder S. HIV infection. Nat Rev Dis Primers 2015;1:15035-.
61. Carrizales-Sepúlveda EF, Ordaz-Farías A, Vera-Pineda R, Flores-Ramírez R. Periodontal disease, systemic inflammation and the risk of cardiovascular disease. Heart Lung Circ 2018;27(11):1327-34.
62. Chen L, Deng H, Cui H, Fang J, Zuo Z, Deng J, et al. Inflammatory responses and inflammation-associated diseases in organs. Oncotarget 2017;9(6):7204-18.
63. Zinöcker MK, Lindseth IA. The western diet-microbiome-host interaction and its role in metabolic disease. Nutrients 2018;10(3):365.
64. Van Gaal LF, Mertens IL, De Block CE. Mechanisms linking obesity with cardiovascular disease. Nature 2006;444(7121):875-80.
65. Meldrum DR, Morris MA, Gambone JC. Obesity pandemic: causes, consequences, and solutions—but do we have the will? Fertil and Steril 2017;107(4):833-9.
66. Gao Z, Chen Z, Sun A, Deng X. Gender differences in cardiovascular disease. Med nov Technol Devices 2019;4:100025.
67. Ministry of Health. Mortality 2017 data tables. Wellington: Ministry of Health; 2019.
68. Lloyd-Jones DM, Larson MG, Beiser A, Levy D. Lifetime risk of developing coronary heart disease. Lancet 1999;353(9147):89-92.
69. Feldman RD. Heart disease in women: unappreciated challenges, GPER as a new target. Int J Mol Sci 2016;17(5):760.
70. Leening MJG, Ferket BS, Steyerberg EW, Kavousi M, Deckers JW, Nieboer D, et al. Sex differences in lifetime risk and first manifestation of cardiovascular disease: prospective population based cohort study. BMJ 2014;349:g5992.
71. Maas AHEM, Appelman YEA. Gender differences in coronary heart disease. Neth Heart J 2010;18(12):598-602.
72. Young L, Cho L. Unique cardiovascular risk factors in women. Heart 2019;105(21):1656-60.
73. Appelman Y, van Rijn BB, Ten Haaf ME, Boersma E, Peters SA. Sex differences in cardiovascular risk factors and disease prevention. Atherosclerosis 2015;241(1):211-8.
74. Colafella KMM, Denton KM. Sex-specific differences in hypertension and associated cardiovascular disease. Nat Rev Nephrol 2018;14(3):185-201.
75. Kip KE, Marroquin OC, Kelley DE, Johnson BD, Kelsey SF, Shaw LJ, et al. Clinical importance of obesity versus the metabolic syndrome in cardiovascular risk in women: a report from the Women's Ischemia Syndrome Evaluation (WISE) study. Circulation 2004;109(6):706-13.
76. Huxley R, Barzi F, Woodward M. Excess risk of fatal coronary heart disease associated with diabetes in men and women: meta-analysis of 37 prospective cohort studies. BMJ 2006;332(7533):73-8.
77. Backholer K, Peters SAE, Bots SH, Peeters A, Huxley RR, Woodward M. Sex differences in the relationship between socioeconomic status and cardiovascular disease: a systematic review and meta-analysis. J Epidemiol Community Health 2017;71(6):550-7.
78. Huxley RR, Woodward M. Cigarette smoking as a risk factor for coronary heart disease in women compared with men: a systematic review and meta-analysis of prospective cohort studies. Lancet 2011;378(9799):1297-305.
79. Gasevic D, Ross ES, Lear SA. Ethnic differences in cardiovascular disease risk factors: a systematic review of North American evidence. Can J Cardiol 2015;31(9):1169-79.

80. Kerr AJ, McLachlan A, Furness S, Broad J, Riddell T, Jackson R, et al. The burden of modifiable cardiovascular risk factors in the coronary care unit by age, ethnicity, and socioeconomic status--PREDICT CVD-9. N Z Med J 2008;121(1285):20-33.
81. Selak V, Poppe K, Grey C, Mehta S, Winter-Smith J, Jackson R, et al. Ethnic differences in cardiovascular risk profiles among 475,241 adults in primary care in Aotearoa, New Zealand. N Z Med J 2020;133(1521):14-27.
82. Ellison-Loschmann L, Pearce N. Improving access to health care among New Zealand's Maori population. Am J Public Health 2006;96(4):612-7.
83. World Health Organization. Global strategy on occupational health for all: The way to health at work. Geneva: World Health Organization; 1994.
84. Leigh JP, Miller TR. Job-related diseases and occupations within a large workers' compensation data set. Am J Ind Med 1998;33(3):197-211.
85. Novak B, Bullen C, Howden-Chapman P, Thornley S. Blue-collar workplaces: a setting for reducing heart health inequalities in New Zealand? N Z Med J 2007;120(1261):U2704.
86. Meneton P, Hoertel N, Wiernik E, Lemogne C, Ribet C, Bonenfant S, et al. Work environment mediates a large part of social inequalities in the incidence of several common cardiovascular risk factors: Findings from the Gazel cohort. Soc Sci Med 2018;216:59-66.
87. Statistics New Zealand. 2013 Census. Wellington: Statistics New Zealand; 2013.
88. Wadsworth E, Dhillon K, Shaw C, Bhui K, Stansfeld S, Smith A. Racial discrimination, ethnicity and work stress. Occup Med 2007;57(1):18-24.
89. Williams DR, Mohammed SA. Racism and health I: Pathways and scientific evidence. Am Behav Sci 2013;57(8).
90. Pereira MA, Kriska AM, Collins VR, Dowse GK, Tuomilehto J, Alberti KG, et al. Occupational status and cardiovascular disease risk factors in the rapidly developing, high-risk population of Mauritius. Am J Epidemiol 1998;148(2):148-59.
91. Nakamura S, Nakamura K, Tanaka M. Increased risk of coronary heart disease in Japanese blue-collar workers. Occup Med 2000;50(1):11-7.
92. Rosvall M, Ostergren PO, Hedblad B, Isacsson SO, Janzon L, Berglund G. Occupational status, educational level, and the prevalence of carotid atherosclerosis in a general population sample of middle-aged Swedish men and women: results from the Malmö Diet and Cancer Study. Am J Epidemiol 2000;152(4):334-46.
93. Gallo LC, Troxel WM, Matthews KA, Jansen-McWilliams L, Kuller LH, Sutton-Tyrrell K. Occupation and subclinical carotid artery disease in women: are clerical workers at greater risk? Health Psychol 2003;22(1):19-29.
94. Engström G, Hedblad B, Rosvall M, Janzon L, Lindgärde F. Occupation, marital status, and low-grade inflammation: mutual confounding or independent cardiovascular risk factors? Arterioscler Thromb Vasc Biol 2006;26(3):643-8.
95. Ainy E, Azizi F. Women, occupation and cardiovascular risk factors: findings from the Tehran Lipid and Glucose Study. Public Health 2007;121(12):950-3.
96. Sánchez Chaparro MA, Calvo Bonacho E, González Quintela A, Cabrera M, Sáinz JC, Fernández-Labander C, et al. High cardiovascular risk in Spanish workers. Nutr Metab Cardiovasc Dis 2011;21(4):231-6.
97. Allman-Farinelli MA, Chey T, Merom D, Bauman AE. Occupational risk of overweight and obesity: an analysis of the Australian Health Survey. J Occup Med Toxicol 2010;5:14.
98. Zimmermann Verdejo M, González Gómez MF, Galán Labaca I. [Cardiovascular risk profiles by occupation in Madrid region, Spain]. Rev Esp Salud Publica 2010;84(3):305-20.

99. Ham DC, Przybeck T, Strickland JR, Luke DA, Bierut LJ, Evanoff BA. Occupation and workplace policies predict smoking behaviors: analysis of national data from the current population survey. J Occup Environ Med 2011;53(11):1337-45.
100. Davis-Lameloise N, Philpot B, Janus ED, Versace VL, Laatikainen T, Vartiainen EA, et al. Occupational differences, cardiovascular risk factors and lifestyle habits in South Eastern rural Australia. BMC Public Health 2013;13:1090.
101. Lewin A, Pannier B, Méline J, Karusisi N, Thomas F, Chaix B. Residential neighborhood, geographic work environment, and work economic sector: associations with body fat measured by bioelectrical impedance in the RECORD Study. Ann Epidemiol 2014;24(3):180-6.
102. Strauss M, Foshag P, Przybylek B, Horlitz M, Lucia A, Sanchis-Gomar F, et al. Occupation and metabolic syndrome: is there correlation? A cross sectional study in different work activity occupations of German firefighters and office workers. Diabetol Metab Syndr 2016;8(1):57.
103. Shockey TM, Sussell AL, Odom EC. Cardiovascular health status by occupational group - 21 States, 2013. MMWR Morb Mortal Wkly Rep 2016;65(31):793-8.
104. Nam JY, Kim J, Cho KH, Choi Y, Choi J, Shin J, et al. Associations of sitting time and occupation with metabolic syndrome in South Korean adults: a cross-sectional study. BMC Public Health 2016;16:943.
105. Akintunde AA, Oloyede TW. Metabolic syndrome and occupation: Any association? Prevalence among auto technicians and school teachers in South West Nigeria. Diabetes Metab Syndr 2017;11 Suppl 1:S223-s7.
106. Lee W, Kim J. Prevalence of metabolic syndrome and related factors in Korean career firefighters in comparisons with other occupational groups. J Occup Environ Med 2017;59(4):384-8.
107. Aginsky KD, Constantinou D, Delport M, Watson ED. Cardiovascular disease risk profile and readiness to change in blue- and white-collar workers. Fam Community Health 2017;40(3):236-44.
108. Prihartono NA, Fitriyani F, Riyadina W. Cardiovascular disease risk factors among blue and white-collar workers in Indonesia. Acta Med Indones 2018;50(2):96-103.
109. Kim SH, Kim W, Yang S, Kwon S, Choi KH. Influence of occupation on sarcopenia, sarcopenic obesity, and metabolic syndrome in men over 65 years of age. J Occup Environ Med 2018;60(10):e512-e7.
110. Mehrdad R, Pouryaghoub G, Moradi M. Association between metabolic syndrome and job rank. Int J Occup Environ Med 2018;9(1):45-51.
111. Kelsall HL, Fernando PHS, Gwini SM, Sim MR. Cardiovascular disease and type 2 diabetes risk across occupational groups and industry in a statewide study of an Australian working population. J Occup Environ Med 2018;60(3):286-94.
112. Shen Y, Wang X, Wang Z, Zhang L, Chen Z, Zhu M, et al. Prevalence, awareness, treatment, and control of hypertension among Chinese working population: results of a workplace-based study. J Am Soc Hypertens 2018;12(4):311-22.e2.
113. Vandersmissen GJM, Schouteden M, Verbeek C, Bulterys S, Godderis L. Prevalence of high cardiovascular risk by economic sector. Int Arch Occup Environ Health 2020;93(1):133-42.
114. Won JU, Hong OS, Hwang WJ. Actual cardiovascular disease risk and related factors: a cross-sectional study of Korean blue collar workers employed by small businesses. Workplace Health Saf 2013;61(4):163-71.
115. Elshatarat RA, Burgel BJ. Cardiovascular risk factors of taxi drivers. J Urban Health 2016;93(3):589-606.
116. Kramer V, Adasme M, Bustamante MJ, Jalil J, Navarrete C, Acevedo M. [Cardiovascular risk factors in a group of health care workers]. Rev Med Chil 2012;140(5):601-8.

117. Haynes SG, Feinleib M. Women, work and coronary heart disease: prospective findings from the Framingham heart study. Am J Public Health 1980;70(2):133-41.
118. Aronson KJ, Howe GR, Carpenter M, Fair ME. Surveillance of potential associations between occupations and causes of death in Canada, 1965-91. Occup Environ Med 1999;56(4):265-9.
119. Calvert GM, Merling JW, Burnett CA. Ischemic heart disease mortality and occupation among 16- to 60-year-old males. J Occup Environ Med 1999;41(11):960-6.
120. Muntaner C, Sorlie P, O'Campo P, Johnson N, Backlund E. Occupational hierarchy, economic sector, and mortality from cardiovascular disease among men and women. Findings from the National Longitudinal Mortality Study. Ann Epidemiol 2001;11(3):194-201.
121. Malinauskiene V, Grazuleviciene R, Nieuwenhuijsen MJ, Azaraviciene A. Myocardial infarction risk and occupational categories in Kaunas 25-64 year old men. Occup Environ Med 2002;59(11):745-50.
122. Finkelstein MM, Verma DK, Sahai D, Stefov E. Ischemic heart disease mortality among heavy equipment operators. Am J Ind Med 2004;46(1):16-22.
123. Luckhaupt SE, Calvert GM. Prevalence of coronary heart disease or stroke among workers aged <55 years--United States, 2008-2012. MMWR Morb Mortal Wkly Rep 2014;63(30):645-9.
124. Fujishiro K, Diez Roux AV, Landsbergis P, Kaufman JD, Korcarz CE, Stein JH. Occupational characteristics and the progression of carotid artery intima-media thickness and plaque over 9 years: the Multi-Ethnic Study of Atherosclerosis (MESA). Occup Environ Med 2015;72(10):690-8.
125. Wada K, Eguchi H, Prieto-Merino D. Differences in stroke and ischemic heart disease mortality by occupation and industry among Japanese working-aged men. SSM Popul Health 2016;2:745-9.
126. Zaitsu M, Kato S, Kim Y, Takeuchi T, Sato Y, Kobayashi Y, et al. Occupational class and risk of cardiovascular disease incidence in Japan: nationwide, multicenter, hospital-based case-control study. J Am Heart Assoc 2019;8(6):e011350.
127. Kim K, Cho SI, Paek D. Association of discrimination and presenteeism with cardiovascular disease: the Fourth Korean Working Conditions Survey. Ann Occup Environ Med 2019;31:e28.
128. Tüchsen F, Andersen O, Costa G, Filakti H, Marmot MG. Occupation and ischemic heart disease in the European Community: a comparative study of occupations at potential high risk. Am J Ind Med 1996;30(4):407-14.
129. Clougherty JE, Souza K, Cullen MR. Work and its role in shaping the social gradient in health. Ann N Y Acad Sci 2010;1186:102-24.
130. Karasek RA, Jr. Job demands, job decision latitude, and mental strain: implications for job redesign. Adm Sci Q 1979;24(2):285-308.
131. Siegrist J. Adverse health effects of high-effort/low-reward conditions. J Occup Health Psychol 1996;1(1):27-41.
132. Kivimaki M, Virtanen M, Elovainio M, Kouvonen A, Vaananen A, Vahtera J. Work stress in the etiology of coronary heart disease--a meta-analysis. Scand J Work Environ Health 2006;32(6):431-42.
133. Kivimäki M, Kawachi I. Work stress as a risk factor for cardiovascular disease. Curr Cardiol Rep 2015;17(9):630.
134. Kivimaki M, Batty GD, Ferrie JE, Kawachi I. Cumulative meta-analysis of job strain and CHD. Epidemiology 2014;25(3):464-5.
135. Taouk Y, Spittal MJ, LaMontagne AD, Milner AJ. Psychosocial work stressors and risk of all-cause and coronary heart disease mortality: A systematic review and meta-analysis. Scand J Work Environ Health 2020;46(1):19-31.

136. Siegrist J, Peter R, Junge A, Cremer P, Seidel D. Low status control, high effort at work and ischemic heart disease: prospective evidence from blue-collar men. Soc Sci Med 1990;31(10):1127-34.
137. Bosma H, Marmot MG, Hemingway H, Nicholson AC, Brunner E, Stansfeld SA. Low job control and risk of coronary heart disease in Whitehall II (prospective cohort) study. BMJ 1997;314(7080):558-65.
138. Kuper H, Marmot M. Job strain, job demands, decision latitude, and risk of coronary heart disease within the Whitehall II study. J Epidemiol Community Health 2003;57(2):147-53.
139. Chandola T, Britton A, Brunner E, Hemingway H, Malik M, Kumari M, et al. Work stress and coronary heart disease: what are the mechanisms? Eur Heart J 2008;29(5):640-8.
140. Eaker ED, Sullivan LM, Kelly-Hayes M, D'Agostino RB, Sr., Benjamin EJ. Does job strain increase the risk for coronary heart disease or death in men and women? The Framingham Offspring Study. Am J Epidemiol 2004;159(10):950-8.
141. Dragano N, Siegrist J, Nyberg ST, Lunau T, Fransson EI, Alfredsson L, et al. Effort-reward imbalance at work and incident coronary heart disease: a multicohort study of 90,164 individuals. Epidemiology 2017;28(4):619-26.
142. Xu W, Zhao Y, Guo L, Guo Y, Gao W. The association between effort-reward imbalance and coronary atherosclerosis in a Chinese sample. Am J Ind Med 2010;53(7):655-61.
143. Eddy P, Wertheim EH, Kingsley M, Wright BJ. Associations between the effort-reward imbalance model of workplace stress and indices of cardiovascular health: A systematic review and meta-analysis. Neurosci Biobehav Rev 2017;83:252-66.
144. Slopen N, Glynn RJ, Buring JE, Lewis TT, Williams DR, Albert MA. Job strain, job insecurity, and incident cardiovascular disease in the Women's Health Study: results from a 10-year prospective study. PLoS One 2012;7(7):e40512.
145. Lee S, Colditz G, Berkman L, Kawachi I. A prospective study of job strain and coronary heart disease in US women. Int J Epidemiol 2002;31(6):1147-54.
146. Theorell T, Jood K, Jarvholm LS, Vingard E, Perk J, Ostergren PO, et al. A systematic review of studies in the contributions of the work environment to ischaemic heart disease development. Eur J Public Health 2016;26(3):470-7.
147. Backé EM, Seidler A, Latza U, Rossnagel K, Schumann B. The role of psychosocial stress at work for the development of cardiovascular diseases: a systematic review. Int Arch Occup Environ Health 2012;85(1):67-79.
148. Belkic KL, Landsbergis PA, Schnall PL, Baker D. Is job strain a major source of cardiovascular disease risk? Scand J Work Environ Health 2004;30(2):85-128.
149. Padkapayeva K, Gilbert-Ouimet M, Bielecky A, Ibrahim S, Mustard C, Brisson C, et al. Gender/sex differences in the relationship between psychosocial work exposures and work and life Stress. Ann Work Expo Health 2018;62(4):416-25.
150. Lucas B, Grayson S, Hamidu H, Han A, No S, Varghese A, et al. Sex differences in heart rate responses to occupational stress. Stress 2020;23(1):13-8.
151. Russell G, Lightman S. The human stress response. Nat Rev Endocrinol 2019;15(9):525-34.
152. Kivimaki M, Steptoe A. Effects of stress on the development and progression of cardiovascular disease. Nat Rev Cardiol 2018;15(4):215-29.
153. Steptoe A, Kivimaki M. Stress and cardiovascular disease. Nat Rev Cardiol 2012;9(6):360-70.
154. Djindjic N, Jovanovic J, Djindjic B, Jovanovic M, Jovanovic JJ. Associations between the occupational stress index and hypertension, type 2 diabetes mellitus, and lipid disorders in middle-aged men and women. Ann Occup Hyg 2012;56(9):1051-62.
155. Feaster M, Krause N. Job strain associated with increases in ambulatory blood and pulse pressure during and after work hours among female hotel room cleaners. Am J Ind Med 2018;61(6):492-503.

156. Gilbert-Ouimet M, Trudel X, Brisson C, Milot A, Vézina M. Adverse effects of psychosocial work factors on blood pressure: systematic review of studies on demand-control-support and effort-reward imbalance models. Scand J Work Environ Health 2014;40(2):109-32.
157. Thosar SS, Butler MP, Shea SA. Role of the circadian system in cardiovascular disease. J Clin Invest 2018;128(6):2157-67.
158. Kivimaki M, Jokela M, Nyberg ST, Singh-Manoux A, Fransson EI, Alfredsson L, et al. Long working hours and risk of coronary heart disease and stroke: a systematic review and meta-analysis of published and unpublished data for 603,838 individuals. Lancet 2015;386(10005):1739-46.
159. Virtanen M, Kivimäki M. Long Working Hours and Risk of Cardiovascular Disease. Curr Cardiol Rep 2018;20(11):123.
160. Kang MY, Park H, Seo JC, Kim D, Lim YH, Lim S, et al. Long working hours and cardiovascular disease: a meta-analysis of epidemiologic studies. J Occup Environ Med 2012;54(5):532-7.
161. Virtanen M, Heikkila K, Jokela M, Ferrie JE, Batty GD, Vahtera J, et al. Long working hours and coronary heart disease: a systematic review and meta-analysis. Am J Epidemiol 2012;176(7):586-96.
162. Rivera AS, Akanbi M, O'Dwyer LC, McHugh M. Shift work and long work hours and their association with chronic health conditions: A systematic review of systematic reviews with meta-analyses. PLoS One 2020;15(4):e0231037.
163. Hannerz H, Larsen AD, Garde AH. Long weekly working hours and ischaemic heart disease: a follow-up study among 145 861 randomly selected workers in Denmark. BMJ Open 2018;8(6):e019807.
164. Alicandro G, Bertuccio P, Sebastiani G, La Vecchia C, Frova L. Long working hours and cardiovascular mortality: a census-based cohort study. Int J Public Health 2020;65(3):257-66.
165. Sokejima S, Kagamimori S. Working hours as a risk factor for acute myocardial infarction in Japan: case-control study. BMJ 1998;317(7161):775-80.
166. Liu Y, Tanaka H. Overtime work, insufficient sleep, and risk of non-fatal acute myocardial infarction in Japanese men. Occup Environ Med 2002;59(7):447-51.
167. Cheng Y, Du CL, Hwang JJ, Chen IS, Chen MF, Su TC. Working hours, sleep duration and the risk of acute coronary heart disease: a case-control study of middle-aged men in Taiwan. Int J Cardiol 2014;171(3):419-22.
168. Lee W, Kang YJ, Kim T, Choi J, Kang MY. The impact of working hours on cardiovascular diseases and moderating effects of sex and type of work: results from a longitudinal analysis of the Korean working population. J Occup Environ Med 2019;61(6):e247-e52.
169. Jackson CL, Redline S, Kawachi I, Williams MA, Hu FB. Racial disparities in short sleep duration by occupation and industry. Am J Epidemiol 2013;178(9):1442-51.
170. Liu H, Chen A. Roles of sleep deprivation in cardiovascular dysfunctions. Life Sciences 2019;219:231-7.
171. Knutson KL, Ryden AM, Mander BA, Van Cauter E. Role of sleep duration and quality in the risk and severity of type 2 diabetes mellitus. Arch Intern Med 2006;166(16):1768-74.
172. Gangwisch JE, Heymsfield SB, Boden-Albala B, Buijs RM, Kreier F, Pickering TG, et al. Short sleep duration as a risk factor for hypertension: analyses of the first National Health and Nutrition Examination Survey. Hypertension 2006;47(5):833-9.
173. Grandner MA, Sands-Lincoln MR, Pak VM, Garland SN. Sleep duration, cardiovascular disease, and proinflammatory biomarkers. Nat Sci Sleep 2013;5:93-107.
174. Reynolds AC, Bucks RS, Paterson JL, Ferguson SA, Mori TA, McArdle N, et al. Working (longer than) 9 to 5: are there cardiometabolic health risks for young Australian

workers who report longer than 38-h working weeks? Int Arch Occup Environ Health 2018;91(4):403-12.

175. Yang H, Schnall PL, Jauregui M, Su TC, Baker D. Work hours and self-reported hypertension among working people in California. Hypertension 2006;48(4):744-50.

176. Kivimäki M, Virtanen M, Kawachi I, Nyberg ST, Alfredsson L, Batty GD, et al. Long working hours, socioeconomic status, and the risk of incident type 2 diabetes: a meta-analysis of published and unpublished data from 222 120 individuals. Lancet Diabetes Endocrinol 2015;3(1):27-34.

177. Yu J. Relationship between long working hours and metabolic syndrome among Korean workers. Asian Nursing Research 2017;11(1):36-41.

178. Schluter PJ, Turner C, Benefer C. Long working hours and alcohol risk among Australian and New Zealand nurses and midwives: A cross-sectional study. Int J Nurs Stud 2012;49(6):701-9.

179. Knutsson A, Akerstedt T, Jonsson BG, Orth-Gomer K. Increased risk of ischaemic heart disease in shift workers. Lancet 1986;2(8498):89-92.

180. Boggild H, Knutsson A. Shift work, risk factors and cardiovascular disease. Scand J Work Environ Health 1999;25(2):85-99.

181. Vyas MV, Garg AX, Iansavichus AV, Costella J, Donner A, Laugsand LE, et al. Shift work and vascular events: systematic review and meta-analysis. BMJ 2012;345:e4800.

182. Torquati L, Mielke GI, Brown WJ, Kolbe-Alexander T. Shift work and the risk of cardiovascular disease. A systematic review and meta-analysis including dose-response relationship. Scand J Work Environ Health 2018;44(3):229-38.

183. Cheng M, He H, Wang D, Xu L, Wang B, Ho KM, et al. Shift work and ischaemic heart disease: meta-analysis and dose-response relationship. Occup Med 2019;69(3):182-8.

184. Kawachi I, Colditz GA, Stampfer MJ, Willett WC, Manson JE, Speizer FE, et al. Prospective study of shift work and risk of coronary heart disease in women. Circulation 1995;92(11):3178-82.

185. Vetter C, Devore EE, Wegrzyn LR, Massa J, Speizer FE, Kawachi I, et al. Association between rotating night shift work and risk of coronary heart disease among women. JAMA 2016;315(16):1726-34.

186. Jørgensen JT, Karlsen S, Stayner L, Andersen J, Andersen ZJ. Shift work and overall and cause-specific mortality in the Danish nurse cohort. Scand J Work Environ Health 2017;43(2):117-26.

187. Wang D, Ruan W, Chen Z, Peng Y, Li W. Shift work and risk of cardiovascular disease morbidity and mortality: A dose-response meta-analysis of cohort studies. Eur J Prev Cardiol 2018;25(12):1293-302.

188. Karlsson B, Alfredsson L, Knutsson A, Andersson E, Torén K. Total mortality and cause-specific mortality of Swedish shift- and dayworkers in the pulp and paper industry in 1952-2001. Scand J Work Environ Health 2005;31(1):30-5.

189. Hublin C, Partinen M, Koskenvuo K, Silventoinen K, Koskenvuo M, Kaprio J. Shift-work and cardiovascular disease: a population-based 22-year follow-up study. Eur J Epidemiol 2010;25(5):315-23.

190. Frost P, Kolstad HA, Bonde JP. Shift work and the risk of ischemic heart disease - a systematic review of the epidemiologic evidence. Scand J Work Environ Health 2009;35(3):163-79.

191. Glen Pyle W, Martino TA. Circadian rhythms influence cardiovascular disease differently in males and females: role of sex and gender. Curr Opin Physiol 2018;5:30-7.

192. Bhatti P, Mirick DK, Davis S. Racial differences in the association between night shift work and melatonin levels among women. Am J Epidemiol 2013;177(5):388-93.

193. Ceïde ME, Pandey A, Ravenell J, Donat M, Ogedegbe G, Jean-Louis G. Associations of short sleep and shift work status with hypertension among black and white Americans. Int J Hypertens 2015;2015:697275.
194. Puttonen S, Harma M, Hublin C. Shift work and cardiovascular disease - pathways from circadian stress to morbidity. Scand J Work Environ Health 2010;36(2):96-108.
195. Amano H, Fukuda Y, Yokoo T, Yamaoka K. Interleukin-6 Level among Shift and Night Workers in Japan: Cross-Sectional Analysis of the J-HOPE Study. J Atheroscler Thromb 2018;25(12):1206-14.
196. Hanprathet N, Lertmaharit S, Lohsoonthorn V, Rattananupong T, Ammaranond P, Jiamjarasrangsi W. Shift work and leukocyte count changes among workers in Bangkok. Ann Work Expo Health 2019;63(6):689-700.
197. Scheer FA, Hilton MF, Mantzoros CS, Shea SA. Adverse metabolic and cardiovascular consequences of circadian misalignment. Proc Natl Acad Sci U S A 2009;106(11):4453-8.
198. Ritonja J, Aronson KJ, Day AG, Korsiak J, Tranmer J. Investigating cortisol production and pattern as mediators in the relationship between shift work and cardiometabolic risk. Can J Cardiol 2018;34(5):683-9.
199. Tucker P, Härmä M, Ojajärvi A, Kivimäki M, Leineweber C, Oksanen T, et al. Associations between shift work and use of prescribed medications for the treatment of hypertension, diabetes, and dyslipidemia: a prospective cohort study. Scand J Work Environ Health 2019;45(5):465-74.
200. Ferguson JM, Costello S, Neophytou AM, Balmes JR, Bradshaw PT, Cullen MR, et al. Night and rotational work exposure within the last 12 months and risk of incident hypertension. Scand J Work Environ Health 2019;45(3):256-66.
201. Antunes LC, Levandovski R, Dantas G, Caumo W, Hidalgo MP. Obesity and shift work: chronobiological aspects. Nutr Res Rev 2010;23(1):155-68.
202. van Drongelen A, Boot CR, Merkus SL, Smid T, van der Beek AJ. The effects of shift work on body weight change - a systematic review of longitudinal studies. Scand J Work Environ Health 2011;37(4):263-75.
203. Hulsegge G, Picavet HSJ, van der Beek AJ, Verschuren WMM, Twisk JW, Proper KI. Shift work, chronotype and the risk of cardiometabolic risk factors. Eur J Public Health 2019;29(1):128-34.
204. Pan A, Schernhammer ES, Sun Q, Hu FB. Rotating night shift work and risk of type 2 diabetes: two prospective cohort studies in women. PLoS Med 2011;8(12):e1001141.
205. Esquirol Y, Perret B, Ruidavets JB, Marquie JC, Dienne E, Niezborala M, et al. Shift work and cardiovascular risk factors: new knowledge from the past decade. Arch Cardiovasc Dis 2011;104(12):636-68.
206. Proper KI, van de Langenberg D, Rodenburg W, Vermeulen RCH, van der Beek AJ, van Steeg H, et al. The relationship between shift work and metabolic risk factors: a systematic review of longitudinal studies. Am J Prev Med 2016;50(5):e147-e57.
207. Wang F, Zhang L, Zhang Y, Zhang B, He Y, Xie S, et al. Meta-analysis on night shift work and risk of metabolic syndrome. Obes Rev 2014;15(9):709-20.
208. Hermansson J, Bøggild H, Hallqvist J, Karlsson B, Knutsson A, Nilsson T, et al. Interaction between shift work and established coronary risk factors. Int J Occup Environ Med 2019;10(2):57-65.
209. Souza RV, Sarmento RA, de Almeida JC, Canuto R. The effect of shift work on eating habits: a systematic review. Scand J Work Environ Health 2019;45(1):7-21.
210. Rosa D, Terzoni S, Dellafiore F, Destrebecq A. Systematic review of shift work and nurses' health. Occup Med 2019;69(4):237-43.
211. Jay SM, Gander PH, Eng A, Cheng S, Douwes J, Ellison-Loschmann L, et al. New Zealanders working non-standard hours also have greater exposure to other workplace hazards. Chronobiol Int 2017;34(4):519-26.

212. Dzhambov AM, Dimitrova DD. Occupational noise and ischemic heart disease: A systematic review. Noise Health 2016;18(83):167-77.
213. Skogstad M, Johannessen HA, Tynes T, Mehlum IS, Nordby KC, Lie A. Systematic review of the cardiovascular effects of occupational noise. Occup Med 2016;66(1):10-6.
214. Teixeira LR, Pega F, Dzhambov AM, Bortkiewicz A, da Silva DTC, de Andrade CAF, et al. The effect of occupational exposure to noise on ischaemic heart disease, stroke and hypertension: A systematic review and meta-analysis from the WHO/ILO Joint Estimates of the Work-Related Burden of Disease and Injury. Environ Int 2021;154:106387.
215. Eriksson HP, Andersson E, Schioler L, Soderberg M, Sjostrom M, Rosengren A, et al. Longitudinal study of occupational noise exposure and joint effects with job strain and risk for coronary heart disease and stroke in Swedish men. BMJ Open 2018;8(4):e019160.
216. Pettersson H, Olsson D, Järvholm B. Occupational exposure to noise and cold environment and the risk of death due to myocardial infarction and stroke. Int Arch Occup Environ Health 2020.
217. Gan WQ, Davies HW, Demers PA. Exposure to occupational noise and cardiovascular disease in the United States: the National Health and Nutrition Examination Survey 1999-2004. Occup Environ Med 2011;68(3):183-90.
218. Davies HW, Teschke K, Kennedy SM, Hodgson MR, Hertzman C, Demers PA. Occupational exposure to noise and mortality from acute myocardial infarction. Epidemiology 2005;16(1):25-32.
219. Virkkunen H, Kauppinen T, Tenkanen L. Long-term effect of occupational noise on the risk of coronary heart disease. Scand J Work Environ Health 2005;31(4):291-9.
220. Gopinath B, Thiagalingam A, Teber E, Mitchell P. Exposure to workplace noise and the risk of cardiovascular disease events and mortality among older adults. Prev Med 2011;53(6):390-4.
221. Münzel T, Gori T, Babisch W, Basner M. Cardiovascular effects of environmental noise exposure. Eur Heart J 2014;35(13):829-36.
222. Tomei G, Fioravanti M, Cerratti D, Sancini A, Tomao E, Rosati MV, et al. Occupational exposure to noise and the cardiovascular system: a meta-analysis. Sci Total Environ 2010;408(4):681-9.
223. Yang Y, Zhang E, Zhang J, Chen S, Yu G, Liu X, et al. Relationship between occupational noise exposure and the risk factors of cardiovascular disease in China: A meta-analysis. Medicine 2018;97(30):e11720.
224. Arlien-Søborg MC, Schmedes AS, Stokholm ZA, Grynderup MB, Bonde JP, Jensen CS, et al. Ambient and at-the-ear occupational noise exposure and serum lipid levels. Int Arch Occup Environ Health 2016;89(7):1087-93.
225. Stokholm ZA, Hansen Å M, Grynderup MB, Bonde JP, Christensen KL, Frederiksen TW, et al. Recent and long-term occupational noise exposure and salivary cortisol level. Psychoneuroendocrinology 2014;39:21-32.
226. Kerns E, Masterson EA, Themann CL, Calvert GM. Cardiovascular conditions, hearing difficulty, and occupational noise exposure within US industries and occupations. Am J Ind Med 2018;61(6):477-91.
227. Lin CY, Tsai PJ, Lin KY, Chen CY, Chung LH, Wu JL, et al. Will daytime occupational noise exposures induce nighttime sleep disturbance? Sleep Med 2018;50:87-96.
228. Bagheri Hosseinabadi M, Khanjani N, Münzel T, Daiber A, Yaghmorloo M. Chronic occupational noise exposure: Effects on DNA damage, blood pressure, and serum biochemistry. Mutat Res 2019;841:17-22.
229. Dzhambov AM, Dimitrova DD. Long-term self-reported exposure to occupational noise is associated with BMI-defined obesity in the US general population. Am J Ind Med 2016;59(11):1009-19.

230. Raum E, Rothenbacher D, Ziegler H, Brenner H. Heavy physical activity: risk or protective factor for cardiovascular disease? A life course perspective. Ann Epidemiol 2007;17(6):417-24.
231. Moe B, Mork PJ, Holtermann A, Nilsen TI. Occupational physical activity, metabolic syndrome and risk of death from all causes and cardiovascular disease in the HUNT 2 cohort study. Occup Environ Med 2013;70(2):86-90.
232. Church TS, Thomas DM, Tudor-Locke C, Katzmarzyk PT, Earnest CP, Rodarte RQ, et al. Trends over 5 decades in U.S. occupation-related physical activity and their associations with obesity. PLoS One 2011;6(5):e19657.
233. van der Ploeg HP, Hillsdon M. Is sedentary behaviour just physical inactivity by another name? Int J Behav Nutr Phys Act 2017;14(1):142.
234. Thorp AA, Owen N, Neuhaus M, Dunstan DW. Sedentary behaviors and subsequent health outcomes in adults a systematic review of longitudinal studies, 1996-2011. Am J Prev Med 2011;41(2):207-15.
235. Biswas A, Oh PI, Faulkner GE, Bajaj RR, Silver MA, Mitchell MS, et al. Sedentary time and its association with risk for disease incidence, mortality, and hospitalization in adults: a systematic review and meta-analysis. Ann Intern Med 2015;162(2):123-32.
236. Wilmot EG, Edwardson CL, Achana FA, Davies MJ, Gorely T, Gray LJ, et al. Sedentary time in adults and the association with diabetes, cardiovascular disease and death: systematic review and meta-analysis. Diabetologia 2012;55(11):2895-905.
237. Pandey A, Salahuddin U, Garg S, Ayers C, Kulinski J, Anand V, et al. Continuous dose-response association between sedentary time and risk for cardiovascular disease: a meta-analysis. JAMA Cardiol 2016;1(5):575-83.
238. Bjørk Petersen C, Bauman A, Grønbæk M, Wulff Helge J, Thygesen LC, Tolstrup JS. Total sitting time and risk of myocardial infarction, coronary heart disease and all-cause mortality in a prospective cohort of Danish adults. Int J Behav Nutr Phys Act 2014;11:13.
239. van Uffelen JG, Wong J, Chau JY, van der Ploeg HP, Riphagen I, Gilson ND, et al. Occupational sitting and health risks: a systematic review. Am J Prev Med 2010;39(4):379-88.
240. Stamatakis E, Chau JY, Pedisic Z, Bauman A, Macniven R, Coombs N, et al. Are sitting occupations associated with increased all-cause, cancer, and cardiovascular disease mortality risk? A pooled analysis of seven British population cohorts. PLoS One 2013;8(9):e73753.
241. Brierley ML, Chater AM, Smith LR, Bailey DP. The effectiveness of sedentary behaviour reduction workplace interventions on cardiometabolic risk markers: a systematic review. Sports Med 2019;49(11):1739-67.
242. Poggio R, Melendi S, Gutierrez L, Elorriaga N, Irazola V. Occupational physical activity and cardiovascular risk factors profile in the adult population of the Southern Cone of Latin America: results from the CESCAS I Study. J Occup Environ Med 2018;60(9):e470-e5.
243. Moller SV, Hannerz H, Hansen AM, Burr H, Holtermann A. Multi-wave cohort study of sedentary work and risk of ischemic heart disease. Scand J Work Environ Health 2016;42(1):43-51.
244. Smith P, Ma H, Glazier RH, Gilbert-Ouimet M, Mustard C. The relationship between occupational standing and sitting and incident Heart disease over a 12-year period in Ontario, Canada. Am J Epidemiol 2018;187(1):27-33.
245. Saidj M, Jørgensen T, Jacobsen RK, Linneberg A, Aadahl M. Separate and joint associations of occupational and leisure-time sitting with cardio-metabolic risk factors in working adults: a cross-sectional study. PLoS One 2013;8(8):e70213.

246. Holtermann A, Marott JL, Gyntelberg F, Søgaard K, Suadicani P, Mortensen OS, et al. Does the benefit on survival from leisure time physical activity depend on physical activity at work? A prospective cohort study. PLoS One 2013;8(1):e54548.
247. Ekelund U, Steene-Johannessen J, Brown WJ, Fagerland MW, Owen N, Powell KE, et al. Does physical activity attenuate, or even eliminate, the detrimental association of sitting time with mortality? A harmonised meta-analysis of data from more than 1 million men and women. Lancet 2016;388(10051):1302-10.
248. Healy GN, Matthews CE, Dunstan DW, Winkler EA, Owen N. Sedentary time and cardio-metabolic biomarkers in US adults: NHANES 2003-06. Eur Heart J 2011;32(5):590-7.
249. Joseph JJ, Echouffo-Tcheugui JB, Golden SH, Chen H, Jenny NS, Carnethon MR, et al. Physical activity, sedentary behaviors and the incidence of type 2 diabetes mellitus: the Multi-Ethnic Study of Atherosclerosis (MESA). BMJ Open Diabetes Res Care 2016;4(1):e000185.
250. Pinto Pereira SM, Ki M, Power C. Sedentary behaviour and biomarkers for cardiovascular disease and diabetes in mid-life: the role of television-viewing and sitting at work. PLoS One 2012;7(2):e31132.
251. Eriksen D, Rosthøj S, Burr H, Holtermann A. Sedentary work--associations between five-year changes in occupational sitting time and body mass index. Prev Med 2015;73:1-5.
252. Thijssen DH, Maiorana AJ, O'Driscoll G, Cable NT, Hopman MT, Green DJ. Impact of inactivity and exercise on the vasculature in humans. Eur J Appl Physiol 2010;108(5):845-75.
253. Carter S, Hartman Y, Holder S, Thijssen DH, Hopkins ND. Sedentary behavior and cardiovascular disease risk: mediating mechanisms. Exerc Sport Sci Rev 2017;45(2):80-6.
254. Blond K, Brinkløv CF, Ried-Larsen M, Crippa A, Grøntved A. Association of high amounts of physical activity with mortality risk: a systematic review and meta-analysis. Br J Sports Med 2019.
255. Myers J, Kokkinos P, Nyelin E. Physical Activity, cardiorespiratory fitness, and the metabolic syndrome. Nutrients 2019;11(7).
256. Hamer M, Stamatakis E. Physical activity and risk of cardiovascular disease events: inflammatory and metabolic mechanisms. Med Sci Sports Exerc 2009;41(6):1206-11.
257. Coenen P, Huysmans MA, Holtermann A, Krause N, van Mechelen W, Straker LM, et al. Do highly physically active workers die early? A systematic review with meta-analysis of data from 193 696 participants. Br J Sports Med 2018;52(20):1320-6.
258. Li J, Loerbroks A, Angerer P. Physical activity and risk of cardiovascular disease: what does the new epidemiological evidence show? Curr Opin Cardiol 2013;28(5):575-83.
259. Krause N, Brand RJ, Kaplan GA, Kauhanen J, Malla S, Tuomainen TP, et al. Occupational physical activity, energy expenditure and 11-year progression of carotid atherosclerosis. Scand J Work Environ Health 2007;33(6):405-24.
260. Allesøe K, Holtermann A, Aadahl M, Thomsen JF, Hundrup YA, Søgaard K. High occupational physical activity and risk of ischaemic heart disease in women: the interplay with physical activity during leisure time. Eur J Prev Cardiol 2015;22(12):1601-8.
261. Wanner M, Lohse T, Braun J, Cabaset S, Bopp M, Krause N, et al. Occupational physical activity and all-cause and cardiovascular disease mortality: Results from two longitudinal studies in Switzerland. Am J Ind Med 2019;62(7):559-67.
262. Harari G, Green MS, Zelber-Sagi S. Combined association of occupational and leisure-time physical activity with all-cause and coronary heart disease mortality among a cohort of men followed-up for 22 years. Occup Environ Med 2015;72(9):617-24.

263. Holtermann A, Mortensen OS, Burr H, Sogaard K, Gyntelberg F, Suadicani P. Physical demands at work, physical fitness, and 30-year ischaemic heart disease and all-cause mortality in the Copenhagen Male Study. Scand J Work Environ Health 2010;36(5):357-65.
264. Holtermann A, Marott JL, Gyntelberg F, Søgaard K, Mortensen OS, Prescott E, et al. Self-reported occupational physical activity and cardiorespiratory fitness: Importance for cardiovascular disease and all-cause mortality. Scand J Work Environ Health 2016;42(4):291-8.
265. Hallman DM, Mathiassen SE, Gupta N, Korshøj M, Holtermann A. Differences between work and leisure in temporal patterns of objectively measured physical activity among blue-collar workers. BMC Public Health 2015;15:976.
266. Holtermann A, Krause N, van der Beek AJ, Straker L. The physical activity paradox: six reasons why occupational physical activity (OPA) does not confer the cardiovascular health benefits that leisure time physical activity does. Br J Sports Med 2018;52(3):149-50.
267. Zelzer S, Tatzber F, Herrmann M, Wonisch W, Rinnerhofer S, Kundi M, et al. Work intensity, low-grade inflammation, and oxidative status: a comparison between office and slaughterhouse workers. Oxid Med Cell Longev 2018;2018:2737563.
268. Steenland K. Epidemiology of occupation and coronary heart disease: research agenda. Am J Ind Med 1996;30(4):495-9.
269. Bulka CM, Daviglus ML, Persky VW, Durazo-Arvizu RA, Lash JP, Elfassy T, et al. Association of occupational exposures with cardiovascular disease among US Hispanics/Latinos. Heart 2019;105(6):439-48.
270. Zhuang X, Guo Y, Ni A, Yang D, Liao L, Zhang S, et al. Toward a panoramic perspective of the association between environmental factors and cardiovascular disease: An environment-wide association study from National Health and Nutrition Examination Survey 1999–2014. Environ Int 2018;118:146-53.
271. Humblet O, Birnbaum L, Rimm E, Mittleman MA, Hauser R. Dioxins and cardiovascular disease mortality. Environ Health Perspect 2008;116(11):1443-8.
272. Gustavsson P, Plato N, Hallqvist J, Hogstedt C, Lewne M, Reuterwall C, et al. A population-based case-referent study of myocardial infarction and occupational exposure to motor exhaust, other combustion products, organic solvents, lead, and dynamite. Stockholm Heart Epidemiology Program (SHEEP) Study Group. Epidemiology 2001;12(2):222-8.
273. Berg ZK, Rodriguez B, Davis J, Katz AR, Cooney RV, Masaki K. Association between occupational exposure to pesticides and cardiovascular disease incidence: the Kuakini Honolulu Heart Program. J Am Heart Assoc 2019;8(19):e012569.
274. Nigra AE, Ruiz-Hernandez A, Redon J, Navas-Acien A, Tellez-Plaza M. Environmental metals and cardiovascular disease in adults: a systematic review beyond lead and cadmium. Curr Environ Health Rep 2016;3(4):416-33.
275. Costello S, Attfield MD, Lubin JH, Neophytou AM, Blair A, Brown DM, et al. Ischemic heart disease mortality and diesel exhaust and respirable dust exposure in the diesel exhaust in miners study. Am J Epidemiol 2018;187(12):2623-32.
276. Brinchmann BC, Le Ferrec E, Podechard N, Lagadic-Gossmann D, Holme JA, Øvrevik J. Organic chemicals from diesel exhaust particles affects intracellular calcium, inflammation and β-adrenoceptors in endothelial cells. Toxicol Lett 2019;302:18-27.
277. Nicolopoulou-Stamati P, Maipas S, Kotampasi C, Stamatis P, Hens L. Chemical pesticides and human health: the urgent need for a new concept in agriculture. Front Public Health 2016;4:148.
278. Hung DZ, Yang HJ, Li YF, Lin CL, Chang SY, Sung FC, et al. The long-term effects of organophosphates poisoning as a risk factor of CVDs: a nationwide population-based cohort study. PLoS One 2015;10(9):e0137632-e.

279. Mostafalou S, Abdollahi M. Pesticides and human chronic diseases: evidences, mechanisms, and perspectives. Toxicol Appl Pharmacol 2013;268(2):157-77.
280. Navas-Acien A, Guallar E, Silbergeld EK, Rothenberg SJ. Lead exposure and cardiovascular disease—a systematic review. Environ Health Perspect 2007;115(3):472-82.
281. Vaziri ND. Mechanisms of lead-induced hypertension and cardiovascular disease. Am J Physiol Heart Circ Physiol 2008;295(2):H454-65.
282. Poręba R, Gać P, Poręba M, Andrzejak R. Environmental and occupational exposure to lead as a potential risk factor for cardiovascular disease. Environ Toxicol Pharmacol 2011;31(2):267-77.
283. Martinelli N, Olivieri O, Girelli D. Air particulate matter and cardiovascular disease: A narrative review. Eur J Intern Med 2013;24(4):295-302.
284. Hamanaka RB, Mutlu GM. Particulate Matter Air Pollution: Effects on the Cardiovascular System. Front Endocrinol (Lausanne) 2018;9:680.
285. Fiordelisi A, Piscitelli P, Trimarco B, Coscioni E, Iaccarino G, Sorriento D. The mechanisms of air pollution and particulate matter in cardiovascular diseases. Heart Fail Rev 2017;22(3):337-47.
286. Westberg H, Elihn K, Andersson E, Persson B, Andersson L, Bryngelsson IL, et al. Inflammatory markers and exposure to airborne particles among workers in a Swedish pulp and paper mill. Int Arch Occup Environ Health 2016;89(5):813-22.
287. Fang SC, Cassidy A, Christiani DC. A systematic review of occupational exposure to particulate matter and cardiovascular disease. Int J Environ Res Public Health 2010;7(4):1773-806.
288. Sjögren B. Occupational exposure to dust: inflammation and ischaemic heart disease. Occup Environ Med 1997;54(7):466-9.
289. Torén K, Bergdahl IA, Nilsson T, Järvholm B. Occupational exposure to particulate air pollution and mortality due to ischaemic heart disease and cerebrovascular disease. Occup Environ Med 2007;64(8):515-9.
290. Krajnak K. Health effects associated with occupational exposure to hand-arm or whole body vibration. J Toxicol Environ Health B Crit Rev 2018;21(5):320-34.
291. Bjor B, Burstrom L, Nilsson T, Reuterwall C. Vibration exposure and myocardial infarction incidence: the VHEEP case-control study. Occup Med 2006;56(5):338-44.
292. Virkkunen H, Harma M, Kauppinen T, Tenkanen L. The triad of shift work, occupational noise, and physical workload and risk of coronary heart disease. Occup Environ Med 2006;63(6):378-86.
293. Selander J, Bluhm G, Nilsson M, Hallqvist J, Theorell T, Willix P, et al. Joint effects of job strain and road-traffic and occupational noise on myocardial infarction. Scand J Work Environ Health 2013;39(2):195-203.
294. Golmohammadi R, Darvishi E. The combined effects of occupational exposure to noise and other risk factors - a systematic review. Noise Health 2019;21(101):125-41.
295. Skogstad M, Mamen A, Lunde LK, Ulvestad B, Matre D, Aass HCD, et al. Shift work including night work and long working hours in industrial plants increases the risk of atherosclerosis. Int J Environ Res Public Health 2019;16(3).
296. Steenland K, Johnson J, Nowlin S. A follow-up study of job strain and heart disease among males in the NHANES1 population. Am J Ind Med 1997;31(2):256-60.
297. Melamed S, Kristal-Boneh E, Froom P. Industrial noise exposure and risk factors for cardiovascular disease: findings from the CORDIS Study. Noise Health 1999;1(4):49-56.
298. Xu S, Huang Y, Xiao J, Zhu W, Wang L, Tang H, et al. The association between job strain and coronary heart disease: a meta-analysis of prospective cohort studies. Ann Med 2015;47(6):512-8.
299. Anand S, Bradshaw C, Prabhakaran D. Prevention and management of CVD in LMICs: why do ethnicity, culture, and context matter? BMC Med 2020;18(1):7.

300. Eng A, 't Mannetje A, Cheng S, Douwes J, Ellison-Loschmann L, McLean D, et al. The New Zealand workforce survey I: self-reported occupational exposures. Ann Occup Hyg 2010;54(2):144-53.
301. 't Mannetje A, Eng A, Douwes J, Ellison-Loschmann L, McLean D, Pearce N. Determinants of non-response in an occupational exposure and health survey in New Zealand. Aust N Z J Public Health 2011;35(3):256-63.
302. Statistics New Zealand. 2018 Census. Wellington: Statistics New Zealand; 2018.
303. Statistics New Zealand. New Zealand standard classification of occupations, 1999. Wellington: Statistics New Zealand; 2001.
304. Statistics New Zealand. Australian and New Zealand standard industrial classification. Wellington: Statistics New Zealand; 1996.
305. Statistics New Zealand. Integrated Data Infrastructure 2017. Available at https://www.stats.govt.nz/integrated-data/integrated-data-infrastructure/ Accessed 6 December 2018.
306. Black A. The IDI prototype spine's creation and coverage. (Statistics New Zealand Working Paper No 16-03). Statistics New Zealand 2016.
307. Milne BJ, Atkinson J, Blakely T, Day H, Douwes J, Gibb S, et al. Data resource profile: the New Zealand integrated data ifrastructure (IDI). Int J Epidemiol 2019;48(3):677-e.
308. Statistics New Zealand. Linking methodology used by Statistics New Zealand in the Integrated Data Infrastructure project. Wellington: Statistics New Zealand; 2014.
309. Ministry of Health. Cardiovascular disease risk assessment and management for primary care. Wellington: Ministry of Health; 2018.
310. National Health Committee. Ischaemic heart disease: a pathway to priorisation. Wellington: National Health Committee; 2014.
311. Kerr A, Exeter D, Hanham G, Grey C, Zhao J, Riddell T, et al. Effect of age, gender, ethnicity, socioeconomic status and region on dispensing of CVD secondary prevention medication in New Zealand: the Atlas of Health Care Variation CVD cohort (VIEW-1). N Z Med J 2014;127(1400):39-69.
312. Wells S, Riddell T, Kerr A, Pylypchuk R, Chelimo C, Marshall R, et al. Cohort profile: The PREDICT cardiovascular disease cohort in New Zealand primary care (PREDICT-CVD 19). Int J Epidemiol 2017;46(1):22.
313. Thornley S, Chan WC, Crengle S, Riddell T, Ameratunga S, Mehta S, et al. Sociodemographic differences in prevalence of diagnosed coronary heart disease in New Zealand estimated from linked national health records. N Z Med J 2011;124(1334):21-34.
314. Statistic New Zealand. IDI Data Dictionary: Chronic conditions/significant health event cohort (November 2015 edition). Wellington: Statistic New Zealand; 2015.
315. Jo EC, Drury PL. Development of a Virtual Diabetes Register using information technology in New Zealand. Healthc Inform Res 2015;21(1):49-55.
316. Peng M, Chen G, Kaplan GG, Lix LM, Drummond N, Lucyk K, et al. Methods of defining hypertension in electronic medical records: validation against national survey data. J Public Health 2016;38(3):e392-e9.
317. Christensen DB, Williams B, Goldberg HI, Martin DP, Engelberg R, LoGerfo JP. Assessing compliance to antihypertensive medications using computer-based pharmacy records. Med Care 1997;35(11):1164-70.
318. Tu K, Campbell NR, Chen ZL, Cauch-Dudek KJ, McAlister FA. Accuracy of administrative databases in identifying patients with hypertension. Open Med 2007;1(1):e18-26.
319. Barnes LA, Eng A, Corbin M, Denison HJ, 't Mannetje A, Haslett S, et al. The prevalence of cardiovascular risk factors in different occupational groups in New Zealand. Ann Work Expo Health 2020;64(6):645-58.
320. Fang N, Jiang M, Fan Y. Ideal cardiovascular health metrics and risk of cardiovascular disease or mortality: A meta-analysis. Int J Cardiol 2016;214:279-83.

321. Gao Y, Xie X, Wang SX, Li H, Tang HZ, Zhang J, et al. Effects of sedentary occupations on type 2 diabetes and hypertension in different ethnic groups in North West China. Diab Vasc Dis Res 2017;14(4):372-5.
322. World Health Organization. Obesity: preventing and managing the global epidemic Report of a WHO Consultation on Obesity, Geneva, 1997 (WHO/NUT/NCD/981). Geneva: World Health Organization; 2000.
323. Pearce N. Effect measures in prevalence studies. Environ Health Perspect 2004;112(10):1047-50.
324. van Kempen EE, Kruize H, Boshuizen HC, Ameling CB, Staatsen BA, de Hollander AE. The association between noise exposure and blood pressure and ischemic heart disease: a meta-analysis. Environ Health Perspect 2002;110(3):307-17.
325. St George A, King L, Newson R, Wells V, Campbell M. Implementing workplace health promotion initiatives: who should we target? Health Promot J Austr 2012;23(2):134-40.
326. Varela-Mato V, Yates T, Stensel DJ, Biddle SJH, Clemes SA. Time spent sitting during and outside working hours in bus drivers: A pilot study. Prev Med Rep 2015;3:36-9.
327. Marqueze EC, Ulhoa MA, Moreno CR. Irregular working times and metabolic disorders among truck drivers: a review. Work 2012;41 Suppl 1:3718-25.
328. Puhkala J, Kukkonen-Harjula K, Aittasalo M, Mansikkamäki K, Partinen M, Hublin C, et al. Lifestyle counseling in overweight truck and bus drivers - effects on dietary patterns and physical activity. Prev Med Rep 2016;4:435-40.
329. Riddell T, Jackson RT, Wells S, Broad J, Bannink L. Assessing Maori/non-Maori differences in cardiovascular disease risk and risk management in routine primary care practice using web-based clinical decision support: (PREDICT CVD-2). N Z Med J 2007;120(1250):U2445.
330. Ljungvall Å, Gerdtham UG, Lindblad U. Misreporting and misclassification: implications for socioeconomic disparities in body-mass index and obesity. Eur J Health Econ 2015;16(1):5-20.
331. Cook C, Simmons G, Swinburn B, Stewart J. Changing risk behaviours for non-communicable disease in New Zealand working men--is workplace intervention effective? N Z Med J 2001;114(1130):175-8.
332. Carnethon M, Whitsel LP, Franklin BA, Kris-Etherton P, Milani R, Pratt CA, et al. Worksite wellness programs for cardiovascular disease prevention: a policy statement from the American Heart Association. Circulation 2009;120(17):1725-41.
333. Chaturvedi N. Ethnic differences in cardiovascular disease. Heart 2003;89(6):681-6.
334. Berdahl JL, Moore C. Workplace harassment: double jeopardy for minority women. J Appl Psychol 2006;91(2):426-36.
335. van Doorn D, Richardson N, Osborne A. Farmers have hearts: the prevalence of risk factors for cardiovascular disease among a subgroup of Irish livestock farmers. J Agromedicine 2017;22(3):264-74.
336. Wiemers PD, Marney L, Yadav S, Tam R, Fraser JF. An overview of Indigenous Australian disadvantage in terms of ischaemic heart disease. Heart Lung Circ 2018;27(11):1274-84.
337. Liebler C. Occupational dissimilarity between the American Indian/Alaska Native workforce and white workforce in the contemporary United States. Am Indian Cult Res J 2018;42(1):41-70.
338. Ministry of Business, Innovation and Employment. Maori in the Labour Market. Wellington: Ministry of Business, Innovation and Employment; 2017.
339. Virtanen M, Nyberg ST, Batty GD, Jokela M, Heikkilä K, Fransson EI, et al. Perceived job insecurity as a risk factor for incident coronary heart disease: systematic review and meta-analysis. BMJ 2013;347:f4746.

340. Landsbergis PA, Grzywacz JG, LaMontagne AD. Work organization, job insecurity, and occupational health disparities. Am J Ind Med 2014;57(5):495-515.
341. Shufelt CL, Pacheco C, Tweet MS, Miller VM. Sex-specific physiology and cardiovascular disease. Adv Exp Med Biol 2018;1065:433-54.
342. Wilcosky TC, Simonsen NR. Solvent exposure and cardiovascular disease. Am J Ind Med 1991;19(5):569-86.
343. Petersen CB, Eriksen L, Tolstrup JS, Søgaard K, Grønbaek M, Holtermann A. Occupational heavy lifting and risk of ischemic heart disease and all-cause mortality. BMC Public Health 2012;12:1070.
344. Statistics New Zealand. Household Labour Force Survey. Wellington: Statistics New Zealand; 2017.
345. 't Mannetje A, Slater T, McLean D, Eng A, Briar B, Douwes J. NOHSAC Technicial Report 13: Women's occupational health and safety in New Zealand. National Occupational Health and Safety Advisory Committee; 2009.
346. Garcia M, Mulvagh SL, Merz CNB, Buring JE, Manson JE. Cardiovascular Disease in Women: Clinical Perspectives. Circulation research 2016;118(8):1273-93.
347. Eng A, 't Mannetje A, Ellison-Loschmann L, McLean D, Cheng S, Pearce N. Ethnic differences in patterns of occupational exposure in New Zealand. Am J Ind Med 2011;54(5):410-8.
348. Kvalvig A, Gibb S, Teng A. Linkage error and linkage bias: a guide for IDI users. University of Otago Wellington; 2019.
349. Chowdhury R, Shah D, Payal AR. Healthy worker effect phenomenon: revisited with emphasis on statistical methods - a review. Indian J Occup Environ Med 2017;21(1):2-8.
350. Magnusson Hanson LL, Rod NH, Vahtera J, Virtanen M, Ferrie J, Shipley M, et al. Job insecurity and risk of coronary heart disease: Mediation analyses of health behaviors, sleep problems, physiological and psychological factors. Psychoneuroendocrinology 2020;118:104706.
351. Olsen O, Kristensen TS. Impact of work environment on cardiovascular diseases in Denmark. J Epidemiol Community Health 1991;45(1):4-9; discussion -10.
352. Ferrario MM, Landsbergis P, Tsutsumi A, Li J, Hynek P, Krause N, et al. Work environment: An opportunity for ground-breaking collaborations in cardiovascular disease prevention. Eur J Prev Cardiol 2017;24(2_suppl):4-6.
353. Fahy KM, Lee A, Milne BJ. Statistics New Zealand. New Zealand socio-economic index 2013; 2017.
354. Ducher M, Cerutti C, Chatellier G, Fauvel JP. Is high job strain associated with hypertension genesis? Am J Hypertens 2006;19(7):694-700.
355. Bourgette-Henry SJ, Davis A, Flood J, Choi SY, Bourgette A. The Wahine Heart Wellness Program: a community approach to reducing women's cardiovascular disease risk. Hawaii J Health Soc Welf 2019;78(11):341-8.
356. Pelletier KR. Clinical and cost outcomes of multifactorial, cardiovascular risk management interventions in worksites: a comprehensive review and analysis. J Occup Environ Med 1997;39(12):1154-69.
357. Miner-Williams W. Racial inequities in cardiovascular disease in New Zealand. Divers Equal Health Care 2017;14(1):23-33.

Appendix

Statements of contribution

DRC 16

MASSEY UNIVERSITY
GRADUATE RESEARCH SCHOOL

STATEMENT OF CONTRIBUTION
DOCTORATE WITH PUBLICATIONS/MANUSCRIPTS

We, the candidate and the candidate's Primary Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of candidate:	Lucy Barnes
Name/title of Primary Supervisor:	Professor Jeroen Douwes
Name of Research Output and full reference:	
The prevalence of cardiovascular risk factors in different occupational groups in New Zealand	
In which Chapter is the Manuscript /Published work:	Chapter 4
Please indicate:	
• The percentage of the manuscript/Published Work that was contributed by the candidate:	90%
and	
• Describe the contribution that the candidate has made to the Manuscript/Published Work:	
Formulated the concept, assisted with data extraction, carried out all statistical analyses and prepared the manuscript.	
For manuscripts intended for publication please indicate target journal:	
Annals of Work Exposures and Health	
Candidate's Signature:	LBarnes
Date:	28/01/2021
Primary Supervisor's Signature:	[signature]
Date:	28/01/21

(This form should appear at the end of each thesis chapter/section/appendix submitted as a manuscript/ publication or collected as an appendix at the end of the thesis)

GRS Version 4– January 2019

DRC 16

MASSEY UNIVERSITY
GRADUATE RESEARCH SCHOOL

STATEMENT OF CONTRIBUTION
DOCTORATE WITH PUBLICATIONS/MANUSCRIPTS

We, the candidate and the candidate's Primary Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of candidate:	Lucy Barnes
Name/title of Primary Supervisor:	Professor Jeroen Douwes
Name of Research Output and full reference:	
A longitudinal linkage study of occupation and ischaemic heart disease in the general and Maori populations of New Zealand	
In which Chapter is the Manuscript /Published work:	Chapter 5
Please indicate:	
• The percentage of the manuscript/Published Work that was contributed by the candidate:	90%
and	
• Describe the contribution that the candidate has made to the Manuscript/Published Work:	
Formulated the concept, assisted with data extraction, carried out all statistical analyses and prepared the manuscript.	
For manuscripts intended for publication please indicate target journal:	
Annals of Work Exposures and Health	
Candidate's Signature:	LBarnes
Date:	28/01/2021
Primary Supervisor's Signature:	[signature]
Date:	28/01/21

(This form should appear at the end of each thesis chapter/section/appendix submitted as a manuscript/ publication or collected as an appendix at the end of the thesis)

GRS Version 4– January 2019

DRC 16

MASSEY UNIVERSITY
GRADUATE RESEARCH SCHOOL

STATEMENT OF CONTRIBUTION
DOCTORATE WITH PUBLICATIONS/MANUSCRIPTS

We, the candidate and the candidate's Primary Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of candidate:	Lucy Barnes
Name/title of Primary Supervisor:	Professor Jeroen Douwes
Name of Research Output and full reference:	
Ischaemic heart disease and occupational exposures - a longitudinal linkage study in the general and Maori populations of New Zealand	
In which Chapter is the Manuscript /Published work:	Chapter 6
Please indicate:	
• The percentage of the manuscript/Published Work that was contributed by the candidate:	90%
and	
• Describe the contribution that the candidate has made to the Manuscript/Published Work:	
Formulated the concept, assisted with data extraction, carried out all statistical analyses and prepared the manuscript.	
For manuscripts intended for publication please indicate target journal:	
Annals of Work Exposures and Health	
Candidate's Signature:	LHBarnes
Date:	28/01/2021
Primary Supervisor's Signature:	[signature]
Date:	28/01/2021

(This form should appear at the end of each thesis chapter/section/appendix submitted as a manuscript/ publication or collected as an appendix at the end of the thesis)

GRS Version 4– January 2019