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A study of the effect of adverse psychosocial work stressors on health and mortality

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"To criticise inequality and to desire equality is not, as is sometimes suggested, to cherish the romantic illusion that men are equal in character and intelligence. It is to hold that, while their natural endowments differ profoundly, it is the mark of a civilised society to aim at eliminating such inequalities as have their source not in individual differences but in (social) organisation."

Richard Henry Tawney 1931, Equality

Michael Marmot 2004, The Status Syndrome: How Social Standing Affects Our Health and Longevity

Abstract

The working environment is central in the lives of individuals in employment, influencing health outcomes including psychological and physical well-being. Psychosocial work stressors are common exposures in the workplace and are important and modifiable determinants of health and health behaviours. There is broad agreement in the literature that exposure to adverse psychosocial work stressors, such as high job demands, low job control, low job security and high effort-reward imbalance are associated with poor health outcomes. All of these exposures are, in turn, associated with unhealthy behaviours such as smoking, alcohol consumption, poor diet and inactivity, and to the development of hypertension, cardiovascular disease, diabetes, musculoskeletal disorders and depressive disorders. Psychosocial work stressors have been identified as significant emerging risks linked to global changes in the structure, organisation and management of work during previous decades, presenting pressing challenges to occupational health and safety and workplace health, in general. Most of the literature in this area assesses disease incidence or other morbidity outcomes, with a growing number of studies focusing on mortality.

However, whether exposure to these psychosocial work stressors associated with adverse health outcomes translates into increased mortality remains unclear, and barriers to making causal interpretations about the relationship between psychosocial work stressors and health persist, mostly due to inherent biases in the methodology across studies. In this thesis, the effect of exposures to psychosocial work stressors in the working environment such as job control, job demands, job strain, long working hours, job insecurity and shift work on health and mortality were investigated. A comprehensive systematic review including meta-analyses of the epidemiological research was conducted. Panel data from the Household, Income and Labour Dynamics in Australia survey was used to capture natural experiments of psychosocial work stressors associated changes in health and well-being, and mortality to investigate

whether and how the effect of psychosocial work stressors on mortality differ in relation to demographic and socioeconomic characteristics. The initial focus of the research included establishing and quantifying the risks associated with adverse psychosocial work stressors, which workers are commonly exposed to in the workplace, on mortality. The final study in the PhD focused on understanding the exposure-outcome dynamics. The dynamics of the connexions between psychosocial work stressor perceived job control and general health, a strong predictor of future morbidity and mortality, were investigated for evidence of a causal relationship.

The first study showed that improving the quality of work might contribute to better health and well-being and decrease the effect of psychosocial work stressors on all-cause mortality and cardiovascular disease mortality. If this observed association is causal, then policy and practice interventions to improve job control could contribute to reductions in mortality. Study II adds to the growing body of evidence showing an effect of adverse psychosocial work stressors on mortality. Study III showed that long-term exposure to low job control and job security was associated with increased risk of all-cause mortality. In Study IV, the dynamics of job control and general health were explored for evidence of a causal relationship using three complementary longitudinal modelling approaches. This study, using improved causal inference methods than previous research, showed that increased job control was strongly associated with increasing general health.

Although the estimates for the single measures suggest modest increases in the risk of death and poor health, this translates to large population impacts because the exposures are relatively common across the working population. Thus, reducing exposure to psychosocial work stressors associated with mortality and poor health could have considerable public health benefit. Awareness by stakeholders, government and organisations of the implications of the adverse effects of psychosocial work stressors on health and mortality in workplaces; and the

availability appropriate, evidence-based work stress interventions to reduce the exposure to work stressors might contribute to better health and well-being, reduce sickness absence and presenteeism to the benefit of workers, workplaces and society.

Declaration

This is to certify that

i. the thesis comprises only my original work towards the PhD, except where indicated in the preface;

ii. due acknowledgment has been made in the text to all other material used;

iii. the thesis is fewer than the maximum 100,000-word limit in length, exclusive of tables, maps, bibliographies and appendices.

_____

Yamna Taouk

21/01/2021

Preface

This thesis is submitted as a thesis with publication. My thesis chapters include three published first author papers and one submitted manuscript. All four papers are primary publications that report on original research conducted by me during my candidature. I was primarily responsible for the planning, execution and preparation of the work for publication. I formed the research question, decided the most suitable statistical methods, performed the data analyses, interpreted results, wrote the first draft of the publication and performed subsequent editing of the publications in response to co-authors' and editors' review, with my contribution exceeding more than 70% of the content of the publication. My co-authors Allison Milner, Anthony LaMontagne, George Disney and Matthew Spittal contributed to the study conception, the analytical plan, the interpretation of results and the critical review of manuscript drafts. I have provided co-author authorisation forms from each co-author. This thesis contains no material which has been submitted for other qualifications and all work included in this thesis was carried out following my enrolment into the PhD.

Publications included in this thesis

This thesis includes three papers that have been published and are in print (Study I, II and III) and one paper that is in the process of submission (Study IV).

Chapter 3 Study I: Y. Taouk, M. J. Spittal, A. D. LaMontagne, A. J. Milner (2020). Psychosocial work stressors and risk of all-cause and coronary heart disease mortality: A systematic review and meta-analysis. *Scandinavian Journal of Work, Environment and Health* 46(1): 19-31.

Author	**Proportion**	**Contributions**
Yamna Taouk	85%	Study conception and design; data searches and extractions; quality

		assessment; data analyses; interpretation of results; draft and review of manuscript
Matthew Spittal	5%	Study conception and design; review of manuscript
Anthony LaMontagne	5%	Study conception and design; review of manuscript
Allison Milner	5%	Study conception and design; review of manuscript

Chapter 4 Study II: Y. Taouk, A. D. LaMontagne, M. J. Spittal, A. Milner (2020). Psychosocial work stressors and risk of mortality in Australia: analysis of data from the Household, Income and Labour Dynamics in Australia survey. *Occupational and environmental medicine* 77(4): 256-264.

Author	**Proportion**	**Contributions**
Yamna Taouk	80%	Study conception and design; data analyses; interpretation of results; draft and review of manuscript
Anthony LaMontagne	5%	Study conception and design; review of manuscript
Matthew Spittal	5%	Study conception and design; review of manuscript
Allison Milner	10%	Study conception and design; data interpretation; review of manuscript

Chapter 5 Study III:

Y. Taouk, M. J. Spittal, A. J. Milner, A. D. LaMontagne (2020). All-cause mortality and the time-varying effects of psychosocial work stressors: a retrospective cohort study using the HILDA survey. *Social Science and Medicine* 266: 113452.

Author	Proportion	Contributions
Yamna Taouk	80%	Study conception and design; data analyses; interpretation of results; draft and review of manuscript
Matthew Spittal	5%	Study conception and design; review of manuscript
Allison Milner	5%	Study conception and design; review of manuscript
Anthony LaMontagne	10%	Study conception and design; data interpretation; review of manuscript

Chapter 6 Study IV: Y. Taouk, M. J. Spittal, G. Disney, A. D. LaMontagne (2020). Changes in job control and perceptions of general health: A longitudinal analysis of Australian workers, 2005-2017 (submission in progress; submitted to Journal of Occupational and Environmental Medicine).

Author	Proportion	Contributions
Yamna Taouk	70%	Study conception and design; data analyses; interpretation of results; draft and review of manuscript
Matthew Spittal	10%	Study conception and design; data interpretation; review of manuscript
George Disney	5%	Study conception and design; review of manuscript
Anthony LaMontagne	15%	Study conception and design; methods and interpretation of results; review of manuscript

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Acknowledgements

The old adage, it takes a village to raise a child, is most apt for describing the progress of this PhD. It has taken the support and kindness of many wonderful people along the way to complete this journey. I am most grateful to my supervisors, the late Allison Milner, Mathew Spittal, George Disney and Tony LaMontagne for their unfailing support, guidance and tutelage. Tragically, Allison's life was cruelly cut short in a freak accident halfway through my PhD. I will be forever thankful to Allison for encouraging me to pursue this PhD and for her utmost confidence in me. I am so grateful for having had the opportunity to embark on this PhD with her. As well as an exceptional mentor and supervisor, Allison was a wonderful friend. She was an exceedingly kind, generous and funny person, and will be forever missed. I would like to express my gratitude to Matt and Tony for their support and contributions to this PhD. I am most thankful for their kindness, and to their commitment to the completion of this PhD. I was fortunate to have George join my supervisory team during the latter part of the PhD and am grateful for his involvement and contributions. I would also like to express my thanks to Professor Anne Kavanagh for chairing the advisory committee throughout my PhD candidature, providing invaluable advice and supporting the progress of this PhD.

I am most grateful to the many wonderful colleagues and fellow PhD students in the Melbourne School of Population and Global Health across the Centres for Health Policy, Health Equity and Mental Health for their friendship, support and kindness. I have been most fortunate to have had the opportunity to work with some amazing people in the school. There are far too many to mention, but Marie Bismark deserves special mention. Marie along with being a wonderful colleague and simply amazing to work with, has been very supportive in accommodating work around the PhD and was a constant source of comfort during some difficult periods in my life.

I am most thankful for the love of my family. This PhD would not have been possible without my husband's unwavering support and belief in my endeavours, the endless cups of tea and words of encouragement he provided, and his unfailing optimism in the outcome. I am grateful and glad that he was with me at every step of this PhD, as well as having been always at my side throughout our life together. To my children, Mona Lisa and Anthony, I dearly hope that this PhD will inspire you both to pursue your dreams as you embark on your own journey in life. Finally, I would like to dedicate this PhD to the memory of my late brother, Joe. His absence is deeply felt by all who had the good fortune to cross paths with him. I was very blessed to have had such an amazing and special brother by my side throughout childhood and adulthood. Joe, the world was indeed a better place with you in it. You will be forever missed and loved.

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List of Abbreviations

ABS	Australian Bureau of Statistics
CI	Confidence interval
CHD	Coronary heart disease
ERI	Effort-reward imbalance
HF	Household form
HR	Hazard ratio
HQ	Household questionnaire
HPA	Hypothalamic-pituitary-adrenal
HILDA	Household, Income and Labour Dynamics in Australia
ICD-10	International Classification of Diseases, Version 10
NDI	National Death Index
NILF	Not in the labour force
OECD	Organisation for economic co-operation and development
OLS	Ordinary least squares
OR	Odds ratio
PQ	Person questionnaire
PRISMA	Preferred reporting items for systematic reviews and meta-analyses
RR	Rate ratio
SCQ	Self-completion questionnaire
SD	Standard deviation
SES	Socioeconomic status
SF-36	Short Form 36
SIGN	Scottish intercollegiate guidelines network

Chapter 1 Introduction

1.1 Work Environment and Health

Work plays a significant role in many people's lives and significantly influences our well-being. Not only does it provide financial independence to support our lifestyles and aspirations, but it also significantly contributes to the development of important social and psychological constructs that shape our identity, social norms and status (Waddell & Burton, 2006). Work provides opportunities for the expansion of social connectivity and community participation, as well as a sense of purpose and identity (Dooley, Fielding, & Levi, 1996; Jahoda, 1981).

Most of our adult life is spent in employment. It is estimated that working adults will have spent over 90,000 hours between the ages of 21 and 65 years in full-time employment and are increasingly expected to work beyond the traditional retirement age (Erdogan, Bauer, Truxillo, & Mansfield, 2012). Australians intend to work longer than ever before. The 2018–19 Multipurpose Household Survey, a supplementary survey to the Australian Bureau of Statistics' (ABS) monthly Labour Force Survey, found that 74% of persons aged 45 years and over had no intention of retiring before 65 years, compared with 48% reported in the initial survey in 2004–05. Furthermore, 23% did not intend to retire before turning 70 years compared with only 8% in the 2004–05 Multipurpose Household Survey (ABS, 2016, 2020). Working longer into older age may adversely affect one's quality of life. Retirement is beneficial to health as retirees exercise more, smoke less, sleep longer, have more time to recover from work-related strain and utilise fewer healthcare services than those who continue to work (Eibich, 2015).

There are many health benefits associated with work and the ill-effects of unemployment on mental and physical health are well documented (Jin, Shah, & Svoboda, 1995; Paul & Moser, 2009; Social Security Advisory Committee, 2016). However, it is not only unemployment that

is associated with poor health. Characteristics of the working environment also influence health outcomes, including psychological and physical well-being (Fishta & Backe, 2015; Goh, Pfeffer, Zenios, & Rajpal, 2015; Karasek, 1979; Nieuwenhuijsen, Bruinvels, & Frings-Dresen, 2010; Nyberg et al., 2014) and poor-quality work may be as bad for health as unemployment (Butterworth, Leach, McManus, & Stansfeld, 2013; Chandola & Zhang, 2018). Individuals in the workplace are subject to a range of physical and psychological exposures. The work environment consists of biological, biomechanical, chemical, ergonomic and radiological hazards, in addition to psychological, social and organisational factors pertaining to the design and management of work, which may affect workers (T. Cox, Griffiths, Rial-González, & European Agency for Safety Health at Work, 2000; T. Cox, Griffiths, & Randall, 2004; Social Security Advisory Committee, 2016). Increasingly, in developed countries, exposure to psychological hazards has dominated the work environment, and exposure to physical hazards has decreased due to increased governance and non-relevance in the post-industrialisation age (Benach, Muntaner, & Santana, 2007). The focus of this thesis is the psychosocial hazards associated with psychological exposure in the workplace. Occupational psychosocial hazards are aspects of work arising from interactions between work design and management, and the social and organisational structure that can cause psychological or physical harm (T. Cox et al., 2004).

1.2 Changing Nature of the Workplace

Over the last decades, the employment landscape has moved away from manually intensive work environments towards professional and service occupations. Accompanying these shifts, physical work became less ubiquitous and increasingly replaced with non-manual cognitive work in developed countries during the post-industrialisation age (Benach et al., 2007). Economic globalisation policies since the latter part of the 20th century have increasingly influenced labour markets and work organisation, and increased work stressors (Benach et al.,

2007). The systematic intensification of work has amplified cognitive and emotional demands in psychosocial working environments as interpersonal and cognitive skills have become increasingly important in modern work life. However, over the same period, job and work security have declined (Kompier, 2006).

Advancement in information technologies has decreased the number of workers required in the manufacturing industry, resulting in increased job insecurity, declining union membership and diminishing labour protection in many industrialised countries (Schnall, Dobson, & Landsbergis, 2017). New work organisation structures including lean production and total quality management have been implemented by companies across manufacturing, healthcare and government sectors throughout the industrialised world, increasing work stressors and influencing the health of workers (Landsbergis, Cahill, & Schnall, 1999). The move towards deregulation and privatisation policies by many governments worldwide might have contributed to an increase in precarious employment and a reduction in job control and social support in the workplace (Benach et al., 2007), disproportionally so for disadvantaged groups over advantaged groups (LaMontagne, Krnjacki, Kavanagh, & Bentley, 2013).

1.3 Psychosocial Working Environment, Psychosocial Work Stressors and Work Stress

All workplaces, regardless of sector or production types, have a physical and psychosocial working environment. The physical working environment relates to the surroundings and elements in the workplace. This environment is closely connected to the work process and the activities of the workplace; hence, individuals are subjected to a range of physical exposures consisting of biological, biomechanical, chemical, ergonomic and radiological hazards (T. Cox et al., 2000). For example, high noise levels may cause deafness, improper use of hazardous chemicals may result in poisoning and poor lighting may affect eyesight. The psychosocial working environment is a consequence of the numerous complex and multifaceted interactions between the workers undertaking the work and the factors pertaining to that work (Hansen et

al., 2015). Specifically, the type of work, organisation and planning of work, qualifications necessary for the job, and perceptions and reactions to the work define the psychosocial working environment embedded in a cultural and social framework (Hansen et al., 2015).

The psychosocial working environment may be either harmful or beneficial. Regardless, this environment influences the health of the worker. Characteristics of the psychosocial working environment related to the organisational, structural and relational aspects of the workplace that may generate a stress response in workers producing physical or psychological harm are known as psychosocial work stressors (Rohmert, 1971). Such psychosocial work stressors comprise a wide range of factors, including risk factors related to the organisation of work (e.g., workload, work autonomy, work demands, time pressure and work schedule); structural factors such as working hours; organisational culture and function including organisational justice; relational factors such as the lack of social support, job insecurity, poor pay and low social value; work, job ambiguity and conflict; and family–work conflicts, associated with work stress and adverse health outcomes (T. Cox et al., 2004; Hansen et al., 2015).

Work stress can be defined as either those characteristics of the psychosocial working environment that may elicit a stress response (work stressors) or the stress response itself (Chandola, 2010). Exposure to psychosocial work stressors can produce perceived stress that, in turn, can give rise to short-term psychological, physiological and behavioural reactions to stress. These short-term reactions can influence health behaviours and ultimately affect health outcomes (Israel, Baker, Goldenhar, Heaney, & Schurman, 1996). Work stress is increasingly recognised as a cause for concern, within various employment sectors and occupational levels, by most developed countries worldwide, and is associated with occupational illness, sickness absence, presenteeism and poor organisational outcomes (LaMontagne, Ostry, & Shaw, 2006). In Europe, work stress is strongly related to almost all health problems and higher levels of sickness absence (Parent-Thirion et al., 2017). In this thesis, psychosocial work stressors,

which are the psychosocial working environment characteristics that may generate stress, will be the focus.

1.4 Work Stress Process

The work stress process begins with exposure to work stressors (G. D. Huang, Feuerstein, & Sauter, 2002; Israel et al., 1996). The process can be explained by the widely used and well-regarded University of Michigan job stress model, which presents a framework for occupational stress, illuminating the complexity and diversity associated with the work environment and health (Israel et al., 1996). Briefly, perceived stress arises from exposure to work stressors and leads to adverse short-term responses. If prolonged durations of perceived stress continue over time, short-term reactions to such stress including physiological (e.g., hypertension), psychological (e.g., tenseness) or behavioural (e.g., smoking and alcohol consumption) could affect persisting health outcomes via physiological (e.g., cardiovascular disease), psychological (e.g., anxiety disorder) or behavioural (e.g., nicotine addiction and alcoholism) health changes (Israel et al., 1996). Furthermore, this model allows for the effect of various modifying variables (social, psychological, biophysical, behavioural and genetic factors) influencing associations during the work stress process.

The work stress process is a multifaceted, dynamic process with numerous interactions, e.g., exposure to stressors, perceived stress, short-term responses and modifying variables affecting each other in addition to long-term health outcomes (Israel et al., 1996). Hence, the work stress process is sequential and involves feedback loops. For example, there may be a direct effect between work stressors and long-term health outcomes and an indirect effect between perceived stress and short-term responses to stress. Persisting long-term health outcomes might contribute to work stressors. In summary, exposure to psychosocial work stressors may affect psychological and physical health and can be moderated by social, psychological, biophysical, behavioural and genetic characteristics (Figure 1.1). In this thesis, I will examine the effect of

psychosocial work stressors on health and mortality. Socio-demographic, health and health behaviours risk factors will be investigated for evidence of confounding and/or effect moderation influencing the relationship.

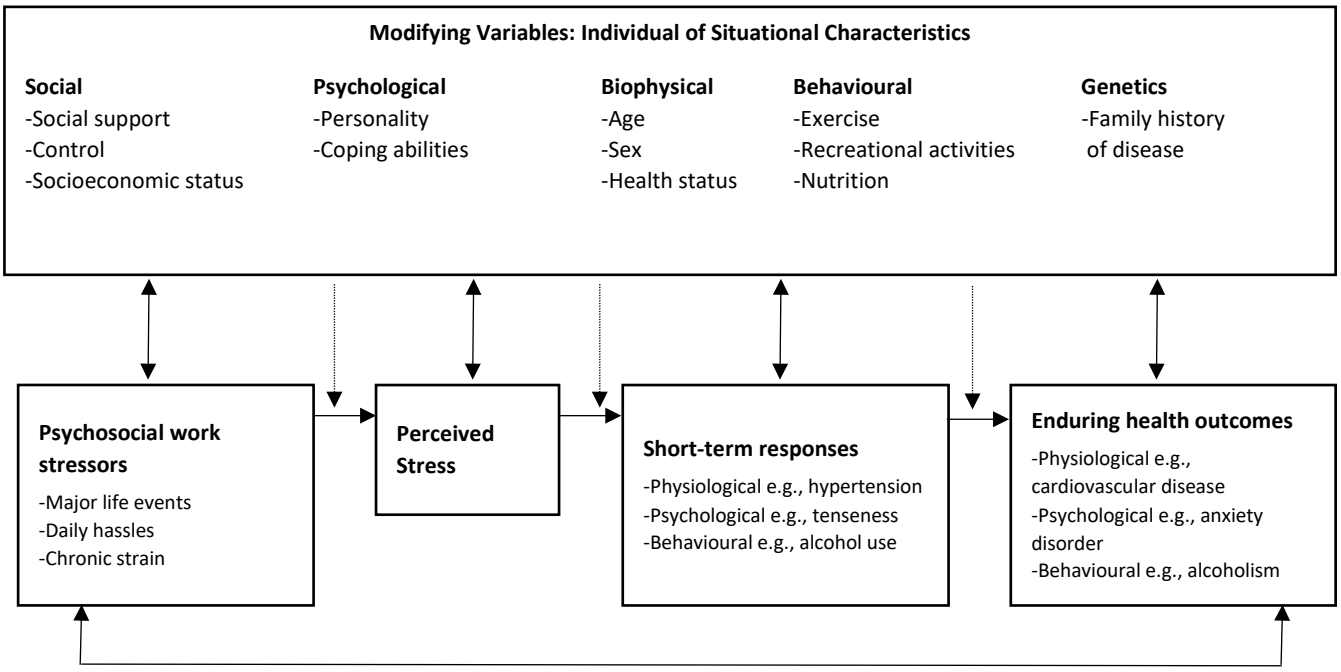

Figure 1.1

Work Stress Process (modified from Israel, Baker, Goldenhar, Heaney, & Schurman, 1996, p. 263)

1.5 Work Stress Models

Various work stress models have been generated to explore the relationship between the psychosocial working environment and the health of the individual. Increased awareness of the effect of psychological conditions and social factors at work on the well-being of employees has resulted in the construction of various theoretical models of work stress measuring psychosocial work stressors. The dominant theoretical models in the study of psychosocial

work stressors include the demand-control or the job strain model (Karasek, 1979; Karasek & Theorell, 1990), the effort-reward imbalance (ERI) model (Siegrist, 1996) and the organisational justice model (Elovainio, Heponiemi, Sinervo, & Magnavita, 2010). These models conceptualise work stressors at a level applicable for generalisation in a myriad of occupations and quantify work stressors responsible for damaging stress in large populations of workers in the workplace (Grosch & Sauter, 2005; G. D. Huang et al., 2002). The most widely used and validated of these work stress models is the two-dimensional demand-control model (Karasek, 1979; Karasek & Theorell, 1990).

1.5.1 Job demand-control model

The job demand-control model has become the main theoretical model for studying stress and strain over the past few decades (Ganster & Rosen, 2013). It was developed for work environments with chronic work stressors resulting from complex and complicated human organisational decision-making and is based on two psychosocial job factors: psychological job demands and decision latitude or job control (Karasek & Theorell, 1990). Karasek (1979) proposed that "strain results not from a single aspect of the work environment, but from the joint effects of the demands of a work situation and the range of decision-making freedom available to the worker facing those demands". Karasek defined job demands as "psychological stressors involved in the realization of workload, stressors related to unexpected tasks and stressors arising from personal conflicts at work". Job demands describe the demands on workers concerning time pressures, concentration requirements and workload. The second component of the model, job control, is characterised by workers' autonomy in the workplace with control over tasks and management of work, and their skills required to perform the work (Karasek, 1979). In the workplace, the controllability of stressors during decision-making becomes increasingly important with the development of complex and integrated social organisations and growing limitations on the behaviour of individuals (Karasek & Theorell,

1990). The demand-control model posits that stress-related ill health stems from job strain due to the combined effects of low job control and high job demands (Karasek & Theorell, 1990). Low job control is defined as the restricted capacity to gain knowledge or acquire skills and the absence of decision-making capability, whereas high job demands are described as excessive workload and job-related pressure. In contrast, low job demands are categorised as passive and low strain in nature (Karasek, 1979) (Figure 1.2).

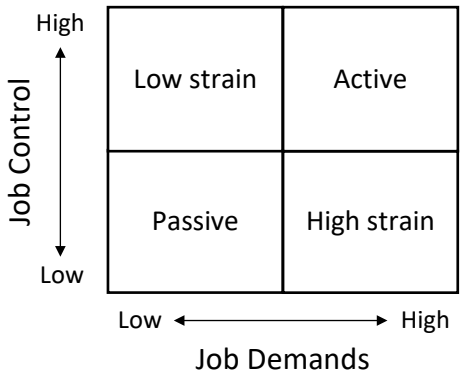

Figure 1.2

Job Demand-Control Model (adapted from Karasek, 1979)

A third component, social support at work, was subsequently integrated into the demand-control model as it was thought that the negative effects of high job demands might be buffered by the influence of social support (Johnson & Hall, 1988). Workers in well-supported working environments benefit from the support and comfort of co-workers and others in the organisation, enabling them to combine their resources to cope with stressors. Social support modulates the effects of job demands and control, either tempering or accentuating their effect, in the model (Johnson & Hall, 1988). The modified demand-control-support model deems the highest risk of poor psychological health and health outcomes to be related to iso-strain jobs, characterised by work environments with high job demands, low job control including low

decision-making latitude, and low social support (Johnson & Hall, 1988). Conversely, in the general population, working environments with low job demands and high job control are the most beneficial to health because increased control at work helps alleviate the adverse effect of high job demands (Karasek & Theorell, 1990) (Figure 1.3).

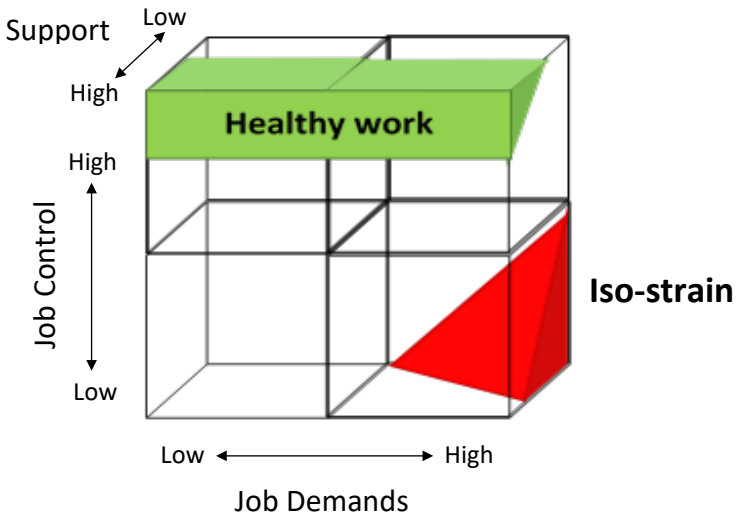

Figure 1.3

Job Demand-Control Support Model (adapted from Karasek & Theorell, 1990)

1.5.2 Effort-reward imbalance (ERI) model

The ERI model, while sharing some elements of the job demand-control-support model, includes aspects of the person and labour market context and job characteristics, and centres on the lack of reciprocity causing stress in working environments (Siegrist, 1996). Two distinct types of efforts, intrinsic and extrinsic, are recognised in the ERI model. Intrinsic effort relates to the motivation level of individuals and their necessity for job control, whereas extrinsic effort pertains to external working conditions such as job demands. ERI resulting in work stress arises from an imbalance between the high extrinsic efforts by workers in demanding jobs and their perceptions correlating with low rewards by way of remuneration, recognition, esteem and career advancements, including job security (Siegrist, 2002a). The model recognises that work

characterised by high efforts and low rewards signifies a lack of reciprocity in terms of high costs and low gains eliciting negative emotions in workers subject to such imbalances (Siegrist, 2002a). Ongoing recurrence of reward deficiency, such as working hard with no or little recognition and being unappreciated, not being treated fairly or experiencing disappointments because of low rewards in the workplace, can elicit negative emotions and generate stress reactions that may be harmful on health in the long-term (Siegrist, 2000). In addition to effort and reward, a third factor, overcommitment, is included in the ERI model to account for differences among individuals when experiencing ERI and managing the effect modification of overcommitment on the relationship between ERI and health (Siegrist et al., 2004). Overcommitment is a personality trait characterised by an excessive commitment to work together with a strong need for approval and respect (Siegrist et al., 2004). It is thought that ERI is intensified by overcommitment, such that excessively overcommitted workers experience greater stress reactions to an ERI than less overcommitted workers. Some similarities exist between the job demand-control and ERI models; however, one key difference is that the job demand-control model is task-focused, whereas the ERI model encompasses broader labour market aspects (e.g., respect for nurses versus supermarket cashiers).

1.5.3 Organisational justice model

As well as the job demand-control and ERI models, additional characteristics of stressful working conditions that describe the psychosocial working environment include organisational and procedural fairness and job insecurity. The organisational justice model expands on equity theory (Adams, 1965) and refers to employees' perceptions of fairness of treatment, outcomes and workplace processes (Elovainio et al., 2010). Organisational justice, or the role of fairness in organisations, refers to workers' perceptions of fairness within organisations (Greenberg, 1987). It is focused on workers' perceptions of their organisation's behaviours, decisions and actions, and the influence these perceptions have on the attitudes and behaviours of workers

(Baldwin, 2006). These perceptions can adversely or favourably affect the attitudes and behaviours of workers and influence their work performance and productivity. The organisational justice model evaluates the quality of social interaction in the workplace and assesses managerial and organisational characteristics of the work environment (Robbins, Ford, & Tetrick, 2012). There are three distinct, though overlapping, elements of the organisational justice model: distributive, procedural and interactional justice.

The first element, distributive justice, is based on Adams's (1965) equity principle and refers to the perceived fairness of the outcomes of organisational processes distributed proportionally to inputs (Baldwin, 2006). The second element, procedural justice, pertains to the perceived fairness of the decision-making processes within organisations, specifically the extent to which these procedures are consistent, ethical, unbiased, appropriate and adaptable and representative (Leventhal, Karuza, & Fry, 1980). The third element, interactional justice, relates to the degree of perceived fairness and respect in interactions between workers and organisational representatives throughout decision-making procedures (Baldwin, 2006; Bies & Moag, 1986). The model proposes that increased perceived unfairness in the working environment may cause experienced stress responses, adversely affecting workers' health as a consequence of physiological and behavioural reactions (Baldwin, 2006; Bergmann, Gyntelberg, & Faber, 2014; Elovainio et al., 2010; Kivimaki, Elovainio, Vahtera, & Ferrie, 2003; Robbins et al., 2012; Theorell et al., 2016).

The experiences of stress reactions are central to these work stress theoretical models. Implicit in all these models is the suggestion that increased psychosocial work stressors, high job demands, low job control, poor reciprocity between costs and rewards or perceived unfairness, result in psychological stress responses including negative emotions, stress, depression and anxiety. These may produce adverse physical health effects mediated by indirect unhealthy behaviours or direct physiological factors (Robbins et al., 2012). As well as the psychosocial

work stressors described in these models, an additional work stressor contributing to the experienced stress in the working environment is job insecurity, a subjective measure of workers' perceptions regarding employment continuation and the consequences of job loss (Bernhard-Oettel, Sverke, & De Witte, 2005; De Witte, 2005). Consequences of perceived job insecurity for the individual can have implications on job attitudes and health outcomes due to the uncertain and uncontrollable nature of the stressor (Sverke, Hellgren, & Naswall, 2002).

1.6 Biology of Stress

Repeated exposure to work stressors may affect the autonomic nervous and metabolic systems, and neuroendocrine activity, resulting in poor physical and emotional health (Kivimaki & Steptoe, 2018; Marmot & Wilkinson, 2006). Stress reactions are a normal response to alarming situations that have evolved in humans as a rapid protective response against external and life-threatening but short-term threats. This reaction, first described by the early 20th-century physiologist Walter Cannon as the fight-or-flight response and further advanced by Hans Selye in the 1930s, is an instinctive and automatic response that alerts the brain about the perceived danger and prepares the body to fight or flee from the threat (Cannon, 1939; Selye, 1970). Nerve and hormone signals, triggered by sensory information, increase awareness and produce physiological changes including increased pulse and blood pressure, deep breathing and muscle tenseness to help the body survive the physical, psychological or biological threats (Marmot & Wilkinson, 2006).

Two main pathways are involved in the fight-or-flight response process. The first pathway, the sympatho-adrenal pathway, involves the rapid activation of the sympathetic autonomic nervous system during which noradrenaline is released at nerve endings and adrenaline is secreted into the bloodstream by the adrenal medulla. Several physiological changes are evoked throughout the body such as accelerated pulse rate, elevated blood pressure, increased metabolism, sweat secretion and sensory vigilance, dilated pupils and airways, constricted or dilated blood vessels

and inhibited salivation. There is much variation in the severity and duration of responses regarding the effects of circulating adrenaline and sympathetic nerve activity on the body due to social and individual differences in psychological coping measures, and the influence of these variations on chronic disease is unclear (Kivimaki & Steptoe, 2018; Marmot & Wilkinson, 2006).

The second much slower response involves the hypothalamic-pituitary-adrenal (HPA) axis pathway. The HPA, triggered by the hypothalamus, releases cortisol into the bloodstream from the adrenal glands. Corticotrophin releasing factor is emitted into the small vessels by the hypothalamus and travels to the pituitary gland, which releases an adrenocorticotrophic hormone into the bloodstream, increasing the levels of the adrenocorticotrophic hormone in the adrenal cortex enough for the release of corticosteroids to target cells in the body. The control and adjustment of circulating cortisol levels due to the experienced stress involve each of these three hormones. Corticosteroids have metabolic and psychological effects. They are important in maintaining and controlling resting and stress-related metabolic functions. During a perceived threat against danger, increased stamina and strength are boosted by increased blood glucose levels and the release of fatty acids from fat tissues. While this may be an appropriate response to life-threatening situations, this excess energy increases blood cholesterol levels in physically inactive situations associated with psychological threats and increases inflammatory protein levels despite the absence of pathogens. Similarly, while cortisol release increases alertness and vigilance in the short-term, prolonged increased cortisol levels can produce paranoia or depression (Kivimaki & Steptoe, 2018; Marmot & Wilkinson, 2006). Stress responses vary depending on an individual's perception and evaluation of the stress-inducing condition, and their ability to cope with stress. Behavioural and biological factors such as personality traits, genes, socioeconomic status (SES), gender and prior experiences of stress

may influence individuals' response to stress (Kivimaki & Kawachi, 2015; Mair, Cutchin, & Kristen Peek, 2011; McEwen & Stellar, 1993).

Since the onset of agriculture over 10,000 years ago and the industrial revolution around 200 years ago, transformations in the material and social environments have greatly affected living conditions over a relatively short period. However, our underlying biology is essentially unchanged from when early *Homo sapiens* roamed the planet approximately 60,000 years ago (Marmot & Wilkinson, 2006). Most humans are rarely exposed to biological and physical threats in modern society; rather contemporary life is filled with psychological demands and ordeals. Consequently, the resultant fight-or-flight response that was so advantageous to the survival of early modern humans is more intense and persists for longer periods than the short-term responses associated with physical and biological dangers (Marmot & Wilkinson, 2006). In the workplace, acute stress associated with high effort linked to high rewards is beneficial to health (Siegrist, 1996). In contrast, chronic stress caused by prolonged exposure to high psychological demands with low control and reward is detrimental to health (Bosma et al., 1997). Repeated activation and the longer durations of the fight-or-flight response might be associated with the development of chronic stress, altering short-term reactivity and normal baseline levels in autonomic, neuroendocrine or metabolic pathways (Brunner, 2017).

1.6.1 Acute and chronic stress

Brunner (2017) and Marmot and Wilkinson (2006) proposed three models of stress reactivity applicable to adrenaline and cortisol and other stress hormones, measuring hormone concentration levels over time. In the first model, hormone concentration increases sharply and returns to the baseline swiftly. This model suggests that physiological changes in the body may not be harmful, provided that there is a fast return to the baseline. In the second model, hormone concentration increases sharply; however, the return to the baseline is delayed, prolonging departure from the resting level. In the third model, hormone concentration is elevated at the

baseline, and there is a blunted response (Figure 1.4). In the latter two models, where stress reaction is more frequent and of longer durations, reactivity is weakened, and abnormal physiological changes persist, which may lead to ill health (Marmot & Wilkinson, 2006). Brain resilience is pivotal in the adaptability of the body's system to stress, influencing variation in the ability of individuals to return to healthy baseline levels or generate unhealthy biological adaptions (McEwen, Gray, & Nasca, 2015). It is generally accepted that early childhood exposures during infancy and childhood, the periods of most significant brain plasticity, determine the resilience of the brain and future health status (Giesinger et al., 2014).

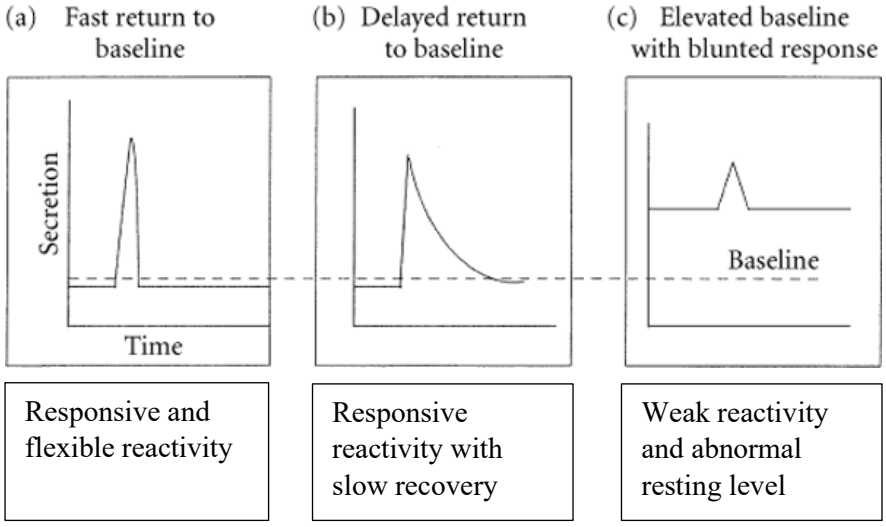

Figure 1.4

Models of Stress Reactivity (modified from Brunner & Marmot, 2006)

1.6.2 Allostatic load model

Stress is not necessarily a detriment to health; for short intervals and in small amounts, stress might be beneficial in certain situations. However, chronic stress, whereby stress responses are prolonged without periods of recovery, is harmful to health (Brunner & Marmot, 2006). The type, intensity and duration of the stress response determine the physiological and

psychological effects of stress (Murison, 2016). Thus, it is important to understand the adaptive and maladaptive consequences of stress responses.

Controlling and stabilising body systems via homeostasis is crucial for optimum function. The process of allostasis, the dynamic maintenance of stability through change, is the adaptive response of the effector systems (e.g., cardiovascular or neuroendocrine) to stresses experienced by the homeostatic systems (e.g., blood pressure, blood oxygen and body temperature) (Ganster & Rosen, 2013; McEwen, 1998). The underlying response of the body's exposure to stress, whether it be physical or psychological, involves two steps: launching the allostatic response involving the sympathetic nervous system and the HPA axis to manage the perceived stress followed by the suspension of the response once the perceived threat has stopped (McEwen, 1998). Allostatic systems function around defined set points, which may be dependent on daily or seasonal rhythms. Exposure to chronic demands that continuously elevate set points above their normal range can reset them (Ganster & Rosen, 2013). In the allostatic load model, the central nervous system is essential in controlling physiological responses by forestalling adaptation through familiarity and awareness of previous episodes in combination with environmental events (Ganster & Rosen, 2013).

During adjusting to stress, the body's systems adapt their psychological and physiological processes to manage the stressors, resulting in chronic over-activity or under-activity of allostatic systems. Different baselines levels for psychological and physiological performances are fixed, resulting in the deterioration of body systems, from repeated and unresolved stressors (McEwen, 1998; Sterling & Eyer, 1988). The interaction of the various adaptation systems is multifaceted and complex and involves a temporal sequence process (Ganster & Rosen, 2013). The first sequence involves the stimuli of primary mediators, consisting of stress hormones and pro- and anti-inflammatory cytokines, in the central nervous system to prepare organisms for

managing disruptions to the homeostatic systems (Ganster & Rosen, 2013). The effects on cellular activities from repeated and persistent activation of primary mediators can compromise physiological systems. This chronic activation initiates secondary mediators, where the adjustment of normal operating ranges in biological systems occur in response to over- or under-activity of primary mediators (Ganster & Rosen, 2013). Secondary mediators involve the metabolic, cardiovascular and immune systems. Dysregulations of these secondary mediators are the primary risk factors for physical and mental ill health. During this phase, indicators exhibit subclinical manifestations. The continued prolongation of abnormal ranges culminates in allostatic overload, the tertiary phase characterised by disease endpoints, psychological disorders and mortality (Ganster & Rosen, 2013) (Figure 1.5).

The allostatic load model suggests repeated and unresolved exposure to stress results in proximal indicators of stress including elevated stress hormones and increased body mass, leading to further distal strain indicators including depression, cardiovascular disease and even death (Juster, McEwen, & Lupien, 2010). There is a growing body of evidence supporting the importance of allostatic load in cardiovascular disease, cancer, infection, cognitive decline and ageing (Marmot & Wilkinson, 2006). In Australia, the top five leading causes of death in 2018 were ischaemic heart disease, dementia and Alzheimer's disease, and cerebrovascular disease including stroke, lung cancer and chronic obstructive pulmonary disease, which accounted for more than one-third (36.6%) of all deaths (ABS, 2019).

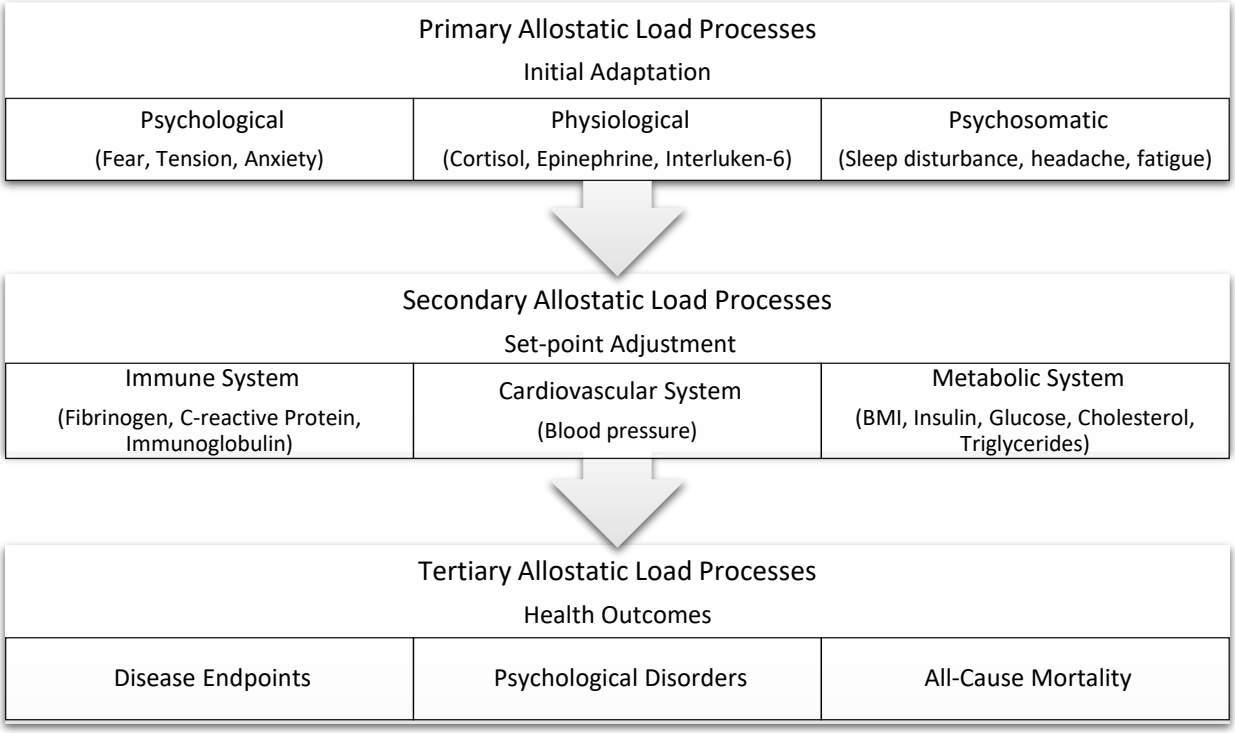

Figure 1.5

Allostatic Load Model of the Stress Process (modified from Ganster & Rosen, 2013)

1.6.3 Infection, inflammation and immunity

Chronic and repeated activation of stress responses in the body may contribute to maladaptive responses of the immune system, including inattentiveness to viral and bacterial infectious agents infiltrating the body and causing disease, and over reactivity such that the immune system itself rather than any biological threat is responsible for causing autoimmune disorders and allergic diseases (Clow, 2001). Infection and immunity have been implicated in the association between psychosocial stressors and physical health (Kivimaki & Steptoe, 2018; Marmot & Wilkinson, 2006). The susceptibility and severity of infection might be affected by chronic stress and infection, contributing to the development of diseases not previously acknowledged as due to infection (Marmot & Wilkinson, 2006).

Immunity has been associated with a range of disorders, including peptic ulcers; gastric, cervical and other cancers; and cardiovascular heart disease (Marmot & Wilkinson, 2006). The brain is important in the function of the immune system. Hormones that affect the immune system and the nerves of the autonomic nervous system are present in every major body tissue, including the bone marrow, thymus, spleen and lymph nodes (Marmot & Wilkinson, 2006). The presence of infection is not detectable by the brain; rather it is the immune system that acts as the brain's sensory system, emitting cytokines into the brain through the bloodstream to alert it to the presence of infection in the body. The immune system relies on cognitive stimuli from the autonomic nervous system and cortisol release from the hypothalamus and pituitary gland (Marmot & Wilkinson, 2006). Stress affects the effectiveness of the body's system defences against infection (Chandola & Zhang, 2018; Marmot & Wilkinson, 2006).

1.7 Psychosocial Work Stressors and Health Outcomes

A significant burden of disease can be credited to health-adverse psychosocial working environments. Psychosocial work stressors are related to many different health outcomes. There is increasing evidence that the exposure to psychosocial work stressors such as high job demands, low job control, high job strain and high ERI might be risk factors for poor mental health (Bentley, Kavanagh, Krnjacki, & LaMontagne, 2015; Bonde, 2008; Harvey et al., 2017; Madsen et al., 2017; Netterstrom et al., 2008; Nieuwenhuijsen et al., 2010; Siegrist, 2008; Stansfeld & Candy, 2006; Theorell et al., 2015); poor cardiovascular health, including stroke (Bishop et al., 2003; Dragano et al., 2017; Fishta & Backe, 2015; Fransson et al., 2015; Y. L. Huang et al., 2015; Kivimaki, Batty, Ferrie, & Kawachi, 2014; Kivimaki & Kawachi, 2015; Kivimaki et al., 2012; Kivimaki, Virtanen, et al., 2006; Kuper, Adami, Theorell, & Weiderpass, 2007; Kuper & Marmot, 2003; Nyberg et al., 2013; Rugulies et al., 2020; Slopen et al., 2012; Toivanen & Hemstrom, 2008; Tsutsumi, Kayaba, Kario, & Ishikawa, 2009), diabetes (Leynen et al., 2003; Nyberg et al., 2014; Nyberg et al., 2013); musculoskeletal disorders (Devereux,

Vlachonikolis, & Buckle, 2002; Hagen, Magnus, & Vetlesen, 1998; Torp, Riise, & Moen, 2001) and poor health behaviours, including alcohol consumption, smoking and lack of physical activity (de Lange, Taris, Kompier, Houtman, & Bongers, 2003; Fransson et al., 2012; Head, Stansfeld, & Siegrist, 2004; Heikkila et al., 2013; Heikkila et al., 2012; Hellerstedt & Jeffery, 1997; Radi, Ostry, & Lamontagne, 2007). Moreover, the degree of available job control partially explains the inverse relationship between the occupational skill level and poor health outcomes (Marmot, Bosma, Hemingway, Brunner, & Stansfeld, 1997; Marmot et al., 1991). Social support in the workplace is beneficial to health via a direct main effect and buffering effect, which reduce the harmful effects of psychological and physical stress (Broadhead et al., 1983; Cohen & Wills, 1985).

There is wide support in the epidemiological literature regarding the health consequences of other broader constructs of psychosocial work stressors, including job insecurity, long working hours and shift work, which are commonly encountered by employees in the workplace. Job insecurity regarding employees' uncertainty about employment continuation adversely affects cardio-metabolic disease, mental and physical health and sickness absence (Ferrie et al., 2016; Kivimaki, Vahtera, Pentti, & Ferrie, 2000; LaMontagne, Too, Laura Punnett, & Milner, 2020; Sverke et al., 2002; Virtanen et al., 2013). The number of hours worked is generally under the control of employers. Some employees can set the number of hours they work; however, most employees have little control over their working hours. Working long hours can result in adverse health behaviours such as alcohol consumption and smoking, and adverse health consequences, including cardio-metabolic disease, musculoskeletal disorders and depression and anxiety (Bannai & Tamakoshi, 2014; Barnett, 2006; Harma, 2006; Johnson & Lipscomb, 2006; Kivimaki, Jokela, et al., 2015; Kivimaki, Virtanen, et al., 2015; Virtanen et al., 2012; Virtanen et al., 2018; Virtanen et al., 2015). Exposure to extended durations of long working hours influences the capability of workers for mental and physiological recovery, which may

cause adverse health behaviours and outcomes (van der Hulst, 2003). Control by workers over their work hours, enabling the adjustment of their hours in response to demands at work and home, might reduce work stress-related health problems. Increased perceived worktime control is associated with good subjective health, less sickness absence and reduced work stress-related absenteeism (Ala-Mursula, Vahtera, Linna, Pentti, & Kivimaki, 2005; Ala-Mursula, Vahtera, Pentti, & Kivimaki, 2004). The effect of insufficient recovery and reduced sleep associated with long work hours is particularly pertinent for workers with high job demands (Harma, 2006).

Shift work is related to lower SES and job control; however, it is the disruption of circadian rhythms and sleep deprivation that is responsible for changes in the physiological, metabolic and endocrinological systems that cause adverse health outcomes associated with night shift work (Harma, 2006; Harma, Kompier, & Vahtera, 2006). Working night shifts was considered as probably carcinogenic to humans by the International Agency for Research on Cancer in 2019 (Ward et al., 2019). An increasing number of longitudinal cohort studies have found that night shift work is associated with an increased risk of cardio-metabolic disease, rheumatoid arthritis, breast cancer and adverse pregnancy-related outcomes (Boggild & Knutsson, 1999; Bonde et al., 2012; Frost, Kolstad, & Bonde, 2009; Garde et al., 2020; Harma, 2006; Kamdar, Tergas, Mateen, Bhayani, & Oh, 2013; Puttonen, Harma, & Hublin, 2010; Puttonen, Oksanen, et al., 2010; Vyas et al., 2012).

1.8 Psychosocial Work Stressors and Mortality

The majority of studies on psychosocial job stressors have focused on mental and physical health outcomes, with most examining the effect of work stressors on cardiovascular morbidity. Research examining the association between psychosocial work stressors and all-cause mortality is limited, and the results are inconsistent. Most studies thus far have examined components of the Karasek's job-demand-control model. Some evidence suggests that

psychosocial work factors, conceptualised and measured based on the jobs-demand-control-support model, are associated with an increased risk of all-cause mortality (Alterman, Shekelle, Vernon, & Burau, 1994; Amick et al., 2002; Falk, Hanson, Isacsson, & Ostergren, 1992; Hibbard & Pope, 1993; Joensuu et al., 2012; Sabbath, Mejia-Guevara, Noelke, & Berkman, 2015) and cardiovascular mortality (Joensuu et al., 2012; Karasek, Baker, Marxer, Ahlbom, & Theorell, 1981; Kivimaki et al., 2002; Toivanen & Hemstrom, 2006). However, other prospective cohort studies examining job strain model factors and mortality have not found an association between psychosocial work stressors and cardiovascular mortality and deaths from coronary heart disease (CHD) (Brunner et al., 2004; Eaker, Sullivan, Kelly-Hayes, D'Agostino, & Benjamin, 2004; Johnson, Stewart, Hall, Fredlund, & Theorell, 1996; Kivimaki, Leino-Arjas, et al., 2006; Lee, Colditz, Berkman, & Kawachi, 2002; Padyab, Blomstedt, & Norberg, 2014; Slopen et al., 2012) or all-cause mortality (Nilsen, Andel, Fritzell, & Kareholt, 2016; Shirom, Toker, Alkaly, Jacobson, & Balicer, 2011; Tobiasz-Adamczyk, Brzyski, Florek, & Brzyska, 2013; Tsutsumi, Kayaba, Hirokawa, Ishikawa, & Jichi Medical School Cohort Study, 2006; von Bonsdorff et al., 2012).

Relatively few prospective cohort studies have assessed the relationship between ERI and mortality, and the findings have been mixed. High ERI was associated with increased cardiovascular mortality in one study (Kivimaki et al., 2002). In another study by Kivimaki et al. (2018), the authors reported that high ERI was associated with increased all-cause mortality in men with no pre-existing cardio-metabolic disease. However, there was no evidence that high ERI was associated with all-cause mortality in two other studies (Brunner et al., 2004; Tobiasz-Adamczyk et al., 2013). The relationship between perceived unfairness in the workplace and all-cause mortality is not clear owing to insufficient evidence (Elovainio, Leino-Arjas, Vahtera, & Kivimaki, 2006; Kivimaki et al., 2005).

There is limited evidence regarding the broader constructs of psychosocial work stressors and all-cause mortality. Several prospective cohort studies have investigated the association between shift work and mortality with inconsistent findings (Akerstedt, Kecklund, & Johansson, 2004; Boggild & Knutsson, 1999; Fujino et al., 2006; Gu et al., 2015; Hublin et al., 2010; Jorgensen, Karlsen, Stayner, Andersen, & Andersen, 2017; Karlsson, Alfredsson, Knutsson, Andersson, & Toren, 2005; McNamee et al., 1996; Natti, Anttila, Oinas, & Mustosmaki, 2012; Vyas et al., 2012; Yadegarfar & McNamee, 2008; Yong, Nasterlack, Messerer, Oberlinner, & Lang, 2014). The limited studies to date investigating the association between long working hours and risk of all-cause mortality were inconclusive in their findings (Holtermann et al., 2010; O'Reilly & Rosato, 2013). A small number of studies have examined the relationship between job insecurity and all-cause mortality, although the results were inconsistent and inconclusive (Amick et al., 2002; Kivimaki, Vahtera, et al., 2003; Laszlo, Engstrom, Hallqvist, Ahlbom, & Janszky, 2013; Natti, Kinnunen, Makikangas, & Mauno, 2009; Perlman & Bobak, 2009; Slopen et al., 2012).

Most prospective cohort studies examining the association between work stressors and mortality have analysed mortality as a secondary outcome, examined cardiovascular deaths only, measured exposure to psychosocial work stressors at the baseline or derived psychosocial work stressors from a job exposure matrix based on occupations (Eaker et al., 2004; Holtermann et al., 2011; Joensuu et al., 2014; Johnson et al., 1996; Karasek et al., 1981; Kivimaki, Elovainio, et al., 2003; Kivimaki, Leino-Arjas, et al., 2006; Kivimaki et al., 2002; Lee et al., 2002; Natti et al., 2009; Nilsen et al., 2016; Padyab et al., 2014; Shirom et al., 2011; Slopen et al., 2012; Tobiasz-Adamczyk et al., 2013; Tsutsumi et al., 2006; von Bonsdorff et al., 2012). The few studies that examined the relationship between cumulative exposure to psychosocial work conditions and all-cause mortality generated exposure histories based on

occupational title data, using a job exposure matrix to impute exposure levels from occupation titles (Amick et al., 2002; Johnson et al., 1996).

Furthermore, in many analyses, the proportion of men and women was unbalanced, with the cohorts studied being predominantly male workers (Alterman et al., 1994; Boggild & Knutsson, 1999; Falk et al., 1992; Fujino et al., 2006; Holtermann et al., 2010; Johnson et al., 1996; Karasek et al., 1981; Karlsson et al., 2005; McNamee et al., 1996; Yadegarfar & McNamee, 2008; Yong et al., 2014) and few studies analysing the effect of psychosocial work stressors on mortality among female workers (Gu et al., 2015; Jorgensen et al., 2017; Lee et al., 2002; Sabbath et al., 2015; Slopen et al., 2012). Therefore, it is pertinent to include female workers during the analysis of work-related psychosocial stressors. Sex disparities in job control across occupations have been well documented and can be explained by labour market segregation of women into disproportionately lower-skilled and precarious employment (LaMontagne et al., 2013). Several studies were affected by small sample sizes (Alterman et al., 1994; Eaker et al., 2004; Joensuu et al., 2012; Johnson et al., 1996; Kivimaki et al., 2002) and many studies focused on single occupations (e.g., nurses and manufacturing workers), limiting the extent of generalisability from these studies to the broader population of professions.

1.9 Gaps in the Literature

Given the increasing evidence for the relationship between psychosocial work stressors and adverse health outcomes, research into work stressors, heath and mortality is warranted, as there is a conspicuous gap in the literature on this topic. It is not yet fully understood the extent that exposure to adverse psychosocial work stressors causes ill health due to several methodological issues commonly found in research (Rugulies et al., 2020). Measurements of psychosocial work stressor exposures have been predominantly self-reported and might have been subjected to differential information bias if the knowledge of the outcome influenced the

exposure measurement. Such a bias is not a problem in mortality outcome research but might be an issue in self-reported health outcome studies. In studies that have used the same method for exposure and outcome measurements, e.g., self-completed questionnaires, the estimated effects might have been vulnerable to common method bias. This bias is caused by individuals systematically overstating their perception of psychosocial work stressors and health assessments due to negative affectivity or the natural propensity for over-evaluation rather than providing an objective representation of the actual environment, thus yielding spurious associations (Jakobsen & Jensen, 2015).

The observed associations between psychosocial work stressors and health may be reciprocal, that is, poor health status may predispose selection into employment with adverse psychosocial work stressors, or workers with poor health may be prone to reporting more negative perceptions of the workplace than might have been the case had they been in better health (Elovainio et al., 2015; Tang, 2014). Related to this is the healthy worker effect that might have attenuated the results of previous studies because unhealthy people are less likely to remain in the workforce and are more likely to be employed in low-status jobs with high stress (Kivimaki et al., 2012; Li & Sung, 1999). This is known as reverse causation, where the outcome causes the exposure rather than the other way around. Thus, it is difficult to determine the directionality of the association between work stressors and health, limiting any causal inference regarding the direction of the relationship. Publication bias is another factor identified as a methodological issue in studies, with studies with results that are in the expected direction more likely to be published (Kivimaki et al., 2012).

Studies may have been affected by confounding bias, that is, common causes of both the exposure and outcome. Associations between psychosocial work stressors and mortality, and health may be confounded by a range of non-workplace health risk factors such as previous illness, personality traits, childhood experiences, pre-employment factors and stressful life

events that are not commonly adjusted for in many studies (Elovainio et al., 2007; Ettner & Grzywacz, 2001; Kivimaki & Steptoe, 2018). Several studies have relied on occupation-based measures of psychosocial work stressors assigned using a job exposure matrix rather than individual data. Thus, they may be susceptible to exposure misclassification bias. In particular, job demands, a subjective measurement, may not be suitable for use in an exposure imputation system designed to measure environmental differences (Alterman et al., 1994; Johnson et al., 1996; Karasek et al., 1981).

There are shortcomings of previous exposure treatments in work stressors and mortality analyses. A baseline measurement assumes that all relevant information pertaining to a person's experience of psychosocial work stressors can be accurately summarised at that one time point and remains constant or represents the life course exposure from that point onwards. It is also unclear how much or how often exposure to work stressors varies over time, given the changing landscape of contemporary workplaces. Changing occupations and career mobility due to digital disruption are becoming ever more commonplace. However, there is some evidence that psychosocial work stressors are not static (Bentley et al., 2015; LaMontagne et al., 2013; LaMontagne et al., 2020). Thus, studies with a single assessment of work characteristics and long follow-up periods without repeated assessment of exposure status over time measuring cumulative exposure may have biased results due to the evaluation of exposure to work stressors temporally distant from the outcome (Chandola et al., 2008).

The strongest evidence for causation is commonly derived from intervention studies with higher causal inference. However, experimental manipulation of psychosocial work stressors and assessing the associated effects on health and mortality is difficult to undertake (Melin, Lundberg, Söderlund, & Granqvist, 1999). Observational studies have provided almost all the evidence on psychosocial work stressors, and health and mortality. A review of prospective cohort studies by Kivimaki and Kawachi (2015) concluded that observational data suggest an

excess risk between 10% and 40% for cardiovascular disease of workers exposed to psychosocial work stressors, including job strain, long working hours and job insecurity. Experimental manipulation of exposure to psychosocial work stressors and assessing mortality outcomes is infeasible and ethically questionable. However, data from some natural experiments have been published. Organisational changes such as downsizing and mergers have been used as proxy measures for work stressors in several studies and are associated with an increased risk of cardiovascular mortality and ill health among the remaining employees in the organisations (Kivimaki et al., 2000; Vahtera et al., 2005; Vahtera et al., 2004; Westerlund et al., 2004).

Evidence regarding mortality and psychosocial work stressors is weak and inconsistent, and research is lacking on the causal mechanisms through which job stressors affect health. The effect of psychosocial work stressors on mortality is multifaceted and involves many pathways, including direct effects via physiological processes and indirect effects via health behaviours. The effect may be spurious due to confounding factors and may be modified by individual differences. There is a lack of large nationally representative working population longitudinal studies of women and men with long follow-up times and repeated measures of exposures over time. Few longitudinal studies to date have comprehensively examined changes in psychosocial work stressors and mortality. These concerns contribute to the confusion regarding the association between psychosocial work stressors and mortality.

It is unclear whether the relationship between psychosocial work stressors and health is mostly contemporaneous or whether cumulative exposure to work stressors over several years is more harmful to health. To appropriately examine whether accumulated or changed exposure affects subsequent health, repeated measurements are required. The long-term effects of psychosocial work stressors on general health are not widely known, because the effect of prior work stressor exposure on current health has rarely been examined. It is also unclear whether previous health

status influences the relationship between psychosocial job stressors and health over the long-term.

Much of the literature to date has concentrated on the harmful effects of adverse psychosocial work stressors, with relatively little research on whether improving work stressors in the workplace could improve health. The scarcity of studies examining the duration and intensity of exposure to psychosocial work stressors has contributed to our lack of understanding of the relationship, including the effect of sex, between psychosocial work stressors and general health, and mortality. More high-quality research is required to elucidate the relationship between psychosocial work stressors and health, and mortality. To address some of the existing research gaps, studies with longer follow-up times and frequent repeat measures that evaluate and consider the potential confounding and other threats to internal validity are required to contribute better evidence regarding change, duration and long-term consequences of exposure to psychosocial work stressors and heath, and mortality outcomes.

1.10 Significance of Research

Currently, there is considerable perplexity and conflicting evidence regarding the relationship between psychosocial work stressors and health, and mortality. Acute stressors such as natural disasters or a loss of a child are known to trigger death (Leor, Poole, & Kloner, 1996; Li, Precht, Mortensen, & Olsen, 2003). However, the extent of the impact of chronic psychosocial work stressors on longevity in a general population is not well known. Prolonged exposure to psychosocial work stressors over time may result in ongoing elevated stress responses in the human body through the prolonged activation of the sympathetic nervous system and the hypothalamic-pituitary-adrenal axis, affecting cardiovascular, metabolic, and immune systems (Chandola et al., 2008; Chandola, Brunner, & Marmot, 2006). Prolonged activation of these systems may lead to stress-related diseases and possibly increase the risk of premature death. Stress may also contribute to a higher frequency of adverse health behaviours, which may affect

health. Assessment of the impact of psychosocial work stressor on all-cause mortality will advance our understanding of the consequences of chronic work stress and its implications for employee health. This thesis aimed to further add to the body of research by reviewing the available evidence of the association between common psychosocial work stressors and all-cause mortality, and mortality due to CHD, and examining the effect of psychosocial work stressors on mortality over time, and the extent to which changes in job control cause changes in general health.

There is considerable evidence that psychosocial work stressors influence a range of physical and psychological health outcomes and may play a role in contributing to the social gradient in ill health (Belkic, Landsbergis, Schnall, & Baker, 2004; Bongers, Kremer, & ter Laak, 2002; Karasek, 1979; LaMontagne, Keegel, Vallance, Ostry, & Wolfe, 2008; Marmot et al., 1997; Marmot & Wilkinson, 2006). Social inequalities in work quality have been documented in the literature, and it is generally accepted that the benefits and harm of the working life are unequally distributed among the population, as is the acceptance of a social gradient of health regarding occupational skill level for mortality, heart disease, psychiatric disorders and respiratory diseases (Siegrist & Marmot, 2004). Given that social factors associated with mortality vary the most during the middle years of an individual's life (Siegrist & Marmot, 2004), there is a need to examine the contribution of psychosocial work stressors to health and mortality outcomes and the underlying causal relationships.

The observational studies that have been conducted to date have mostly examined baseline exposure to psychosocial work stressors and have used relatively blunt job exposure matrix measures to estimate psychosocial work stressors. However, the duration and intensity of psychosocial work stressors vary during the working life course (Elder Jr, George, & Shanahan, 1996). Dynamics of the connexions between the exposure and outcome are not well known, yet it is of interest to determine whether the prior exposure to psychosocial work stressors has

continuing long-term effects on health or whether the relationship is mostly contemporaneous (Amick et al., 2002; Milner, Aitken, Kavanagh, LaMontagne, & Petrie, 2016). Additionally, there might be persistent indirect effects of lagged general health over time on health. Therefore, the dynamics of the relationship should be measured to mitigate the bias present in the averaged effects of exposure to psychosocial work stressors on general health. To better understand the role of work stressors, multiple measurements of exposure and designs that allow us to examine various causal relationships using conceptually advanced frameworks are required (Frese & Zapf, 1988). Measurements of psychosocial work stressor can be influenced by an individual's cognitive and emotional processing, as well as by their subjective perception of the stressors. The influence of cognitive and emotional processing on the subjective measurements of job control, however, appears not to be as considerable as for other work stressors. Different dynamic models can be developed of the psychosocial work stressors and health relationship using observational data to integrate different results to better examine the causal relationship.

The dynamics of the relationship between the commonly examined psychosocial work stressor perceived job control, a valid appraisal of the objective stressor, and general health, a strong predictor of future morbidity and mortality (DeSalvo, Bloser, Reynolds, He, & Muntner, 2006; Idler & Benyamini, 1997; Larsson, Hemmingsson, Allebeck, & Lundberg, 2002) was investigated in the present study to provide evidence of a causal relationship between job control and general health (as a proxy measurement for mortality) to compliment mortality studies that are customarily subject to a lack of precision and systematic error. Data were used from a repeated measures longitudinal study with time-varying psychosocial work stressors to elucidate the involvement of cumulative psychosocial work stressors on health and mortality. The observation and examination of exposure to work stressors over time in the working population present a useful supplement to intervention studies. The influences of deleterious

and positive work stressors, which are generally not feasible or ethical to randomise in intervention studies, were identified and examined in less constrained natural experiments.

The use of repeated measures panel data allowed for a robust examination of causal inferences. Statistical methods were utilised to observe the effects of different influences on life outcomes while controlling for the effects of fixed characteristics that cannot be ordinarily measured or even known, and that might otherwise confound the measurements of causal effects. Furthermore, in working population representative samples, precise questions regarding subtleties of the relationship between exposure and outcome factors could be addressed and resolved, and conclusions extrapolated to the general working populations. In contrast, randomised studies are likely to be conducted in selected organisations and worker groups limiting external validity. The Household, Income and Labour Dynamics in Australia (HILDA) survey (Wilkins, Laß, Butterworth, & Vera-Toscano, 2019) is an example of an existing panel data source that provides a considerable amount of information on a wide range of life domains. The family, health, income and labour dynamics, and data relating to individual behaviours and outcomes in the HILDA survey allow for examining the many linkages between various life domains. Thus, a detailed analysis of how mortality risk and poor health outcomes depend on an individual's psychosocial work stressors is feasible.

Psychosocial work stressors have been identified as significant risks linked to global changes in the structure, organisation and management of work during previous decades, presenting new challenges to occupational health and safety (Landsbergis, Grzywacz, & LaMontagne, 2014; Leka, Jain, & World Health Organization, 2010). Improving our understanding of the relationship between psychosocial work stressors and the risk of poor health, and mortality would have considerable public health implications and would be of benefit to the workers themselves as well as the various stakeholders, including organisations, workplaces, and society.

1.11 Aims, Objectives and Research Questions

The relationships among psychosocial work stressors, poor health and mortality require further investigation. Our understanding of exposure-outcome dynamics is poor, and it remains unclear whether the association between adverse psychosocial work stressors in the working environment and poor mental and physical well-being translates into an increased mortality risk. The purpose of this research was to investigate the effect of exposures to psychosocial work stressors in the working environment such as job control, job demands, job strain, long working hours, job insecurity and shift work on health and mortality. The research applied a repeated measures panel study to perform natural experiments of changes in psychosocial work stressors associated variations in health and well-being, and mortality to investigate whether and how the effect of psychosocial job stressors on health and mortality differed in relation to demographic and socioeconomic characteristics.

The specific research questions were:

1. What is the evidence regarding the effect of psychosocial work stressors on the risk of all-cause and cardiovascular mortalities? Answering this question involved undertaking a systematic review of the available literature and undertaking a meta-analysis of prospective cohort studies of psychosocial job stressors and mortality.

2. What is the effect of adverse psychosocial work stressors, namely high job demands, low job control, job insecurity and low fairness of pay, on all-cause mortality? Semi-parametric Cox proportional hazards regression models were used to examine the association between psychosocial work stressors and mortality, and a series of models that adjusted for demographic, socioeconomic, health and behavioural risk factors.

3. What is the effect of repeated exposure to adverse psychosocial work stressors, namely high job demands, low job control, job insecurity and effort-reward imbalance over time on all-

cause mortality? Fully parametric survival analysis models were used to examine the association between psychosocial work stressors over time and mortality.

4. Are changes in job control over time associated with changes in the perceptions of general health? Dynamic fixed-effects regression models controlling for time-invariant confounding factors were performed to investigate the effect of changes in job control on general health, and ordinary least squares regression models with cumulative job control exposure were used to analyse cumulative exposure to low job control.

Chapter 2 General Methods

2.1 Methods for Aim 1

To address the first research aim of this thesis, a systematic review and meta-analysis were undertaken in Study I. I conducted a comprehensive systematic review of the available literature from conception until the end of 2017 for studies examining the risk of mortality regarding psychosocial job stressors, in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines (Shamseer et al., 2015). A search strategy was formulated to systematically identify studies that answered the research question "What is the effect of psychosocial work stressors on the risk of all-cause and cardiovascular mortalities?" The key research question was specified by defining the 'PICOS' modules as follows: Population (P) = workers; Intervention or exposure (I) = exposed to psychosocial work stressors (low job control, high job demands, high job strain, low social support at work, high ERI, high job insecurity, shift work, long working hours); Comparisons (C) = contrasting exposure levels (high job control, low job demands, high social support at work, low ERI, low job insecurity, no shift work, ordinary working hours); Outcome (O) = mortality and Study type (S) = longitudinal studies.

2.1.1 Search strategy

I searched seven electronic databases covering a range of disciplines, including Medline, Pubmed, EMBASE and Web of Science (medical science databases); Scopus (social science database); PsycINFO (psychology database) and Global Health (public health database) for eligible literature published from their respective commencement date to the present. A three-tier search strategy was used to identify eligible studies. The first stage combined search terms with keywords inclusive of work stress and strain. The second stage combined search terms with keywords inclusive of psychosocial work stressors (job control, job demands, social support at work, ERI, job insecurity, shift work and working hours). The third stage combined

search terms with keywords inclusive of mortality. The Boolean operator "OR" was used to link terms together from the first and second stage searches and these were combined with the third stage search results using the "AND" Boolean operator (e.g., first stage results OR second stage results AND third stage results) (Table 2.1). No restrictions were placed on the date, status or language of publications. A secondary search included examination of the reference list of all studies identified for potential inclusion in the systematic review.

Table 2.1

Search Terminology and Strategy for Systematic Review

First stage	Second stage	Third stage
Search terms for work stress and work strain	Search terms for psychosocial work stressors	Search terms for mortality
"psychosocial job stress*" OR "working condition*" OR "psychosocial work*" OR "psychosocial job*" OR "occupation* stress*" OR "job stress" OR "job strain" OR "work stress" OR "work strain"	"job control" OR "job demands" OR "job secur*" OR "job insecur*" OR "work insecur*" OR "work secur*" OR "precarious work" OR "precarious employ" OR "precarious job" OR "decision latitude" OR "skill discretion" OR "decision authority" OR "psychosocial job demands" OR "job social support" OR "work social support" OR "workload" OR "effort reward imbalance*" OR "organisation* justice" OR "organisation* injustice" OR "organisation* fairness" OR "organisation* unfairness" OR "organization* justice" OR "organization* injustice" OR "organization* fairness" OR "organization* unfairness" OR "shift work" OR "work* hour*" OR "work* time" OR "work* span" OR "underemployment" OR (("work" OR "business" OR "employ*") and ("management style" OR "leadership")) OR (("temporary" OR "casual") and ("employ*"))	"mortality" OR "death"

2.1.2 Eligibility criteria

Original articles limited to the English language (or available translations in English) that had the key search terms in the title or abstract and mortality as an outcome variable were considered for inclusion in the systematic review. Reviews, letters, editorials, case reports,

book chapters and conference abstracts were excluded. Prospective population-level (cohort studies) and case-control studies that contained quantitative estimates and 95% confidence intervals (CIs) of the relative risk, rate ratio, odds ratio or hazard ratio (HR) for mortality associated with psychosocial job stressors were included in the systematic review. Studies investigating physical exposure to work-related factors including physical, chemical, ergonomic and biological factors were excluded from the systematic review as the focus of the review was the exposure to psychosocial job stressors associated with mortality. Studies were screened for eligibility using a two-stage process. Titles and abstracts of studies with the key search terms in the title or abstract, retrieved using the search strategy, were screened to identify studies that potentially met the inclusion criteria. Following a review of the full text, studies that met the eligibility criteria were retained.

2.1.3 Data extraction

Methodological details and data including study description, author, cohort, year, country, population (sample size, gender and age), exposure assessment and level, description of mortality (method of assessment and incidence), study design, duration of follow-up, number of events, confounders adjusted for in the analyses, effect size for mortality and CIs were extracted from each study selected for inclusion in the systematic review.

2.1.4 Quality assessment

For bias risk assessment, the quality of studies included in the systematic review was assessed using the Scottish Intercollegiate Guidelines Network (SIGN) methodological checklist for cohort and case-control studies with 14 criteria assessing the quality of evidence (Sanderson, Tatt, & Higgins, 2007). This checklist is recognised as a useful assessment tool for assessing quality and bias susceptibility in observational studies The checklist for observational studies is based on the rigorous and widely validated Method for Evaluating Research and Guideline Evidence checklist developed by the New South Wales Department of Health (Liddle,

Williamson, & Irwig, 1996) and further adapted by the Scottish Intercollegiate Guidelines Network to improve functionality while maintaining methodological rigour (Harbour & Miller, 2001). Studies that agreed with much of the guidelines with little or no bias risk were rated as high-quality. Studies that mostly agreed with the guidelines but contained some flaws with an associated bias risk were rated as acceptable. Studies that did not agree with the criteria or contained significant flaws with respect to study design were rated as low-quality.

2.1.5 Meta-analysis

Quantitative analyses of homogeneous studies, with sufficiently similar exposure, outcome and study population, were performed incorporating summary data into meta-analyses. For multiple studies reporting on the same study data, the most recent study with a longer follow-up period was included in the quantitative analysis. Multiple studies with the same author/s were included in the analyses insofar as the study sample, exposures or outcomes differed. Studies were included if age-adjusted effect estimates were available. Studies with unadjusted crude measurements were excluded because age is such a strong predictor of mortality. As the studies included in this review addressed the same outcomes and were measured in the same way, albeit with different approaches to analysis, and the outcome of interest was rare, in which case indices were approximately equal, studies with summary data on odds, risk and HRs were combined (Borenstein, Hedges, Higgins, & Rothstein, 2011). Reciprocals of effect estimates and CIs were calculated for risk estimates involving reversed exposure levels, and all effect estimates and CIs were log-transformed (Borenstein et al., 2011; Tierney, Stewart, Ghersi, Burdett, & Sydes, 2007) prior to entering into the meta-analysis software.

Separate inverse variance random-effects meta-analyses were conducted for each psychosocial work stressor with more than two studies and the primary outcome all-cause mortality; and the secondary outcome deaths due to CHD. For studies reporting multiple levels within an exposure, the referent group was only included once in the meta-analyses. Hence, the highest

job demands level was compared to lowest job demands level, job strain was compared to no job strain and lowest job control level was compared to highest job control level. For studies reporting multiple shift work categories (e.g., evening shift work, night shift work and rotating shift work), the category with the highest frequency was compared to the referent category of no shift work. In addition to the pooled effect size for individual psychosocial job stressors, separate subgroup random-effects meta-analyses were conducted for studies where effect estimates were stratified by gender. Pooled results, including pooled subgroup results, were presented on the exponential scale.

The I^2 statistic was used to calculate the heterogeneity between studies measuring variability due to between-study factors rather than chance alone. Funnel plots were used to assess the precision of estimates and publication bias (Sterne & Harbord, 2004) and a modified Egger's test was performed to test for small study effects by regressing effect estimates on their standard errors weighted by the reciprocal of the variance of intervention effect estimates (Harbord, Harris, & Sterne, 2009).

2.2 Data Source and Study Population for Research Aims 2, 3 and 4

2.2.1 HILDA survey

Data from the HILDA survey were used to address research aims 2, 3 and 4. HILDA is a nationally representative, household-based longitudinal study of Australian residents and households that collects detailed information about economic and personal well-being, labour market dynamics and family life from a combination of interviews (mostly face-to-face) and self-completion questionnaires (Wilkins et al., 2019).

Commencing in 2001 with a large national probability sample of Australian households residing in private residences, HILDA follows over 17,000 Australian lives annually. Residents of households comprise the panel data source and are interviewed in each wave of data

collection, with each wave being approximately 1 year apart. For Wave 1, HILDA reported data on 13,969 respondents from 7,682 households (66% response rate). Retention of these individuals during Wave 2 was 87% and greater than 90% for subsequent waves. The total number of respondents in each later wave was greater than the number of respondents in Wave 1 because during later waves: (i) some non-respondents in Wave 1 were subsequently successfully interviewed; (ii) all persons in sample households who turned 15 years of age were sought to be interviewed; and (iii) additional persons were added to the panel due to changes in household composition (Summerfield et al., 2019). In Wave 11, the sample was topped-up with the inclusion of an additional 3,652 people from 2,153 households into the study to correct for population under-coverage and retain cross-sectional representativeness in the sample. Specifically, the HILDA survey was extended to include representation of migrant populations and help alleviate biases from non-random attrition (Watson, 2006, 2011).

The advantages of using the HILDA dataset include the large sample size and representativeness of the Australian population. The panel data allows for the assessment of cumulative psychosocial job stressors and changes in socioeconomic, occupational and well-being characteristics over time. Health-related questions enable scrutiny of changes in self-reported health status, long-term disabilities and impairments and health behaviours. The HILDA survey sample is linked to the National Death Index (NDI); hence, details of deaths, including the date and cause of death, which occurred in the survey samples are recorded in the data files, allowing for the opportunity to examine the changes in work and life course preceding death (Watson & Summerfield, 2014).

2.2.2 HILDA questionnaires and data collection

The HILDA survey comprises data from four separate questionnaires. The Household Form (HF), Household Questionnaire (HQ), and Person Questionnaire (PQ) are administered via face-to-face interviews, whereas the Self-Completion Questionnaire (SCQ) is completed by

participants. The HF records basic information about the household structure directly after making contact and is mainly used to determine who is interviewed, the management of new members of the household and those who have left the household, and to record phone information and reasons for non-interviews. The HQ collects information about the household and is typically completed by a single member of the household, although more than one household member may contribute to the interview if so desired or required. The HQ records details regarding childcare, housing and household spending and wealth. The PQ is administered to all household members over the age of 15 years, although parental consent is obtained prior to interviewing children aged under 18 years who are still living at home. The PQ collects biographical data about the country of birth and language, family background, educational attainment, employment history, marital history, visa category for immigrants and parents' education. Together with the PQ, an SCQ is also completed by respondents, which is comprised of mainly attitudinal questions covering sensitive issues that may cause some discomfort or embarrassment for respondents to answer in a face-to-face interview. Data collected in the SCQ cover topics such as general health and well-being, lifestyle and living situation, personal and household finances, attitudes and values, job and workplace issues and parenting (Summerfield et al., 2019).

From 2009, the change of fieldwork data collection from Nielson to Roy Morgan Research resulted in a change in the data collection method to computer-assisted personal interviewing from pen and paper. The introduction of the computer-assisted personal interviewing instrument allowed for the use of dependent data from previous interviews to be preloaded into the HF. Additional information from the previous interview was also included as proactive dependent data to remind respondents of their responses from the previous interview, allowing them to amend the data if necessary or skip a sequence of questions answered previously. Most

of the data were collected through face-to-face interviews, although some telephone interviews were conducted as a last resort to ensure a high response rate (Summerfield et al., 2019).

2.2.3 Attrition in HILDA

The re-interview rate in the HILDA survey is lowest for participants in the 15 to 24 years age group, non-natives born in non-English speaking countries, Aboriginal or Torres Strait Islander persons, single people and those employed in low occupational skill level jobs or unemployed. Over time, missing data in the HILDA survey correlates with age, country of birth, labour force and occupation factors. Nonetheless, groups with low re-interview rates across waves remain engaged with the study. The difference in the re-interview rates for respondents from Wave 1 who participated in the most recent wave across characteristics does not vary greatly from those who participated during every wave of data collection. Furthermore, retention across the characteristics of the top-up sample from Wave 11, to adjust for population under-coverage and cross-sectional representativeness, are considerably higher than those reported for previous waves (Summerfield et al., 2019).

2.2.4 Ethical considerations in HILDA

The HILDA survey is conducted by the Melbourne Institute of Applied Economic and Social Research at the University of Melbourne on behalf of the Australian Commonwealth Government Department of Social Services. All relevant ethical issues associated with this research project (including consent) were addressed by the Melbourne Institute of Applied Economic and Social Research at the University of Melbourne, which operates within the university's Ethics Guidelines. All processes involving consent have been approved by both the University of Melbourne's Human Research Ethics Committee and the Australian Institute of Health and Welfare's Ethics Committee. All members of this group and researchers involved in the HILDA project must comply with the *Privacy Act (1988)*. The research concerning the HILDA survey data contained in this PhD received ethical approval from the Melbourne

School of Population and Global Health's Human Ethics Advisory Group (Application Reference Number: 1750707.2). The authors were provided with de-identified data by the organisational data custodian, in which the names and addresses had been removed upon submission of a signed and completed Confidentiality Deed Poll and approval from the Australian Government Department of Social Services (Australian Government Department of Social Services, 2019).

All participants are informed prior to commencing the HILDA survey (through a primary approach letter) about the objectives of the survey. Their participation is voluntary, with the ability to decline participation in the current or any future wave. They are informed about the purpose, management and access of the data provided, and guaranteed that their identity will be protected. Prior to the authorised linkage of the HILDA data to other administrative datasets, it is required that participants are fully informed about the reasons for linking data and provide informed consent prior to any undertaking. The HILDA survey dataset is provided under strict privacy and undertakings conditions. The data are made available to authorised users under strict terms and conditions regarding use, privacy and security set out in a confidentiality deed. Authorised users are not permitted to publish any information that could lead to the identification of the participants. To mitigate against the identification of the study participants, cell suppression is recommended, where the cell count is less than four and the inclusion of small cell sizes threatens anonymity. Furthermore, items that provide more detailed data such as geography, specific employment characteristics and occupational codes, date of birth, hours and types of childcare, housing payment amounts, interview outcome codes, and interview start- and end-times are only available in the Restricted Release HILDA datasets. Approval for seeking access requires strong justification (Australian Government Department of Social Services, 2019).

2.3 Methods Overview of Studies II, III and IV in Thesis

An overview of Studies II, III and IV in this thesis with data from the HILDA survey including sample population, exposure, outcome and confounder variables, and the statistical methods used are presented in Table 2.2.

Table 2.2

Overview of the Methods for Studies II, III and IV

	Study II	**Study III**	**Study IV**
Title	Psychosocial work stressors and risk of mortality in Australia: analysis of data from the HILDA survey	All-cause mortality and the time-varying effects of psychosocial work stressors: survival analysis of data from the HILDA survey	Changes in job control and perceptions of general health: A longitudinal analysis of Australian workers 2005–2017
Sample population	18,444 HILDA participants; Waves 1–15	20,423 HILDA participants; Waves 1–15	105,017 observations; 18,574 participants in HILDA; Waves 5–17
Inclusion	Participants aged ≥ 15 years who were employed with data on psychosocial work stressors at baseline	Participants aged ≥ 15 years who were employed with data on psychosocial work stressors	Participants aged 15-64 years who were employed with data on job control and general health
Exposure(s)	Job demands and complexity Job control Job security Fairness of pay	Job demands and complexity Job control Job security Fairness of pay	Job control
Outcome	All-cause mortality	All-cause mortality	Self-reported perception of general health
Potential confounders	Gender, calendar year, country of birth, education, income, occupation level, employment arrangement, household structure, long-term health condition, alcohol consumption, smoking, physical activity	Age, calendar year, gender, labour force status, country of birth, education, income, occupation level, employment arrangement, household structure, long-term health condition	Age, calendar year, gender, education, income, occupational level, employment arrangement, household structure, significant life events, long-term disability, health condition, alcohol consumption, smoking, physical activity,1-year lag of job control, 3-year lag of general health
Statistical methods	Cox proportional hazards regression models serially adjusted for each subgroup of demographic, socioeconomic, health and behavioural risk factors	Fully parametric time-varying covariates survival analysis with baseline hazard specified using the Weibull distribution	Fixed-effects regression models; Ordinary least squares regression with cumulative job control exposure;

2.4 Psychosocial Work Stressors Exposure Variables

In every wave of the HILDA survey, measures of psychosocial work characteristics, based on a variety of economic and social surveys, were available. Participants in paid employment were initially asked to respond to 12 items about job control, demands and complexity, job insecurity and perceptions of unfair pay on a Likert scale ranging from 1 (strongly disagree) to 7 (strongly agree) in the SCQ. Many of the questions were based on the Karasek job demand-control model; however, four items were taken directly from the International Social Science Surveys Australia series. From Wave 5 onwards, an additional nine items concerning job demands and control and workload with responses ranging from 1 (strongly disagree) to 7 (strongly agree) were added to the SCQ. These items were taken from the Personality and Total Health (PATH) Through Life study (Summerfield et al., 2019).

Constructs of psychosocial work stressor exposures in this thesis are based on the work stress models, including the Karasek job demand-control and ERI models using the available items from the HILDA survey which limited the psychosocial work stressor exposures to those scales and items available in the HILDA survey. Three different measures of the psychosocial work environment, previously identified through factor analysis and structural equation modelling, were constructed: job demands and complexity, job control and perceived job security (Leach, Butterworth, Rodgers, & Strazdins, 2010). An additional single item that assessed whether respondents believed they were paid fairly for their work efforts was considered a measure of effort-reward unfairness (Siegrist, 1996) (Table 2.3). One measure of job demands and complexity, “my job is more stressful than I had ever imagined”, was not included in the job demands and complexity construct due to confirmatory factor analyses indicating that removing it from the jobs demand and complexity factor improved the modelling (Leach et al.,

2010). These individual scales have demonstrated predictable associations with more widely used measures including high job demands, low job control, low job security, effort-reward unfairness and employment status (Butterworth et al., 2011; Leach et al., 2010). The job security item "I worry about the future of my job" was reverse coded so that high scores represented higher security for all items. Scores for individual construct scales were calculated as the sum of item scores. As there are no normative data for these scales, scores were dichotomised at the median value into high and low levels, consistent with the most common operationalisation of psychosocial work factors, to enable comparison of results to previous studies (Courvoisier & Perneger, 2010; de Lange et al., 2003). At the median and above, exposure was defined as low, whereas below the median was high exposure for job control, job security and effort-reward unfairness and the reverse for job demands. For the few participants with missing data for some items (< 1%), scale scores were based on completed items and weighted up to the expected total had all items been answered.

In Study III, time-varying measures of each of these psychosocial work stressors were constructed by considering each stressor at each time point as a separate observation. All items were recoded in the same direction, such that high scores represented higher control, greater demand and complexity, greater security and higher fairness of pay, and scores for each scale were summed. For those participants with missing data for some items (1.6%), scale scores were based on the completed items and weighted up to the expected total had all items been answered. As the psychosocial work stressors exposure is a relative measure (Karasek et al., 1981; Stansfeld, Fuhrer, Shipley, & Marmot, 1999), the choice of cut points was constructed to identify approximately one-third of the participants as experiencing the most adverse levels of each psychosocial work stressor. Scales were, therefore, divided into tertiles. For each psychosocial stressor exposure measured, participants with scores in the tertile of the distribution corresponding to the most adversity (lowest job control, highest job demands,

lowest job security and lowest perceptions of fair pay) were categorised as exposed. The remaining two tertiles for each scale were combined to create the reference category.

Table 2.3

Construct of Psychosocial Work Stressors Examined in Study II and Study III

Construct name	Items in the construct (from 1 = strongly disagree to 7 = strongly agree)
Job demands and complexity	My job is complex and difficult My job often requires me to learn new skills I use many of my skills and abilities in my current job
Job control	I have a lot of freedom to decide how I do my own work I have a lot of say about what happens in my job I have a lot of freedom to decide when I do my work
Job security	I have a secure future in my job The company I work for will still be in business 5 years from now I worry about the future of my job *(reverse coded so that 1 = strongly agree to 7 = strongly disagree)*
Fairness of pay	I get paid fairly for the things I do in my job

For Study IV, a job control measure based on the Karasek job demand-control model was constructed (Table 2.4). Initially, in the HILDA survey, three items assessed decision authority and two items assessed skill discretion. However, from Wave 5 onwards, four additional items about decision authority and two items about skill discretion were added to the SCQ. The construct of the job control measure in Study IV involved the 11 items available from Wave 5 onwards. The skill discretion item "my job requires me to do the same things over and over again" was reverse coded so that high scores represented higher control for all items. Separate scales were constructed for skill discretion and decision authority and the scores were calculated as the sum of the item scores. For participants with missing data for some items (decision authority 1.0%; skill discretion 0.6%), the scale scores were based on completed items and weighted up to the expected total had all items been answered. Summary scales for decision authority and skill discretion were combined into an equally weighted scale of job

control. Quintiles of job control were defined with the 'xtile' command in Stata 14.2 using the data from Wave 5, with the same cut-off scores applied to each subsequent wave of the survey.

Table 2.4

Construct of Job Control Exposure Examined in Study IV

Construct name	Items in the construct (from 1 = strongly disagree to 7 = strongly agree)
Decision authority	I have a lot of freedom to decide how I do my own work I have a lot of say about what happens in my job I have a lot of freedom to decide when I do my work I have a lot of choice in deciding what I do at work My job requires me to take initiative My working times can be flexible I can decide when to take a break
Skill discretion	My job often requires me to learn new skills I use many of my skills and abilities in my current job My job provides me with a variety of interesting things to do My job requires me to do the same things over and over again (*reverse coded so that 1 = strongly agree to 7 = strongly disagree)*
Job control	Combined decision authority and skill discretion scales (range 11 – 77)

2.5 Mortality Outcome

The entire HILDA survey sample was matched to the NDI in 2014. In summary, 1,238 deaths had already been identified through the HILDA survey fieldwork and an additional 333 deaths were identified by the data linkage. A total of 64 deaths (4%) identified via fieldwork could not be matched to the NDI. A further 212 deaths identified by fieldwork as occurring after the data linkage were also included in the data files. One death was identified as having occurred prior to the data linkage and incorporated into an earlier wave in HILDA (Watson & Summerfield, 2014).

The date of death of HILDA participants reported to interviewers was frequently incomplete. At the time of data linkage, only 62% of deaths reported contained both the month and year of death and 3% had data only on the year of death. For the remaining 35% of reported deaths, it

was assumed that the death had occurred between the current wave when the death was notified and the wave prior to the notification of death when it was known that the patient was not deceased. Where both month and year of death was available in HILDA, there was an 87% exact match to the death register and two-thirds of those that did not match had an earlier date of death according to the death register. This is most likely due to forward telescoping bias, where individuals are more likely to remember the event occurring more recently than it had happened (Watson & Summerfield, 2014). The cause of death was classified according to the International Classification of Diseases, Version 10 (ICD-10); however, the cause of death information is not very complete. Approximately 40% of deaths reported in HILDA are missing the cause of death information due to processing delays in the NDI (18.0%), cause of death being withheld in the NDI (4.9%), no match identified in the NDI (3.6%) and deaths reported in HILDA after the data linkage (13.6%).

The causes of death data in the HILDA survey aligned reasonably well to the distribution of deaths in the Australian population (Table 2.5). The leading causes of death in HILDA due to ischaemic heart disease (17.0%), cerebrovascular disease (7.5%), lung cancer (5.5%), dementia and Alzheimer's disease (4.7%) and chronic respiratory disease (5.0%) corresponded with the leading causes of death in the Australian population even though the HILDA deaths were observed over 13 years and all deaths in Australia were based on deaths registered in a single year in 2007, the midway point of the HILDA survey (ABS, 2014; Watson & Summerfield, 2014).

Table 2.5

Leading Cause of Death (adapted from Watson & Summerfield, 2014)

Leading cause of death (ICD-10 codes)	All deaths in Australia in 2007		HILDA survey deaths during 2001-2013 (n=1,069 deaths with cause of death information)	
	Ranking	Percent	Number of cases	Percent
Ischaemic heart diseases (I20-I25)	1	16.7	182	17.0
Cerebrovascular diseases (I60-I69)	2	8.3	80	7.5
Trachea, bronchus and lung cancer (C33-C34)	3	5.5	59	5.5
Dementia and Alzheimer's disease (F01, F03, G30)	4	5.3	50	4.7
Chronic lower respiratory diseases (J40-J47)	5	4.2	53	5.0
Colon, sigmoid, rectum and anus cancer (C18-C21)	6	3.0	33	3.1
Diabetes (E10-E14)	7	2.8	25	2.3
Blood and lymph cancer (C81-C96)	8	2.6	43	4.0
Heart failure (I50-I51)	9	2.5	21	2.0
Diseases of the urinary system (N00-N39)	10	2.3	23	2.2
Other causes of death		46.7	500	46.8

2.6 General Health Outcome

The HILDA survey includes the Short Form 36 (SF-36) instrument in the SCQ in every wave of data collection. The SF-36 is an internationally recognised diagnostic tool for evaluating mental and physical health. It is widely used as a self-completion measure of health status and includes items regarding physical, psychological and social functioning, symptoms experienced and limitations due to health. There are 36 items with eight subscales of distinct health domains: mental health, general health perceptions, physical functioning, role limitations due to physical health, bodily pain, vitality, social functioning and role limitations due to emotional problems (Ware, 2002). All subscales are scored from 0 to 100, with higher scores indicating better health. The SF-36 has undergone an extensive evaluation of content, construct, criterion and predictive validity, and proven test-retest reliability and good psychometric properties (Ware & Gandek, 1998).

The SF-36 measures from the HILDA survey are psychometrically sound, with good internal consistency, discriminant validity and high reliability, and scales are significantly associated with social status measures (Butterworth & Crosier, 2004). General health was ascertained from the five-item General Health subscale, which measures individual perceptions of general health with evaluations for rating own health, comparison with others' health and with others' proneness to illness. Items are rated on a 5-point Likert scale (four items ranging from 1 (definitely true) to 5 (definitely false) and one item ranging from 1 (excellent) to 5 (poor) in the SCQ). Three items were reverse coded so that higher scores represented better general health for all items (Table 2.6). Raw scale scores were calculated by summing the items and the raw score was transformed to a 0–100 scale. A person-specific raw score was estimated if valid responses were available for more than half the items in the scale, the average of which was calculated and applied to the missing data (Ware, Snow, Kosinski, & Gandek, 1997).

This measure of self-related health is a strong predictor of future morbidity and mortality (DeSalvo, Bloser, Reynolds, He, & Muntner, 2006; Idler & Benyamini, 1997; Larsson, Hemmingsson, Allebeck, & Lundberg, 2002) and is used in health research and large-scale surveys (Subramanian, Huijts, & Avendano, 2010). Although there is no universally accepted translation of the general health score difference to clinical meaningfulness, it has been suggested that a difference of three points on the norm-based scale reflects a minimal clinically important difference (Ware, 2000) and a difference of four or more on the unstandardised scale represents a moderate clinically significant effect (Contopoulos-Ioannidis, Karvouni, Kouri, & Ioannidis, 2009).

Table 2.6

Construct of General Health Outcome Examined in Study IV

Construct name	Items in the construct
General health	In general, would you say your health is: 1 = excellent, 2 = very good, 3 = good, 4 = fair, 5 = poor; reverse coded so that 1 = poor to 5 = excellent How TRUE or FALSE is the following statement for you? I seem to get sick a little easier than other people: 1 = definitely true, 2 = mostly true, 3 = don't know, 4 = mostly false, 5 = definitely false How TRUE or FALSE is the following statement for you? I am as healthy as anybody I know: 1 = definitely true, 2 = mostly true, 3 = don't know, 4 = mostly false, 5 = definitely false; reverse coded so that 1 = definitely false to 5 = definitely true How TRUE or FALSE is the following statement for you? I expect my health to get worse: 1 = definitely true, 2 = mostly true, 3 = don't know, 4 = mostly false, 5 = definitely false How TRUE or FALSE is the following statement for you? My health is excellent: 1 = definitely true, 2 = mostly true, 3 = don't know, 4 = mostly false, 5 = definitely false; reverse coded so that 1 = definitely false to 5 = definitely true

2.7 Covariates

Information on demographic, socioeconomic, health and behavioural factors, identified a priori, obtained from SCQs were included and examined (Figure 2.1).

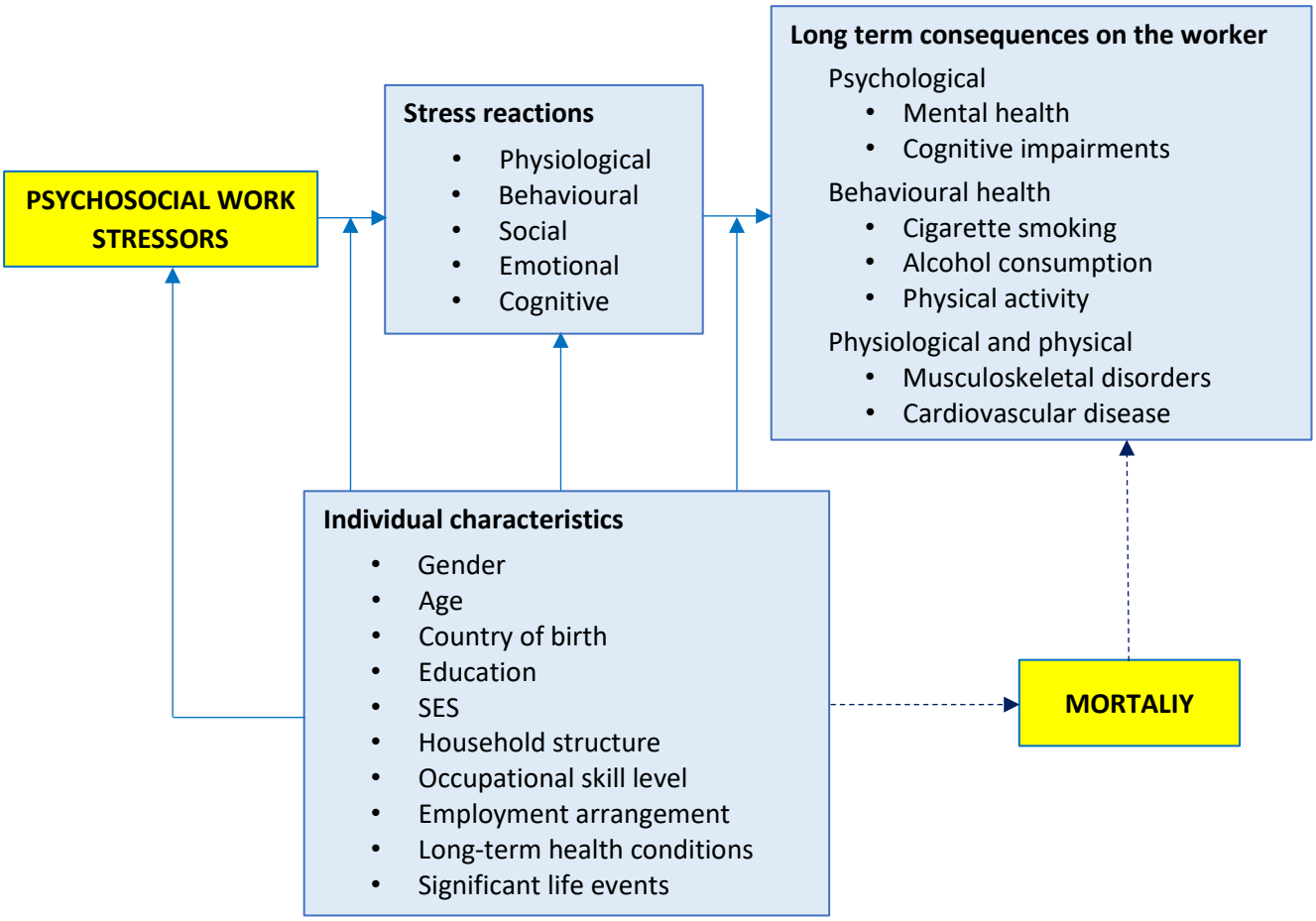

Figure 2.1

Directed Acyclic Graph for Psychosocial Work Stressors and Mortality

2.7.1 Demographic factors

Calendar year of study entry was controlled for in the baseline survival analysis in Study II. For the time-varying covariate survival analysis in Study III, the wave of first entry into the HILDA survey was grouped into two levels (2001–2010 and 2011–2015) to control for the top-up sampling that occurred in 2011. In the final study, the calendar year of the HILDA survey was included in the analyses as a continuous variable. Similar to Study II, Cox proportional hazards regression models with age as the time metric were used; age was calculated in 10-

year bands (15–24, 25–34, 35–44, 45–54, 55–64 and 65+) and fixed at the time of study entry. In Study III, age at each wave of the survey was measured continuously. For Study IV, 10-year bands (15–24, 25–34, 35–44, 45–54 and 55–64 years) were calculated for every wave. Further demographic factors controlled for in all studies included sex (male or female), country of birth (Australia, other English-speaking country and other non-English speaking country) and household structure (couple with no children, couple with children, lone parent with children, lone person and other). In Study IV, labour status was ascertained yearly from a work-status question in the HILDA survey and participants were classified as employed, unemployed or not in the labour force/retired for the year preceding the current interview year.

2.7.2 Socioeconomic factors

Typically, three variables, income, occupation and education, are used to describe SES when examining health inequalities and although they are interconnected, they are not equal (Gallo & Matthews, 2003). Each variable encapsulates the resource and status aspects of SES, with higher levels coveted in society, and each variable contributing to the study of SES-based health distinctions. Income and occupation might be strongly connected to social status as an adult; however, the association between education and health is less likely to be affected by reverse causation because education is generally completed prior to the development of significant health problems. Hence, later in life, income and occupation may best describe SES and education becomes less relevant. However, examining education and health can assist help segregate the directional effects of SES on health because inequalities in health in adulthood may be attributed to experiences in early life, and SES may both precede and respond to health conditions (Christie & Barling, 2009).

The following socioeconomic factors were controlled for in the studies: educational attainment (no completion of high school, completion of high school or vocational certificate/diploma and bachelor's degree or above), occupational skill level (low, medium and high) according to

Australian and New Zealand Standard Classification of Occupational groupings (ABS, 2013), employment arrangement (permanent, casual or labour hire, fixed term and self-employed) and income (quintiles of the population distribution of disposable household income with the highest quintile used as the referent group). In the HILDA survey, household disposable income is calculated by summing the income components for the previous financial year for all adults in the household (gross regular income minus taxes). Consequently, the disposable income was equivalised using the modified Organisation for Economic Co-operation and Development scale (Hagenaars, Vos, Zaidi, & Statistical Office of the European, 1994). For each wave of data, I converted nominal household income values to quintiles of the Australian population distribution using percentile statistics for the corresponding financial year released by the ABS from the biennial Survey of Income and Housing (ABS, 2017). The average household incomes for the calendar years not included in the biennial survey were estimated from the mean of the years on either side.

2.7.3 Health factors

Analyses were adjusted for disabilities and health conditions, including mental disorders. The long-term health condition binary variable used was based on the following question from the PQ in the HILDA survey: "Do you have any of the following disabilities/health conditions that have lasted, or are likely to last, 6 months or more, restrict everyday activity and cannot be corrected by medication or medical aids including sight problems not corrected by glasses or lenses; hearing problems; speech problems; blackouts, fits or loss of consciousness; difficulty learning or understanding things; limited use of arms or fingers, difficulty gripping things, limited use of feet or legs; nervous or emotional condition that requires treatment; condition that restricts physical activity or physical work; disfigurement or deformity; mental illness that requires help or supervision; shortness of breath or difficulty breathing; chronic or recurring pain; long-term effects as a result of a head injury, stroke or other brain damage; long-term

condition or ailment that is still restrictive even though it is being treated or medication is being taken for it; other long-term condition such as arthritis, asthma, heart disease, Alzheimer's disease or dementia?"

2.7.4 Health behavioural factors

Health behavioural factors are strong predictors of mortality and are associated with work stress (Chandola, Brunner, & Marmot, 2006). Adjustment for these risk factors may control for selection into stressful work environments because of early risk factors and adverse environments during childhood and adolescence (Hughes et al., 2017). Therefore, adjustments for cigarette smoking, alcohol consumption and physical activity were included in Studies II–IV. Participants were asked whether they smoked cigarettes or other tobacco products using a five-point response scale (1 = No, I have never smoked, 2 = No, I no longer smoke, 3 = Yes, I smoke daily, 4 = Yes, I smoke at least weekly (but not daily) and 5 = Yes, I smoke less often than weekly). Responses were recoded to construct a smoking variable with "0" coded as "non-smoker" if initial responses ranged from 1 to 2 inclusive and "1" coded as "current smoker" if initial responses ranged from 3 to 5 inclusive.

The HILDA survey contains two questions relating to alcohol consumption in the SCQ intended to capture current drinking patterns. Participants were asked how often they currently consume alcohol with seven response categories ranging from never to every day (I have never drunk alcohol, I no longer drink, I drink alcohol every day, 5–6 days/week, 3–4 days/week, 1–2 times/week, 2–3 times/month and rarely). Participants were then asked how many standard drinks (approximately 10 g of alcohol or equivalent to 12.5 mL of pure alcohol) they consumed on any usual drinking occasion with seven response categories (1–2, 3–4, 5–6, 7–8, 9–10,11–12 and 13+ standard drinks) (NHMRC, 2009). The estimated standard alcoholic drinks per week were calculated by multiplying the median number of standard drinks/occasion (e.g., 1–2 standard drinks = 1.5, 9–10 standard drinks = 9.5 and the highest category 13+ standard

drinks conservatively capped as 13.5) by the frequency/week reported consuming alcohol, again using the median for each category (e.g., 1–2 = 1.5 days/week, 5–6 = 5.5 days/week, daily = 7 days/week). The 2–3 times/month category was converted to a weekly estimate using 2.5 times/months and 365.25 days per year (365.25/12 = 4.35 weeks per month) = 0.575 times/week. "Rarely" was conservatively estimated at zero times/week. Participants were grouped into four categories: abstinence, low consumption (less than 1 drink per week), moderate consumption (women: 1–20 drinks per week and men: 1–27 drinks per week) and heavy consumption (women: 21+ drinks per week and men: 28+ drinks per week) (Hakulinen et al., 2015).

Data on physical activity was based on a single question from the SCQ in the HILDA survey regarding moderate- and vigorous-intensity activity. Participants were asked "In general, how often do you participate in moderate or intensive physical activity for at least 30 minutes?" with six response categories ranging from not at all to every day (not at all, less than once a week, 1 to 2 times a week, 3 times a week, more than 3 times a week and every day). Responses were recoded to construct a physical activity variable containing three categories (> 3 times per week, 1–3 times per week and less than once a week/never) for inclusion in the analyses in Studies II, III and IV.

2.7.5 Significant life events

At some stage during their life, most individuals will obtain a job, with some entering part-time employment while still at school and full-time employment eventually taking over from study during young adulthood. The achievement of these various milestones contributes to the timing of further transitions, including leaving home, partnering and becoming a parent. The timing and sequencing of transitions together with the circumstances that contribute to their occurrence influence well-being. Such transitions are known as 'life events' because physical and mental adjustments to these demands are often required (Moloney, Weston, Qu, & Hayes,

2012). These are not the only experiences that will occur in life. Other circumstances in life may be the result of previous life events or may occur independently. Some experiences may relate to other people, but nonetheless have a profound impact on the health and well-being of the individual (e.g., sickness or injury of a close friend or family member, or the death of a family member). Many of these events, such as marriage and parenthood, are usually celebratory; however, others, for example, the death of a friend, are unfavourably perceived. Regardless, life events are disruptive and generally involve some sense of loss associated with the change in life caused by the event (Moloney et al., 2012). Therefore, an adjustment for life events is required when examining the perceptions of general health. The life events examined in Study IV included the following events strongly predicting declines in well-being among those affected: separated from a spouse or long-term partner, serious personal injury/illness, serious injury/illness to a close relative/family member, death of spouse or child, death of close relative/family member, victim of physical violence and major worsening in finances (Qu, Baxter, Weston, Moloney, & Hayes, 2012). The events formed part of a list of 21 events included in the SCQ distributed to all HILDA household members aged 15 years and older. The number of significant life events was derived based on the responses of respondents regarding having experienced the aforementioned life events in the previous 12 months and categorised as no events, one event, two events, or three or more events.

2.8 Statistical Methods

In this thesis, exposure and covariate data measured at the baseline in the HILDA surveys during Waves 1–15 for Study II and measured repeatedly during Waves 1–15 for Study III were modelled, and the associations between psychosocial work stressors and all-cause mortality examined using survival analyses. For Study IV, fixed-effects regression analyses and ordinary least squares regression with cumulative job control exposure were used to examine whether the change in job control was associated with the change in the perceptions

of general health. The statistical analyses applied in the studies were performed using STATA/SE versions 14 and 16 (StataCorp, College Station, Texas).

2.8.1 Descriptive analyses

Counts and percentages for categorical data and the mean and standard deviation for continuous data were used to describe characteristics of the study sample. In Study IV, additional descriptive analyses were conducted to assess the frequencies of job control and mean of self-rated health by each variable of interest, plotting their year-to-year changes, and summarising the numbers of contributed survey waves per participant.

2.8.2 Cox proportional hazards model

In Study II, the Cox proportional hazard regression model was used to examine the effect of baseline exposure to psychosocial work stressors on survival. The effect measure is the hazard rate that is the risk of failure of an event conditional on survival up to a specific time. The hazard represents the expected number of events per interval of time. Cox proportional hazard regressions are semi-parametric models where time is divided into reasonably small intervals and the baseline hazard is assumed to be constant for each time interval, resulting in a piece-wise exponential model. The HR is the ratio of hazards rates between two or more groups. While there are no assumptions about the shape of the baseline hazard function, the key assumption of the Cox model is the proportional hazards assumption, i.e. it is assumed that each variable has a multiplicative effect in the hazards function that is proportional over time (D. R. Cox & Oakes, 1984). Tests are available to assess this assumption, although it is highly sensitive to sample size, meaning that even small departures from proportionality may be flagged in a large sample.

In Study II, participants first became at risk of death at date of birth, and came under observation at the time of study entry (time of first employment recorded in the HILDA survey

data). The scale for time-since-birth (analysis time) was measured in calendar years; specifically change in age in years. Age in years was used as the time metric rather than number of days since time of study entry to control for left censoring associated with increased mortality risk with increasing age. Participants were right censored at the age at last interview for participants who remained alive. Baseline measures of exposures and covariates at the time of first employment recorded in the HILDA survey data were included in Cox proportional hazard regressions.

2.8.3 Weibull model

The Cox proportional hazard regression does not consider the actual time between the baseline and the event, only the sequence of events; hence, there are no assumptions regarding the shape of the baseline hazard function. In Study III, it was not feasible to model repeated exposures and covariates over time without specifying the baseline hazard in the models. Preliminary analyses were conducted to test for the proportionality assumption. Examining the relationship between the scaled Schoenfeld residuals and survival time scaled concluded that the assumption of proportional hazards was violated for exposure variables (Cleves, Gould, & Marchenko, 2016). Consequently, a fully parametric survival analysis model with a Weibull distribution specified for the baseline hazard was fitted to the data to calculate HR and 95% CI for the association between psychosocial work stressors and mortality.

This parametric approach assumes a specific functional form for the baseline hazard and models based on the Weibull distribution. With a parametric specification of the baseline hazard, the risk of death is assumed to increase exponentially over time with age. As time progresses, it becomes more and more likely that mortality will occur in the next period given that one has lived until the current period. Therefore, the hazard function steadily increases as it becomes less likely that one will still be alive after each successive period, which is why the survival function decreases as time goes on. The Weibull distribution can accurately model the

time-to-failure of the mortality event and is sufficiently flexible despite having only two parameters: the shape and scale parameters. The shape and scale parameters that best fit the data are found using the maximum likelihood method. The Weibull model is known to approximates the distribution of time to mortality reasonably well (Kalbfleisch & Prentice, 2002).

A survival analysis dataset with multiple observations per person was constructed. To be considered eligible for inclusion in the analysis, participants must have been employed (and exposure measured) for at least one wave in the HILDA survey. Time at risk commenced on inclusion into the study and ceased at the time of death or year of the last interview in the HILDA survey. In Study III, participants who transitioned into long-term unemployment or retirement, or who left the labour force due to study, caring responsibility or disability, but continued in the survey, were censored 5 years following their last year of employment and psychosocial work stressors exposures were fixed at the time of the last interview with the employment data. This was performed because I assumed that the psychosocial work stressors exposures effects would persist for up to 5 years after a person exited the labour force (Amick et al., 2002; McDonough, Duncan, Williams, & House, 1997). To control for the effects of unemployment and retirement, these variables were measured yearly and included in the models. The occupation and employment arrangement data at the time of last employment of the participants were carried forward to the time of the last interview or death for participants who ceased to work during the study.

2.8.4 Within-person analysis

For Study IV, within-person fixed-effects regression analysis was used to study the association between differences in job control and general health. Repeated measures analysis can control for unmeasured time-invariant confounding, which commonly affects observational studies. Data from 13 waves were pooled into a common pool of observations and within-person fixed-

effects estimates were calculated. Examining the within-person association is an effective strategy for the analyses of changes in the outcome related to changes in the exposure.

Typically, within-person analysis is modelled using random-effects, as these models assess the combined influence of time-invariant variables and variables that change over time in panel data (Wooldridge, 2016). In random-effects models, the unobserved heterogeneity is controlled because the introduction of an error term at each level of the analysis divides the unexplained variance into distinct hierarchical levels, which controls for the possibility that residuals in the different levels may not be distributed with the same variance (Gardiner, Luo, & Roman, 2009). However, the random-effects model is underpinned by the assumption that residuals are uncorrelated with the observed covariates, that is, the variation between persons is random and no unmeasured time-invariant factors are associated with the exposure and outcome (Gunasekara, Richardson, Carter, & Blakely, 2014). Violation of this central assumption may introduce confounding bias into the model estimates.

Fixed-effects models are designed to study the causes of changes within a person; hence, they are not affected by confounding from unmeasured time-invariant personal, demographic and environmental factors. Such models are a more robust test of the causal nature of the relationship between job control and general health, and are particularly useful where time-invariant confounding is likely to create bias in causal estimates (Gardiner et al., 2009; Kohler & Kreuter, 2012). Fixed-effects regression modelling makes use of repeated measures, allowing each individual to act as their own control; therefore, the exchangeability of exposed and unexposed observations is enhanced, time-invariant individual factors are controlled for and causal inference improved (Gunasekara et al., 2014). Fixed-effects models do not estimate time-invariant characteristics, because factors that do not change over time drop out due to mean-centring. Rather, fixed-effects estimators rely only on the variation within individuals. For each person in the dataset, the exposure-outcome association was compared to non-

exposure and outcome association at each wave and data were pooled to calculate average differences within-persons. Therefore, for a given person, the coefficients represent the difference in general health during the years in which they were exposed to low job control compared to those years where they were not exposed to low job control.

The reduction in bias using a fixed-effects model may come at the expense of precision, particularly if there is little change in exposures over time. Therefore, a random-effects model may be preferable where confounding bias is minimal. To test that the central assumptions of the random-effects model were not violated, biasing results, the Hausman specification test was used to assess the consistency of the fixed-effects and random-effects models (Hausman, 1978). The Hausman specification test compares the parameter estimates of the fixed-effects and random-effects models using a Wald test of the difference between the coefficient estimates vectors (Wooldridge, 2016). If a systematic difference exists in the estimates, the random-effects estimator likely violates the underlying assumptions, and the fixed-effects model is considered the more consistent and reliable model.

2.8.5 Dynamic fixed-effects models with lagged effects

Neither fixed-effects nor random-effects models control for unmeasured time-varying confounding or reverse causation, and directionality cannot be established when exposure and outcome are modelled contemporaneously (Gunasekara et al., 2014). The effect of psychosocial work stressors might affect general health in the current year and have a carry-on effect in subsequent years. Fixed-effects models do not control for lagged effects, which may bias effect estimates. Inserting a lagged exposure variable into the fixed-effect model controls for the direct effect of lagged job control on current health, allowing for the examination of the dynamics of the exposure-outcome relationship; specifically, whether prior (i.e. time lagged) exposure to low job control has a continuing long-term effect on health (Ahlin, LaMontagne, & Magnusson Hanson, 2019; Milner et al., 2016). General health in the years prior to the year

of measurement may also be a persistent effect on general health that may bias effect estimates if not controlled for in the analysis. To mitigate these biases, dynamic fixed-effects models with lags of prior general health, current job control exposure and a 1-year lag of job control exposure were performed using Arellano-Bond regression analyses (Arellano & Bond, 1991).

Typically, the inclusion of a lagged outcome variable from future periods is dependent on the outcome variable from earlier periods, and in a fixed-effects model negates the assumption of independence inherent in within-persons data, hence exchangeability of exposed and unexposed observations over the periods is compromised. The longer the duration of time under observation, the less related the lagged and current outcome variables, and the less bias observed. Thus, results will be biased if fixed-effects modelling is used to estimate the persistence parameter. To manage this concern, Arellano and Bond (1991) proposed employing a generalised method of moments estimator to first-difference models, where instrumental variables for the lagged change in the outcome variable including earlier lagged values of the explanatory and outcome variables are used. However, this method holds true only if the instrumental variables are correlated with the first difference of the lagged outcome variable but not with the first differenced outcome variable other than through the lagged difference itself. To test the validity of the principal assumptions in the model, the Arellano-Bond test for serial correlation in first differenced errors was used to determine the accurate lags for outcome persistence included in the model (Arellano & Bond, 1991).

2.8.6 Cumulative exposure approach

To complement the preceding analyses and strengthen the causal inference, I also conducted a prospective exposure to outcome analysis. A cumulative exposure approach was applied such that I could also assess the potential for dose-response. I assessed the cumulative exposure as a function of both exposure duration and intensity and examined the directionality between job control and general health. Cumulative exposure over consecutive waves was assessed to

determine whether job control predicted subsequent general health. A cumulative exposure approach assessing the potential for exposure duration and intensity to predict future outcome might improve causal inference by establishing temporal precedence and allowing for the assessment of dose-response (Amick et al., 2002). Because participants contributed, on average, to four consecutive waves in HILDA, the cumulative job control exposure variable was derived from any four multiple consecutive waves of employment with job control data during the total period of observation in the HILDA survey. Including only observations of four consecutive waves was done to control for duration in assessing dose-response. The cumulative exposure was calculated as the job control quintile ($t_1 + t_2 + t_3 + t_4$). The general health outcome was measured in the subsequent wave to the last observed job control exposure (t_5); that is, in the fifth consecutive wave for each contributing participant.

The relationship between cumulative job control and general health was modelled using ordinary least squares (OLS) multiple linear regression (between persons). The OLS method based on the least squares principle minimises the sum of square differences between the observed dependent variable and predicted values. The OLS multiple regression model is centred on the following assumptions: linearity in parameters, absence of multicollinearity, observations randomly selected, residuals normally distributed and homoskedasticity of errors. According to the Gauss-Markov theorem, under these assumptions, OLS parameters of the regression are true estimates of the real values of the explanatory and dependent variables (Wooldridge, 2016). The OLS regression results can be interpreted as the mean change in outcome per level of the explanatory variable.

Chapter 3 Study I - Psychosocial work stressors and risk of all-cause and coronary heart disease mortality: A systematic review and meta-analysis

This chapter is presented as the peer-reviewed research article published in the Scandinavian Journal of Work, Environment and Health (2020; 46(1):19-31).

3.1 Overview

The published study which this chapter is based on (with supplementary material) presents the findings of the first detailed and comprehensive systematic review of the available literature, including quality assessment and meta-analyses based on prospective cohort studies of common psychosocial work stressors and mortality, focusing on all-cause and CHD mortalities. Pooling results from individual studies together, there is fairly strong evidence that low job control is a risk for premature mortality from CHD, or premature mortality overall, from any cause, even after controlling for other risk factors. The lack of high-quality studies included in the meta-analysis suggests the results should be interpreted with a degree of caution. Nonetheless, pooling results from all the previous studies on this topic helps explain why there is an association between psychosocial work stressors and mortality and contribute to the evidence between low job control and mortality. This is an important finding, because if we can improve the way in which people work, then there is the potential to reduce those workers' risk of premature mortality.

3.2 Research Questions and Objectives

3.2.1 Research questions

The overall research question was what is the evidence regarding the effect of psychosocial work stressors on the risk of all-cause and cardiovascular mortalities?

The specific research questions are:

1. What is the epidemiological evidence on the effect of the psychosocial work stressors which workers are commonly exposed to in the workplace on all-cause mortality?
2. What is the epidemiological evidence on the effect of the psychosocial work stressors which workers are commonly exposed to in the workplace on mortality due to CHD?
3. Does the association between common psychosocial work stressors and mortality, including mortality due to CHD, differ between males and females?
4. What is the effect of excluding studies with low-quality assessment on the association between common psychosocial work stressors and all-cause mortality, including mortality due to CHD?

3.2.2 Research objectives

1. To perform a quality assessment and quantitative synthesis of the available studies on psychosocial work stressors and all-cause mortality.
2. To perform a quality assessment and quantitative synthesis of the available studies on psychosocial work stressors and mortality due to CHD.
3. To perform separate subgroup random-effects meta-analyses for studies where effect estimates are stratified by gender for psychosocial work stressors and all-cause mortality, including mortality due to CHD.
4. To perform separate random-effects meta-analyses for common psychosocial work stressors and mortality, including all-cause mortality, in studies with acceptable quality assessment.

3.3 Publication

3.3.1 Main document and data supplement

Taouk Y., Spittal M. J., LaMontagne A. D., Milner A. J. Psychosocial work stressors and risk of all-cause and coronary heart disease mortality: A systematic review and meta-analysis.

Scandinavian Journal of Work, Environment and Health. 46(1):19-31, 2020. doi:10.5271/sjweh.3854.

Review

Scand J Work Environ Health Online-first -article

doi:10.5271/sjweh.3854

Psychosocial work stressors and risk of all-cause and coronary heart disease mortality: A systematic review and meta-analysis

by Taouk Y, Spittal MJ, LaMontagne AD, Milner AJ

This study represents the first systematic review and meta-analysis on the relationship between psychosocial work stressors and mortality. Across 32 studies, it finds that workers with low job control are at increased risk of all-cause and CHD mortality. Improving the psychosocial quality of work may contribute to better health and wellbeing, reducing the effect of psychosocial work stressors on mortality.

Key terms: all-cause mortality; cardiovascular disease mortality; CHD; coronary heart disease; coronary heart disease mortality; death; job control; meta-analysis; mortality; occupational stress; psychological stress; psychosocial; psychosocial work stressor; stress; systematic review; work stress; work stressor

Additional material

Please note that there is additional material available belonging to this article on the *Scandinavian Journal of Work, Environment & Health* -website.

Original article

Scand J Work Environ Health – online first. doi:10.5271/sjweh.3854

Psychosocial work stressors and risk of all-cause and coronary heart disease mortality: A systematic review and meta-analysis

by Yamna Taouk, MPH,[1] Matthew J Spittal, PhD,[1] Anthony D LaMontagne, ScD,[2] Allison J Milner, PhD [1, 3]

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* – online first. doi:10.5271/sjweh.3854

Objectives Psychosocial work stressors are common exposures affecting the working population, and there is good evidence that they have adverse health consequences. There is some evidence that they may impact on mortality, but this has not been systematically examined. We performed a systematic review, including risk of bias, and meta-analyses of observational studies to examine the association between psychosocial work stressors and all-cause mortality and death due to coronary heart disease (CHD).

Methods Electronic databases were searched to identify studies and information on study characteristics and outcomes extracted in accordance with PRISMA guidelines. Risk estimates of outcomes associated with psychosocial work stressors: specifically, all-cause mortality, and death due to CHD were pooled using inverse variance weighted random effects meta-analysis.

Results We identified 45 eligible cohort studies, of which 32 were included in the quantitative analyses of psychosocial work stressors and mortality. Low job control was associated with an increased risk of all-cause mortality [hazard ratio (HR) 1.21, 95% confidence interval (CI) 1.07–1.37, minimally-adjusted; HR 1.05, 95% CI 1.01–1.10, multivariable-adjusted; HR 1.03, 95% CI 1.00–1.06 exclusion of low quality studies and multivariable-adjusted] and CHD mortality [HR 1.50, 95% CI 1.42–1.58, minimally-adjusted; HR 1.23, 95% CI 1.17–1.30, multivariable-adjusted; HR 1.19, 95% CI 1.01–1.40, exclusion of low quality studies and multivariable-adjusted].

Conclusions Workers with low job control are at increased risk of all-cause and CHD mortality compared to workers with high job control. Policy and practice interventions to improve job control could contribute to reductions in all-cause and CHD mortality.

Key terms all-cause mortality; cardiovascular disease mortality; CHD; death; job control; occupational stress; psychological stress; stress; work stress.

Psychosocial work stressors represent the objective characteristics of the work environment, including design, organization and context of work, which may elicit stress response in workers and cause physiological or psychological harm (1). Exposure to psychosocial work stressors have been associated with coronary heart disease (CHD), diabetes, clinical depression, as well as a range of other physical and mental health outcomes (2–18). Work stressors have also been associated with poor organizational outcomes, including sickness absence and presenteeism (19–21).

Research into the effect of psychosocial work stressors on mortality is sparse and has produced inconsistent results. Most studies thus far have examined components of the Karasek's job-demand-control model, which is composed of the psychosocial factors job demands which refer to the pace and intensity of work, and job control comprising decision authority and skill discretion (22, 23). The model posits that job strain which results from the combined effects of low job control and high job demands may cause stress-related ill-health (23). The model was further extended to include an additional component representing social support in the workplace (24). Work stressors conceptualized and measured according to the job-demand-control-support model has been found to increase the risk of mortality in

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3 Dr Allison Milner tragically died in an accident on 12 August 2019. Please see Acknowledgement section for details.

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some studies (25–31), but not in others (32–43).

The concept of work stressors was broadened beyond proximal job task characteristics to include organizational factors in the effort-reward imbalance (ERI) model which recognizes both the effort and the reward structure of work (44). The model is based upon the premise that work-related benefits depend upon a reciprocal relationship between efforts and rewards at work and that lack of reciprocity due to an imbalance between high effort and low rewards is stressful to workers and may result in adverse health outcomes (44). Cohort studies examining the effect of high ERI on mortality have had mixed findings (28, 34, 39). A further work stressor model, the organizational justice model, which measures employees' degree of perceived fairness of treatment in the workplace proposes that increased perceived unfairness may lead to increased stress responses, resulting in physiological and behavioral reactions adversely impacting on workers' health and well-being (45, 46). The effect of increased perceived unfairness on mortality is not clear (47, 48). More recently, broader constructs of work stressors encompassing labor market arrangements including long working hours, shift work and job insecurity have been examined as risk factors for mortality with mixed results (11, 26, 36, 49–55).

Given these mixed findings and the fact that there have been no previous reviews of evidence about psychosocial work stressors and mortality, a comprehensive systematic review and meta analyses is needed to clarify the relationship. The objective of this study was to evaluate the evidence for the association between (i) psychosocial work stressors and all-cause mortality and (ii) psychosocial work stressors and mortality due to CHD. We included CHD mortality as a secondary outcome because the association with job stressors is biologically plausible, and it is one of the most frequently studied mortality outcomes (56).

Methods

A comprehensive systematic review of the available literature until the end of 2017 for studies providing information on the risk of mortality in relation to psychosocial work stressors was conducted as per the Preferred Reporting Items for Systematic Reviews and Meta-analyses (PRISMA) guidelines (57). A search strategy was formulated to identify studies systematically that answer the research question "What is the effect of psychosocial work stressors on mortality?". The question was specified by defining the "PICOS' modules as follows: Population (P) = workers; Intervention or exposure (I) = exposed to the psychosocial work stressors: low job control, high job demands, high job strain, low support at work, job insecurity, organization injustice, ERI, long working hours, shift work; Outcome (O) = total mortality, mortality due to cardiovascular heart disease; and Study type (S) = longitudinal studies.

Search strategy

A search of seven electronic databases covering a range of disciplines − including Medline, PubMed, EMBASE, Web of Science (medical science database), Scopus (social science database), PsycINFO (psychology database), and Global Health (public health database) − for eligible literature published from their respective commencement date to present was undertaken. A three-tier search strategy was used to identify eligible studies (see table 1 and supplementary material www.sjweh.fi/show_abstract.php?abstract_id=3854, table S1). The first and the second stages were combined with an "or" operator and matched with the third using the "and" operator. No restrictions were placed on the date, status, or language of publications. A secondary search included examination of the reference list of all studies identified for potential inclusion in the systematic review. The first author conducted the initial data searches and, together with the last author, independently screened all potentially relevant titles and abstracts to retrieve relevant articles to minimize the possibility of selective selection.

Eligibility criteria

Original articles that had the key search terms in the title or abstract and mortality as an outcome variable were considered for inclusion in the systematic review. We excluded reviews, letters, editorials, case reports, book chapters, studies with no English translation, and conference abstracts. Studies investigating physical exposure to work-related factors including physical, chemical, ergonomic, and biological factors were excluded as the focus of the review is exposure to psychosocial work stressors in association with mortality. Studies examining deaths from *karoshi,* the Japenese term for death by overwork, were excluded from the review as karoshi conflates the exposure and outcome making it difficult to assign a common cause of death. Prospective population-level studies (cohort studies) and case control studies that contained quantitative estimates and 95% confidence intervals (CI) of the relative risk, rate ratio (RR), odds ratio (OR, or hazard ratio (HR) for mortality associated with psychosocial work stressors were included in the systematic review.

Studies were screened for eligibility using a two-stage process. Titles and abstracts of studies with the key search terms in the title or abstract, retrieved using

Table 1. Search terminology and strategy for systematic review.

First stage	Second stage	Third stage
Search terms for job stress and job strain	Search terms for psychosocial work stressors	Search terms for mortality
"psychosocial job stress*" OR "working condition*" OR "psychosocial work*" OR "psychosocial job*" OR "occupation* stress*" OR "job stress" OR "job strain" OR "work stress" OR "work strain"	"job control" OR "job demands" OR "job secur*" OR "job insecur*" OR "work insecur*" OR "work secur*" OR "precarious work" OR "precarious employ" OR "precarious job" OR "decision latitude" OR "skill discretion" OR "decision authority" OR "psychosocial job demands" OR "job social support" OR "work social support" OR "workload" OR "effort reward imbalance*" OR "organisation* justice" OR "organisation* injustice" OR "organisation* fairness" OR "organisation* unfairness" OR "organization* justice" OR "organization* injustice" OR "organization* fairness" OR "organization* unfairness" OR "shift work" OR "work* hour*" OR "work* time" OR "work* span" OR "underemployment" OR (("work" OR "business" OR "employ*") and ("management style" OR "leadership")) OR (("temporary" OR "casual") and ("employ*"))	"mortality" OR "death"

the search strategy, were screened independently by the two review authors to identify studies that potentially met the inclusion criteria. Following review of the full text, studies that met the eligibility criteria were retained and any disagreements were resolved by consensus or through discussion with an independent researcher.

Data extraction

Methodological details and data including study description, author, cohort, year, country, population (sample size, gender, age), exposure assessment and exposure level, description of mortality (method of assessment and incidence), study design, duration of follow-up, number of events, confounders adjusted for in the analyses, effect size for mortality, and CI were extracted from each included study.

Quality assessment

The quality of studies was assessed by the same two independent researchers using the Scottish Intercollegiate Guidelines Network (SIGN) methodological checklist for cohort and case-control studies version 3 (58) (supplementart table S2). Studies with little or no risk of bias were rated as high quality. Studies that mostly agreed with the guidelines but containing some flaws with an associated risk of bias were rated as acceptable. Studies that did not agree with the criteria or contained significant flaws with respect to study design were rated as low quality. Disagreements over quality assessment were resolved by discussion, with involvement of a third independent researcher where necessary.

Data analysis

There was limited scope for quantitative synthesis because of the heterogeneity in exposures measured across a small number of studies. But where possible, we conducted quantitative analyses of studies with sufficiently similar exposures, outcomes and study populations. For multiple studies reporting on the same study data, only the most recent study with longer follow up period was included in the quantitative analysis. Multiple studies with the same author/s were included in the meta-analysis insofar as the study sample, exposures, or outcomes differed. Studies were included if age-adjusted effect estimates were available, otherwise we excluded studies with unadjusted crude measurements because age is such a strong predictor of mortality. OR, RR, and HR were combined to estimate pooled effect sizes (59).

We conducted inverse variance random-effects meta-analyses to assess the association between each psychosocial work stressor and the primary outcome: all-cause mortality; and the secondary outcome: deaths due to CHD, in minimally- and multivariable-adjusted analyses. Our analyses were restricted to combining estimates when more than two studies were available for pooling. For studies reporting multiple levels of an exposure (eg, job strain quartiles), the referent group was only included once in the meta-analyses. Hence highest job demands level was compared to lowest job demands level, job strain was compared to no job strain, and lowest job control level was compared to highest job control level. For studies reporting multiple categories of shift work (eg, evening shift work, night shift work, rotating shift work), the category with the highest frequency was compared to the referent category, no shift work.

In addition to the pooled effect size for individual psychosocial work stressors, we undertook separate subgroup random-effects meta-analyses for studies where effect estimates were stratified by gender. The I^2 statistic was used to measure the heterogeneity between studies. Funnel plots were used to assess the precision of estimates and publication bias (60) and a modified Egger's test was performed to test for small study effects by regressing effect estimates on their standard errors weighted by the reciprocal of the variance of intervention effect estimates (61).

Two sensitivity analyses were conducted. The first sensitivity analysis tested differences between studies without low quality assessment compared with the inclusion of all studies irrespective of quality assessment. The second sensitivity analysis tested differences between studies that assessed relatively healthy study samples (participants with pre-existing diseases and/or cancer other than melanoma were excluded from the

study analysis or the study controlled for health status) compared with studies that did not exclude participants due to ill health from their analysis or control for health status.

All analyses were conducted using Stata 14.2 (Stata Corp, College Station, TX).

Results

A total of 13 237 studies were initially identified (figure 1). After removing duplicates, 6778 records were screened by the title and abstract and 146 were further screened by full text for eligibility assessment and the reference lists were examined. 102 studies were excluded (supplementary table S3) resulting in 45 eligible studies for inclusion in the systematic review (11, 25–43, 47, 49–55, 62–78). Too few studies examined the risk of mortality and low social support at work, high ERI, or long working hours hence 32 studies were included in the quantitative analyses for low job control, high job demands, high job strain, shift work or job insecurity and mortality (11, 25–42, 50–54, 65, 66, 71–74, 76, 77).

Quality assessment

Twenty-seven studies were assessed to be of acceptable quality and eighteen studies were assessed as low quality (supplementary table S4). No study was judged to be of high quality.

Characteristics and results of reviewed studies

Twenty-seven studies were from Finland, Sweden and Denmark (11, 25, 28–30, 35, 37–39, 42, 43, 47, 50, 51, 53, 63, 64, 67–74, 76, 78). A further nine studies came from the USA (26, 27, 31, 32, 36, 41, 62, 65, 75), two studies from UK (54, 66) and Japan (40, 77), and one study each in Poland (34), Israel (33), France (49), Ireland (55), and Russia (52). The earliest study was published in 1981 (30) and the most recent published in 2017 (62, 74). The 45 articles included in the review used data from thirty-seven different studies. Four studies used data from the Valmet study (28, 39, 43, 47); and the Nurses' Health Study (41, 75), Copenhagen Male Study (69, 70), Stockholm Heart Epidemiology Program (67, 68), Statistics Finland Quality of Work Life Surveys (50, 51), and UK industrial cohort (54, 66) data were used in two studies each (supplementary table S5).

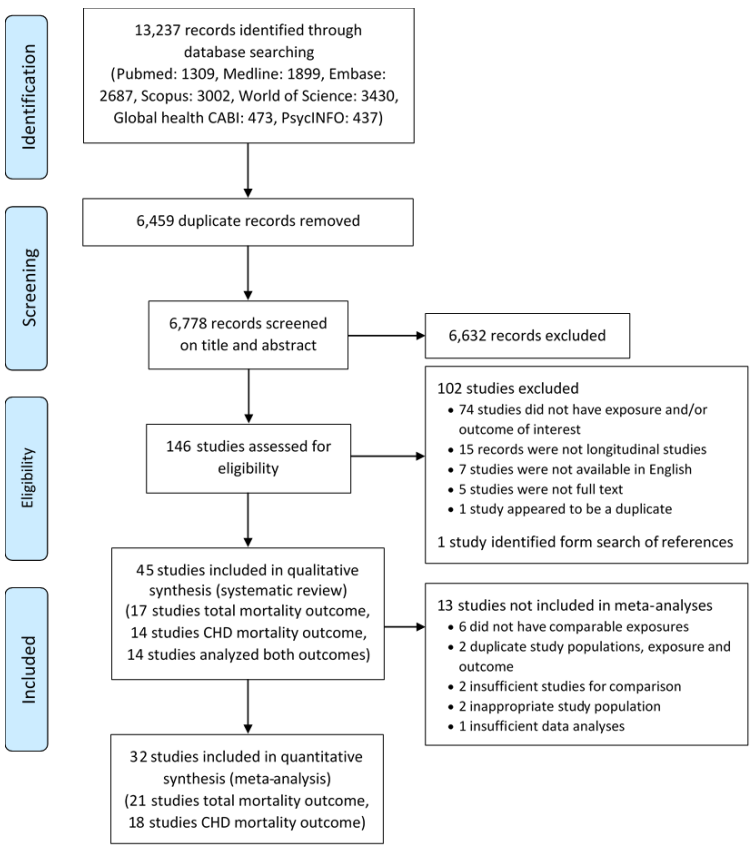

Figure 1. Prisma Flowchart depicting the article search and selection procedure

The effect of psychosocial work stressors on mortality was based on males in thirteen studies (11, 25, 30, 42, 54, 64–66, 69, 70, 73, 76, 77), and females in five studies (27, 36, 41, 74, 75). Twenty-six studies examined psychosocial work stressors and mortality in all persons (26, 28, 29, 31–35, 37–40, 43, 47, 49–51, 53, 55, 62, 63, 67, 68, 71, 72, 78). Components of the main job-strain model used to measure work stress (22–24), including job demands, job control, job strain, support at work and/or iso-strain and the risk of mortality were investigated in twenty-seven studies (25–43, 49, 51, 62, 64, 65, 67, 71, 72). Eleven studies examined shift work and mortality (11, 50, 53, 54, 63, 66, 73–77), six studies looked at job insecurity and mortality (26, 36, 51, 52, 68, 78), three studies explored ERI and mortality (28, 34, 39), two studies included working hours and mortality (55, 69), one study inspected organizational justice and mortality (47), and another psychological pressure and mortality (70). Most studies used self-reported exposure measures, however five studies used a psychosocial job exposure matrix to assign job demands and job control values for each occupation (26, 27, 29, 42, 65), one study used both self-reported and occupation-based measures of work-related stress (38), four studies assessed shift work exposure from company records (54, 66, 73, 76), and one study used company records to assess employment status (78).

Median follow up in these studies ranged from 4–30 years. The outcome, all-cause mortality, was identified independently of the exposure from official national or regional death registers in almost all studies. One study did not report on outcome ascertainment (32), and three others relied on outcome status information provided from relatives and friends (26, 52, 62). Deaths due to CHD were ascertained from International Classifications of Diseases codes assigned for cardiovascular disease, ischemic heart disease, myocardial infarction and/or strokes on death certificates. Seventeen studies reported on specific working populations (industrial workers (28, 39, 43, 47, 54, 65, 66, 71, 73, 76), health professionals (36, 41, 74, 75), municipal and/or hospital employees (35, 72, 78)), two studies used non-fatal acute myocardial infarction cases (67, 68), and one study examined graduates from Wisconsin high schools in 1957 (62). The other twenty-five studies used a random working population sample drawn from the general population.

Various effect sizes were reported across studies. Thirty studies used HR (27, 28, 31, 33–39, 43, 47, 49–53, 55, 63, 67–75, 77, 78), nine studies used RR (11, 25, 29, 32, 40–42, 64, 65), four studies used OR (30, 54, 62, 66), and one study used standardized relative rates (76). The referent group for components of the job-strain model, job demands, job control and job strain differed across studies due to variation in exposure measurements and classifications between studies. Some studies reported results as continuous measures (31–33, 43, 62, 65) and various categorical measures were derived across studies. Different referent groups were used among studies with shift work exposure (supplementary table S6).

Quantitative analysis

Individual meta-analysis results for each of the five exposures, and risk of all-cause mortality and risk of CHD mortality are shown in supplementary figures S1–S17, and pooled results are depicted in figures 2–5. Pooled results for psychosocial work stressors and mortality in minimally adjusted analysis showed that employees in low control jobs had a significantly higher mortality risk than those in high control jobs (all-cause mortality: HR 1.21, 95% CI 1.07–1.37, k=3; CHD mortality: HR 1.50, 95% CI 1.42–1.58, k=5). The increased risk for low job control and mortality persisted in multivariable-adjusted analysis (all-cause mortality: HR 1.05, 95% CI 1.01–1.10, k=10; CHD mortality: HR 1.23, 95% CI 1.17–1.30, k=6), although risk estimates were attenuated.

Pooled results for high job demands, job strain, and shift work were not associated with risk of all-cause mortality (HR 0.93, 95% CI 0.81–1.08, k=3; HR 1.07, 95% CI 0.82–1.39, k=3; and HR 1.01, 95% CI 0.96–1.07, k=5 respectively) or CHD mortality (HR 0.89, 95% CI 0.61–1.29, k=4; HR 1.31, 95% CI 0.91–1.88, k=7; and HR 1.08, 95% CI 0.94–1.25, k=7 respectively) in minimally adjusted analysis.

The pooled result for high job demands was not found to be associated with all-cause mortality in multivariable-adjusted analysis (HR 0.96, 95% CI 0.90–1.02, k=8) and differed little from pooled results for job demands and mortality in minimally adjusted analysis. Pooled results for job strain and risk of all-cause mortality (HR 1.04, 95% CI 0.92–1.18, k=7) and risk of CHD mortality (HR 1.26, 95% CI 0.82–1.94, k=4) included the null. An effect of shift work on all-cause mortality (HR 0.97, 95% CI 0.82–1.14, k=5) and CHD mortality (HR 1.06, 95% CI 0.87–1.29, k=6) was not observed, though the risk estimates were in opposite directions. Results for high job demands and CHD mortality were not able to be pooled due to insufficient number of studies available for analysis.

The pooled results for job insecurity and all-cause mortality were inconclusive (minimally adjusted analysis HR 1.33, 95% CI 0.94–1.88, k=3 and multivariable-adjusted analysis HR 1.26, 95% CI 0.92–1.73, k=3). Pooled estimates for job insecurity and CHD mortality were not able to be calculated due to insufficient number of individual studies.

Some heterogeneity was observed in studies pooled for examination of low job control and all-cause mortal-

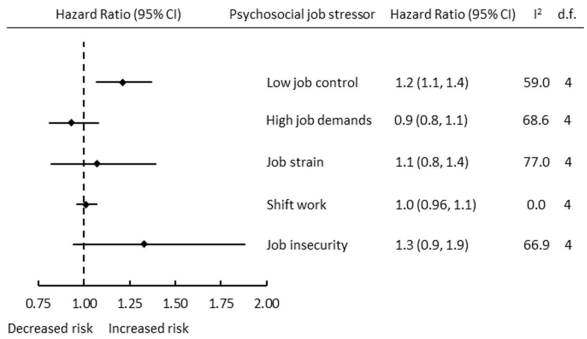

Figure 2. Summary of pooled effect estimates for psychosocial work stressors and all-cause mortality minimally adjusted analysis.

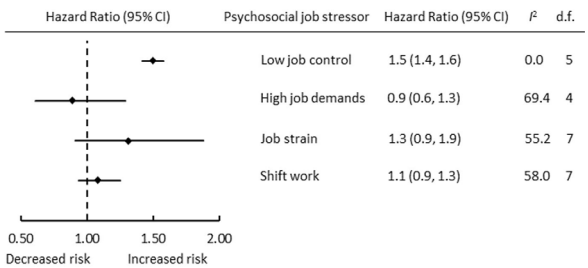

Figure 3. Summary of pooled effect estimates for psychosocial work stressors and CHD mortality minimally adjusted analysis.

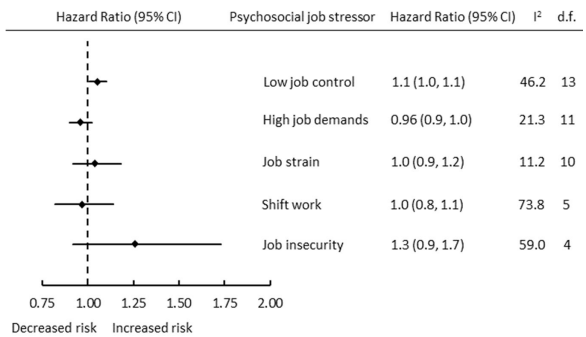

Figure 4. Summary of pooled effect estimates for psychosocial work stressors and all-cause mortality multivariable-adjusted analysis.

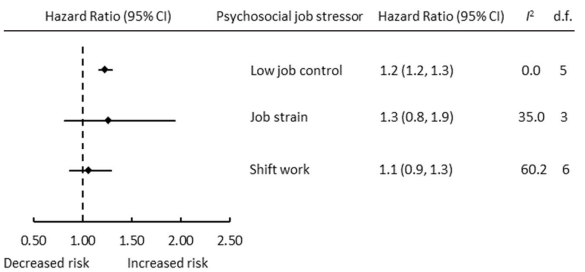

Figure 5. Summary of pooled effect estimates for psychosocial work stressors and CHD mortality multivariable-adjusted analysis.

ity (I^2=59.0%, P=0.045), high job demands and mortality (all-cause mortality I^2=68.6%, P=0.013; CHD mortality I^2=69.4%, P=0.011), job strain and mortality (all-cause mortality I^2=77.0%, p=0.002; CHD mortality I^2=55.2%, P=0.029), shift work and CHD mortality (I^2=58.0%, P=0.020), and job insecurity and all-cause mortality (I^2=66.9%, P=0.017) in minimally adjusted analysis. Heterogeneity was also seen in studies pooled for analysis of shift work and mortality (all-cause mortality I^2=73.8%, P=0.002; CHD mortality I^2=60.2%, P=0.020), low job control and all-cause mortality (I^2=46.2%, P=0.030), and job insecurity and all-cause mortality (I^2=59.0%, P=0.045) in multivariable-adjusted analysis.

No heterogeneity was observed in studies pooled for analysis of low job control and CHD mortality (I^2=0.0%, P=0.543) or shift work and all-cause mortality (I^2=0.0%, P=0.707) in minimally adjusted analyses, or in studies pooled for analysis of low job control and mortality (CHD mortality I^2=0.0%, P=0.983), high job demands and all-cause mortality (I^2=21.3%, P=0.234), or job strain and mortality (all-cause mortality I^2=11.2%, P=0.337; CHD mortality I^2=35.0%, P=0.202) in multivariable-adjusted analysis.

Investigation of funnel plots (supplementary figures S18–S21) suggested some degree of asymmetry. Almost all studies had small standard errors and risk estimates ranging from under one to approximately two. The few studies with larger risk estimates approximating four had larger standard errors estimates possibly due to small study effects or methodological limitations. Due to the small number of studies included in the meta-analyses, it is difficult to reliably assess publication bias solely based on the funnel plots (79), hence formal testing for asymmetry was performed using Egger's test (61). Formal statistical testing did not detect strong evidence of small study effects and risk of publication bias in the studies included in quantitative analyses, however there was some suggestion of small study effects for job insecurity and mortality (minimally adjusted analysis P=0.09 and multivariable-adjusted analysis P=0.08).

Sensitivity analyses excluding studies with low quality assessment resulted in attenuated pooled estimates and wider confidence intervals, probably due to validity issues with low quality studies. However, the association between exposure to low job control and mortality remained the same (minimally adjusted analysis: all-cause mortality HR 1.16, 95% CI 1.06–1.26, k=2; CHD mortality HR 1.36, 95% CI 1.19–1.56, k=4 and multivariable-adjusted analysis: all-cause mortality HR 1.03, 95% CI 1.00–1.06, k=5; and CHD mortality HR 1.19, 95% CI 1.01–1.40, k=4). As to be expected there was almost perfect overlap between studies with low quality assessment and studies that did not exclude or adjust for unhealthy participants at baseline, hence results of the sensitivity analyses examining differ-

ences between studies that assessed relatively healthy participants and studies that did not exclude unhealthy participants or control for health status were very similar to the aforementioned results.

Discussion

Main findings

This systematic review and meta-analysis examined the association between mortality and psychosocial work stressor exposures. Our findings show that workers with low job control have a 21% increased risk of all-cause mortality, and 50% increased risk of CHD mortality, compared with workers with high job control. Even after adjustment for relevant confounders in the multivariable-adjusted analysis, the increased risk of low job control and CHD mortality persisted as did the risk of all-cause mortality remain elevated, albeit attenuated, for workers with low job control. Excluding studies with low quality assessment attenuated the risk estimates but the association between low job control and mortality persisted. Low job control increased the risk of all-cause mortality by 3% and the risk of CHD mortality by 19% in the multivariable-adjusted analyses. A decreased risk of mortality for workers with exposure to high job demands was persistent across all subgroup analyses but was not statistically significant. Similarly, an elevated, but not significant, risk of mortality was observed for job strain and job insecurity. There did not appear to be any evidence supporting an association between exposure to shift work, and CHD mortality or all-cause mortality.

Comparison with other studies

This is the first review to synthesize systematically and quantitatively the respective epidemiological evidence for psychosocial work stressor exposures and CHD mortality or all-cause mortality. The observed association between low job control and increased all-cause mortality, in addition to CHD mortality, in minimally adjusted and multivariable-adjusted analyses strengthens the argument that job control is predictive of mortality. Many individual studies observe an elevated risk of mortality and low job control, but the association was not supported following adjustments for relevant covariates (28, 32, 35, 42, 65, 71, 72). However, these studies were generally small and underpowered to detect associations between mortality and job control, with most reporting less than several hundred deaths (28, 32, 42, 65, 71, 72), thus combining studies in a meta-analysis greatly increased the power to detect an association. A few studies detected decreased or no risk of mortality and low job control (31, 33, 38, 40). Variations in the underlying methodological design across studies, including differences in the measurement of the exposure and referent group, and assessment of potential confounders, may have contributed to inconsistent results.

Results for high job demands and mortality are consistent with results of individual studies. Most studies observed a reduced but non-significant risk of mortality and high job demands (26, 27, 33, 40, 51, 65), in contrast to Karasek (30) who found that high job demands increased the risk of CHD mortality. Self-reported assessments of job demands may not be a homogeneous measure across different occupations as job demands are experienced and interpreted differently across various occupational groups. There is a such a diversity of demands that responses might represent a range of job demands, such as psychological, physical, emotional, social, or organizational. It may also be that perceptions of job demands have changed over time. This along with inconsistent harmonization of scales across studies may explain the inconsistency in the literature.

Analyses of job strain and mortality were suggestive of a relationship between job strain and CHD mortality, but results were not conclusive. There is growing evidence that job strain and increased risk of mortality may be more pertinent in workers with existing cardiometabolic disease than for healthy workers. A study of patients with CHD based on three small prospective studies (67, 80, 81) reported a 60% increased risk of recurrent CHD events associated with job strain (82), and a more recent multicohort study examining work stress and mortality in workers with and without cardiometabolic disease reported a 70% increased risk of mortality in men with prevalent cardiometabolic disease (83). Analyses of job strain and CHD mortality were for healthy participants in all individual studies included in this review.

Results of the effects of shift work are consistent with the limited epidemiologic evidence that is available (12). Studies investigating shift work exposure can be prone to healthy shift work hire worker and healthy shift worker survivor selection bias due to unhealthy workers being less likely to take up shift work and healthier workers being more likely to remain in shift work employment compared to less healthy workers. This may explain the variation in mortality rates across studies. The evidence for job insecurity and mortality was inconclusive due to the small number of published studies available for inclusion in this review.

Biological plausibility

The observed associations are biologically plausible. Changes in brain stress-responsive neurocircuitry stimulate peripheral physiological responses in the sympa-

thetic autonomic nervous system and hypothalamus–pituitary–adrenal (HPA) through the release and control of adrenaline and stress hormone cortisol throughout the body, and increase inflammatory protein levels despite the absence of pathogens (56). To cope with stress, the body's systems adjust psychological and physiological processes, resulting in chronic over-activity or under-activity of allostatic systems, fixing new baselines levels for psychological and physiological performances, causing deterioration of body systems from repeated and unresolved stressors (84). The cardiovascular system is particularly affected by stress. Combined effects of dysfunction of both the autonomic nervous system and HPA-axis responses over time can result in increases in platelet activation, blood fibrinogen levels and blood pressure levels, accelerating the atherosclerotic process and increasing the likelihood of fatal and non-fatal cardiovascular or cerebrovascular events (56, 84).

Strengths and limitations of the study

The review has several strengths, including thorough systematic review conducted according to PRISMA guidelines (57) of 45 studies and the inclusion of 32 studies from Europe, United States, Israel, and Japan, most of which were assessed as acceptable quality. Sensitivity analyses attenuated results, but still excluded the null, with estimates remaining consistent even when omitting studies with low quality assessment or studies that did not exclude or adjust for unhealthy participants at baseline. Assessment of study quality was conducted using a rigorously validated scoring tool recognized as a useful assessment tool for assessing quality and susceptibility to bias in observational studies (58, 85).

There was some heterogeneity in pooled study results, ranging from 0–77% across the analyses, likely due to the variation in composition and classification of psychosocial work stressor exposures and referent groups between studies, and variation in composition of study sample (eg, general working population, specific industries, gender-specific), as well as due to variations in study period and geography. Furthermore, participants with previous history of CHD and/or cancer were excluded in some studies but not by others.

There is a concern about insufficient variance for psychosocial work stressor exposures when using generic work strain instruments in single occupation studies that may have biased our results towards the null. These generic measures although well-validated may not have measured psychosocial work stressors pertinent to some occupations, eg, healthcare workers' exposure to violence. It should be also mentioned that the design of the review precluded assessment of the relation between single stressful occupations and mortality.

Nearly all studies measured exposure to psychosocial work stressors along with occupational, behavioral and/or biological risk factors at baseline and estimated their association with mortality during lengthy follow-up periods. Studies with single assessment of job characteristics and long follow-up periods without repeated assessment of exposure status over time measuring cumulative exposure, may bias results to null due to assessment of exposure to work stressors temporally distant from the outcome (86). Measurements of psychosocial work stressor exposures were self-reported in most individual studies and may have been subjected to reporting bias, however external measurements of work stress, like organizational downsizing, have been found to be associated with an increased risk of cardiovascular mortality (87). A few of the studies included in the review used occupation-based measures of psychosocial work stressors based on a job exposure matrix and may be susceptible to nondifferential exposure misclassification bias, biasing toward the null. In particular, job demands, a subjective measurement, may not be suitable for use in an exposure imputation system designed to measure environmental differences (30, 42, 65).

The individual studies had some degree of potential selection bias, including selective attrition due to cessation of employment as a result of job stressor exposure during the follow up period, and survivor bias (healthy worker effect) due to healthier participants being more willing to participate in health surveys, and less likely to be in low status jobs with high stress, that may have attenuated results. Of concern in shift work studies, the composition of the reference category may have influenced mortality risk estimates as healthier workers are more likely to take up shift work than less healthy workers and remain in shift work employment compared to those who develop debilitating conditions and depart shift work or retire from work altogether.

The lack of high-quality studies for inclusion in the meta-analyses is a limitation that may have potentially biased the results of our study towards the null. However, our analyses represent a summary of the existing knowledge. The fact that no high-quality study has been conducted is significant. More recent epidemiological methods including marginal structural methods and causal mediation analysis should be considered in future studies examining mortality outcomes in longitudinal studies with long periods of follow-up. It must be noted that this review's narrow focus on psychosocial work stressors conceptualized from well-studied and validated work stress models is a limitation. Other conceptualizations of psychosocial work stressors for example burnout due to chronic exhaustion in combination with a negative attitude and diminished efficacy at work as a result of imbalance between job demands and resources (88, 89), and cognitive ergonomics such as the mental burden of specific tasks (90) and extra burdens created by time

pressure and barriers hindering performance (91), were not included in this review. However, research into these work stressors and mortality is limited and their broad psychosocial framework may not be consistent with assumptions of homogeneity and variability between studies required for meta-analyses (92).

The outcomes, all-cause mortality and mortality due to CHD, were ascertained through record linkage and ICD codes for most studies included in the review following the assessment of exposure to psychosocial work stressors. Potential reverse causation due to confounding was considered by study authors, and controlled for in the analysis, or at the design stage of the study by the exclusion of participants at baseline based on health status. Despite the evidence for psychosocial work stressors and mental health disorders (10), suicide-related mortality was not included as a third outcome in this study, as several of the authors involved in this review had previously undertaken a comprehensive systematic review and meta-analysis of psychosocial job stressors and suicidality (93). The association between psychosocial work stressors and mortality may have been modified by gender, but we were unable to assess this effect moderation due to an insufficient number of studies presenting gender-stratified results. Nonetheless, we did provide pooled results by gender in supplementary figures S1–S17.

Concluding remarks

In conclusion, the results of the systematic review and meta-analyses suggest that workers with low job control are at increased risk of all-cause and CHD mortality compared to workers with high job control. Given the observed association between low job control and mortality along with the inconclusive findings for the other work stressors, further research is recommended to examine mechanisms underlying the observed effect between low job control and mortality and to elucidate the relationship between psychosocial work stressors and mortality. The strongest effect observed was that of deaths due to CHD and low job control, even after controlling for relevant confounders and the exclusion of low-quality studies. If the observed association is causal, then policy and practice interventions to improve job control could contribute to reductions in all-cause and CHD mortality.

Acknowledgement

We would like to pay our gratitude and our respects to our co-author and beloved colleague, Associate Professor Allison Milner who was tragically killed in August of 2019. She was Deputy Director of the Disability and Health Unit at the Centre for Health Equity, Melbourne School of Population and Global Health, at the University of Melbourne, with a passion for research pertaining to mental health and the workplace. Allison Milner was a tireless and prolific researcher and had made significant progress in broadening our understanding of psychological health at work and suicides and our understanding of the work stressors affecting workers health. She is remembered as an outstanding scholar, an exceptional mentor, and a loving and generous person with great affection by all who knew her. She certainly was in our thoughts whilst preparing this paper for publication.

Ethical approval

The Melbourne School of Population and Global Health Human Ethics Advisory Group approved this study (ethics application ID: 1750707.1).

Conflicts of Interest

The authors declare no conflicts of interest.

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Psychosocial work stressors and risk of all-cause and coronary heart disease mortality: A systematic review and meta-analysis [1]

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Figure S20: Funnel plots of psychosocial work stressors and CHD mortality minimally adjusted analysis

Figure S21: Funnel plots of psychosocial work stressors and CHD mortality multivariable-adjusted analysis

Table S1: Search strategies

Search strategy in Embase

#	Query
#1	(psychosocial job stress* or working condition* or (psychosocial adj2 work*) or (psychosocial adj2 job*) or (occupation* adj2 stress*)).ti,ab.
#2	((job or work) adj5 (strain or stress)).ti,ab.
#3	(job control or job demand* or job secur* or job insecure* or work insecure* or work secur* or decision latitude or skill discretion or decision authority or ((precarious adj2 work) or (precarious adj2 employ*) or (precarious adj2 job))).ti,ab.
#4	(psychosocial job demands or workload or effort reward imbalance or (organisation* adj2 justice) or (organisation* adj2 injustice) or (organisation* adj2 fairness) or (organisation* adj2 unfairness) or (organization adj2 justice) or (organization adj2 injustice) or (organization* adj2 fairness) or (organization* adj2 unfairness) or (shift adj work) or ((job or work) adj5 social support)).ti,ab.
#5	((work* adj2 hour*) or (work* adj2 time) or (work* adj2 span) or underemployment).ti,ab.
#6	((work or business or employ*) adj5 (management style or leadership)).ti,ab.
#7	((temporary or casual) adj5 employ*).ti,ab.
#8	(mortality or death).ti,ab.
#9	#1 OR #2 OR #3 OR #4 OR #5 OR #6 OR #7
#10	#8 AND #9

Search strategy in Medline

#	Query
#1	(psychosocial job stress* or working condition* or (psychosocial adj2 work*) or (psychosocial adj2 job*) or (occupation* adj2 stress*)).ti,ab.
#2	((job or work) adj5 (strain or stress)).ti,ab.
#3	(job control or job demand* or job secur* or job insecure* or work insecure* or work secur* or decision latitude or skill discretion or decision authority or ((precarious adj2 work) or (precarious adj2 employ*) or (precarious adj2 job))).ti,ab.
#4	(psychosocial job demands or workload or effort reward imbalance or (organisation* adj2 justice) or (organisation* adj2 injustice) or (organisation* adj2 fairness) or (organisation* adj2 unfairness) or (organization adj2 justice) or (organization adj2 injustice) or (organization* adj2 fairness) or organization* adj2 unfairness) or (shift adj work) or ((job or work) adj5 social support)).ti,ab.
#5	((work* adj2 hour*) or (work* adj2 time) or (work* adj2 span) or underemployment).ti,ab.
#6	((work or business or employ*) adj5 (management style or leadership)).ti,ab.
#7	((temporary or casual) adj5 employ*).ti,ab.
#8	(mortality or death).ti,ab.
#9	#1 OR #2 OR #3 OR #4 OR #5 OR #6 OR #7
#10	#8 AND #9

Search strategy in PubMed (read from bottom to top)

#	Query
#12	#10 AND #11
#11	mortality[Title/Abstract] OR death[Title/Abstract]

#10	#1 OR #2 OR #3 OR #4 OR #5 OR #6 OR #7 OR #8 OR #9
#9	temporary employ*[Title/Abstract] OR casual employ*[Title/Abstract]
#8	employ* management style[Title/Abstract] OR employ* leadership[Title/Abstract]
#7	business management style[Title/Abstract] OR business leadership[Title/Abstract]
#6	work management style[Title/Abstract] OR work leadership[Title/Abstract]
#5	work* hour*[Title/Abstract] OR work* time[Title/Abstract] OR work* span[Title/Abstract] OR underemployment[Title/Abstract]
#4	psychosocial job demands[Title/Abstract] OR workload[Title/Abstract] OR effort reward imbalance[Title/Abstract] OR organisation* justice[Title/Abstract] OR organisation* injustice[Title/Abstract] OR organisation* fairness[Title/Abstract] OR organisation* unfairness[Title/Abstract] OR organization justice[Title/Abstract] OR organization injustice[Title/Abstract] OR organization* fairness[Title/Abstract] OR organization* unfairness[Title/Abstract] OR work* social support[Title/Abstract] OR shift work[Title/Abstract] OR employ* social support[Title/Abstract] OR job social support[Title/Abstract]
#3	job control[Title/Abstract] OR job demand*[Title/Abstract] OR job secur*[Title/Abstract] OR job insecure*[Title/Abstract] OR work insecure*[Title/Abstract] OR work secur*[Title/Abstract] OR decision latitude[Title/Abstract] OR skill discretion[Title/Abstract] OR decision authority[Title/Abstract] OR precarious work[Title/Abstract] OR precarious employ*[Title/Abstract] OR precarious job[Title/Abstract]
#2	job strain[Title/Abstract] OR job stress[Title/Abstract] OR work strain[Title/Abstract] OR work stress[Title/Abstract]
#1	psychosocial job stress*[Title/Abstract] OR working condition*[Title/Abstract] OR psychosocial work*[Title/Abstract] OR psychosocial job*[Title/Abstract] OR occupation* stress*[Title/Abstract]

Search strategy in SCOPUS

#1	TITLE-ABS((“psychosocial job stress*”) or (“working condition*”) or (psychosocial W/2 work*) or (psychosocial W/2 job*) or (occupation* W/2 stress*))
#2	TITLE-ABS((job W/2 strain or job W/2 stress or work W/2 strain or work W/2 stress)
#3	TITLE-ABS((“job control”) or (“job demand*”) or (“job secur*”) or (“job insecure*”) or (“work insecure*”) or (“work secur*”) or (“decision latitude”) or (“skill discretion”) or (“decision authority”) or (precarious W/2 work) or (precarious W/2 employ*) or (precarious W/2 job))
#4	TITLE-ABS((“psychosocial job demands”) or (workload) or (“effort reward imbalance”) or (organisation* W/2 justice) or (organisation* W/2 injustice) or (organisation* W/2 fairness) or (organisation* W/2 unfairness) or (organization W/2 justice) or (organization W/2 injustice) or (organization* W/2 fairness) or (organization* W/2 unfairness) or (shift W/1 work))
#5	TITLE-ABS((job W/5 “social support”) or (work W/5 “social support”))
#6	TITLE-ABS((work* W/2 hour*) or (work* W/2 time) or (work* W/2 span) or underemployment)
#7	TITLE-ABS(work W/5 “management style” or work W/5 leadership)
#8	TITLE-ABS(business W/5 “management style” or business W/5 leadership)
#9	TITLE-ABS(employ* W/5 “management style” or employ* W/5 leadership)
#10	TITLE-ABS(temporary W/5 employ* or casual W/5 employ*)
#11	TITLE-ABS(mortality or death)
#12	#1 OR #2 OR #3 OR #4 OR #5 OR #6 OR #7 OR #8 OR #9 OR #10
#13	#12 and #11

Search strategy in PsycINFO

#	**Query**
#1	(psychosocial job stress* or working condition* or (psychosocial adj2 work*) or (psychosocial adj2 job*) or (occupation* adj2 stress*)).ti,ab.
#2	((job or work) adj5 (strain or stress)).ti,ab.

#3	(job control or job demand* or job secur* or job insecure* or work insecure* or work secur* or decision latitude or skill discretion or decision authority or ((precarious adj2 work) or (precarious adj2 employ*) or (precarious adj2 job))).ti,ab.
#4	(psychosocial job demands or workload or effort reward imbalance or (organisation* adj2 justice) or (organisation* adj2 injustice) or (organisation* adj2 fairness) or (organisation* adj2 unfairness) or (organization adj2 justice) or (organization adj2 injustice) or (organization* adj2 fairness) or (organization* adj2 unfairness) or (shift adj work) ((job or work) adj5 social support)).ti,ab.
#5	((work* adj2 hour*) or (work* adj2 time) or (work* adj2 span) or underemployment).ti,ab.
#6	((work or business or employ*) adj5 (management style or leadership)).ti,ab.
#7	((temporary or casual) adj5 employ*).ti,ab.
#8	(mortality or death).ti,ab.
#9	#1 OR #2 OR #3 OR #4 OR #5 OR #6 OR #7
#10	#8 AND #9

Search strategy in Web of Science

#1	(TS=("psychosocial job stress*" or "working condition*" or "psychosocial near/2 work*" or "psychosocial near/2 job*" or "occupation* near/2 stress*"))
#2	(TS=(job near/2 strain or job near/2 stress or work near/2 strain or work near/2 stress))
#3	(TS=("job control" or "job demand*" or "job secur*" or "job insecure*" or "work insecure*" or "work secur*" or "decision latitude" or "skill discretion" or "decision authority" or precarious near/2 work or precarious near/2 employ* or precarious near/2 job))
#4	(TS=(psychosocial job demands or workload or effort reward imbalance or (organisation* near/2 justice) or (organisation* near/2 injustice) or (organisation* near/2 fairness) or (organisation* near/2 unfairness) or (organization near/2 justice) or (organization near/2 injustice) or (organization* near/2 fairness) or (organization* near/2 unfairness) or (shift near/1 work)))
#5	(TS=((job near/5 "social support") or (work near/5 "social support")))
#6	(TS=((work* near/2 hour*) or (work* near/2 time) or (work* near/2 span) or underemployment))
#7	(TS=(work near/5 "management style" or work near/5 leadership))
#8	(TS=(business near/5 "management style" or business near/5 leadership))
#9	(TS=(employ* near/5 "management style" or employ* near/5 leadership))
#10	(TS=(temporary near/5 employ* or casual near/5 employ*))
#11	(TS=(mortality or death))
#12	#1 OR #2 OR #3 OR #4 OR #5 OR #6 OR #7 OR #8 OR #9 OR #10
#13	#11 AND #12

Search strategy in Global Health (CABI)

#1	("psychosocial job stress*" or "working condition*" or "psychosocial near/2 work*" or "psychosocial near/2 job*" or "occupation* near/2 stress*")
#2	("job near/2 strain" or "job near/2 stress" or "work near/2 strain" or "work near/2 stress")
#3	("job control" or "job demand*" or "job secur*" or "job insecure*" or "work insecure*" or "work secur*" or "decision latitude" or "skill discretion" or "decision authority" or "precarious near/2 work" or "precarious near/2 employ*" or "precarious near/2 job")
#4	("psychosocial job demands" or "workload" or "effort reward imbalance" or ("organisation* near/2 justice") or ("organisation* near/2 injustice") or ("organisation* near/2 fairness") or ("organisation* near/2 unfairness") or ("organization near/2 justice") or ("organization near/2 injustice") or ("organization* near/2 fairness") or ("organization* near/2 unfairness") or ("shift near/1 work")
#5	(job near/5 "social support") or (work near/5 "social support")
#6	(work* near/2 hour* or work* near/2 time or work* near/2 span or underemployment)
#7	(work near/5 "management style" or work near/5 leadership)

#8	(business near/5 "management style" or business near/5 leadership)
#9	(employ* near/5 "management style" or employ* near/5 leadership)
#10	(temporary near/5 employ* or casual near/5 employ*)
#11	(mortality or death)
#12	#1 OR #2 OR #3 OR #4 OR #5 OR #6 OR #7 OR #8 OR #9 OR #10
#13	#11 AND #12

Table S2: Quality assessment

SIGN Methodology Checklist 3: Cohort studies *(version 3, 20 November 2012)*

	CRITERIA	**Yes**	**No**	**Cannot Say**
1.	The study addresses an appropriate and clearly focused question?			
2.	The two groups being studied are selected from source populations that are comparable in all respects other than the factor under investigation?			
3.	The study indicates how many of the people asked to take part did so, in each of the groups being studied?			
4.	The likelihood that some eligible subjects might have the outcome at the time of enrolment is assessed and taken into account in the analysis?			
5.	The percentage of individuals or clusters recruited into each arm of the study whom dropped out before the study was completed is reported?			
6.	If applicable, comparison is made between full participants and those lost to follow up, by exposure status?			
7.	The outcomes are clearly defined?			
8.	The assessment of outcome is made blind to exposure status? (if the study is retrospective this may not be applicable)			
9.	Where blinding was not possible, there is some recognition that knowledge of exposure status could have influenced the assessment of outcome?			
10.	The method of assessment of exposure is reliable?			
11.	Evidence from other sources is used to demonstrate that the method of outcome assessment is valid and reliable?			
12.	Exposure level or prognostic factor is assessed more than once?			
13.	The main potential confounders are identified and taken into account in the design and analysis? *			
14.	Have confidence intervals been provided?			
	OVERALL			
	How well was the study done to minimise the risk of bias or confounding? (High quality/acceptable/low quality)			

*Limited was included as an additional response for item 13.

1. A well-defined question is a prerequisite in assessing the objectives and relevance of the study

2. Comparable groups should be similar in all characteristics except for their exposure status

3. Large differences in participation rates between groups may be due to selection bias hence study results should be treated with considerable caution

4. If the outcome is present at baseline, results may be subject to performance bias

5. A high drop-out rate in the study may indicate attrition bias and the study should be examined as to why people dropped out and whether drop-out rates were comparable in the exposed and unexposed groups

6. Attrition bias may occur if the participants who dropped out of the study differed significantly from those whom remained and any unexplained differences warrants study results to be treated with caution

7. Outcomes and the criteria used for measuring them should be clearly defined and participants should be followed until specified end points or outcomes are reached to minimise the risk of detection bias

8. If outcome assessment is made blind to exposure status, minimising the risk of detection bias and increasing the likelihood of unbiased results, the study should be rated more highly than those where it is not done, or not done adequately

9. Blinding is not possible in many cohort studies so assessment of extent of detection bias present in the study allows results to be regarded with more confidence

10. Clearly described, reliable measures minimise the risk of detection bias in the study and should increase the confidence in the quality of the study

11. The primary outcome measures used should be clear to minimise the risk of detection bias. If outcome measures are not stated, or the study bases its main conclusions on secondary outcomes, the study should be rejected

12. Confidence in data quality should be increased and the risk of detection bias minimised if exposure level is measured more than once and independent assessment is made by more than one investigator

13. The study should report on potential confounders considered and how they have been assessed or allowed for in the analysis. Judgement should be made as to the inclusion of all likely confounders. If the measures used to address confounding are inadequate, the study should be downgraded or rejected. A study that does not address the risk of confounding should be rejected

14. Confidence limits are the preferred method for indicating the precision of statistical results, and can be used to differentiate between an inconclusive study and a study that shows no effect. Studies that report a single value with no assessment of precision should be treated with extreme caution

SIGN Methodology Checklist 4: Case-control studies *(version 2, 28 May 2012)*

(Used for nested case control studies within longitudinal studies)

	CRITERIA	**Yes**	**No**	**Cannot Say**
1.	The study addresses an appropriate and clearly focused question?			
2.	The cases and controls are taken from comparable populations?			
3.	The same exclusion criteria are used for both cases and controls?			
4.	Percentage of each group (cases and controls) that participated in the study reported?			
5.	Comparison is made between participants and non-participants to establish their similarities or differences?			
6.	Cases are clearly defined and differentiated from controls?			
7.	It is clearly established that controls are non-cases?			
8.	Measures will have been taken to prevent knowledge of primary exposure influencing case ascertainment?			
9.	Exposure status is measured in a standard, valid and reliable way?			
10.	The main potential confounders are identified and taken into account in the design and analysis? *			
11.	Confidence intervals are provided?			
	OVERALL			
	How well was the study done to minimise the risk of bias or confounding? (High quality/acceptable/low quality)			

*Limited was included as an additional response for item 10.

1. A well-defined question is a prerequisite in assessing the objectives and relevance of the study

2. If the study does not include clear definitions of the source population it should be rejected

3.Failure to apply selection and exclusion criteria equally to cases and controls may introduce a significant degree of bias into the results of the study

4. Low participation rates, or large difference between cases and controls, may bias the study results due to differences between participants and non-participants

5. Well conducted case-control study will examine non-participants among the source population to ensure that the participants are a truly representative sample and do not differ from other members of the source population in some significant way

6. The method of selection of cases is of critical importance to the validity of the study. It must be clear that cases are truly cases and are representative of the eligible population

7. Controls must not have the outcome under investigation and the exposure status assessed in a similar way to that used for the selection of cases

8. If there is a possibility that case ascertainment can be influenced by knowledge of exposure status, assessment of any association is likely to be biased

9. The primary outcome measures used should be clear to minimise the risk of detection bias. If outcome measures are not stated, or the study bases its main conclusions on secondary outcomes, the study should be rejected

10. The study should report on potential confounders considered and how they have been assessed or allowed for in the analysis. Judgement should be made as to the inclusion of all likely confounders. If the measures used to address confounding are inadequate, the study should be downgraded or rejected. A study that does not address the risk of confounding should be rejected

11. Confidence limits are the preferred method for indicating the precision of statistical results, and can be used to differentiate between an inconclusive study and a study that shows no effect. Studies that report a single value with no assessment of precision should be treated with extreme caution.

The overall methodological quality of the study is rated as i) High quality (++): majority of criteria met (little or no risk of bias and results unlikely to be changed by further research), ii) Acceptable (+): most criteria met (some flaws in the study with an associated risk of bias and conclusions may change in the light of further studies), iii) Low quality (0): (either most criteria not met, or significant flaws relating to key aspects of study design and conclusions likely to change in the light of further studies).

Table S3: Excluded articles by reason for exclusion

	Authors	Journal	Title	Volume; (Issue): pages	Reason for exclusion
1	Aasland, O. G., Falkum, E.	Tidsskrift for Den Norske Laegeforening	How are we today? On physicians' health, well-being and job satisfaction	1992; 112(30):3818-3823	No full text
2	Aboa-Eboule, C., Brisson, C., Maunsell, E., Masse, B., Bourbonnais, R., Vezina, M., Milot, A., Theroux, P., Dagenais, GR.	Jama-Journal of the American Medical Association	Job strain and risk of acute recurrent coronary heart disease events	2007; 298(14):1652-1660	Exposure and/or outcome not suitable
3	Ahlbom, A., Karasek, R., Theorell, T.	Lakartidningen	Psychosocial stress during work and the risk of cardiovascular death	1980; 77(46):4243-4245	No English translation
4	Ahola, K., Vaananen, A., Koskinen, A., Kouvonen, A., Shirom, A.	Journal of Psychosomatic Research	Burnout as a predictor of all-cause mortality among industrial employees: a 10-year prospective register-linkage study	2010; 69(1):51-57	Exposure and/or outcome not suitable
5	Alfredsson, L., Hammar, N., Hogstedt, C.	International Journal of Epidemiology	Incidence of myocardial infarction and mortality from specific causes among bus drivers in Sweden	1993; 22(1):57-61	Exposure and/or outcome not suitable
6	Alfredsson, L., Karasek, R., Theorell, T.	Social Science and Medicine	Myocardial infarction risk and psychosocial work environment: an analysis of the male Swedish working force	1982; 16(4):463-467	Exposure and/or outcome not suitable
7	Amagasa, T., Nakayama, T., Takahashi, Y.	Journal of Occupational Health	Karojisatsu in Japan: characteristics of 22 cases of work-related suicide	2005; 47(2):157-164	Exposure and/or outcome not suitable
8	Andersen, I., Burr, H., Kristensen, T. S., Gamborg, M., Osler, M., Prescott, E., Diderichsen, F.	Occupational and Environmental Medicine	Do factors in the psychosocial work environment mediate the effect of socioeconomic position on the risk of myocardial infarction? Study from the Copenhagen Centre for Prospective Population Studies	2004; 61(11):886-892	Exposure and/or outcome not suitable
9	Andreev, E., Hoffmann, R., Carlson, E., Shkolnikov, V., Kharkova, T.	European Societies	Concentration of working-age male mortality among manual workers in urban Latvia and Russia, 1970-/1989	2009; 11(1):161-185	Exposure and/or outcome not suitable
10	Astrand, NE., Hanson, BS., Isacsson, SO.	British Journal of Industrial Medicine	Job demands, job decision latitude, job support, and social	1989; 46(5):334-340	Exposure and/or outcome not suitable

			network factors as predictors of mortality in a Swedish pulp and paper company		
11	Backe, EM., Seidler, A., Latza, U., Rossnagel, K., Schumann, B.	International Archives of Occupational and Environmental Health	The role of psychosocial stress at work for the development of cardiovascular diseases: a systematic review	2012; 85(1):67-79	Not longitudinal study
12	Bannai, A., Tamakoshi, A.	Scandinavian Journal of Work, Environment and Health	The association between long working hours and health: a systematic review of epidemiological evidence	2014; 40(1):5-18	Not longitudinal study
13	Baumert, J., Schneider, B., Lukaschek, K., Emeny, RT., Meisinger, C., Erazo, N., Dragano, N., Ladwig, KH.	Journal of Psychiatric Research	Adverse conditions at the workplace are associated with increased suicide risk	2014; 57():90-95	Exposure and/or outcome not suitable
14	Biering, K., Andersen, JH., Lund, T., Hjollund, NH.	Journal of Occupational Rehabilitation	Psychosocial working environment and risk of adverse cardiac events in patients treated for coronary heart disease	2015; 25(4):770-775	Exposure and/or outcome not suitable
15	Boggild, H., Knutsson, A.	Scandinavian Journal of Work, Environment and Health	Shift work, risk factors and cardiovascular disease	1999; 25(2):85-99	Exposure and/or outcome not suitable
16	Brown, DL., Feskanich, D., Sanchez, BN., Rexrode, KM., Schernhammer, ES., Lisabeth, LD.	American Journal of Epidemiology	Rotating night shift work and the risk of ischemic stroke	2009; 169(11):1370-1377	Exposure and/or outcome not suitable
17	Diene, E., Fouquet, A., Esquirol, Y.	Archives of cardiovascular diseases	Cardiovascular diseases and psychosocial factors at work	2012; 105(1):33-39	Exposure and/or outcome not suitable
18	Dragano, N., Siegrist, J., Nyberg, S., Kivimaki, M.	European Journal of Preventive Cardiology	Effort-reward imbalance at work and job strain as risk factors for incident coronary heart disease: results from the multicohort IPD-work consortium	2017; 24(2 Supplement 1):11	No full text
19	Dragano, N., Siegrist, J., Nyberg, ST., Lunau, T., Fransson, EI., Alfredsson, L., Bjorner, JB., Borritz, M., Burr, H., Erbel, R., Fahlen, G., Goldberg, M., Hamer, M., Heikkila, K., Jockel, KH., Knutsson, A., Madsen, IEH., Nielsen, ML., Nordin, M., Oksanen, T., Pejtersen, JH., Pentti, J., Rugulies, R., Salo, P., Schupp, J., Singh-Manoux, A., Steptoe, A., Theorell, T., Vahtera, J., Westerholm, PJM., Westerlund, H., Virtanen, M., Zins, M., Batty, GD., Kivimaki, M.	Epidemiology	Effort-Reward imbalance at work and incident coronary heart disease: a multicohort study of 90,164 Individuals	2017; 28(4):619-626	Exposure and/or outcome not suitable
20	Emeny, RT., Zierer, A., Lacruz, ME., Baumert, J., Herder, C., Gornitzka, G., Koenig, W., Thorand, B., Ladwig, KH.	Psychosomatic Medicine	Job strain-Associated inflammatory burden and long-term risk of coronary events:	2013; 75(3):317-325	Exposure and/or outcome not suitable

			findings from the MONICA/KORA Augsburg Case-Cohort Study		
21	Ferrada-Noli, M.	Lakartidningen	Is work-related stress the primary cause of sudden death? [Swedish]	2000; 97(51-52):6108-6110	No English translation
22	Ferrada-Noli, M.	Lakartidningen	Work-related stress and sudden death epidemiology [Swedish]	2000; 97(50):5946-5947	No English translation
23	Ferrario, M. M., Veronesi, G., Chambless, L. E., Sega, R., Fornari, C., Bonzini, M., Cesana, G.	Occupational and Environmental Medicine	The contribution of major risk factors and job strain to occupational class differences in coronary heart disease incidence: the MONICA Brianza and PAMELA population-based cohorts	2011; 68(10):717-722	Exposure and/or outcome not suitable
24	Ferrario, M., Porati, S., Chiodini, P., Taborelli, S., Toso, C., Borchini, R., Maretti, A., Cesana, G.	Giornale Italiano di Medicina del Lavoro Ed Ergonomia	Differences of mortality risk for all causes and for cardiovascular diseases among occupational classes in men living the Northern Italy	2003; 25(3):426-427	No English translation
25	Fishta, A., Backe, EM.	International Archives of Occupational and Environmental Health	Psychosocial stress at work and cardiovascular diseases: an overview of systematic reviews	2015; 88(8):997-1014	Not longitudinal study
26	Frost, P., Kolstad, HA., Bonde, JP.	Scandinavian Journal of Work, Environment and Health	Shift work and the risk of ischemic heart disease - a systematic review of the epidemiologic evidence	2009; 35(3):163-179	Exposure and/or outcome not suitable
27	Fujishiro, K., Diez Roux, AV., Landsbergis, P., Baron, S., Barr, RG., Kaufman, JD., Polak, JF., Stukovsky, KH.	Occupational and Environmental Medicine	Associations of occupation, job control and job demands with intima-media thickness: the Multi-Ethnic Study of Atherosclerosis (MESA)	2011; 68(5):319-326	Exposure and/or outcome not suitable
28	Fujishiro, K., Hajat, A., Landsbergis, PA., Meyer, JD., Schreiner, PJ., Kaufman, JD.	SSM - Population Health	Explaining racial/ethnic differences in all-cause mortality in the Multi-Ethnic Study of Atherosclerosis (MESA): substantive complexity and hazardous working conditions as mediating factors	2017; 3:497-505	Exposure and/or outcome not suitable
29	Gallo, LC., Bogart, LM., Vranceanu, AM., Walt, LC.	Annals of Behavioral Medicine	Job characteristics, occupational status, and ambulatory cardiovascular activity in women	2004; 28(1):62-73	Exposure and/or outcome not suitable

30	Ganster, DC., Rosen, CC.	Journal of Management	Work stress and employee health: a multidisciplinary review	2013; 39(5):1085-1122	Not longitudinal study
31	Goh, J., Pfeffer, J., Zenios, SA.	Management Science	The relationship between workplace stressors and mortality and health costs in the United State	2016; 62(2):608-628	Not longitudinal study
32	Golubic, R., Ekelund, U., Luben, R., Khaw, K., Wareham, N., Brage, S.	Journal of Science and Medicine in Sport	Does total physical activity modify the association between working hours and all-cause mortality? The EPIC-Norfolk cohort	2012; 15:S28	No full text
33	Hammar, N., Alfredsson, L., Johnson, JV.	Occupational and Environmental Medicine	Job strain, social support at work, and incidence of myocardial infarction	1998; 55(8):548-553	Exposure and/or outcome not suitable
34	Hammar, N., Alfredsson, L., Theorell, T.	International Journal of Epidemiology	Job characteristics and the incidence of myocardial infarction	1994; 23(2):277-284	Exposure and/or outcome not suitable
35	Hannerz, H., Larsen, AD., Garde, AH.	Jmir Research Protocols	Working time arrangements as potential risk factors for ischemic heart disease among workers in Denmark: a study protocol	2016; 5(2):e130	Exposure and/or outcome not suitable
36	Heikkila, K., Nyberg, ST., Madsen, IE., de Vroome, E., Alfredsson, L., Bjorner, JJ., Borritz, M., Burr, H., Erbel, R., Ferrie, JE., Fransson, EI., Geuskens, GA., Hooftman, WE., Houtman, IL., Jockel, KH., Knutsson, A., Koskenvuo, M., Lunau, T., Nielsen, ML., Nordin, M., Oksanen, T., Pejtersen, JH., Pentti, J., Shipley, MJ., Steptoe, A., Suominen, SB., Theorell, T., Vahtera, J., Westerholm, PJ., Westerlund, H., Dragano, N., Rugulies, R., Kawachi, I., Batty, GD., Singh-Manoux, A., Virtanen, M., Kivimaki, M., Consortium, I. PD-Work	British Journal of Cancer	Long working hours and cancer risk: a multi-cohort study	2016; 114(7):813-818	Exposure and/or outcome not suitable
37	Hemmingsson, T., Lundberg, I.	International Journal of Epidemiology	Is the association between low job control and coronary heart disease confounded by risk factors measured in childhood and adolescence among Swedish males 40-53 years of age?	2006; 35(3):616-622	Exposure and/or outcome not suitable
38	Heslop, P., Smith, G. D., Metcalfe, C., Macleod, J., Hart, C.	Social Science and Medicine	Change in job satisfaction, and its association with self-reported stress, cardiovascular risk factors and mortality	2002; 54(10):1589-1599	Exposure and/or outcome not suitable

39	Hibbard, JH., Pope, CR.	Women and Health	Women's employment, social support, and mortality	1992; 18(1):119-133	Duplicate study
40	House, JS., Strecher, VJ., Metzner, HL., Robbins, CA.	Journal of Health and Social Behavior	Occupational stress and health among men and women in the Tecumseh Community Health Study	1986; 27(1):62-77	Exposure and/or outcome not suitable
41	Huang, Y. L., Xu, SX., Hua, JH., Zhu, DJ., Liu, CH., Hu, YZ., Liu, TB., Xu, DL.	Neurology	Association between job strain and risk of incident stroke: a meta-analysis	2015; 85(19):1648-1654	Not longitudinal study
42	Inoue, M., Nishikitani, M., Tsurugano, S., Yano, E.	Sangyo Eiseigaku Zasshi	The health of permanent workers and workers with precarious employment: a literature review	2011; 53(4):117-139	No English translation
43	Jarvholm, B., Reuterwall, C., Bystedt, J.	Scandinavian Journal of Work, Environment and Health	Mortality attributable to occupational exposure in Sweden	2013; 39(1):106-111	Exposure and/or outcome not suitable
44	Ke, DS.	Acta Neurologica Taiwanica	Overwork, stroke, and karoshi-death from overwork	2012; 21(2):54-59	Exposure and/or outcome not suitable
45	Kivimaki, M., Batty, GD., Hamer, M., Ferrie, JE., Vahtera, J., Virtanen, M., Marmot, MG., Singh-Manoux, A., Shipley, MJ.	Annals of Internal Medicine	Using additional information on working hours to predict coronary heart disease: a cohort study	2011; 154(7):457-463	Exposure and/or outcome not suitable
46	Kivimaki, M., Ferrie, JE., Brunner, E., Head, J., Shipley, MJ., Vahtera, J., Marmot, MG.	Archives of Internal Medicine	Justice at work and reduced risk of coronary heart disease among employees: the Whitehall II Study	2005; 165(19):2245-2251	Exposure and/or outcome not suitable
47	Kivimaki, M., Gimeno, D., Ferrie, JE., Batty, GD., Oksanen, T., Jokela, M., Virtanen, M., Salo, P., Akbaraly, TN., Elovainio, M., Pentti, J., Vahtera, J.	International Journal of Epidemiology	Socioeconomic position, psychosocial work environment and cerebrovascular disease among women: the Finnish public sector study	2009; 38(5):1265-1271	Exposure and/or outcome not suitable
48	Kivimaki, M., Head, J., Ferrie, JE., Brunner, E., Marmot, MG., Vahtera, J., Shipley, MJ.	Psychosomatic Medicine	Why Is evidence on job strain and coronary heart disease mixed? An illustration of measurement challenges in the Whitehall II study	2006; 68(3):398-401	Exposure and/or outcome not suitable
49	Kivimaki, M., Leino-Arjas, P., Kaila-Kangas, L., Luukkonen, R., Vahtera, J., Elovainio, M., Harma, M., Kirjonen, J.	Psychosomatic Medicine	Is incomplete recovery from work a risk marker of cardiovascular death? Prospective evidence from industrial employees	2006; 68(3):402-407	Exposure and/or outcome not suitable
50	Kivimaki, M., Nyberg, ST., Batty, G., Fransson, EI., Heikkila, K., Alfredsson, L., Bjorner, JB., Borritz, M., Burr, H., Casini, A., Clays, E., De Bacquer, D., Dragano, N., Ferrie, JE., Geuskens, GA.,	The Lancet	Job strain as a risk factor for coronary heart disease: a	2012; 380(9852):1491-1497	Not longitudinal study

	Goldberg, M., Hamer, M., Hooftman, WE., Houtman, IL., Joensuu, M., Jokela, M., Kittel, F., Knutsson, A., Koskenvuo, M., Koskinen, A., Kouvonen, A., Kumari, M., Madsen, IE., Marmot, MG., Nielsen, ML., Nordin, M., Oksanen, T., Pentti, J., Rugulies, R., Salo, P., Siegrist, J., Singh-Manoux, A., Suominen, SB., Vaananen, A., Vahtera, J., Virtanen, M., Westerholm, PJ., Westerlund, H., Zins, M., Steptoe, A., Theorell, T.		collaborative meta-analysis of individual participant data		
51	Kivimaki, M., Nyberg, ST., Batty, GD., Shipley, MJ., Ferrie, JE., Virtanen, M., Marmot, MG., Vahtera, J., Singh-Manoux, A., Hamer, M.	International Journal of Epidemiology	Does adding information on job strain improve risk prediction for coronary heart disease beyond the standard Framingham risk score? The Whitehall II study	2011; 40(6):1577-1584	Exposure and/or outcome not suitable
52	Kivimaki, M., Nyberg, ST., Fransson, EI., Heikkila, K., Alfredsson, L., Casini, A., Clays, E., De Bacquer, D., Dragano, N., Ferrie, JE., Goldberg, M., Hamer, M., Jokela, M., Karasek, R., Kittel, F., Knutsson, A., Koskenvuo, M., Nordin, M., Oksanen, T., Pentti, J., Rugulies, R., Salo, P., Siegrist, J., Suominen, SB., Theorell, T., Vahtera, J., Virtanen, M., Westerholm, PJ., Westerlund, H., Zins, M., Steptoe, A., Singh-Manoux, A., Batty, G.	Canadian Medical Association Journal	Associations of job strain and lifestyle risk factors with risk of coronary artery disease: a meta-analysis of individual participant data	2013; 185(9):763-769	Exposure and/or outcome not suitable
53	Kivimaki, M., Theorell, T., Westerlund, H., Vahtera, J., Alfredsson, L.	Journal of Epidemiology and Community Health	Job strain and ischaemic disease: Does the inclusion of older employees in the cohort dilute the association? The WOLF Stockholm Study	2008; 62(4):372-374	Exposure and/or outcome not suitable
54	Kivimaki, M., Virtanen, M., Elovainio, M., Kouvonen, A., Vaananen, A., Vahtera, J.	Scandinavian Journal of Work, Environment and Health	Work stress in the etiology of coronary heart disease - a meta-analysis	2006; 32(6):431-442	Not longitudinal study
55	Kjeldsen, SE., Knudsen, K., Ekrem, G., Fure, TO., Movinckel, P., Erikssen, JE.	Blood Pressure	Is there an association between severe job strain, transient rise in blood pressure and increased mortality?	2006; 15(2):93-100	Exposure and/or outcome not suitable
56	Knutsson, A.	Scandinavian Journal of Social Medicine. Supplementum	Shift work and coronary heart disease	1989; 44:1-36	Exposure and/or outcome not suitable
57	Knutsson, A., Hammar, N., Karlsson, B.	Chronobiology International	Shift workers' mortality scrutinized	2004; 21(6):1049-1053	No full text
58	Kopp, M., Skrabski, A.	Neuropsychopharmacologia Hungarica	Why do Hungarian men die early?	2009; 11(3):141-149	No English translation
59	Kopp, M., Skrabski, A., Szanto, Z., Siegrist, J.	Journal of Epidemiology and Community Health	Psychosocial determinants of premature cardiovascular	2006; 60(9):782-788	Not longitudinal study

			mortality differences within Hungary		
60	Kopp, MS., Skrabski, A., Szekely, A., Stauder, A., Williams, R.	Csermely, Peter [Ed]; Korcsmaros, Tamas [Ed]; Sulyok, Katalin (2007) Stress responses in biology and medicine: Stress of life in molecules, organisms, and psychosocial communities (pp 325-338) xvii, 366 pp Malden: Blackwell Publishing	Chronic stress and social changes: socioeconomic determination of chronic stress	2007; :325-338	Not longitudinal study
61	Kornitzer, M., Desmet, P., Sans, S., Dramaix, M., Boulenguez, C., Debacker, G., Ferrario, M., Houtman, I., Isacsson, SO., Ostergren, PO., Peres, I., Pelfrene, E., Romon, M., Rosengren, A., Cesana, G., Wilhelmsen, L.	European Journal of Preventive Cardiology	Job stress and major coronary events: results from the job stress, absenteeism and coronary heart disease in Europe study	2006; 13(5):695-704	Exposure and/or outcome not suitable
62	Kristenson, M., Kucinskiene, Z., Bergdahl, B., Calkauskas, H., Urmonas, V., Orth-Gomer, K.	Psychosomatic Medicine	Increased psychosocial strain in Lithuanian versus Swedish men: the LiVicordia study [Erratum appears in Psychosom Med. 2003 May-Jun;65(3):346]	1998; 60(3):277-282	Not longitudinal study
63	Kuper, H., Marmot, M.	Journal of Epidemiology and Community Health	Job strain, job demands, decision latitude, and risk of coronary heart disease within the Whitehall II study	2003; 57(2):147-153	Exposure and/or outcome not suitable
64	Li, J., Zhang, M., Loerbroks, A., Angerer, P., Siegrist, J.	International Journal of Occupational Medicine and Environmental Health	Work stress and the risk of recurrent coronary heart disease events: a systematic review and meta-analysis	2015; 28(1):8-19	Not longitudinal study
65	Lynch, J., Krause, N., Kaplan, GA., Tuomilehto, J., Salonen, JT.	American Journal of Public Health	Workplace conditions, socioeconomic status, and the risk of mortality and acute myocardial infarction: the Kuopio ischemic heart disease risk factor study	1997; 87(4):617-622	Exposure and/or outcome not suitable
66	Macleod, J., Davey Smith, G., Metcalfe, C.,Hart, C.	Social Science and Medicine	How are we today? On physicians' health, well-being and job satisfaction	2005; 61(9):1916-1929	Exposure and/or outcome not suitable
67	Marmot, M. G., Bosma, H., Hemingway, H., Brunner, E. and Stansfeld, S.	Lancet	Contribution of job control and other risk factors to social	1997; 350(9073):235-239	Exposure and/or outcome not suitable

			variations in coronary heart disease incidence		
68	Matthews, KA., Gump, BB.	Archives of Internal Medicine	Chronic work stress and marital dissolution increase risk of posttrial mortality in men from the Multiple Risk Factor Intervention Trial	2002; 162(3):309-315	Exposure and/or outcome not suitable
69	Michaels, D., Zoloth, SR.	International Journal of Epidemiology	Mortality among urban bus drivers	1991; 20(2):399-404	Exposure and/or outcome not suitable
70	Milner, A., Spittal, MJ., Pirkis, J., Chastang, JF., Niedhammer, I., LaMontagne, AD.	Psychosomatic Medicine	Low control and high demands at work as risk factors for suicide: an Australian national population-level case-control study	2017; 79(3):358-364	Exposure and/or outcome not suitable
71	Nabi, H., Kivimaki, M., Batty, G. D., Shipley, M. J., Britton, A., Brunner, E. J., Vahtera, J., Lemogne, C., Elbaz, A., Singh-Manoux, A.	European Heart Journal	Increased risk of coronary heart disease among individuals reporting adverse impact of stress on their health: the Whitehall II prospective cohort study	2013; 34(34):2697-2705	Exposure and/or outcome not suitable
72	Netterstrom, B., Juel, K.	Scandinavian Journal of Work, Environment and Health	Impact of work-related and psychosocial factors on the development of ischemic heart disease among urban bus drivers in Denmark	1988; 14(4):231-238	Exposure and/or outcome not suitable
73	Netterstrom, B., Kristensen, TS., Jensen, G., Schnor, P.	International Journal of Occupational Medicine and Environmental Health	Is the demand-control model still a usefull tool to assess work-related psychosocial risk for ischemic heart disease? Results from 14 year follow up in the Copenhagen City Heart study	2010; 23(3):217-224	Exposure and/or outcome not suitable
74	Netterstrom, B., Kristensen, TS., Sjol, A.	European Journal of Cardiovascular Prevention and Rehabilitation	Psychological job demands increase the risk of ischaemic heart disease: a 14-year cohort study of employed Danish men	2006; 13(3):414-420	Exposure and/or outcome not suitable
75	Netterstrom, B., Suadicani, P.	International Journal of Epidemiology	Self-assessed job satisfaction and ischaemic heart disease mortality: a 10-year follow-up of urban bus drivers	1993; 22(1):51-56	Exposure and/or outcome not suitable

76	Nielsen, NR., Kristensen, TS., Schnohr, P., Gronbaek, M.	American Journal of Epidemiology	Perceived stress and cause-specific mortality among men and women: results from a prospective cohort study	2008; 168(5):481-491	Exposure and/or outcome not suitable
77	Nyberg, A., Alfredsson, L., Theorell, T., Westerlund, H., Vahtera, J., Kivimaki, M.	Occupational and Environmental Medicine	Managerial leadership and ischaemic heart disease among employees: the Swedish WOLF study	2009; 66(1):51-55	Exposure and/or outcome not suitable
78	Nylen, L., Voss, M., Floderus, B.	Occupational and Environmental Medicine	Mortality among women and men relative to unemployment, part time work, overtime work, and extra work: a study based on data from the Swedish twin registry	2001; 58(1):52-57	Exposure and/or outcome not suitable
79	Oberlinner, C., Ott, MG., Nasterlack, M., Yong, M., Messerer, P., Zober, A., Lang, S.	Scandinavian Journal of Work, Environment and Health	Medical program for shift workers - impacts on chronic disease and mortality outcomes	2009; 35(4):309-318	Exposure and/or outcome not suitable
80	Ostry, A., Maggi, S., Tansey, J., Dunn, J., Hershler, R., Chen, L., Louie, AM., Hertzman, C.	Scandinavian Journal of Public Health	The impact of psychosocial work conditions on attempted and completed suicide among western Canadian sawmill workers	2007; 35(3):265-271	Exposure and/or outcome not suitable
81	Peter, R., Siegrist, J.	International Archives of Occupational and Environmental Health	Psychosocial work environment and the risk of coronary heart disease	2000; 73:S41-S45	Not longitudinal study
82	Schioler, L., Soderberg, M., Rosengren, A., Jarvholm, B., Toren, K.	Scandinavian Journal of Work, Environment and Health	Psychosocial work environment and risk of ischemic stroke and coronary heart disease: a prospective longitudinal study of 75 236 construction workers	2015; 41(3):280-287	Exposure and/or outcome not suitable
83	Steenland, K., Fine, L.	American Journal of Industrial Medicine	Shift work, shift change, and risk of death from heart disease at work	1996; 29(3):278-281	Exposure and/or outcome not suitable
84	Taylor, PJ., Pocock, SJ.	British Journal of Industrial Medicine	Mortality of shift and day workers 1956-68	1972; 29(2):201-207	Incomplete data
85	Theorell, T., Tsutsumi, A., Hallquist, J., Reuterwall, C., Hogstedt, C., Fredlund, P., Emlund, N., Johnson, JV., Grp, Sheep Study	American Journal of Public Health	Decision latitude, job strain, and myocardial infarction: a study of working men in Stockholm	1998; 88(3):382-388	Exposure and/or outcome not suitable

86	Tobiasz-Adamczyk, B., Bartoszewska, E., Brzyski, P., Kopacz, M.	International Journal of Occupational Medicine and Environmental Health	Long-term consequences of education, working conditions, and health-related behaviors on mortality patterns in older age. A 17-year observational study in Kraków, Poland	2007; 20(3):247-256	Exposure and/or outcome not suitable
87	Toivanen, S.	Scandinavian Journal of Work Environment and Health	Job control and the risk of incident stroke in the working population in Sweden	2008; 34(1):40-47	Exposure and/or outcome not suitable
88	Toivanen, S., Griep, RH., Mellner, C., Vinberg, S., Eloranta, S.	Occupational and Environmental Medicine	Mortality differences between self-employed and paid employees: a 5-year follow-up study of the working population in Sweden	2016; 73(9):627-636	Exposure and/or outcome not suitable
89	Toivanen, S., Hemstrom, O.	Stroke	Is the impact of job control on stroke independent from socioeconomic status? A large-scale study of the Swedish working population	2008; 39(4):1321-1323	Exposure and/or outcome not suitable
90	Toren, K., Schioler, L., Giang, WK., Novak, M., Soderberg, M., Rosengren, A.	BMJ Open	A longitudinal general population-based study of job strain and risk for coronary heart disease and stroke in Swedish men	2014; 4(3):e004355	Exposure and/or outcome not suitable
91	Toren, K., Schioler, L., Soderberg, M., Giang, KW., Rosengren, A.	Occupational and Environmental Medicine	The association between job strain and atrial fibrillation in Swedish men	2015; 72(3):177-180	Exposure and/or outcome not suitable
92	Trudel-Fitzgerald, C., Poole, EM., Idahl, A., Lundin, E., Sood, AK., Kawachi, I., Kubzansky, LD., Tworoger, SS.	Psychosomatic Medicine	The association of work characteristics with ovarian cancer risk and mortality	2017; 79(9):1059-1067	Exposure and/or outcome not suitable
93	Tsutsumi, A.	European Journal of Preventive Cardiology	Work environment and strokes: an update of the most recent findings	2017; 24(2 Supplement 1):12-13	No full text
94	Tsutsumi, A., Kayaba, K., Ishikawa, S.	Social Science and Medicine	Impact of occupational stress on stroke across occupational classes and genders	2011; 72(10):1652-1658	Exposure and/or outcome not suitable
95	Tsutsumi, A., Kayaba, K., Kario, K., Ishikawa, S.	Archives of Internal Medicine	Prospective study on occupational stress and risk of stroke	2009; 169(1):56-61	Exposure and/or outcome not suitable

96	Tsutsumi, A., Kayaba, K., Ojima, T., Ishikawa, S., Kawakami, N.	Psychotherapy and Psychosomatics	Low control at work and the risk of suicide in Japanese men: a prospective cohort study	2007; 76(3):177-185	Exposure and/or outcome not suitable
97	Tuyishimire, D., Brisson, C., Milot, A., Vezina, M., Gilbert-Ouimet, M.	European Journal of Preventive Cardiology	Combined effect of job strain and psychological distress on the risk of recurrent myocardial infarction	2017; 24(2 Supplement 1):19	No full text
98	Uchiyama, S., Kurasawa, T., Sekizawa, T., Nakatsuka, H.	Journal of Science of Labour	Work and hypertension	2004; 80(5):213-219	No English translation
99	Uchiyama, S., Kurasawa, T., Sekizawa, T., Nakatsuka, H.	Journal of Occupational Health	Job strain and risk of cardiovascular events in treated hypertensive Japanese workers: hypertension follow-up group study	2005; 47(2):102-111	Exposure and/or outcome not suitable
100	Virtanen, SV., Notkola, V.	International Journal of Epidemiology	Socioeconomic inequalities in cardiovascular mortality and the role of work: a register study of Finnish men	2002; 31(3):614-621	Exposure and/or outcome not suitable
101	von Bonsdorff, M. E., Kokko, K., Seitsamo, J., von Bonsdorff, M. B., Nygard, C. H., Ilmarinen, J., Rantanen, T.	Scandinavian Journal of Work, Environment and Health	Work strain in midlife and 28-year work ability trajectories	2011; 37(6):455-463	Exposure and/or outcome not suitable
102	Yong, M., Nasterlack, M., Germann, C., Lang, S., Oberlinner, C.	International Archives of Occupational and Environmental Health	Shift work and risk of non-cancer mortality in a cohort of German male chemical workers	2014; 87(7):763-773	Exposure and/or outcome not suitable

Table S4: Quality assessment of studies for all-cause and CHD mortality outcome included in systematic review per criteria described in Table S2

First author; Year	Item 1	Item 2	Item 3	Item 4	Item 5	Item 6	Item 7	Item 8	Item 9	Item 10	Item 11	Item 12	Item 13	Item 14	Overall Score[#]
All-cause mortality outcome (n=17)															
Hibbard 1993 (1)	Cannot say	Yes	Yes	Yes	Cannot say	Cannot say	Yes	Cannot say	No	No	Yes	No	Limited	Yes	0
Eaker 2004 (2)	Yes	Yes	Yes	Yes	Cannot say	Cannot say	No	Cannot say	No	Yes	Cannot say	No	Yes	Yes	+
Falk 1992 (3)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Gonzalez-Mule 2017 (4)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	No	Yes	Yes	No	0
Natti 2009 (5)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	Yes	No	Limited	Yes	0
Natti 2012 (6)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Niedhammer 2011 (7)	Yes	Yes	Yes	Yes	Yes	No	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Nilsen 2016 (8)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	Yes	Yes	Limited	Yes	0
O'Reily 2013 (9)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Shirom 2011 (10)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	Yes	Yes	Yes	Yes	+
Tobiasz-Adamczyk 2013 (11)	Yes	Yes	No	Yes	Cannot say	Cannot say	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	0
Von Bonsdorff 2012 (12)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+

Amick 2002 (13)	Yes	Yes	Yes	No	Yes	No	Yes	Cannot say	No	No	No	No	Yes	Yes	0
Boggild 1999 (14)	Yes	Yes	Yes	No	Cannot say	Cannot say	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	0
Åkerstedt 2004 (15)	Yes	Yes	Yes	No	Yes	No	Yes	Cannot say	No	Yes	Yes	No	Limited	Yes	0
Sabbath 2015 (16)	Yes	Yes	No	No	Cannot say	Cannot say	Yes	Cannot say	No	Yes	Yes	No	Limited	Yes	0
Perlman 2009 (17)	Yes	Yes	Yes	No	Yes	Yes	Yes	Cannot say	No	Yes	No	No	Limited	Yes	0
CHD mortality outcome (n=14)															
Johnson 1989 (18)	Yes	Yes	Yes	No	Yes	Yes	Yes	Cannot say	No	Yes	Yes	No	No	Yes	0
Kivimaki 2006 (19)	Yes	Yes	Yes	Yes	Yes	No	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Kivimaki 2002 (20)	Yes	Yes	No	Yes	Cannot say	No	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Elovainio 2006 (21)	Yes	Yes	No	Yes	Cannot say	No	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Lee 2002 (22)	Yes	Yes	Yes	Yes	Yes	No	Yes	Cannot say	No	Yes	Yes	No	No	Yes	0
Slopen 2012 (23)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Toivanen 2006 (24)	Yes	Yes	Yes	No	Yes	Yes	Yes	Cannot say	No	Yes	Yes	No	No	Yes	0
Alterman 1994 (25)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Padyab 2014 (26)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Hublin 2010 (27)	Yes	Yes	No	Yes	Yes	No	Yes	Cannot say	No	Yes	Yes	Yes	Yes	Yes	+
Johnson 1996* (28)	Yes	Yes	Yes	Yes	No	Yes	Yes	Cannot say	Yes	Limited	Yes				+
Karasek 1981* (29)	Yes	Yes	Yes	Yes	No	Yes	Yes	Cannot say	Yes	Yes	Yes				+

Yadegarfar 2008* (30)	Yes	Yes	Yes	Yes	No	Yes	Yes	Yes	N/A	Limited	Yes				+
McNamee 1996* (31)	Yes	Yes	Yes	No	No	Yes	Yes	Yes	N/A	Limited	Yes				+
All-cause mortality and CHD mortality outcomes (n=14)															
Brunner 2004 (32)	Yes	Yes	No	Yes	Cannot say	No	Yes	Cannot say	No	Yes	Yes	No	Limited	Yes	+
Laszlo 2010 (33)	Yes	Yes	No	Yes	Yes	No	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Laszlo 2013 (34)	Yes	Yes	No	Yes	Yes	No	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Holtermann 2010 (35)	Yes	Yes	Yes	Yes	Cannot say	Cannot say	Yes	Cannot say	No	Yes	Yes	No	No	Yes	0
Holtermann 2011 (36)	Yes	Yes	Yes	Yes	Cannot say	Cannot say	Yes	Cannot say	No	Yes	Yes	No	Limited	Yes	0
Joensuu 2012 (37)	Yes	Yes	Yes	Yes	Cannot say	Cannot say	Yes	Cannot say	No	Yes	Yes	No	Limited	Yes	0
Joensuu 2014 (38)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Tsutsumi 2006 (39)	Yes	Yes	Yes	Yes	Yes	No	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Yong 2014 (40)	Yes	Yes	No	Yes	Yes	No	Yes	Yes	N/A	Yes	Yes	Yes	Yes	Yes	+
Kivimaki 2003 (41)	Yes	Yes	No	No	Cannot say	Cannot say	Yes	Cannot say	No	Yes	Yes	No	Limited	Yes	0
Jorgensen 2017 (42)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Gu 2015 (43)	Yes	Yes	Yes	Yes	Yes	Yes	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+
Karlsson 2005 (44)	Yes	Yes	Yes	No	Yes	Yes	Yes	Cannot say	No	Yes	Yes	Yes	No	Yes	0
Fujino 2006 (45)	Yes	Yes	Yes	Yes	Yes	No	Yes	Cannot say	No	Yes	Yes	No	Yes	Yes	+

* SIGN Methodology Checklist 4: Case-control studies *(version 2, 28 May 2012)* used for nested case control studies within longitudinal studies

[#] The overall methodological quality of the study is rated as i) High quality (++): majority of criteria met (little or no risk of bias and results unlikely to be changed by further research), ii) Acceptable (+): most criteria met (some flaws in the study with an associated risk of bias and conclusions may change in the light of further studies), iii) Low quality (0): (either most criteria not met, or significant flaws relating to key aspects of study design and conclusions likely to change in the light of further studies).

Table S5: Characteristics of 45 epidemiological studies of work stressors and risk of mortality

First author & year; cohort & country	Study population	Participants (n)	Men (%)	Age (years)	Work stressor exposure and measurement	Outcome	Measurement of Outcome	Follow up duration
All-cause mortality outcome (n=17)								
Hibbard 1993 (1) Household interview survey, United States	general population	1502	61	18-65	Work support: self-reported at baseline; summary of 3 items on continuous scale Job control: self-reported at baseline; summary of 8 items on continuous scale	All-cause mortality	Health plan records, National Death Index and vital records	15
Eaker 2004 (2) Framingham Offspring Study, United States	general population	3039	56	18-77	Job demands: self-reported 3rd examination cycle 1984-1987; summary of 5 items scored on Likert scale from 1 to 4 on continuous scale Decision latitude: self-reported 3rd examination cycle 1984-1987; summary of subscales decision authority (3 items) and skill discretion (6 items) scored on Likert scale from 1 (strongly disagree) to 4 (strongly agree) on continuous scale Job strain derived from job demands and decision latitude high job strain: high job demands (above median score) and low decision latitude (at or below median score), low job strain: low job demands and high decision latitude, passive job strain: low job demands and low decision latitude, active job strain: high job demands and high decision latitude	All-cause mortality	No information	10
Falk 1992 (3) Malmo study, Sweden	general population	477	100	68-69	Job demands: self-reported 3 years after participants' retirement 1982-1983; affirmative answers to two items on workload and time pressure used to define high job demands Personal schedule freedom: self-reported 3 years after participants' retirement 1982-1983; low personal schedule freedom defined as negative answers to at least two of three items Job strain derived from job demands and personal schedule freedom; job strain: high job demands and low personal schedule freedom, no job strain: all other job demands and personal schedule freedom groups	All-cause mortality	Cause of Death Register (Department of Community Health Sciences in Malmo and Swedish National Central Bureau of Statistics)	6
Gonzalez-Mule 2017 (4) Wisconsin Longitudinal Study, United States	Wisconsin high schools'	2363	50	63-67	Job demands: self-reported from questionnaire administered in 2004; 8 items scored on Likert scale from 1 (strongly disagree) to 4 (strongly agree) and 1 item scored	All-cause mortality	Relatives and Death certificate	7

	graduates in 1957				on Likert scale from 1 (never) to 5 (always), and scores standardised then averaged Job control: self-reported from questionnaire administered in 2004; 6 items scored on Likert scale from 1 (strongly disagree) to 4 (strongly agree) and scores standardised then averaged			
Natti 2009 (5) Statistics Finland Quality of Work Life Surveys, Finland (Study 1)	Finnish employees	4502	51*	15-64	Physical demands: self-reported at baseline; 1 item scored on Likert scale from 1 (very undemanding) to 4 (very demanding) and dichotomised into low (very or quite undemanding) or high (very or rather demanding) levels of physical demands Psychological demands: self-reported at baseline; 1 item scored on Likert scale from 1 (very undemanding) to 4 (very demanding) dichotomised into low (very or quite undemanding) or high (very or rather demanding) levels of psychological demands Demand for learning: self-reported at baseline; 1 item on Likert scale from 1 (not at all) to 4 (very much) dichotomised into low (not at all or some) or high (high or very high) demands for learning new things Job insecurity: self-reported at baseline; based on 2 items and classified as permanent employees, temporary employees satisfied and temporary employees unsatisfied	All-cause mortality	Death certificates supplemented with data from the population information system of the Population Register Centre	16
Natti 2009 (5) Statistics Finland Quality of Work Life Surveys, Finland (Study 2)	Finnish employees	3345	49*	15-64	Physical demands: self-reported at baseline; 1 item scored on Likert scale from 1 (very undemanding) to 4 (very demanding) dichotomised into low (very or quite undemanding) or high (very or rather demanding) levels of physical demands Psychological demands: self-reported at baseline; 1 item scored on Likert scale from 1 (very undemanding) to 4 (very demanding) dichotomised into low (very or quite undemanding) or high (very or rather demanding) levels of psychological demands Demand for learning at work: self-reported at baseline; 1 item on Likert scale from 1 (not at all) to 4 (very much) dichotomised into low (not at all or some) or high (high or very high) demands for learning new things	All-cause mortality	Death certificates supplemented with data from the population information system of the Population Register Centre	10

					Job insecurity: self-reported at baseline; based on 2 items and classified as permanent employees, voluntary temporary employees and involuntary permanent employees			
Natti 2012 (6) Statistics Finland Quality of Work Life Surveys, Finland	Finnish employees	3095	47	15-64	Night work: self-reported at baseline; employees classified as night workers who worked most often (on a weekly basis) at night or dayworkers who did not work at night	All-cause mortality	Death certificates supplemented with data from the population information system of the Population Register Centre	23
Niedhammer 2011 (7) Lorhandicap study, France	general population	4118	53	15-70	Work support: self-reported at baseline; 1 item based on Likert scale from 1 (very unsatisfied) to 5 (very satisfied) dichotomised into low (very unsatisfied or unsatisfied) or high (neither satisfied nor unsatisfied, satisfied or very satisfied) social support from colleagues	All-cause mortality	National computerised database and registry offices	12.5
Nilsen 2016 (8) Level of Living Survey, Sweden	general working population	1393	50	42-65	Job demands: 1. self-reported at baseline; affirmative answers to two items on workload and time pressure used to define high job demands 2. JEM based on occupation-by-industry data at baseline and dichotomised into low or high job demands using median split Job control: 1. self-reported at baseline; scores from 4 items on intellectual discretion (low, medium, high categories) and 2 items on personal schedule freedom on Likert scale from 0 (very high) to 4 (not at all) summed and dichotomised into low or high job control using median split 2. JEM based on occupation-by-industry data at baseline and dichotomised into low or high job control using median split Job strain derived from job demands and job control high job strain: high job demands and low job control, low job strain: low job demands and high job control, passive job strain: low job demands and low job control, active job strain: high job demands and high job control	All-cause mortality	Official Swedish personal identification number linked to National Death Register	23
O'Reily 2013 (9) Northern Ireland Mortality Study, Ireland	Employees	414949	65	m: 20-64 f: 20-59	Working weekly hours: self-reported at baseline 2001 Census; response to weekly number of hours usually worked in main job in past 4 weeks classified into 4 groups (35-40, 41-48, 49-54, ≥ 55 hours)	All-cause mortality	2001 Census data for the whole enumerated population linked to registered deaths up to 2009	8.7

Shirom 2011 (10) Periodic employee health examination and Clalit Health Services (CHS) data, Israel	adult Jewish workforce	820	67	mean 41.6 (SD 9.34)	Job demands: self-reported at baseline; scores from 8 items on 5-point Likert scale summarised and mean centred (high scores reflecting high job demands) Job control: self-reported at baseline; scores from 5 items on 5-point Likert scale summarised and mean centred (high scores reflect high job control) Supervisor support: self-reported at baseline; scores from 2 items on 5-point Likert scale summarised and mean centred (high scores reflect high supervisor support) Peer support: self-reported at baseline; scores from 2 items on 5-point Likert scale summarised and mean centred (high scores reflect high peer support)	All-cause mortality	CHS's medical records (deaths officially notified to CHS by hospitals and the Ministry of the Interior)	20
Tobiasz-Adamczyk 2013 (11) Community dwelling citizens of Krakow, Poland	general population aged 65 years	727	44	65	Psychological demands and control: self-reported at baseline; subscales for psychological effort/demands (3 items) and job control (2 items) summarised and dichotomised into low or high psychological demands and low or high job control using median split, and classified into 4 groups (low psychological demands and high control, high psychological demands and high control, low psychological demands and low control, high psychological demands and low control) Psychological efforts and rewards: self-reported at baseline; subscales for psychological effort/demands (3 items) and rewards (4 items) summarised and dichotomised into low or high psychological effort and low or high rewards using median split, and classified into 4 groups (low psychological effort and high reward, high psychological effort and high reward, low psychological effort and low reward, high psychological efforts and high reward)	All-cause mortality	City Vital Records	7
Von Bonsdorff 2012 (12) Finnish Longitudinal Study on Municipal Employees, Finland	municipal employees	5731	45	44-58	Job demands: self-reported at baseline; scores from 5 items on Likert scale from 0 (not at all) to 3 (a lot) summed and dichotomised into low or high job demands using median split Job control: self-reported at baseline; scores from 10 items on Likert scale from 0 (not at all) to 3 (enough) summed and dichotomised into low or high job control using median split Job strain derived from job demands and job control	All-cause mortality	Finnish National Population Register	25.3

					high job strain: high job demands and low job control, low job strain: low job demands and high job control, passive job strain: low job demands and low job control, active job strain: high job demands and high job control			
Amick 2002 (13) Panel Study of Income Dynamics, United States	general population	7746	52	18-62	Job demands: JEM based on occupational data imputed for 1968 to 1991; yearly percentages of working life spent in each quartile of the distribution (high, medium high, medium low, low) calculated to estimate cumulative lifetime job demands exposure as time-varying measure Job decision latitude: JEM based on occupational data imputed for 1968 to 1991; yearly percentages of time spent in each quartile of the distribution (high, medium high, medium low, low) calculated to estimate cumulative lifetime job control exposure as time-varying measure Job strain: job demands divided by job decision latitude calculated to create a continuous measure (high values representing high strain) and yearly percentages of time spent in each quartile of the distribution (high, medium high, medium low, low) calculated to estimate cumulative lifetime job strain exposure as time-varying measure Work support: JEM based on occupational data imputed for 1968 to 1991; yearly percentages of time spent in each quartile of the distribution (high, medium high, medium low, low) calculated to estimate cumulative lifetime work support exposure as time-varying measure Job security: JEM based on occupational data imputed for 1968 to 1991; yearly percentages of time spent in each quartile of the distribution (high, medium high, medium low, low) calculated to estimate cumulative lifetime job security exposure as time-varying measure	All-cause mortality	Yearly reports of the reasons for nonresponse	24
Boggild 1999 (14) Copenhagen Male Study, Denmark	general population	5207	100	40-59	Shift work: self-reported at baseline; response to question on working time (worked irregular hours, shift work, often had night work, day time work only) dichotomised into day worker (day time work only) and shift worker (all other working hours)	All-cause mortality	Danish Central Population Register	22
Åkerstedt 2004 (15)	general population	22411	48	25-64	Shift work: self-reported at baseline; response to question on working time (3-shift work, night work, evening work,	All-cause mortality	Swedish Cause-of-Death register	11.8

National Survey of Living Conditions, Sweden					roster work, other work, day time work only) dichotomised into day workers (day time work only) and shift workers (all other work time)			
Sabbath 2015 (16) Health and Retirement Study (HRS), United States	working mothers	7352	0	18-50	Job demands: JEM based on occupation-by-industry data at baseline; imputed scores summed and trichotomized at 33rd and 67th percentiles into low, medium or high tertiles of job demands Job control: JEM based on occupation-by-industry data at baseline; imputed scores summed and trichotomized at 33rd and 67th percentiles into low, medium or high levels of job control	All-cause mortality after age 50	National death Index (NDI) and supplemented by proxy reports for post-2008 deaths, for which NDI data were not yet available	Not reported
Perlman 2009 (17) Russia Longitudinal Monitoring Survey (RLMS)	general population	8723	52	18-59	Job insecurity: self-reported at baseline; responses from 1 item on concern about loss of job graded on 5-point scale (very concerned, a little concerned, yes and no, not very concerned, not at all concerned) grouped as very concerned versus moderately concerned or unconcerned about job loss	All-cause mortality	Household members were asked if absence of non-responders due to death	8
CHD mortality outcome (n=14)								
Johnson 1989 (18) Central Bureau of Statistics' survey of living conditions, Sweden	general population	7219	100	25-65	iso-strain (combination of social isolation and job strain): self-reported at baseline; subscales for job demands (2 items on binary scale), job control (11 items on 3-point Likert scale), and work social support (5 items on binary scale) scored such that high score represented adverse condition, standardised to mean (SD) 0 (1), and scaled to above one, and multiplied together. Quintiles calculated from frequency distribution and grouped into high (5th quintile), medium (2nd, 3rd, 4th quintiles), low (1st quintile) levels of iso-strain	CVD mortality	Official Swedish personal identification number linked to National Death Register (*ICD-8* codes 400-404, 410-414, 427, 429-436, 440-445, 454-458)	9
Kivimaki 2006 (19) METELI study, Finland	Valmet factories industrial employees	788	68	17-65	Job demands: self-reported at baseline; responses from 4 items on 5-point Likert scale averaged and standardised to mean (SD) 0 (1) Job control: self-reported at baseline; responses form 12 items on 5-point Likert scale averaged and standardised to mean (SD) 0 (1)	CVD mortality	Official Finnish personal identification number linked to Statistics Finland National Mortality Register; *ICD-8, ICD-9, ICD-10* classifications converted to *ICD-10* codes and deaths due to CVD identified from codes I20-I25, I30-I52, I60-I69, I00-I19, I26-I29, I70-I99	25.6
Kivimaki 2002 (20) METELI study, Finland	Valmet factories	812	67	17-65	Job demands: self-reported at baseline; responses from questions about responsibility, task difficulty and mental load on 5-point Likert scale summed and trichotomized at	CVD mortality	Official Finnish personal identification number linked to Statistics Finland	25.6

	industrial employees				33rd and 67th percentiles into low, intermediate or high levels of job demands Job control: self-reported at baseline; responses from questions about decision authority and skill discretion with 5-point Likert scale summed and trichotomized at 33rd and 67th percentiles into low, intermediate or high levels of job control Job strain derived from job demands and job control high job strain: high/intermediate demands and low control, low job strain: low demands and high/intermediate control, intermediate job strain: all other combinations of job demands and job control Effort reward imbalance: questions on effort at work (pace of work, physical and mental load) and rewards (satisfaction with income, fairness, job security, promotion) on 5-point Likert scale self-reported at baseline; Effort and reward imbalance created by calculating ratio between sum response score in the effort scale and sum response score in reward scale and trichotomizing quotient at 33rd and 67th percentiles into low, intermediate or high levels of effort reward imbalance		National Mortality Register and all deaths due to CVD diseases pooled from the register	
Elovainio 2006 (21) Valmet study, Finland	Valmet factories industrial employees	804	67	17-65	Justice at work: self-reported at baseline; response to one question on justice at work dichotomised into high or low/intermediate levels of justice at work	CVD mortality	Official Finnish personal identification number linked to Statistics Finland National Mortality Register and all deaths due to CVD disease pooled to indicate death due to cardiovascular diseases from *ICD-10* codes I20-I25, I30-I52, I60-I69, I00-I19, I26-I29, I70-I99	25.6
Lee 2002 (22) Nurses' Health Study, United States	Registered nurses	35038	0	46-71	Job strain: derived from self-reported job demands and job control at baseline; responses for subscales for job demands (5 items) and job control (9 items) on 4-point Likert scale summed and dichotomised into low or high levels using median split, and classified into 4 groups (low job strain: low demands and high control, active job strain: high demands and high control, passive job strain: low demands and low control, high job strain: high demands and low control)	Fatal CHD	Information from next of kin confirmed by National Death Index and Fatal CHD defined as fatal MI confirmed by hospital records or at autopsy or CHD recorded on death certificate	4

Slopen 2012 (23) Women's Health Study, United States	Health professionals	22068	0	mean 57.2 (SD 5.2)	Job strain: derived from self-reported job demands and job control at baseline; responses for subscales for job demands (5 items) and job control (9 items) on 4-point Likert scale summed and dichotomised into low or high levels using median split, and classified into 4 groups (low job strain: low demands and high control, active job strain: high demands and high control, passive job strain: low demands and low control, high job strain: high demands and low control) Job insecurity: self-reported at baseline; response from 1 item scored on Likert scale from 1 (strongly disagree) to 4 (strongly agree) dichotomised into job insecure (strongly disagree and disagree) or job secure (agree and strongly agree) levels	CVD death	National Death Index, reports from family members or the postal service. CVD deaths confirmed by medical record review included as events	10
Toivanen 2006 (24) Statistics Sweden Surveys of Living Conditions and Swedish Census, Sweden	general population	1858373	50	40-64	Job control: JEM based on occupational data from census data imputed at baseline; 5 job control items with scores ranging from 0 to 5 dichotomised into low or high levels of job control using median split Job control index: JEM based on occupational data from census data imputed at baseline; 5 job control items with scores ranging from 0 to 5 dichotomised into low or high levels of job control using median split and each group further split into two equal size groups creating 4 levels of job control (lowest, low, intermediate, high)	CVD mortality	Swedish Census data linked to National cause of death registry	5
Alterman 1994 (25) Chicago Western Electric Study, United States	Caucasian middle-aged men employed at Hawthorne Works	1683	100	38-56	Decision latitude: JEM based on occupational data from census data imputed at baseline; weighted sum of responses for job control (10 items) on continuous scale Psychologic demand: JEM based on occupational data from census data imputed at baseline; sum of responses for job demands (5 items) on continuous scale Job strain derived from psychologic demand and decision latitude; scores for psychologic demand trichotomized into lower quarter, middle half, and upper quarter groups of the distribution, decision latitude scores divided into tertiles, and classified into high (1st tertile decision latitude and upper quarter group psychologic demand) or no (all other combinations of decision latitude and psychologic demand) levels of job strain	CHD mortality	Death certificates and deaths due to CHD based on *ICD-8* codes 410-414	25

Padyab 2014 (26) Vasterbotten Intervention Programme, Sweden	general population	74988	49	40-60	Psychological demands: self-reported at baseline; scores from 4 items on 4-point Likert scale summed and dichotomised into low or high job demands using median split Decision latitude: self-reported at baseline; scores from 6 items on 4-point Likert scale summed and dichotomised into low or high job control using median split Job strain derived from job demands and job control high job strain: high job demands and low job control, low job strain: low job demands and high job control, passive job strain: low job demands and low job control, active job strain: high job demands and high job control Iso-strain (combination of social isolation and job strain): Social support at work self-reported at baseline, based on 5 items regarding contacts with co-workers during work and leisure time, dichotomised into low or high social support at work using gender-specific median split. Iso-strain (low social support and high job strain) or no iso-strain (all other combinations of social support and job strain)	CVD mortality	Official Swedish personal identification number linked to National Death Register. Deaths due to stroke identified from *ICD-9* codes 431, 434, 436 and *ICD-10* codes I61, I63, I64; d deaths due to myocardial infarction identified from *ICD-9* codes: 410, 411, 412, 414, 427F and *ICD-10* codes I21, I22, I23, I25, I46	m: 11.3 f: 11.8
Hublin 2010 (27) Finnish Twin Cohort, Finland	general population	20142	49	24-70	Type of working time: self-reported (1975 and 1981); response to question on working time (mainly day, mainly night, mainly shift work, never worked) classified into 5 level categorical variable (daytime work both in 1975 & 1981, night-time work either in 1975 or 1981, shift-work in 1975 & daytime work in 1981, daytime work in 1975 & shift-work in 1981, shift-work both in 1975 & 1981)	CHD mortality	Population Register Centre of Finland and Statistics Finland. Deaths due to CHD identified from *ICD-8* and *ICD-9* codes 410–414 and *ICD-10* codes I20–I25	22
Johnson 1996 (28) Statistics Sweden, Sweden	general population	2943	100	25-74	Psychological job demands: JEM based on occupational data used to assign scores for job demands to each year of employment as time-varying measure to estimate cumulative exposure; scores from 2 items for job demands summed and divided into quartiles of high, medium high, medium low and low levels of job demands Job control: JEM based on occupational data used to assign scores for job control to each year of employment as time-varying measure to estimate cumulative exposure; scores from 12 items for job control summed, and divided into quartiles of high, medium high, medium low, and low levels of job control	CVD mortality	Official Swedish personal identification number linked to National Death Register. Deaths due to CVD from *ICD-8* codes 400-404, 410-414, 427, 430-436, 440-445	14

					Social support at work: JEM based on occupational data used to assign scores for social support at work to each year of employment as time-varying exposure to estimate cumulative exposure; scores from 4 items for social support summed and divided into quartiles of high, medium high, medium low, and low levels of social support at work			
Karasek 1981 (29) Level of Living Survey, Sweden	general population	88	100	15-67	Intellectual Discretion: self-reported in 1974 at second interview; response for 1 item on combination of skill level of job and level of repetitious/monotonous work classified into low (repetitive jobs requiring minimum education), medium low (non-repetitive jobs requiring minimum education or repetitive jobs requiring additional training), medium high (non-repetitive jobs requiring at least 1-4 years additional training, or high levels of intellectual discretion), or high (non-repetitive jobs requiring > 4 years additional training) levels of intellectual discretion Personal Schedule Freedom: self-reported in 1974 at second interview; responses for 3 items on binary scale classified into low (no to all 3 questions), medium low (no to 2 questions), medium high (yes to 2 questions), or high (yes to all questions) levels of personal schedule freedom Job demands: self-reported in 1974 at second interview; responses for 2 items on binary scale classified into low (no to both questions), medium (yes to 1 question), or high (yes to both questions) levels of job demands	cardiovascular-cerebrovascular deaths	verified death certificates cardiovascular-cerebrovascular deaths identified from *ICD* codes 400-404, 410-414, 427, 430-436, 440-445.	6
Yadegarfar 2008 (30) Industrial cohort, UK	male industrial workers	1270	100	<50	Shift work: assessed from company records; workers classified as shift worker (shift work > 1 month) or day workers (all other workers)	IHD mortality ≤ 75 years	UK Office of National Statistics cause of death classification on death certificate *ICD* codes 410-414	25.5
McNamee 1996 (31) Industrial cohort, UK	male industrial workers	934	100	<50	Shift work: assessed from company records; workers classified as shift worker (shift work > 1 month) or day workers (all other workers)	IHD mortality ≤ 75 years	UK Office of National Statistics cause of death classification on death certificate *ICD* codes 410-414	23
All-cause and CHD mortality outcomes (n=14)								

Brunner 2004 (32) Valmet study, Finland	Valmet factories industrial employees	812	67	17-65	Job strain: derived from self-reported job demands and job control at baseline; responses for subscales for job demands and job control on 5-point Likert scale summed and trichotomized into low, intermediate or high levels using tertile split, and classified into 3 groups (low job strain: low demands and high/intermediate control, high job strain: high/intermediate demands and low control, and intermediate job strain: all other combinations of job demands and control) Effort reward imbalance: questions on effort at work (pace of work and physical and mental load) and rewards (satisfaction with income, fairness, job security and promotion) on 5-point Likert scale self-reported at baseline. Effort and reward imbalance derived by calculating ratio between summed response scores in effort and reward scales; and trichotomizing quotient into low, intermediate or high levels or effort reward imbalance using tertile split	All-cause mortality; CVD mortality	Official Finnish personal identification number linked to Statistics Finland National Mortality Register and all deaths due to CVD diseases pooled from the register	25.6
Laszlo 2010 (33) Stockholm Heart Epidemiology Program (SHEEP), Sweden	Non-fatal AMI incident cases employed and < 65 years	674	79	40-65	Job demands: self-reported at baseline; responses from 5 items on 4-point Likert scale (almost always to almost never) summed and divided into quartiles Job control: self-reported at baseline; responses from 6 items on 4-point Likert scale (almost always to almost never) summed and divided into quartiles Job strain: job demands and job control scores dichotomized using median split and classified into high (high demands and low control), low (low demands and high control), passive (low demands and low control), or active (job demands and high control) levels of job strain	All-cause mortality; Cardiac mortality	Official Swedish personal identification number linked to National Death Register for death and cause of death data	8.5
Laszlo 2013 (34) Stockholm Heart Epidemiology Program (SHEEP), Sweden	Non-fatal AMI incident cases employed and < 65 years	676	79	40-65	Job insecurity: self-reported at baseline; response to 1 item assessing concerns at work dichotomised into job insecurity (concerns about dismissal from work) and no job insecurity (all other responses)	All-cause mortality; Cardiac mortality	Official Swedish personal identification number linked to National Death Register and deaths due to cardiac mortality identified from *ICD-9* codes 410, 412, 414, 420–429; and *ICD-10* codes I20, I21, I25, I38, I48, I50	8.5
Holtermann 2010 (35)	general population	4943	100	40-59	Working weekly hours: self-reported at baseline; response to one question about weekly number of work hours	All-cause mortality;	Official national registers	30

Copenhagen Male Study, Denmark					classified into 3 groups (≤ 40, 41-45, and ≥ 46 hours per week)	IHD mortality	Deaths due to IHD identified from *ICD-8* codes 410-414, and *ICD-10* codes I20-I25	
Holtermann 2011 (36) Copenhagen Male Study, Denmark	general population	4931	100	40-59	Psychological pressure at work: self-reported at baseline; response to one question about psychological pressure when performing work (rarely, regularly) dichotomised into rarely or regularly levels of perceived exposure to psychological work pressure	All-cause mortality; IHD mortality	Official national registers Deaths due to IHD identified from *ICD-8* codes 410-414, and *ICD-10* codes I20-I25	30
Joensuu 2012 (37) Still Working Study, Finland	private sector industrial employees in multinational forest industry corporation	13510	77	16-65	Skill discretion: self-reported at baseline; responses to 5 items on 5-point Likert scale trichotomized into low, intermediate, or high levels of skill discretion using tertile split Decision authority: self-reported at baseline; responses to 5 items on 5-point Likert scale trichotomized into low, intermediate, or high levels of decision authority using tertile split	All-cause mortality; CVD mortality	Official Finnish personal identification number linked to Statistics Finland National Mortality Register; *ICD-8, ICD-9, ICD-10* classifications converted to *ICD-9* codes and deaths due to CVD identified from *ICD-9* codes 390-459, and *ICD-10* codes I00-I99	15.5
Joensuu 2014 (38) Finnish Public Sector study, Finland	municipal and public hospitals employees	60202	20	17-69	Skill discretion: self-reported at baseline; responses to 6 items on 5-point Likert scale trichotomized into low, intermediate, or high levels of skill discretion using tertile split Decision authority: self-reported at baseline; responses to 3 items on 5-point Likert scale trichotomized into low, intermediate, or high levels of decision authority using tertile split	All-cause mortality; CVD mortality	Statistics Finland National Death Registry; deaths due to cardiovascular diseases identified from *ICD-9* codes 390-459, and *ICD-10* I00-I99	8.8
Tsutsumi 2006 (39) Jichi Medical School Cohort Study, Japan	working population aged 65 years and younger	6509	49	18-65	Job demands: self-reported at baseline; scores from 5 items on 4-point Likert scale summarised and divided into quartiles (highest, higher middle, lower middle, lowest) Job control: self-reported at baseline; subscales for skill discretion (4 items) on 4-point Likert scale and autonomy for decision making (2 items) on 4-point Likert scale summarised and divided into quartiles (highest, higher middle, lower middle, lowest) Job strain derived from job demands and job control dichotomies of low and high levels using median split high job strain: high job demands and low job control,	All-cause mortality; CVD mortality	Cause-of-Death Register at public health centres in each community; deaths due to cardiovascular diseases identified from *ICD-10* codes I00-I52, I60-I69	9

					low job strain: low job demands and high job control, passive job strain: low job demands and low job control, active job strain: high job demands and high job control			
Yong 2014 (40) BASF Ludwigshafen, Sweden	production workers in chemical factory	31143	100	mean 41 (SD 11)	Shift work: assessed from company records; workers classified as rotating shift worker (rotating shift work > 1 year) or day workers (all other workers)	All-cause mortality; IHD mortality	Death certificates; deaths due to IHD identified from *ICD* 10 codes I20.0-I25.9	10
Kivimaki 2003 (41) 10-Town Study, Finland	municipal employees and long-term unemployed	92351	29	18-63	Employment status: assessed from employers' records from January 1990, to December 2000; permanent employees (permanent job contract between 1990 & 2000), employees who moved from a temporary to a permanent job, temporarily employed workers (fixed-term contract, but not permanent employment), long-term unemployed persons who obtained subsidised work contracts offered by municipalities	All-cause mortality; CVD mortality	Official Finnish personal identification number linked to Statistics Finland National Mortality Register; deaths from CVD identified from *ICD-9* codes 390-459, *ICD-10* codes I00-I99	7.7
Jorgensen 2017 (42) Danish nurse cohort, Denmark	female nurses	18015	0	≥44	Shift work: self-reported at baseline; one question on shift work status with 4 categories (day, evening, night, rotating shifts)	All-cause mortality; CVD mortality	Danish Register of Causes of Death and Civil Registration System; deaths due to CVD identified from *ICD-10* codes I00-99, *ICD-8* codes 4010, 4100, 4129, 4279, 4339, 4369, 4412, 4500	17.6
Gu 2015 (43) Nurses' Health Study, United States	female nurses	74862	0	30-55	Night shift work duration: self-reported at baseline (1988 survey); response to question on total number of years worked rotating night shifts at least 3 nights/month in addition to day or evening shifts (never, 1-2, 3-5, 6-9, 10-14, 15-19, 20-29, and ≥30 years) summarised into 4 categories (never, 1-5 years, 6-14 years, ≥15 years)	All-cause mortality; CVD mortality	Reports from next of kin and postal authorities. Non-responders searched in the NDI; cause of death based on medical records and death certificates, and deaths due to CVD identified from *ICD-8* codes 390-459, 795	22
Karlsson 2005 (44) pulp and paper manufacturing employees, Sweden	male pulp and paper employees	5442	100	<60	Shift work: assessed from company records; total duration of years shift work summarised into 6 categories (never, <5 years, 5-9 years, 10-19 years, 20-29 years, ≥30 years)	All-cause mortality; CHD mortality	Official Swedish personal identification number linked to National Cause of Death Register; deaths due to CHD identified from *ICD-6* codes 4200, 4201, 4202, 4203, 4209; *ICD-7* codes 4200, 4201, 4202; *ICD-8*	30

							and *ICD-9* codes 410, 411, 412, 413, 414; *ICD-10* codes I20, I21, I22, I23, I24	
Fujino 2006 (45) Japan Collaborative Cohort Study, Japan	general population males	17649	100	40-59	Shift work: self-reported at baseline; response to question about patterns of shift work during working life (mainly daytime, mainly night, alternate night and daytime work)	All-cause mortality; CVD mortality	Regional research centres' population registration sheets; deaths due to CVD identified from *ICD-10* codes I00-I99	13.3

* Descriptive statistics for gender not reported hence the percentage of males in the study estimated from sub group analyses

Table S6: Results of 45 epidemiological studies of work stressors and risk of mortality

First author & year	Events (n)	Exposure comparisons	Minimal model Effect estimate and confidence intervals	Fully adjusted model Effect estimate and confidence intervals (highest category and lowest category)	Fully adjusted model Adjustment confounders
All-cause mortality outcome (n=17)					
Hibbard 1993 (1)	138	High social support at work		f: HR 0.8 (95% CI 0.7 - 1.0) m: HR 1.0 (95% CI: 0.8 - 1.1)	age, education, health status, parental role characteristics, marital role characteristics
		High job control		f: HR 1.0 (95% CI 0.9 - 1.2) m: HR 1.0 (95% CI: 0.9 - 1.1)	
Eaker 2004 (2)	214	High job demands (1 SD change in continuous measure)	f: RR 0.75 (95% CI 0.55 - 1.02) m: RR 1.01 (95% CI 0.85 - 1.21) (adjusted for age)	f: RR 0.96 (95% CI 0.91 - 1.01) m: RR 1.05 (95% CI 0.78 - 1.41)	age, SBP, ratio of total cholesterol to HDL cholesterol, BMI, smoking, diabetes
		High decision latitude (1 SD change in continuous measure)	f: RR 0.86 (95% CI 0.63 - 1.18) m: RR 0.91 (95% CI 0.76 - 1.08) (adjusted for age)	f: RR 0.98 (95% CI 0.96 - 1.02) m: RR 0.97 (95% CI 0.81 - 1.16)	
		Low job strain compared with high job strain		f: RR 0.98 (95% CI 0.76 - 2.42) m: RR 0.85 (95% CI 0.48 - 1.50)	
Falk 1992 (3)	87	High job demands compared with low job demands	RR 1.3 (95% CI 0.8 - 2.0) (restricted to 68-69 years at baseline)		restricted to 68-69 years at baseline, social class, cardiovascular risk factors
		Low personal schedule freedom compared with high personal schedule freedom	RR 0.7 (95% CI 0.5 - 1.2) (restricted to 68-69 years at baseline)		
		High job strain compared with no job strain	RR 1.7 (95% CI 1.0 - 2.3) (restricted to 68-69 years at baseline)	RR 1.6 (95% CI 1.0 - 2.5)	
Gonzalez-Mule 2017 (4)	189	High job demands		OR 0.92 (CI not reported)	age, sex, marital status, smoking, overall health, income, net worth, education, job prestige, managerial job, positive affect, negative affect**, job demands, job control, interaction between job demands and job control**
		High job control		OR 0.94 (CI not reported)	
		Interaction between high job demands and job control		OR 0.72 (CI not reported)	

Natti 2009 (5) (Study 1)	255	High physical demands compared with low physical demands		HR 0.97 (95% CI 0.74 - 1.26)	age, sex, marital status, education, county, long-standing illness, pain symptoms, psychological symptoms, smoking, **physical work demands, psychological demands, demand for learning at work,** employment relationship
		High psychological demands compared with low psychological demands		HR 0.92 (95% CI 0.70 - 1.19)	
		High demand for learning at work compared with low demand for learning		HR 0.88 (95% CI 0.67 - 1.16)	
		Temporary unsatisfied /involuntary (job insecurity) compared with permanent employees (no job insecurity)	HR 2.10 (95% CI 1.23 - 3.59) (adjusted for sex, age, marital status, education, country)	HR 1.95 (95% CI 1.13 - 3.35)	
Natti 2009 (5) (Study 2)	82	High physical demands compared with low physical demands		HR 1.47 (95% CI 0.91 - 2.37)	age, sex, marital status, education, county, long-standing illness, pain symptoms, psychological symptoms, smoking, **physical work demands, psychological demands, demand for learning at work,** employment relationship
		High psychological demands compared with low psychological demands		HR 1.12 (95% CI 0.70 - 1.80)	
		High demand for learning at work compared with low demand for learning		HR 1.05 (95% CI 0.66 - 1.68)	
		Temporary unsatisfied /involuntary (job insecurity) compared with permanent employees (no job insecurity)	HR 2.62 (95% CI 1.19–5.80) (adjusted for sex, age, marital status, education, country)	HR 2.59 (95% CI 1.16 - 5.80)	
Natti 2012 (6)	326	Worked most often (on a weekly basis) at night compared with dayworkers who did not work at night	f: HR 2.10 (95% CI 1.13 -3.93) m: HR 1.11 (95% CI 0.70 - 1.75) (no adjustments)	f: HR 2.25 (95% CI 1.20 -4.20) m: HR 1.16 (95% CI 0.73 - 1.84)	age, family situation (among men), longstanding illness, smoking
Niedhammer 2011 (7)	291	Low social support at work compared with high social support at work	HR 2.63 (95% CI 2.08 - 3.31) *f: HR 2.72 (95% CI 1.76 - 4.20)* *m: HR 2.71 (95% CI 2.06 - 3.57)* (no adjustments)	HR 1.28 (95% CI 1.00 - 1.65) *f: HR 1.41 (95% CI 0.86 - 2.30)* *m: HR 1.23 (95% CI 0.91 - 1.66)*	age, sex, SES, smoking, alcohol abuse, BMI, biomechanical exposure, physical exposure, temporary contract, social support
Nilsen 2016 (8)	221	High job demands compared with low job demands		Self-reported: HR 1.28 (95% CI 0.97 - 1.70) *f: HR 1.55 (95% CI 1.00 - 2.38)* *m: HR 1.10 (95% CI 0.75 - 1.61)* JEM: HR 1.41 (95% CI 1.04 - 1.92)	age, sex, physical work environment, education, occupation-based social class

				f: HR 1.54 (95% CI 0.95 - 2.50) *m: HR 1.30 (95% CI 0.84 - 2.03)*	
		Low job control compared with high job control		Self-reported: HR 1.00 (95% CI 0.97 - 1.04) *f: HR 0.97 (95% CI 0.92 - 1.03)* *m: HR 1.04 (95% CI 0.99 - 1.09)* JEM: HR 1.07 (95% CI 0.73 - 1.57) *f: HR 1.17 (95% CI 0.61 - 2.25)* *m: HR 1.00 (95% CI 0.62 - 1.61)*	
		High job strain compared with no job strain (all other job strain categories combined)		Self-reported: HR 1.02 (95% CI 0.71 - 1.48) *f: HR 1.21 (95% CI 0.74 - 1.98)* *m: HR 0.83 (95% CI 0.46 - 1.50)* JEM: HR 1.41 (95% CI 1.01 - 1.96) *f: HR 1.54 (95% CI 0.94 - 2.52)* *m: HR 1.26 (95% CI 0.79 - 2.03)*	
O'Reily 2013 (9)	5590	Number of working hours per week (41-48, 49-54, >55 hours) compared with normal weekly working hours (35-40 hours)	f 41-48 hrs/wk: HR 0.93 (95% CI 0.75 - 1.14) f 49-54 hrs/wk: HR 1.03 (95% CI 0.78 - 1.38) f ≥55 hrs/wk: HR 0.80 (95% CI 0.59 - 1.08) m 41-48 hrs/wk: HR 0.92 (95% CI 0.85 - 1.00) m 49-54 hrs/wk: HR 0.91 (95% CI 0.82 - 1.01) m ≥55 hrs/wk: HR 0.88 (95% CI 0.80 - 0.96) (adjusted for age, marital status)	f 41-48 hrs/wk: HR 0.98 (95% CI 0.80 - 1.21) f 49-54 hrs/wk: HR 1.17 (95% CI 0.87 - 1.57) f ≥55 hrs/wk: HR 0.86 (95% CI 0.63 - 1.17) m 41-48 hrs/wk: HR 0.96 (95% CI 0.88 - 1.05) m 49-54 hrs/wk: HR 1.02 (95% CI 0.92 - 1.13) m ≥55 hrs/wk: HR 0.97 (95% CI 0.88 - 1.07)	age, marital status, SES, dependent children, caregiver, limiting long-term illness, general health
Shirom 2011 (10)	53	High job demands		HR 0.80 (95% CI 0.56 - 1.13)	age, sex, education, workload, control, supervisor support, peer support
		High job control		HR 1.28 (95% CI 0.89 - 1.88) *f: HR 1.70 (95% CI 1.07 - 2.71)* *m: HR 0.48 (95% CI 0.21 - 0.99)*	
		High supervisor support		HR 0.79 (95% CI 0.61 - 1.03)	
		High peer support		HR 0.72 (95% CI 0.55 - 0.94)	
Tobiasz-Adamczyk 2013 (11)	59	High psychological demands and low control compared with low psychological demands and high control	f: HR 2.73 (95% CI 0.35 -21.5) m: HR 2.18 (95% CI 0.50 - 9.55) (no adjustments)	f: HR 3.38 (95% CI 0.42 - 27.1) m: HR 2.33 (95% CI 0.53 - 10.3)	self-rated health, number of chronic conditions, independence in functional status, psychological well-being, depression, supervisor position, income
		High psychological efforts and low rewards compared with low psychological efforts and high rewards	f: HR 1.63 (95% CI 0.30 - 8.91) m: HR 1.27 (95% CI 0.29 - 5.58) (no adjustments)	f: HR 2.10 (95% CI 0.38 - 11.8) m: HR 1.53 (95% CI 0.34 - 6.88)	
Von Bonsdorff 2012 (12)	1836	High job demands compared with low job demands	f: HR 0.82 (95% CI 0.71 - 0.95) m: HR 1.10 (95% CI 0.98 - 1.23) (adjusted for age)	f: HR 0.82 (95% CI 0.71 - 0.95) m: HR 1.05 (95% CI 0.93 - 1.18)	age, occupation, smoking, alcohol, physical activity, CVD, metabolic disorders, cancer
		Low job control compared with high job control	f: HR 1.07 (95% CI 0.93 - 1.24) m: HR 1.26 (95% CI 1.12 - 1.42)	f: HR 1.06 (95% CI 0.91 - 1.23) m: HR 1.08 (95% CI 0.95 - 1.22)	

			(adjusted for age)		
		High job strain compared with low job strain	f: HR 0.88 (95% CI 0.72 - 1.08) m: HR 1.36 (95% CI 1.16 - 1.60) (adjusted for age)	f: HR 0.89 (95% CI 0.72 - 1.09) m: HR 1.14 (95% CI 0.96 - 1.35)	
Amick 2002 (13)	10-year employment lag: 726 5-year employment lag: 571	Low job demands (1th quartile) compared with high job demands (4th quartile)	10-year lag: HR 1.07 (95% CI 0.80 - 1.42) 5-year lag: HR 1.13 (95% CI 0.85 - 1.53) (adjusted for age, race, gender, current interview year)	10-year lag: HR 1.02 (95% CI 0.76 - 1.37) 5-year lag: HR 1.12 (95% CI 0.82 - 1.53)	age, race, gender, current interview year, family income, family size, retirement, unemployment, retirement by age interaction, race by age interaction, baseline disability
		Low job decision latitude (1st quartile) compared with high decision latitude (4th quartile)	10-year lag: HR 1.55 (95% CI 1.26 - 1.92) 5-year lag: HR 1.50 (95% CI 1.20 - 1.88) adjusted for age, race, gender, current interview year)	10-year lag: HR 1.43 (95% CI 1.13 - 1.81) 5-year lag: HR 1.50 (95% CI 1.18 - 1.91)	
		High job strain compared with low job strain	10-year lag: HR 1.42 (95% CI 1.10 - 1.84) 5-year lag: HR 1.36 (95% CI 1.03 - 1.80) adjusted for age, race, gender, current interview year)	10-year lag: HR 1.29 (95% CI not reported) 5-year lag: HR 1.33 (95% CI not reported)	
		Low work support (1st quartile) compared with high low work support (4th quartile)	10-year lag: HR 0.84 (95% CI 0.62 - 1.14) 5-year lag: HR 0.78 (95% CI 0.56 - 1.09) (adjusted for age, race, gender, current interview year)	10-year lag: HR 0.86 (95% CI 0.64 - 1.17) 5-year lag: HR 0.81 (95% CI 0.57 - 1.14)	
		Low job security (1st quartile) compared with high job security (4th quartile)	10-year lag: HR 0.90 (95% CI 0.67 - 1.20) 5-year lag: HR 0.95 (95% CI 0.70 - 1.30) (adjusted for age, race, gender, current interview year)	10-year lag: HR 0.98 (95% CI 0.73 - 1.32) 5-year lag: HR 0.98 (95% CI 0.70 - 1.35)	
Boggild 1999 (14)	1679	Shift worker (worked irregular hours, shift work, often had night work) compared with day worker (day time work only)	RR 1.10 (95% CI 0.90 - 1.20) (adjusted for age)	RR 0.90 (95% CI 0.80 - 1.10)	age, social class, sleep deviation, smoking, weight, height, fitness
Åkerstedt 2004 (15)	864	Shift worker (3-shift work, night work, evening work, roster work, other work) compared with day worker (day time work only)	**Blue Collar** f: HR 0.72 (95% CI 0.45 - 1.14) m: HR 0.86 (95% CI 0.68 - 1.01) **White Collar** f: HR 1.91 (95% CI 0.94 - 3.88) m: HR 0.95 (95% CI 0.58 - 1.56)	**Blue Collar** f: HR 0.79 (95% CI 0.50 - 1.26) m: HR 1.04 (95% CI 0.82 - 1.33) **White Collar** f: HR 2.61 (95% CI 1.26 - 5.41) m: HR 1.23 (95% CI 0.75 - 1.26)	age, stress, physically strenuous work, smoking, long-term disease
Sabbath 2015 (16)	933	Low job demands (1st tertile) compared with high job demands (3rd tertile)		HR 1.07 (95% CI 0.90 - 1.26)	age at HRS baseline, birth cohort, year of entry into HRS, race/ethnicity, region of birth in the US, parental education, own education, family status
		Low job control (1st tertile) compared with high job control (3rd tertile)		HR 1.31 (95% CI 1.10 - 1.56)	

Perlman 2009 (17)	285	Job insecure compared with job secure	f: HR 1.31 (95% CI 0.66 - 2.58) m: HR 1.08 (95% CI 0.83 - 1.40) (adjusted for age)	f: HR 1.15 (95% CI 0.58 - 2.30) m: HR 0.99 (95% CI 0.75 - 1.29)	age, education, occupation, alcohol, smoking, material goods
CHD mortality outcome (n=14)					
Johnson 1989 (18)	193	High iso-strain (5th quintile) compared with low iso-strain (1st quintile)	RR 1.92 (95% CI 1.15 - 3.21) (adjusted for age)		
Kivimaki 2006 (19)	67	High job demands		HR 1.13 (95% CI 0.86 - 1.47)	age, sex, education, occupational status, salary, second job, shift work, cholesterol, SBP, BMI, smoking, physical activity, alcohol, depression, lack of energy, fatigue, **job control**, **job demands**, insufficient recovery from work
		High job control		HR 0.69 (95% CI 0.51 - 0.93)	
Kivimaki 2002 (20)	73	High job demands (3rd tertile) compared with low job demands (1st tertile)	HR 1.35 (95% CI 0.77-2.36) (adjusted for age and sex)		age, sex, occupation group, smoking, physical activity, SBP, cholesterol, BMI
		Low job control (1st tertile) compared with high job control (3rd tertile)	HR 1.90 (95% CI 1.08-3.37) (adjusted for age and sex)	HR 1.42 (95% CI 0.72-2.82)	
		High job strain compared with low job strain	HR 2.20 (95% CI 1.16-4.17) (adjusted for age and sex)	HR 2.22 (95% CI 1.04-4.73)	
		High effort reward imbalance (3rd tertile) compared with low effort reward (1st tertile)	HR 2.36 (95% CI 1.26-4.42) (adjusted for age and sex)	HR 2.42 (95% CI 1.02-5.73)	
Elovainio 2006 (21)	73	High justice compared with low/intermediate justice	HR 0.55 (95% CI 0.34-0.88) (adjusted for age and sex)	HR 0.61 (95% CI 0.36-1.00)	age, sex, occupational group, smoking, physical activity, SBP, cholesterol, BMI, job strain, effort–reward imbalance
Lee 2002 (22)	38	High job strain compared with low job strain	RR 1.09 (95% CI 0.40 - 2.92) (adjusted for age)		
Slopen 2012 (23)	52	High job strain compared with low job strain	HR 1.07 (95% CI 0.45 - 2.55) (adjusted for age, race, study drug randomisation)	HR 0.84 (95% CI 0.35 - 2.06)	age, race, study drug randomisation, education, income, coronary revascularisation
		Job insecure compared with job secure	HR 1.52 (95% CI 0.81 - 2.85)	HR 1.41 (95% CI 0.75 - 2.65)	

			(adjusted for age, race, study drug randomisation)		
Toivanen 2006 (24)	10916	Low job control compared with high job control	RR 1.32 (95% CI 1.27 - 1.38) *f: RR 1.28 (95% CI 1.18 - 1.40)* *m: RR 1.34 (95% CI 1.28 -1.40)* (adjusted for age)	RR 1.15 (95% CI 1.10 - 1.19); *f: RR 1.20 (95% CI 1.10 - 1.31)* *m: RR 1.14 (95% CI 1.08 -1.19)*	age, sex, income
		Low level of job control index compared to high level of job control index	RR 1.52 (95% CI 1.44 - 1.61) *f: RR 1.60 (95% CI 1.35 - 1.89)* *m: RR 1.49 (95% CI 1.40 -1.59)* (adjusted for age)	RR 1.24 (95% CI 1.17 - 1.31) *f: RR 1.40 (95% CI 1.18 - 1.67)* *m: RR 1.21 (95% CI 1.13 -1.30)*	
Alterman 1994 (25)	283	Decision latitude (RR decision latitude calculated for 20-point change in scale score)	RR 0.76 (95% CI 0.59 - 0.97) (adjusted for age)	RR 0.85 (95% CI 0.70 - 1.03)	age, SBP, cholesterol, smoking, alcohol, family history CVD, education, occupational class
		Psychologic demand (RR for psychologic demand calculated were calculated for 10-point change in scale score)	RR 0.79 (95% CI 0.48 - 1.28) (adjusted for age)	RR 0.76 (95% CI 0.55 - 1.05)	
		High job strain (scores for decision latitude in lowest category and psychologic demand in highest category) compared to no job strain	RR 1.48 (95% CI 0.98 - 2.24) (adjusted for age)	RR 1.03 (95% CI 0.75 - 1.41)	
Padyab 2014 (26)	595	High psychological demands compared with low psychological demands	f: HR 0.58 (95% CI 0.39 - 0.86) m: HR 0.73 (95% CI 0.59 - 0.89) (adjusted for age)	f: HR 0.75 (95% CI 0.47 - 1.19) m: HR 0.81 (95% CI 0.64 - 1.03)	age, work-stress (**psychological demands**, **decision latitude**), non-work stress (emotional support and social network), BMI, alcohol, physical activity, marital status, education, smoking
		Low decision latitude compared with high decision latitude	f: HR 1.21 (95% CI 0.83 - 1.76) m: HR 1.37 (95% CI 1.12 - 1.67) (adjusted for age)	f: HR 0.91 (95% CI 0.58 - 1.43) m: HR 1.07 (95% CI 0.85 - 1.36)	
		High job strain compared with low job strain	f: HR 0.66 (95% CI 0.34 - 1.33) m: HR 0.87 (95% CI 0.61 - 1.24) (adjusted for age)		
		Iso-strain (low social support and high job strain) compared with no iso-strain	f: HR 0.51 (95% CI 0.21 - 1.26) m: HR 1.18 (95% CI 0.81 - 1.72) (adjusted for age)		
Hublin 2010 (27)	857	Type of working time (night-time work 1975 or 1981, shift-work 1975 and daytime work 1981, daytime work 1975 and shift work 1981, shift-work 1975 and	**Night-time work 1975 or 1981** f: HR: 1.38 (95% CI 0.71, 2.69) m: HR 1.75 (95% CI 0.92, 3.33) **Shift-work 1975, daytime work 1981** f: HR 1.16 (95% CI 0.66, 2.04)	**Night-time work 1975 or 1981** f: HR 0.90 (95% CI 0.36, 2.23) m: HR 1.82 (95% CI 0.97, 3.41) *healthy f: not computable* *healthy m: HR 1.17 (95% CI 0.35, 3.90)*	age, marital status, social class, education, smoking, binge drinking, grams of alcohol consumed daily, hypertension, BMI, conditioning physical

		1981 compared with daytime work 1975 and 1981)	m: HR 1.09 (95% CI 0.68, 1.76) **Daytime work 1975, shift work 1981** f: HR 1.55 (95% CI 0.88, 2.74) m: HR 0.94 (95% CI 0.56, 1.56) **Shift-work 1975 and 1981** f: HR 1.22 (95% CI 0.83, 1.79) m: HR 1.09 (95% CI 0.82, 1.44) (adjusted for age)	**Shift-work 1975, daytime work 1981** f: HR 1.08 (95% CI 0.56, 2.08) m: HR 0.86 (95% CI 0.48, 1.54) *healthy f: HR 0.67 (95% CI 0.09, 4.96)* *healthy m: HR 0.14 (95% CI 0.02, 1.05)* **Daytime work 1975, shift work 1981** f: HR 1.52 (95% CI 0.82, 2.82) m: HR 0.79 (95% CI 0.44, 1.41) *healthy f: HR 2.16 (95% CI 0.62, 7.55)* *healthy m: HR 0.91 (95% CI 0.39, 2.12)* **Shift-work 1975 and 1981** f: HR 1.21 (95% CI 0.75, 1.93) m: HR 1.06 (95% CI 0.75, 1.50) *healthy f: HR 1.02 (95% CI 0.27, 3.95)* *healthy m: HR 0.77 (95% CI 0.42, 1.39)*	activity, life satisfaction, diurnal type, sleep length, use of hypnotics and/or tranquillizers, physical load of work, working pace
Johnson 1996 (28)	521	High psychosocial demands (4th quartile) compared with low psychological demands (1st quartile)		**5-year cumulative exposure period** RR 0.76 (95% CI 0.52 - 1.13) **26+ years cumulative exposure period** RR 0.55 (95% CI 0.34 - 0.88)	age, survey year, year last worked, social class, education, smoking, physical exercise, nationality
		Low job control (1st quartile) compared with high job control (4th quartile)		**5-year cumulative exposure period** RR 1.46 (95% CI 0.95 - 2.25) **26+ years cumulative exposure period** RR 1.09 (95% CI 0.68 - 1.74)	
		Low social support (1st quartile) compared with high social support (4th quartile)		**5-year cumulative exposure period** RR 0.96 (95% CI 0.68 - 1.37) **26+ years cumulative exposure period** RR 1.06 (95% CI 0.74 - 1.51)	
		Combined low, medium low, medium high job control compared with high job control		**5-year cumulative exposure period** RR 1.60 (95% CI 1.06 - 2.41) **26+ years cumulative exposure period** RR 1.10 (95% CI 0.67 - 1.80)	age, survey year, year last worked, social class, education, smoking, physical exercise, nationality, psychological demands, job control, social support, hazards, physical demands
		Combined medium low, medium high, high psychological demands compared with low psychological demands		**5-year cumulative exposure period** RR 0.95 (95% CI 0.71 - 1.24) **26+ years cumulative exposure period** RR 0.76 (95% CI 0.47 - 1.02)	
		Combined low, medium low, medium high social support compared with high social support		**5-year cumulative exposure period** RR 1.00 (95% CI 0.75 - 1.34) **26+ years cumulative exposure period** RR 1.10 (95% CI 0.79 - 1.53)	

Karasek 1981 (29)	22	Low/medium low intellectual discretion compared with high/medium high intellectual discretion	OR 1.5 (95% CI 0.4 - 5.1) (cases matched with controls for age, smoking, education, CHD symptoms)		
		Low/medium low personal schedule freedom compared with high/medium high personal schedule freedom	OR 1.7 (95% CI 0.6 - 4.7) (cases matched with controls for age, smoking, education, CHD symptoms)		
		High job demands compared with medium or low job demands	OR 4.0 (95% CI 1.2 - 13.9) (cases matched with controls for age, smoking, education, CHD symptoms)		
		Low personal schedule freedom and high job demands	OR 4.0 (95% CI 1.1 - 14.4) (cases matched with controls for age, smoking, education, CHD symptoms)		
Yadegarfar 2008 (30)	635	Shift worker (shift work > 1 month) compared with day workers (all other workers)	OR 1.09 (90% CI 0.91 - 1.32) (cases matched with controls for age, year of starting work)	**Total cohort** OR 1.03 (90% CI 0.83 - 1.28) ***Surviving at least 10 years after hire*** *OR 1.04 (90% CI 0.83 - 1.30)*	SBP, DBP, BMI, smoking, height, duration of employment, social class
McNamee 1996 (31)	467	Shift worker (shift work > 1 month) compared with day workers (all other workers)	**Total cohort** OR 0.79 (90% CI 0.62 - 1.01) ***Deaths <60 years of age*** *OR 0.72 (90% CI 0.49 - 1.04)* ***Deaths ≥60 years of age*** *OR 0.85 (90% CI 0.58 - 1.26)* (cases matched with controls for age, year of starting work)	**Total cohort** OR 0.85 (90% CI 0.65 - 1.12) ***Surviving at least 10 years after first shift*** *OR 0.90 (90% CI 0.68 - 1.21)*	SBP, DBP, BMI, smoking, height, job status, duration of employment
All-cause and CHD mortality outcomes (n=14)					
Brunner 2004 (32)	Total: 180 CHD: 73	High job strain compared with low job strain		**Total mortality** HR 0.95 (95% CI 0.6 - 1.5)	age, sex, height, father's occupational group, education, occupational group, income
			CHD mortality HR 2.20 (95% CI 1.2 - 4.2) (adjusted for age, sex)	**CHD mortality** HR 2.04 (95% CI 1.0 - 4.3)	
				Total mortality HR 1.08 (95% CI 0.7 - 1.8)	

		High effort reward imbalance compared with low effort-reward imbalance	**CHD mortality** HR 2.36 (95% CI 1.3 - 4.4) (adjusted for age, sex)	**CHD mortality** HR 2.54 (95% CI 1.1 - 5.9)	
Laszlo 2010 (33)	Total: 96 CHD: 52	High job demands (4th quartile) compared with low job demands (1st quartile)		**Total mortality** HR 1.13 (95% CI 0.62 - 2.08)	age, sex, education, occupational class, managerial status, overtime work, shift work, household work, household work by age interaction
				CHD mortality HR 1.31 (95% CI 0.59 - 2.89)	
		Low job control (1st quartile) compared with high job control (4th quartile)		**Total mortality** HR 1.20 (95% CI 0.63 - 2.30)	
				CHD mortality HR 1.35 (95% CI 0.51 - 3.56)	
		High job strain compared with low job strain	**Total mortality** HR 1.38 (95% CI 0.80 - 2.39) (no adjustments)	**Total mortality** HR 1.65 (95% CI 0.91 - 2.98)	
			CHD mortality HR 2.20 (95% CI 0.99 - 4.90) (no adjustments)	**CHD mortality** HR 2.81 (95% CI 1.16 - 6.82)	
Laszlo 2013 (34)	Total: 96 CHD: 52	job insecurity (concerns about dismissal from work) compared with no job insecurity	**Total mortality** HR 1.57 (95% CI 0.99 - 2.49) (no adjustments)	**Total mortality** HR 1.69 (95% CI 1.04 - 2.75)	age, sex, education, occupational class, employment type, managerial status, experience of unemployment, dismissal or unsuccessful job searching, marital status, diabetes mellitus, earlier hospitalisation for depression, history of chest pain, previous stroke or heart failure
			CHD mortality HR 1.52 (95% CI 0.81 - 2.85) (no adjustments)	**CHD mortality** HR 1.57 (95% CI 0.80 - 3.09)	
Holtermann 2010 (35)	Total: 2675 CHD: 587	Weekly number of work hours (41-45, and ≥46 compared with ≤40 hours per week)	**Total mortality** 41-45 hrs/wk: HR 1.07 (95% CI 0.95 - 1.20) ≥46 hrs/wk: HR 0.91 (95% CI 0.79 - 1.05) (adjusted for age)	**Total mortality** **Lowest physical fitness 41-45 hrs/wk** HR 1.00 (95% CI 0.76 - 1.30) **Lowest physical fitness ≥46 hrs/wk** HR 1.08 (95% CI 0.79 - 1.49) **Medium physical fitness 41-45 hrs/wk** HR 0.93 (95% CI 0.79 - 1.09) **Medium physical fitness ≥46 hrs/wk**	BMI, systolic blood pressure, diastolic blood pressure, diabetes (treatment for), hypertension (treatment of), alcohol use, smoking (current, never, previous), physical work

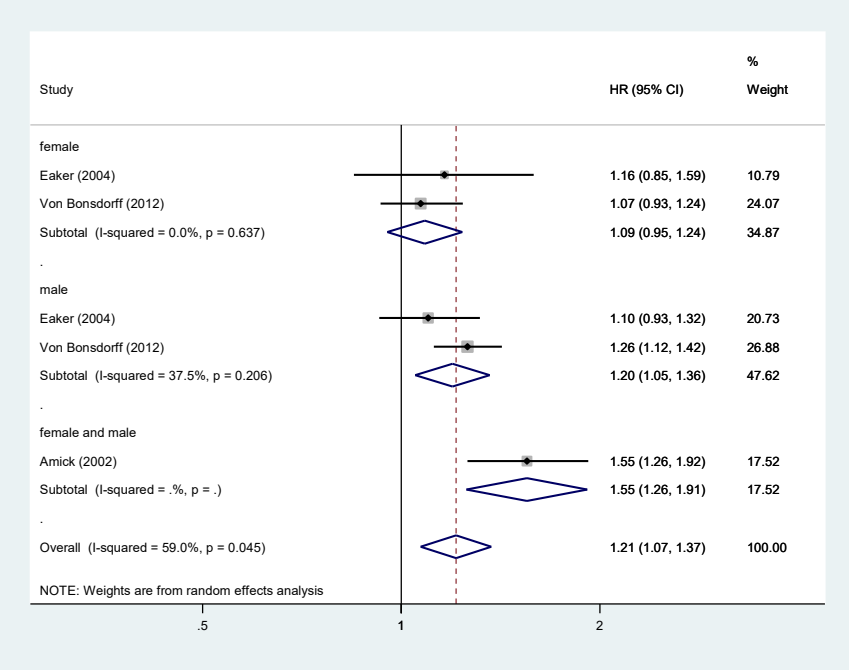
				HR 0.79 (95% CI 0.69 - 0.95) **Highest physical fitness 41-45 hrs/wk** HR 0.85 (95% CI 0.64 - 1.14) **Highest physical fitness ≥46 hrs/wk** HR 0.87 (95% CI 0.61 - 1.23)	
			CHD mortality 41-45 hrs/wk: HR 1.59 (95% CI 1.20 - 2.11) ≥46 hrs/wk: HR 1.28 (95% CI 0.91 - 1.78) (adjusted for age)	**CHD mortality** **Lowest physical fitness 41-45 hrs/wk** HR 1.49 (95% CI 0.76 - 2.89) **Lowest physical fitness ≥46 hrs/wk** HR 2.28 (95% CI 1.10 - 4.73) **Medium physical fitness 41-45 hrs/wk** HR 1.37 (95% CI 0.93 - 2.03) **Medium physical fitness ≥46 hrs/wk** HR 0.94 (95% CI 0.59 - 1.51) **Highest physical fitness 41-45 hrs/wk** HR 0.80 (95% CI 0.41 - 1.57) **Highest physical fitness ≥46 hrs/wk** HR 0.91 (95% CI 0.41 - 2.02)	
Holtermann 2011 (36)	Total: 2656 CHD: 585	Regular psychological pressure at work compared with rare psychological pressure at work	**Total mortality** HR 0.90 (95% CI 0.82 - 0.99) (adjusted for age)	**Total mortality** HR 0.98 (95% CI 0.89 - 1.07)	age, social class, work hours, physical work demands
			CHD mortality HR 0.87 (95% CI 0.71 - 1.07) (adjusted for age)	**CHD mortality** HR 0.94 (95% CI 0.75 - 1.17)	
Joensuu 2012 (37)	Total: 981 CHD: 375	High skill discretion (3rd tertile) compared with low skill discretion (1st tertile)	**Total mortality** HR 1.15 (95% CI 0.99 - 1.35) (no adjustments)	**Total mortality** HR 0.84 (95% CI 0.69 - 1.02)	both job control variables, age, sex, occupational status, physical health (hypertension, diabetes), supervisor position, supervisor support, co-worker support
			CHD mortality HR 1.22 (95% CI 0.94 - 1.58) (no adjustments)	**CHD mortality** HR 0.80 (95% CI 0.58 - 1.09)	
		High decision authority (3rd tertile) compared with low decision authority (1st tertile)	**Total mortality** HR 1.29 (95% CI 1.11 - 1.51) (no adjustments)	**Total mortality** HR 1.28 (95% CI 1.06 - 1.54)	
			CHD mortality HR 1.47 (95% CI 1.14 - 1.90) (no adjustments)	**CHD mortality** HR 1.49 (95% CI 1.11 - 2.02)	

Joensuu 2014 (38)	Total: 696 CHD: 128	High skill discretion (3rd tertile) compared with low skill discretion (1st tertile)	**Total mortality** HR 0.66 (95% CI 0.55 - 0.80) (no adjustments)	**Total mortality** HR 0.86 (95% CI 0.68 - 1.08)	both job control variables, age, sex, SES, physical health (hypertension, diabetes)
			CHD mortality HR 0.56 (95% CI 0.36 - 0.87) (no adjustments)	**CHD mortality** HR 0.80 (95% CI 0.47 - 1.36)	
		High decision authority (3rd tertile) compared with low decision authority (1st tertile)	**Total mortality** HR 0.85 (95% CI 0.69 - 1.04) (no adjustments)	**Total mortality** HR 0.93 (95% CI 0.74 - 1.17)	
			CHD mortality HR 0.76 (95% CI 0.47 - 1.23) (no adjustments)	**CHD mortality** HR 0.86 (95% CI 0.50 - 1.48)	
Tsutsumi 2006 (39)	Total: 221 CHD: 35	High job demands (4th quartile) compared with low job demands (1st quartile)		**Total mortality** f: RR 0.79 (95% CI 0.35 - 1.74) m: RR 0.76 (95% CI 0.48 - 1.19)	age, educational, occupation, smoking, alcohol, physical activity, BMI, cholesterol, hypertension, diabetes, community
		Low job control (1st quartile) compared with high job control (4th quartile)		**Total mortality** f: RR 0.82 (95% CI 0.33 - 2.04) m: RR 1.11 (95% CI 0.61 - 2.01)	
		High job strain compared with low job strain	**Total mortality** f: RR 0.76 (95% CI 0.38 - 1.51) m: RR 0.79 (95% CI 0.50 - 1.26) (adjusted for age)	**Total mortality** f: RR 0.72 (95% CI 0.32 - 1.64) m: HR 0.79 (95% CI 0.47 - 1.31)	
			CHD mortality RR 2.47 (95% CI 0.81 - 7.51) (adjusted for age and sex)	**CHD mortality** RR 1.98 (95% CI 0.59 - 6.70)	age, sex, educational, occupation, smoking, alcohol, physical activity, BMI, cholesterol, hypertension, diabetes, community
Yong 2014 (40)	Total: 1062 CHD: 122	Rotating shift work (rotating shift work > 1 year) compared with day workers (all other workers)	**Total mortality** HR 0.96 (95% CI 0.84 - 1.10) (adjusted for age and manual work)	**Total mortality** HR 0.73 (95% CI 0.62 - 0.85)	age, manual work, smoking, alcohol, job duration, cancer, epilepsy, cerebral vascular disease, diseases of the liver, diabetes, IHD, COPD, conductive heart disorders, hypertensive diseases
			CHD mortality HR 0.77 (95% CI 0.52 - 1.14) (adjusted for age and manual work)	**CHD mortality** HR 0.62 (95% CI 0.39 - 0.99)	age, manual work, smoking, job duration, BMI, diseases of the liver, diabetes, hypertensive diseases
Kivimaki 2003 (41)	Total: 1332			**Total mortality**	age, occupational status, salary

	CHD: 300	Employment status (temporary to permanent, temporary, unemployed workers compared with permanent employees)		**temporary to permanent** f: HR 0.63 (95% CI 0.44 - 0.89) m: HR 0.87 (95% CI 0.53 - 1.44) **temporary** f: HR 1.24 (95% CI 1.01 - 1.54) m: HR 1.61 (95% CI 1.25 - 2.09) **unemployed** f: HR 2.91 (95% CI 2.16 - 3.91) m: HR 2.81 (95% CI 2.18 - 3.64)	
				CHD mortality **temporary to permanent** f: HR 0.35 (95% CI 0.11 - 1.11) m: HR 1.10 (95% CI 0.45 - 2.72) **temporary** f: HR 1.42 (95% CI 0.86 - 2.35) m: HR 0.99 (95% CI 0.55 - 1.80) **unemployed** f: HR 4.23 (95% CI 2.29 - 7.81) m: HR 2.36 (95% CI 1.43 - 3.89)	
Jorgensen 2017 (42)	Total: 1616 CHD: 217	Shift work (evening, night, rotating shifts compared with day shift)	**Total mortality** evening shifts: HR 1.53 (95% CI 1.33 - 1.77) night shifts: HR 1.74 (95 % CI 1.48 - 2.07) rotating shifts: HR 0.98 (95% CI 0.86 - 1.12) (adjusted for age)	**Total mortality** evening shifts: HR 1.29 (95% CI 1.11 - 1.49) night shifts: HR 1.26 (95 % CI 1.05 - 1.51) rotating shifts: HR 1.00 (95% CI 0.88 - 1.15)	age, smoking, physical activity, BMI, alcohol, diet, pre-existing diseases, self-reported health, stressful work environment, marital status, female reproductive factors (birth, use of hormone therapy, oral contraceptives)
			CHD mortality evening shifts: HR 1.74 (95% CI 1.18 - 2.57) night shifts: HR 2.42 (95 % CI 1.58 - 3.71) rotating shifts: HR 1.21 (95% CI 0.86 - 1.72) (adjusted for age)	**CHD mortality** evening shifts: HR 1.47 (95% CI 0.98 - 2.18) night shifts: HR 1.71 (95 % CI 1.09 - 2.69) rotating shifts: HR 1.24 (95% CI 0.87 - 1.77)	
Gu 2015 (43)	Total: 14181 CHD: 3062	Night shift work duration (1-5, 6-14, ≥15 years compared with never)	**Total mortality** 1-5 years: HR 0.97 (95% CI 0.94 - 1.01) 6-14 years: HR 1.19 (95% CI 1.13 - 1.25) ≥15 years: HR 1.24 (95% CI 1.17 - 1.32) (adjusted for age)	**Total mortality** 1-5 years: HR 1.01 (95% CI 0.97 - 1.05) 6-14 years: HR 1.11 (95% CI 1.06 - 1.17) ≥15 years: HR 1.11 (95% CI 1.05 - 1.18)	age, alcohol, physical exercise, multivitamins, menopausal status, postmenopausal hormones, physical exam in the past 2 years, healthy eating score, smoking, pack years, BMI, husband's education
			CHD mortality 1-5 years: HR 0.97 (95% CI 0.90 - 1.06) 6-14 years: HR 1.30 (95% CI 1.16 - 1.45) ≥15 years: HR 1.45 (95% CI 1.29 - 1.63) (adjusted for age)	**CHD mortality** 1-5 years: HR 1.02 (95% CI 0.94 - 1.11) 6-14 years: HR 1.19 (95% CI 1.07 - 1.33) ≥15 years: HR 1.23 (95% CI 1.09 - 1.38)	
Karlsson 2005 (44)	Total: 760		**Total mortality**		

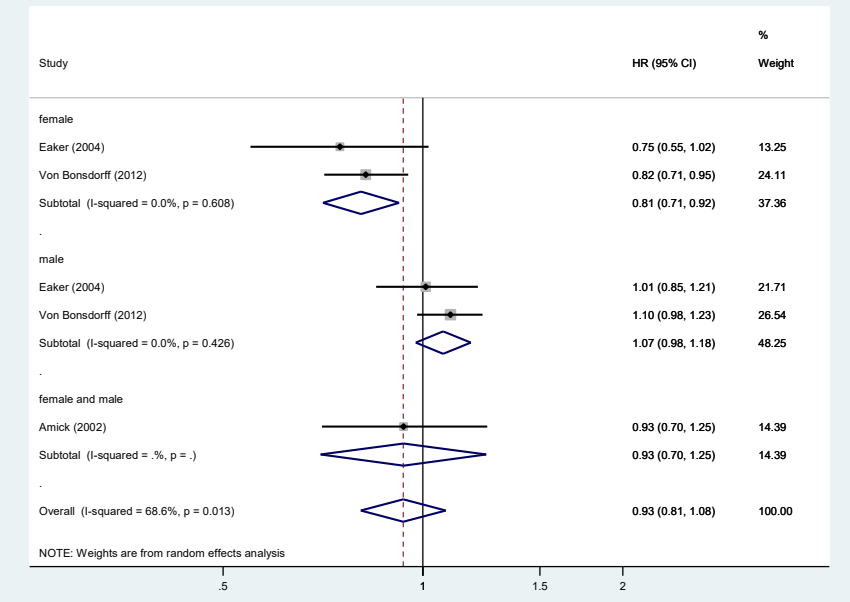
	CHD: 287	Total duration of years shift work (<5, 5-9, 10-19, 20-29, ≥30 years compared with dayworkers)	All shiftworkers: SRR 1.02 (95% CI 0.93 - 1.11) *<5 years: SRR 0.99 (95% CI 0.65 - 1.50)* *5-9 years: SRR 0.94 (95% CI 0.65 - 1.36)* *10-19 years: SRR 0.93 (95% CI 0.76 - 1.14)* *20-29 years: SRR 1.06 (95% CI 0.90 - 1.25)* *≥30 years: SRR 0.98 (95% CI 0.88 - 1.10)* (SRR calculated based on weights derived from unexposed group adjusted for age and calendar time)		
			CHD mortality All shiftworkers: SRR 1.11 (95% CI 0.95 - 1.30) *<5 years: SRR 0.85 (95% CI 0.30 - 2.38)* *5-9 years: SRR 0.97 (95% CI 0.56 - 1.67)* *10-19 years: SRR 0.83 (95% CI 0.58 - 1.19)* *20-29 years: SRR 1.02 (95% CI 0.77 - 1.36)* *≥30 years: SRR 1.24 (95% CI 1.04 - 1.49)* (SRR calculated based on weights derived from unexposed group adjusted for age and calendar time)		
Fujino 2006 (45)	Total: 1363 CHD: 304	Shift work (fixed-night, rotating-shift workers compared with daytime workers)	**Total mortality** fixed-night: HR 1.13 (95% CI 0.90 - 1.42) rotating-shift: HR 1.00 (95% CI 0.84 - 1.19) (adjusted for age)	**Total mortality** fixed-night: HR 1.06 95% CI (0.85 - 1.34) rotating-shift: HR 0.98 95% CI (0.82 - 1.17)	age, smoking, alcohol, education, perceived stress, past medical history, BMI, hours of walking, hours of exercise, job type
			CHD mortality fixed-night: HR 1.41 (95% CI 0.90 - 2.21) rotating-shift: HR 1.62 (95% CI 1.19 - 2.21) (adjusted for age)	**CHD mortality** fixed-night: HR 1.29 (95% CI 0.82 - 2.03) rotating-shift: HR 1.59 (95% CI 1.16 - 2.18)	

Figure S1: Forest plot of the effect of low job control compared to high job control on all-cause mortality minimal analysis



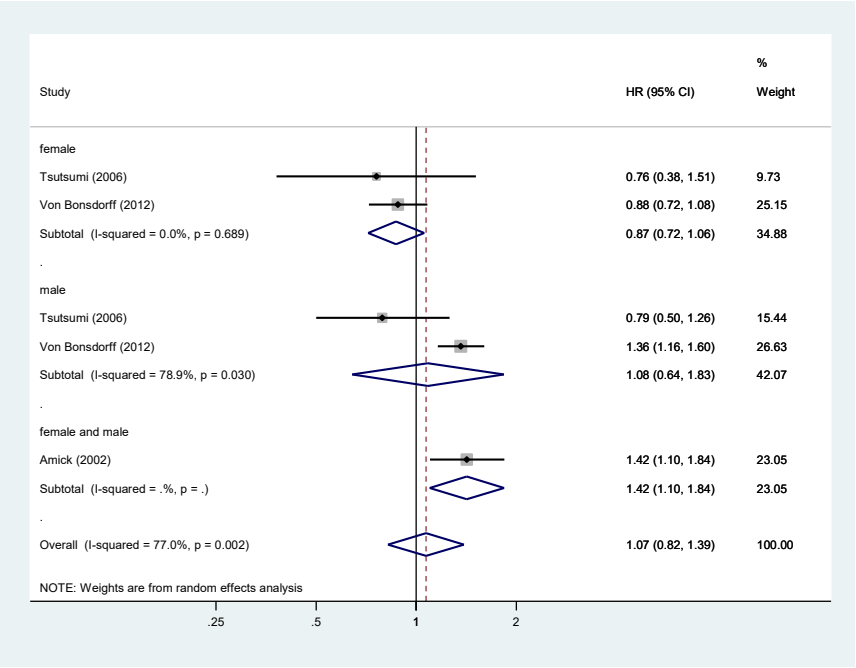
female Heterogeneity: $Tau^2 = 0.00$; test statistic = 0.22, df = 1 (p = 0.637); $I^2 = 0.0\%$; Test for overall effect: z = 1.23 (p = 0.218)
male Heterogeneity: $Tau^2 = 0.004$; test statistic = 1.60, df = 1 (p = 0.206); $I^2 = 37.5\%$; Test for overall effect: z = 2.69 (p = 0.007)
Total Heterogeneity: $Tau^2 = 0.01$; test statistic = 9.76, df = 4 (p = 0.045); $I^2 = 59.0\%$; Test for overall effect: z = 3.03 (p = 0.002)

Figure S2: Forest plot of the effect of high job demands compared to low job demands on all-cause mortality minimal analysis



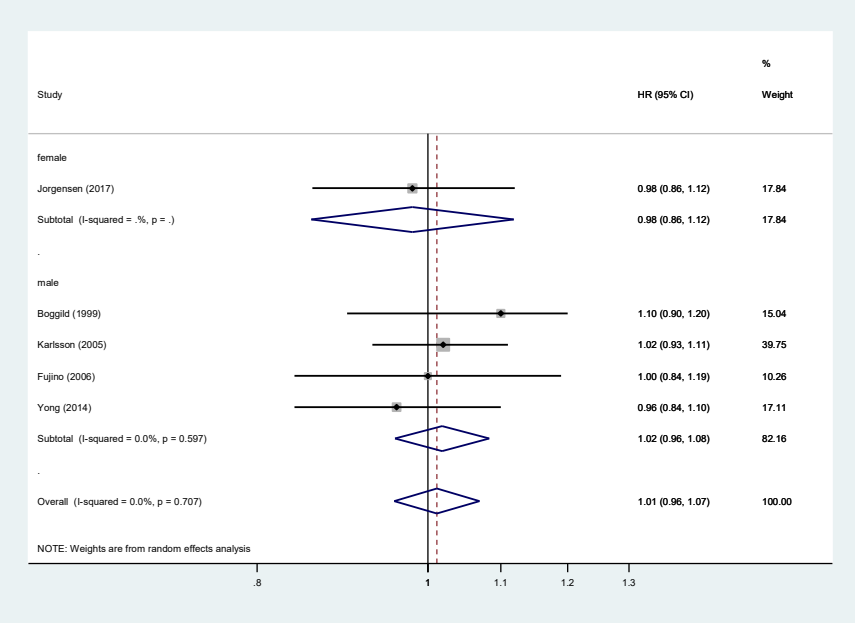
female Heterogeneity: $Tau^2 = 0.00$; test statistic = 0.26, df = 1 (p = 0.608); $I^2 = 0.0\%$; Test for overall effect: z = 3.19 (p = 0.001)
male Heterogeneity: $Tau^2 = 0.00$; test statistic = 0.63, df = 1 (p = 0.426); $I^2 = 0.0\%$; Test for overall effect: z = 1.44 (p = 0.149)
Total Heterogeneity: $Tau^2 = 0.02$; test statistic = 12.8, df = 4 (p = 0.013); $I^2 = 68.6\%$; Test for overall effect: z = 0.91 (p = 0.365)

Figure S3: Forest plot of the effect of job strain compared to no job strain on all-cause mortality minimal analysis



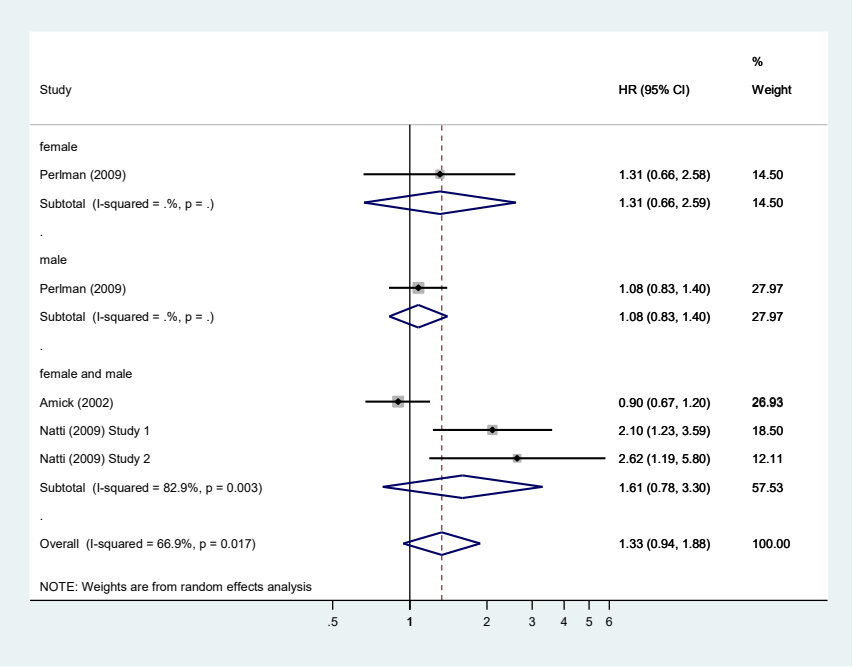
female Heterogeneity: Tau^2 = 0.00; test statistic = 0.16, df = 1 (p = 0.689); I^2 = 0.0%; Test for overall effect: z = 1.41 (p = 0.160)
male Heterogeneity: Tau^2 = 0.12; test statistic = 4.73, df = 1 (p = 0.030); I^2 = 78.9%; Test for overall effect: z = 0.30 (p = 0.763)
Total Heterogeneity: Tau^2 = 0.06; test statistic = 17.4, df = 4 (p = 0.002); I^2 = 77.0%; Test for overall effect: z = 0.50 (p = 0.615)

Figure S4: Forest plot of the effect of shift workers compared to day workers on all-cause mortality minimal analysis



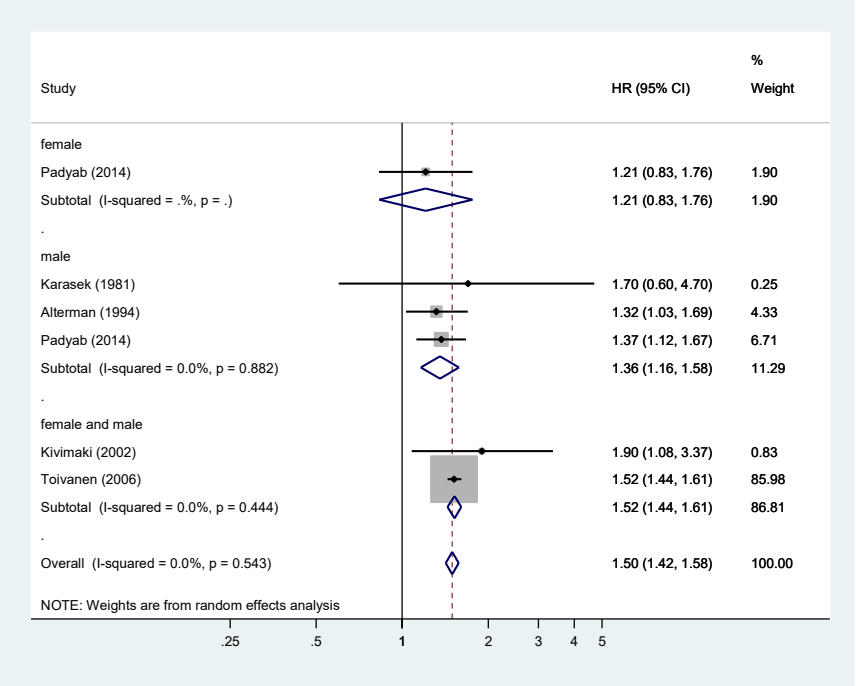
male Heterogeneity: Tau^2 = 0.00; test statistic = 1.88, df = 3 (p = 0.597); I^2 = 0.0%; Test for overall effect: z = 0.59 (p = 0.555)
Total Heterogeneity: Tau^2 = 0.00; test statistic = 2.15, df = 4 (p = 0.707); I^2 = 0.0%; Test for overall effect: z = 0.41 (p = 0.683)

Figure S5: Forest plot of the effect of job insecurity compared to no job insecurity on all-cause mortality minimal analysis



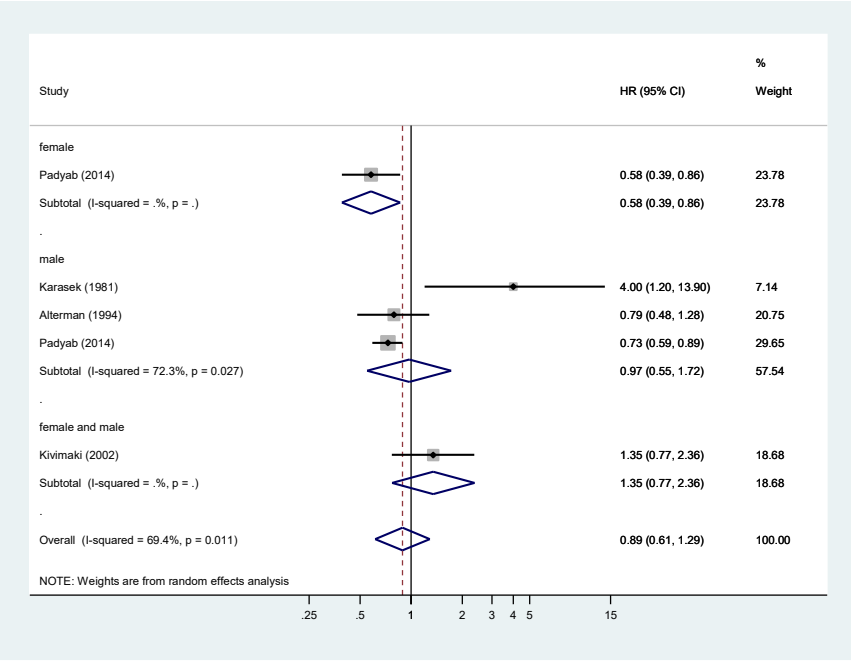
female and male Heterogeneity: $Tau^2 = 0.32$; test statistic = 11.66, df = 2 (p = 0.003); $I^2 = 82.9\%$; Test for overall effect: z = 1.29 (p = 0.196)
Total Heterogeneity: $Tau^2 = 0.09$; test statistic = 12.1, df = 4 (p = 0.017); $I^2 = 66.9\%$; Test for overall effect: z = 1.62 (p = 0.104)

Figure S6: Forest plot of the effect of low job control compared to high job control on CHD mortality minimal analysis



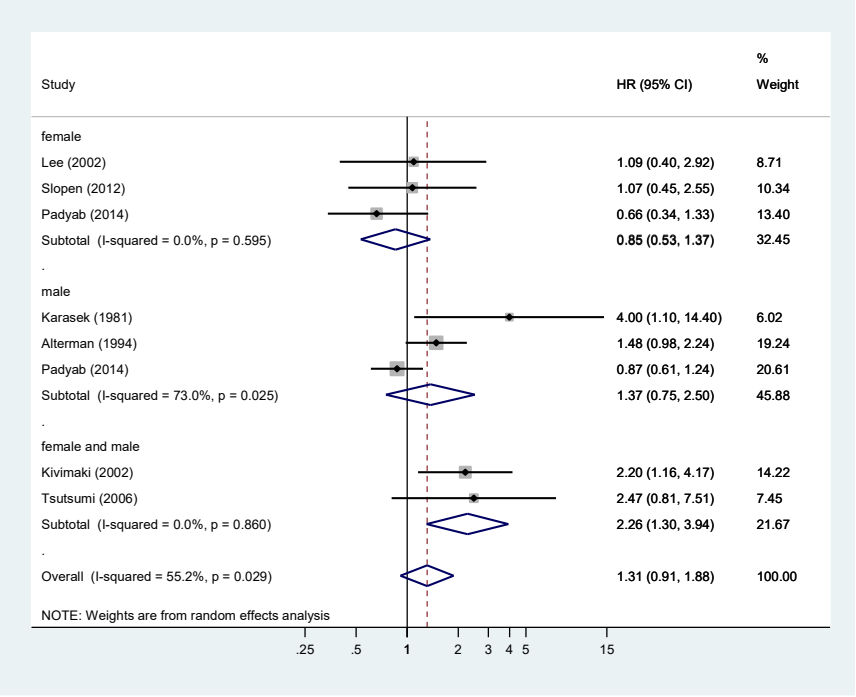
male Heterogeneity: $Tau^2 = 0.00$; test statistic = 0.25, df = 2 (p = 0.882); $I^2 = 0.0\%$; Test for overall effect: z = 3.87 (p < 0.001)
female and male Heterogeneity: $Tau^2 = 0.00$; test statistic = 0.59, df = 1 (p = 0.444); $I^2 = 0.0\%$; Test for overall effect: z = 14.9 (p < 0.001)
Total Heterogeneity: $Tau^2 = 0.00$; test statistic = 4.04, df = 5 (p = 0.543); $I^2 = 0.0\%$; Test for overall effect: z = 15.3 (p < 0.001)

Figure S7: Forest plot of the effect of high job demands compared to low job demands on CHD mortality minimal analysis



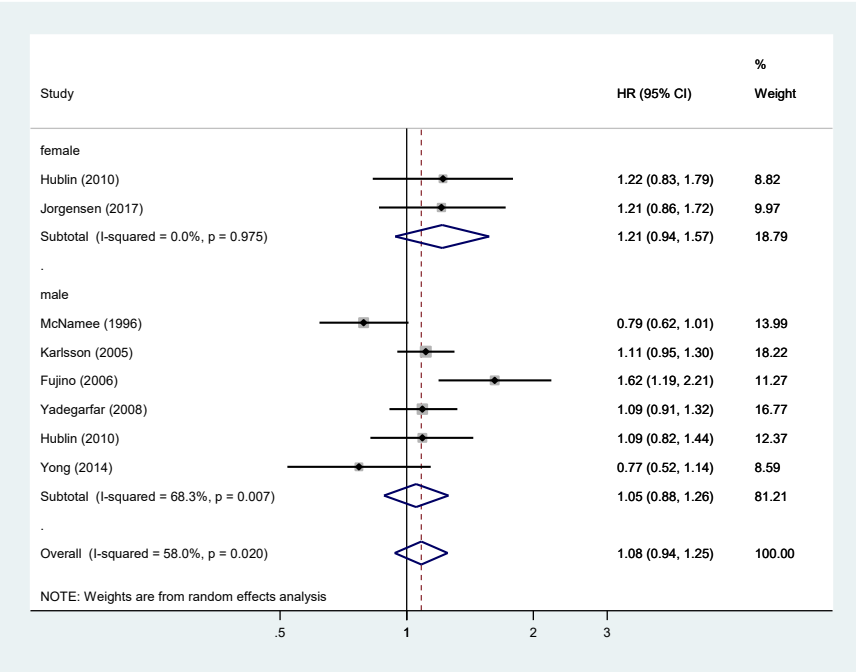
male Heterogeneity: $Tau^2 = 0.17$; test statistic = 7.22, df = 2 (p = 0.027); $I^2 = 72.3\%$; Test for overall effect: z = 0.09 (p = 0.925)
Total Heterogeneity: $Tau^2 = 0.11$; test statistic = 13.1, df = 4 (p = 0.011); $I^2 = 69.4\%$; Test for overall effect: z = 0.62 (p = 0.537)

Figure S8: Forest plot of the effect of job strain compared to no job strain on CHD mortality minimal analysis



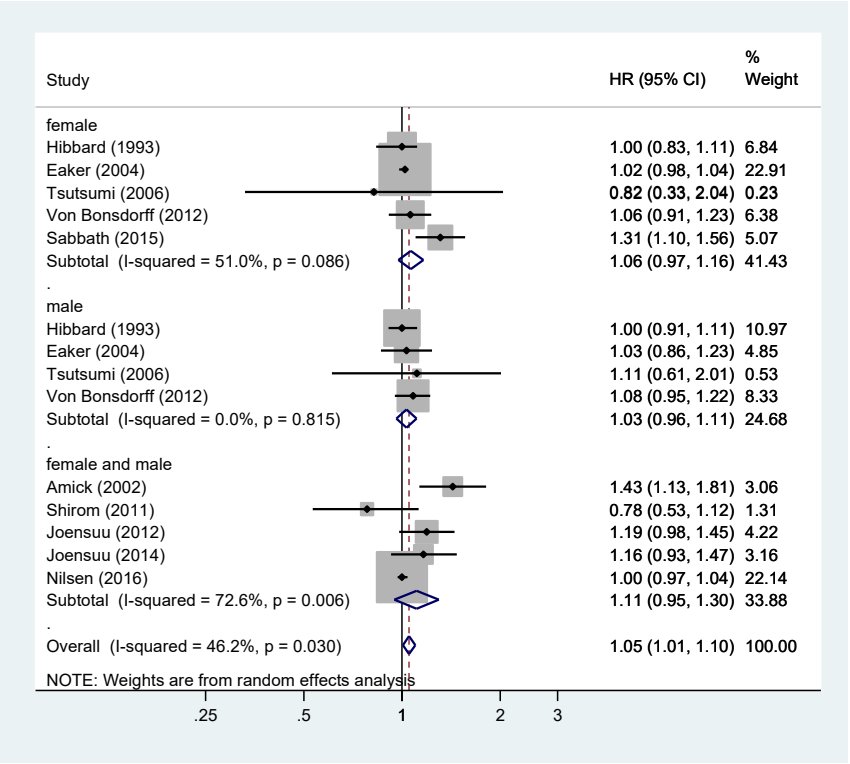
female Heterogeneity: $Tau^2 = 0.00$; test statistic = 1.04, df = 2 (p = 0.595); $I^2 = 0.0\%$; Test for overall effect: z = 0.66 (p = 0.508)
male Heterogeneity: $Tau^2 = 0.19$; test statistic = 7.42, df = 2 (p = 0.025); $I^2 = 73.0\%$; Test for overall effect: z = 1.02 (p = 0.307)
female and male Heterogeneity: $Tau^2 = 0.00$; test statistic = 0.03, df = 1 (p = 0.860); $I^2 = 0.0\%$; Test for overall effect: z = 2.89 (p = 0.004)
Total Heterogeneity: $Tau^2 = 0.13$; test statistic = 15.6, df = 7 (p = 0.029); $I^2 = 55.2\%$; Test for overall effect: z = 1.46 (p = 0.144)

Figure S9: Forest plot of the effect of shift workers compared to day workers on CHD mortality minimal analysis



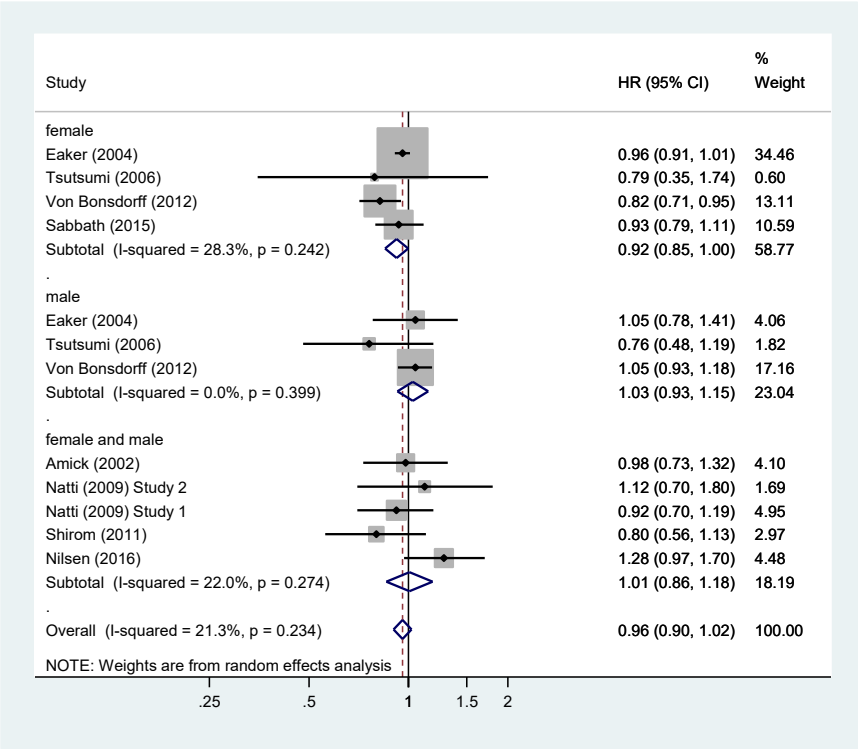
female Heterogeneity: $Tau^2 = 0.00$; test statistic = 0.00, df = 1 (p = 0.975); $I^2 = 0.0\%$; Test for overall effect: z = 1.48 (p = 0.139)
male Heterogeneity: $Tau^2 = 0.03$; test statistic = 15.8, df = 5 (p = 0.007); $I^2 = 68.3\%$; Test for overall effect: z = 0.58 (p = 0.563)
Total Heterogeneity: $Tau^2 = 0.02$; test statistic = 16.7, df = 7 (p = 0.020); $I^2 = 58.0\%$; Test for overall effect: z = 1.08 (p = 0.282)

Figure S10: Forest plot of the effect of low job control compared to high job control on all-cause mortality multivariable-adjusted analysis



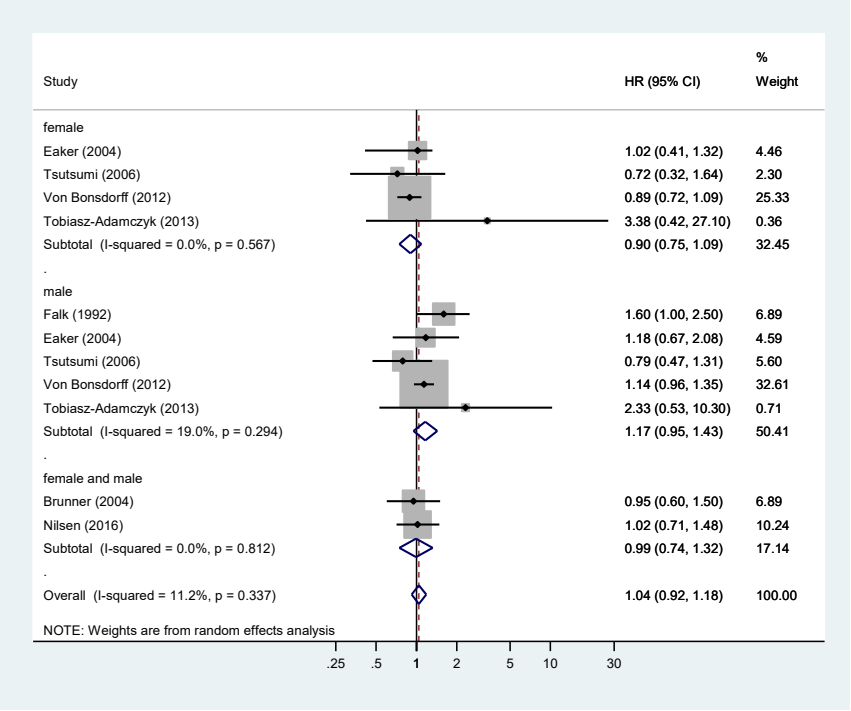
female Heterogeneity: $Tau^2 = 0.005$; test statistic = 8.16, df = 4 (p = 0.086); $I^2 = 51.0\%$; Test for overall effect: z = 1.38 (p = 0.169)
male Heterogeneity: $Tau^2 = 0.00$; test statistic = 0.94, df = 3 (p = 0.815); $I^2 = 0.0\%$; Test for overall effect: z = 0.86 (p = 0.390)
female and male Heterogeneity: $Tau^2 = 0.02$; test statistic = 14.6, df = 4 (p = 0.006); $I^2 = 72.6\%$; Test for overall effect: z = 1.28 (p = 0.202)
Total Heterogeneity: $Tau^2 = 0.002$; test statistic = 24.2, df = 13 (p = 0.030); $I^2 = 46.2\%$; Test for overall effect: z = 2.23 (p = 0.026)

Figure S11: Forest plot of the effect of high job demands compared to low job demands on all-cause mortality multivariable-adjusted analysis



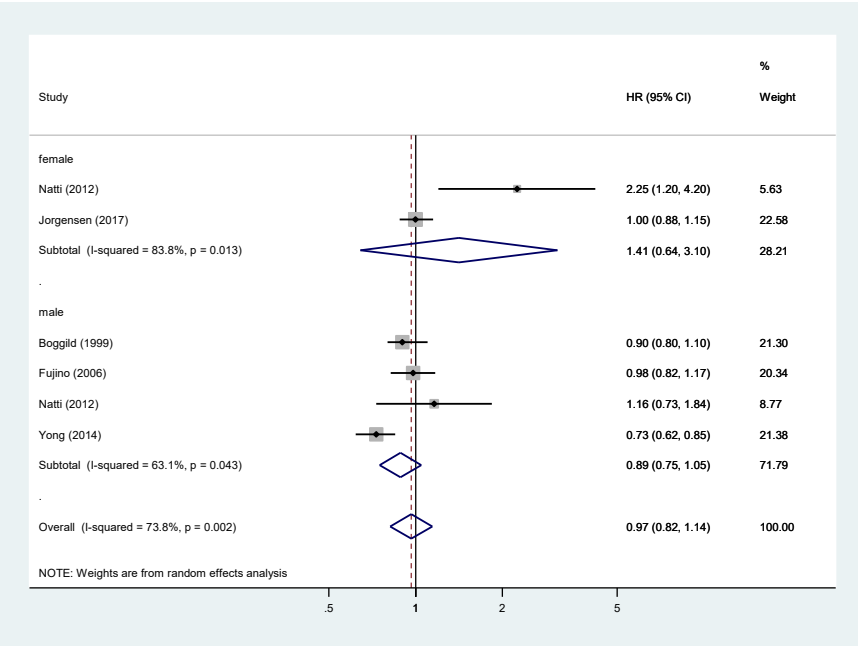
female Heterogeneity: $Tau^2 = 0.002$; Test statistic = 4.19, df = 3 (p = 0.242); $I^2 = 28.3\%$; Test for overall effect: z = 2.00 (p = 0.045)
male Heterogeneity: $Tau^2 = 0.00$; Test statistic = 1.84, df = 2 (p = 0.399); $I^2 = 0.0\%$; Test for overall effect: z = 0.56 (p = 0.575)
female and male Heterogeneity: $Tau^2 = 0.01$; Test statistic = 5.1, df = 4 (p = 0.274); $I^2 = 22.0\%$; Test for overall effect: z = 0.08 (p = 0.933)
Total Heterogeneity: $Tau^2 = 0.002$; Test statistic = 14.0, df = 11 (p = 0.234); $I^2 = 21.3\%$; Test for overall effect: z = 1.31 (p = 0.189)

Figure S12: Forest plot of the effect of job strain compared to no job strain on all-cause mortality multivariable-adjusted analysis



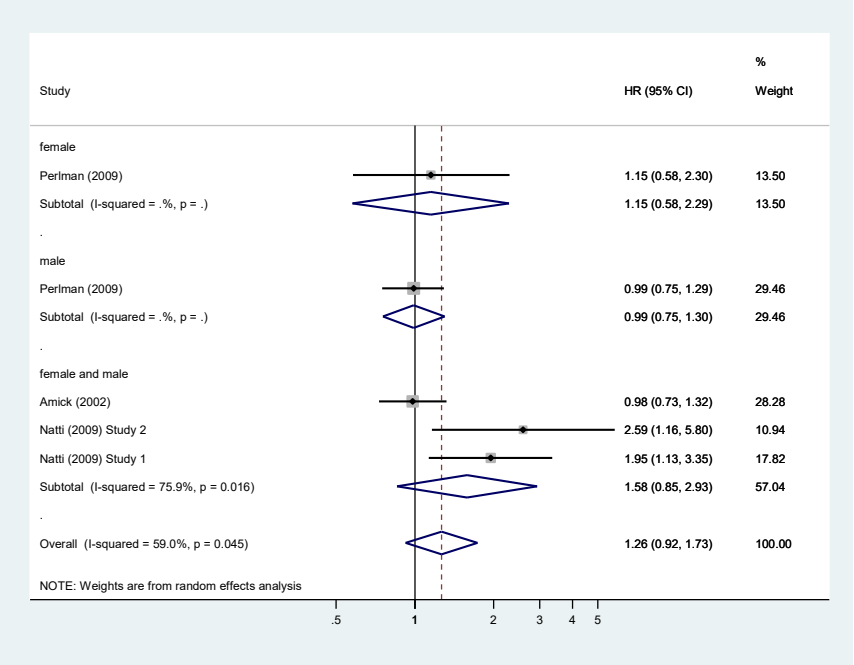
female Heterogeneity: Tau^2 = 0.00; Test statistic = 2.03, df = 3 (p = 0.567); I^2 = 0.0%; Test for overall effect: z = 1.06 (p = 0.289)
male Heterogeneity: Tau^2 = 0.01; Test statistic = 4.94, df = 4 (p = 0.294); I^2 = 19.0%; Test for overall effect: z = 1.46 (p = 0.144)
female and male Heterogeneity: Tau^2 = 0.00; Test statistic = 0.06, df = 1 (p = 0.812); I^2 = 0.0%; Test for overall effect: z = 0.05 (p = 0.956)
Total Heterogeneity: Tau^2 = 0.005; Test statistic = 11.3, df = 10 (p = 0.337); I^2 = 11.2%; Test for overall effect: z = 0.66 (p = 0.511)

Figure S13: Forest plot of the effect of shift workers compared to day workers on all-cause mortality multivariable-adjusted analysis



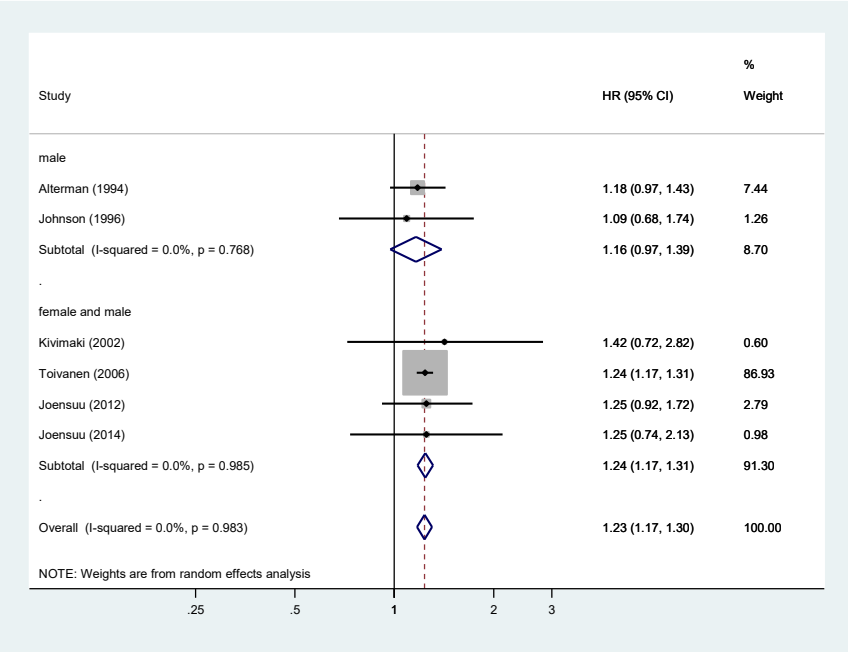
female Heterogeneity: Tau^2 = 0.28; test statistic = 6.16, df = 1 (p = 0.013); I^2 = 83.8%; Test for overall effect: z = 0.86 (p = 0.389)
male Heterogeneity: Tau^2 = 0.02; test statistic = 8.13, df = 3 (p = 0.043); I^2 = 63.1%; Test for overall effect: z = 1.42 (p = 0.154)
Total Heterogeneity: Tau^2 = 0.03; test statistic = 19.1, df = 5 (p = 0.002); I^2 = 73.8%; Test for overall effect: z = 0.41 (p = 0.681)

Figure S14: Forest plot of the effect of job insecurity compared to no job insecurity on all-cause mortality multivariable-adjusted analysis



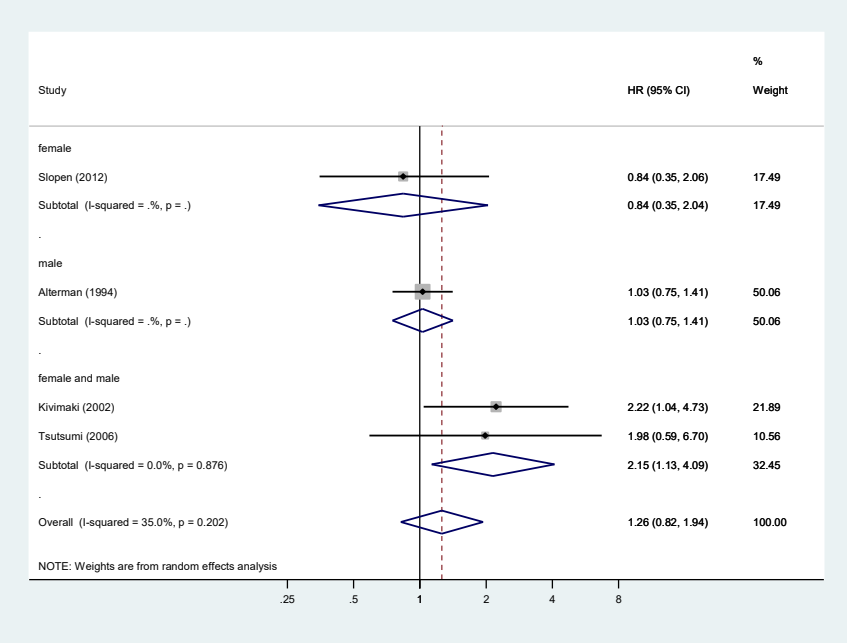
female and male Heterogeneity: $Tau^2 = 0.22$; Test statistic = 8.31, df = 2 (p = 0.016); $I^2 = 75.9\%$; Test for overall effect: z = 1.46 (p = 0.145)
Total Heterogeneity: $Tau^2 = 0.07$; Test statistic = 9.75, df = 4 (p = 0.045); $I^2 = 59.0\%$; Test for overall effect: z = 1.45 (p = 0.148)

Figure S15: Forest plot of the effect of low job control compared to high job control on CHD mortality multivariable-adjusted analysis



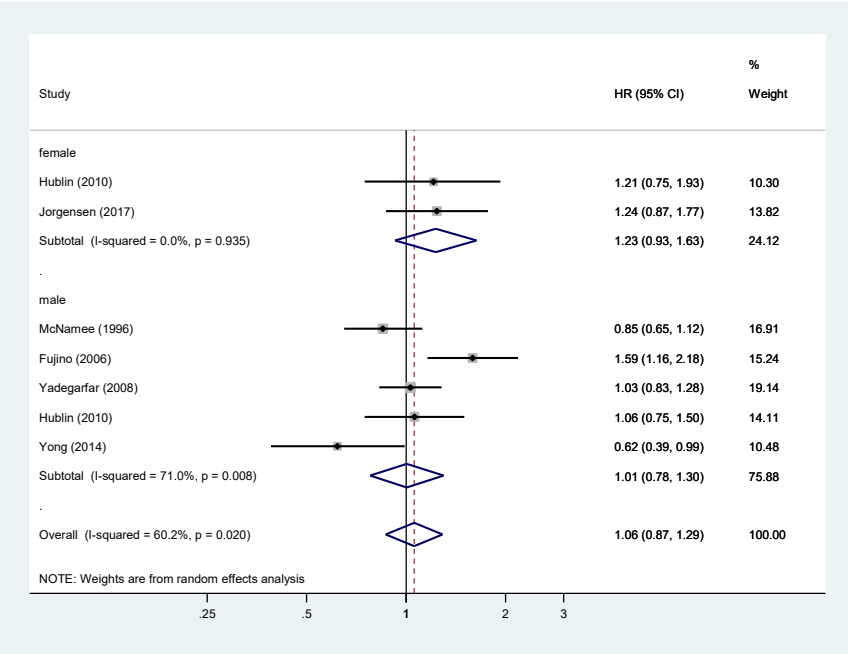
male Heterogeneity: $Tau^2 = 0.00$; Test statistic = 0.09, df = 1 (p = 0.768); $I^2 = 0.0\%$; Test for overall effect: z = 1.66 (p = 0.096)
female and male Heterogeneity: $Tau^2 = 0.00$; Test statistic = 0.15, df = 3 (p = 0.985); $I^2 = 0.0\%$; Test for overall effect: z = 7.69 (p < 0.001)
Total Heterogeneity: $Tau^2 = 0.00$; Test statistic = 0.70, df = 5 (p = 0.983); $I^2 = 0.0\%$; Test for overall effect: z = 7.84 (p < 0.001)

Figure S16: Forest plot of the effect of job strain compared to no job strain on CHD mortality multivariable-adjusted analysis



female and male Heterogeneity: Tau^2 = 0.00; Test statistic = 0.02, df = 1 (p = 0.876); I^2 = 0.0%; Test for overall effect: z = 2.33 (p = 0.020)
Total Heterogeneity: Tau^2 = 0.07; Test statistic = 4.62, df = 3 (p = 0.202); I^2 = 35.0%; Test for overall effect: z = 1.02 (p = 0.308)

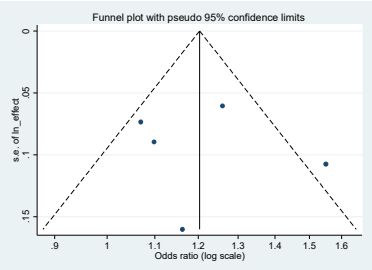
Figure S17: Forest plot of the effect of shift workers compared to day workers on CHD mortality multivariable-adjusted analysis



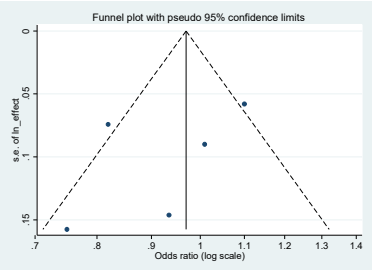
female Heterogeneity: Tau^2 = 0.00; test statistic = 0.01, df = 1 (p = 0.935); I^2 =0.0%; Test for overall effect: z = 1.42 (p = 0.154)
male Heterogeneity: Tau^2 = 0.06; test statistic = 13.8, df = 4 (p = 0.008); I^2 = 71.0%; Test for overall effect: z = 0.04 (p = 0.967)
Total Heterogeneity: Tau^2 = 0.04; test statistic = 15.1, df = 6 (p = 0.020); I^2 = 60.2%; Test for overall effect: z = 0.56 (p = 0.578)

Figure S18: Funnel plots of psychosocial work stressors and all-cause mortality minimally adjusted analysis

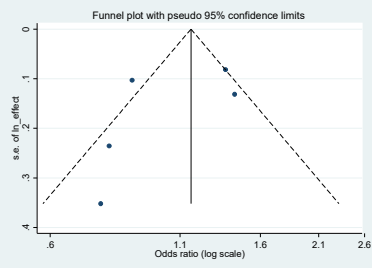
a) Job control (Egger's test p value = 0.825)



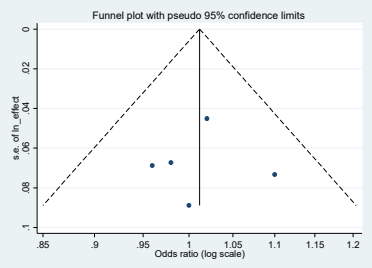
b) Job demands (Egger's test p value = 0.343)



c) Job strain (Egger's test p value = 0.403)



d) Shift work (Egger's test p value = 0.928)



e) Job insecurity (Egger's test p value = 0.092)

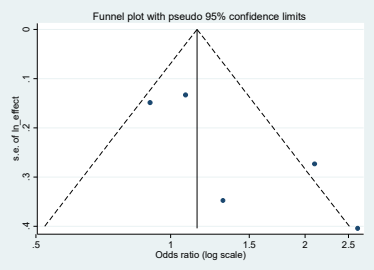
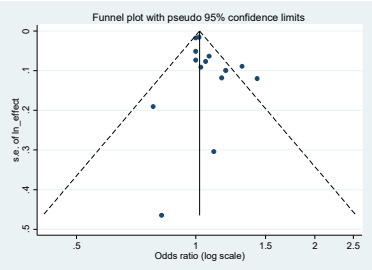
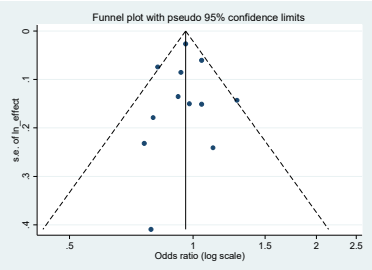


Figure S19: Funnel plots of psychosocial work stressors and all-cause mortality multivariable-adjusted analysis

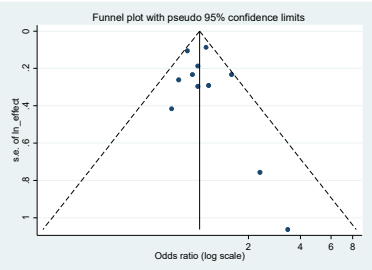
a) Job control (Egger's test p value = 0.116)



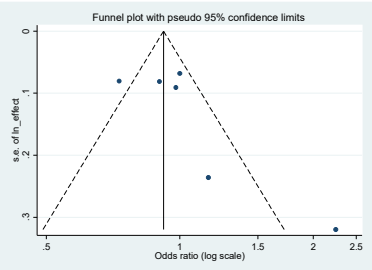
b) Job demands (Egger's test p value = 0.886)



c) Job strain (Egger's test p value = 0.532)



d) Shift work (Egger's test p value = 0.217)



e) Job insecurity (Egger's test p value = 0.081)

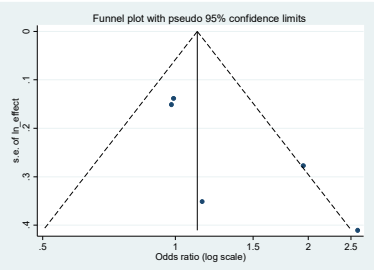
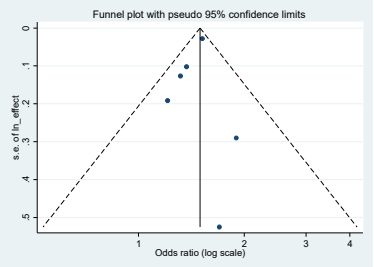
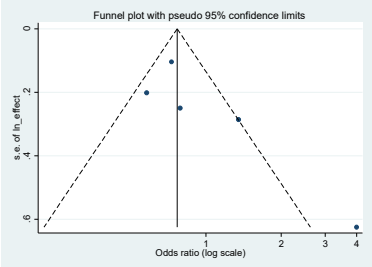


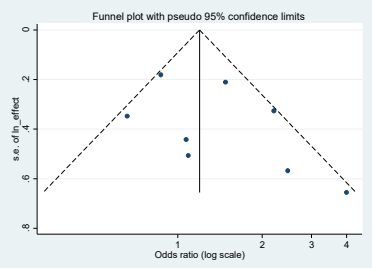
Figure S20: Funnel plots of psychosocial work stressors and CHD mortality minimally adjusted analysis

a) Job control (Egger's test p value = 0.450) *b) Job demands (Egger's test p value = 0.171)*





c) Job strain (Egger's test p value = 0.264) *d) Shift work (Egger's test p value = 0.878)*



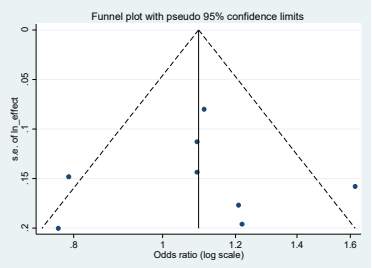
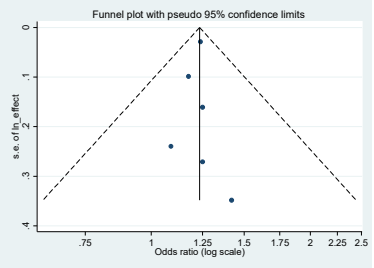
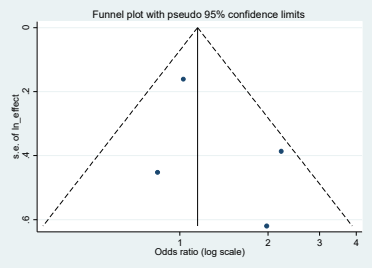


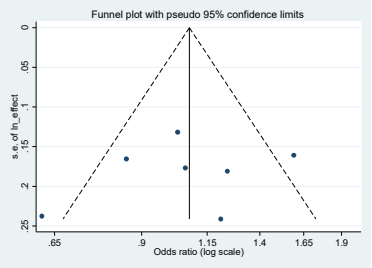
Figure S21: Funnel plots of psychosocial work stressors and CHD mortality multivariable-adjusted analysis

a) Job control (Egger's test p value = 0.678)



c) Job strain (Egger's test p value = 0.440) *d) Shift work (Egger's test p value = 0.598)*





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Chapter 4 Study II - Psychosocial work stressors and risk of mortality in Australia: analysis of data from the HILDA survey

This chapter is presented as the peer-reviewed research article published in the BMJ Journal of Occupational and Environmental Medicine (2020; 77(4):256-64).

4.1 Overview

This published study which this chapter is based on (with supplementary material) analyses the association between psychosocial work stressors and mortality using a nationally representative sample of people in work. Data collected from over 18,000 working participants from the HILDA survey with self-reported job demands, job control, job security and fair pay psychosocial work stressors exposures at baseline and followed over time for up to 15 years were analysed in this first Australian study of adverse psychosocial work stressors exposures and mortality. Low job control was associated with a 39% increased risk of all-cause mortality after accounting for demographic, socioeconomic, health and behavioural factors. This study adds to a growing body of evidence showing an effect of deleterious psychosocial work stressors on mortality.

4.2 Research Questions and Objectives

4.2.1 Research questions

The overall research question was what is the effect of adverse psychosocial work stressors in working environments on all-cause mortality?

The specific research questions are:

1. What is the effect of adverse psychosocial work stressors in working environments, such as high job demands, low job control, job insecurity and low fairness of pay, on all-cause mortality?

2. Is the relationship between adverse psychosocial work stressors and all-cause mortality confounded by demographic, socioeconomic, health and behavioural risk factors?
3. Does gender modify the relationship between psychosocial work stressors and all-cause mortality?

4.2.2 Research objectives

1. To examine the association between psychosocial work stressors including job demands, job control, job insecurity and fairness of pay measured at baseline, and all-cause mortality.
2. To control for the effect of demographic, socioeconomic, health and behavioural risk factors on the associations between adverse psychosocial work stressors and all-cause mortality.
3. To investigate if gender is an effect-modifier for the relationship between adverse psychosocial work stressors in working environments and all-cause mortality.

4.3 Publication

4.3.1 Main document and data supplement

Taouk Y., LaMontagne A. D., Spittal M. J., Milner A. Psychosocial work stressors and risk of mortality in Australia: analysis of data from the Household, Income and Labour Dynamics in Australia survey. Occupational and environmental medicine. 77(4):256-264, 2020. https://doi.org/10.1136/oemed-2019-106001.

ORIGINAL RESEARCH

Psychosocial work stressors and risk of mortality in Australia: analysis of data from the Household, Income and Labour Dynamics in Australia survey

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ABSTRACT

Objective To examine the association between exposures to psychosocial work stressors and mortality in a nationally representative Australian working population sample.

Methods 18 000 participants from the Household, Income and Labour Dynamics in Australia survey with self-reported job demands, job control, job security and fair pay psychosocial work stressors exposures at baseline were followed for up to 15 waves. Cox proportional hazards regression models were used to examine the association between psychosocial work stressors and mortality. Models were serially adjusted for each subgroup of demographic, socioeconomic, health and behavioural risk factors.

Results Low job control was associated with a 39% increase in the risk of all-cause mortality (HR 1.39; 95% CI 1.04 to 1.85), controlling for demographic, socioeconomic, health and behavioural factors. A decreased risk of mortality was observed for workers with exposure to high job demands (HR 0.76; 95% CI 0.60 to 0.96, adjusted for gender and calendar), but the risk was attenuated after serially adjusting for socioeconomic status, health (HR=0.84; 95% CI 0.65 to 1.08) and behavioural (HR=0.79; 95% CI 0.60 to 1.04) factors. There did not appear to be an association between exposure to job insecurity (HR 1.03; 95% CI 0.79 to 1.33) and mortality, or unfair pay and mortality (HR 1.04; 95% CI 0.80 to 1.34).

Conclusions Low job control may be associated with an increased risk of all-cause mortality. Policy and practice interventions that reduce the adverse impact of low job control in stressful work environments could be considered to improve health and decrease risk of mortality.

Key messages

What is already known about this subject?

- Previous research has shown that psychosocial work stressors are associated with adverse health outcomes including physical and mental ill health.
- The relationship between psychosocial work stressors and mortality is less clear-cut due to the paucity of studies and the inconsistency of results to date.

What are the new findings?

- Low job control was associated with a 39% increase in the risk of all-cause mortality independent of demographic, socioeconomic, health and behavioural factors.
- High job demand was associated with a 24% decrease in the risk of all-cause mortality; however, the risk was attenuated after adjustments for socioeconomic, health and behavioural factors.

How might this impact on policy or clinical practice in the foreseeable future?

- Given the high prevalence of exposed individuals, job control-associated increase in mortality may have considerable public health consequences.
- Intervention studies to improve job control and target job demands in stressful work environments could be implemented to gather evidence to improve health and decrease risk of mortality.

INTRODUCTION

The working environment is central to many people's lives, influencing both psychological and physical health and well-being outcomes.[1] Psychosocial work stressors are increasingly recognised as cause for concern by most developed countries around the world because of their association with occupational illness, sickness absence, presenteeism and poor organisational outcomes.[2 3]

Traditionally, research has focused on common psychosocial work stressors using the Karasek's job demand-control model, which identifies two psychosocial factors: high job demands (the need to work quickly and intensely) and low job control (lack of control over skill use, time allocation and organisational decisions).[1 4] The model posits that stress-related ill health stems from job strain as a result of the combined effects of low job control and high job demands in the work environment.[4] The effort–reward imbalance (ERI) model, another work stress model, recognises both the effort and reward structure of work, and postulates that the lack of reciprocity due to the imbalance between low rewards and high efforts at work is a cause of stress-related diseases.[5] Both globalisation and advancements in technology have meant that work intensity, job insecurity and organisational injustice

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(all components of the ERI model) have become more common in contemporary workplaces. Job insecurity and organisational injustice have also been shown to be important influences on mental and physical health.[6 7]

A number of studies have demonstrated the adverse effects of exposure to psychosocial work stressors, such as high job demands, low job control, low job security and high ERI, on health.[8–12] There is some evidence that the effects of psychosocial work stressors on health are stronger among men than women.[13] Whether exposure to these psychosocial work stressors associated with adverse health outcomes translates into increased mortality is unclear. Relatively few studies have examined this, and among those that have the results, including gender effects, have been inconsistent.[14–27] This may be due to different study designs and methodological approaches. Most studies were generally small and underpowered to detect associations,[14 17 19 22 26] or mortality outcome was restricted to deaths from cardiovascular disease.[14–16 18 19 21 26] In some studies, the exposure status was estimated and assigned with objective data based on occupation using a job exposure matrix.[23 25 26] Other studies were restricted to narrow occupational groups[14 19 22 26] and under-represented employed women.[15 20 24 26] There were inconsistencies in the measurements of psychosocial work stressors and adjustments for confounders across studies.

The Household, Income and Labour Dynamics in Australia (HILDA) survey, a nationally representative longitudinal cohort of Australian households,[28] can potentially address these inconsistencies. The HILDA survey contains extensive data on employment and labour-force characteristics of working Australians with over 15 annual waves of data and has been linked to the National Death Index (NDI). The study is a prospective cohort design with a national sampling frame and a large sample size, and includes measures of a large number of socioeconomic, health and behavioural indicators.

In the present study we analysed the association of the various measures of psychosocial work stressors with all-cause mortality and tested whether gender modified the relationship between psychosocial work stressors and mortality.

METHODS

Study population

Commenced in 2001, HILDA follows over 17 000 Australians annually.[28] In brief, households comprise the source of the panel data and are interviewed in each wave of data collection from a combination of face-to-face interviews and self-completion questionnaires. In wave 1, HILDA had 13 969 participants from 7682 households (66% response rate). Retention at wave 2 was 87% and greater than 90% at subsequent waves. In wave 11, the sample was topped up by 4009 participants from 2153 households to correct for population undercoverage and retain cross-sectional representativeness in the sample to include representation of migrant populations and help alleviate biases from non-random attrition.[29]

Sample

There were 29 810 participants aged over 15 years during waves 1–15 in the HILDA survey (figure 1). Participants were excluded if they were not in paid work at any time during the survey (n=7621) and if they did not have data on any psychosocial work stressors at baseline (n=3745). Of these, 99.7% had

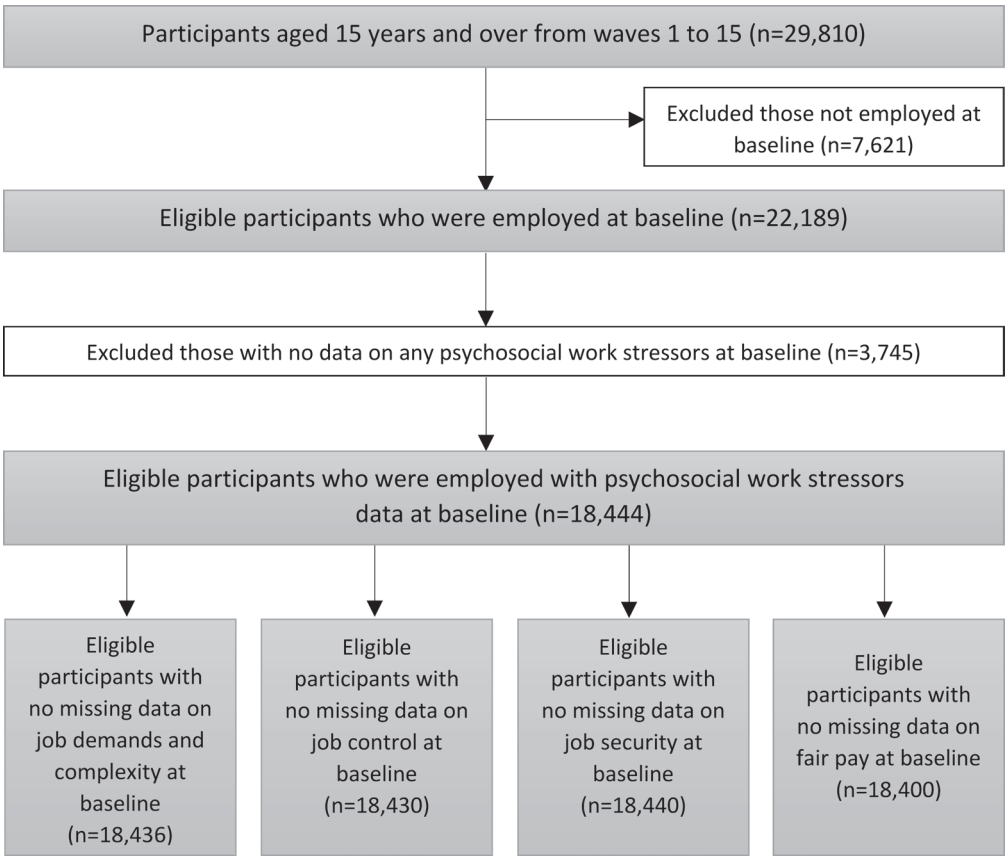

Figure 1 Flow chart showing the selection of HILDA participants for the analyses. HILDA, Household, Income and Labour Dynamics in Australia.

information on socioeconomic status and 96.0% had information on behavioural factors.

All-cause mortality outcome

The entire HILDA survey sample was matched to the NDI in 2014. In summary, 1238 deaths had already been identified through the HILDA survey fieldwork and an additional 333 deaths were identified by the data linkage. Sixty-four deaths (4%) identified via fieldwork could not be matched to the NDI. A further 213 deaths have been identified since the data linkage.[30]

Measures

Psychosocial work stressors

Self-reported psychosocial work stressors were measured at baseline. The items and scales available in all waves of the HILDA survey were based on a variety of economic and social surveys,[31] and three separate measures of the psychosocial work environment, previously identified through factor analysis and structural equation modelling, were constructed: job demands and complexity (three items, $\alpha=0.70$), job control (three items, $\alpha=0.82$) and perceived job security (three items, $\alpha=0.64$).[32 33] The ERI model maintains that work stressors result from a perceived inequality by workers between their efforts at work and a range of rewards[5]; however, in HILDA only a single item assessing financial rewards relative to effort is available for assessment of any aspects of ERI. These individual scales have strong associations with more widely used measures, including high job demands, low job control, low job security, effort–reward unfairness and employment status.[32 34]

All items were recoded in the same direction, such that high scores represented higher control, greater demand and complexity, greater security, and higher fairness of pay, and scores for each scale were summed. For those participants with missing data for some items, scale scores were based on completed items and weighted up to the expected total had all items been answered. As there are no normative data for these scales, scores were dichotomised at the median value into high and low levels, consistent with the most common operationalisation of psychosocial work factors,[35 36] enabling comparison of results of the current study with previous research. At the median and above, exposure was defined as low, while below the median was high exposure for control, security and fairness and the reverse for demands.

Covariates

Potential confounders were identified a priori, including demographic, socioeconomic, health and behavioural factors.

- Demographic factors: age, gender, country of birth (Australia, English-speaking country other than Australia, other country), educational attainment (no completion of high school, completion of high school or vocational certificate/diploma, bachelor's degree or above) and household structure (couple with no children, couple with children, lone parent with children, lone person, other).
- Socioeconomic factors: occupational skill level (low, medium, high),[37] employment arrangement (permanent, casual or labour hire, fixed term, self-employed) and income (quintiles of the population distribution of disposable household income with the highest quintile used as the referent). Household disposable income, calculated by summing the income components for the previous financial year for all adults in the household, was equivalised using the modified Organisation for Economic Co-operation and Development (OECD) scale.[38] For each wave of data, nominal household income values were converted to quintiles of the Australian population distribution using percentile statistics for the corresponding financial year from the Australian Bureau of Statistics biennial Survey of Income and Housing.[39]
- Health factors: any disability or mental or physical health condition which has lasted, or is likely to last, 6 months or more, restrict everyday activity, and cannot be corrected by medication or medical aids (no, yes).
- Behavioural factors: cigarette smoking (never smoker, ex-smoker, current smoker) and frequency of physical activity (<1 time/week, 1–3 times/week, >3 times/week). Alcohol consumption categories: abstinence, low (<1 drink/week), moderate (women: 1–20 drinks/week; men: 1–27 drinks/week) and heavy (women: 21+ drinks/week; men: 28+ drinks/week).[40]

Statistical analysis

Cox proportional hazards regression models with age as the time metric were used to calculate HR and 95% CI. Robust SEs were used to account for clustering within households. Time of study entry was age of first employment and ended at age of death, or age at last interview for participants who remained alive. For each variable, the value representing the lowest risk was chosen as the reference category. Given the rare outcome, the main analyses in this study were based on dichotomous measures to improve statistical power and accuracy. However, models with ordinal exposures were also included for completeness of results. Sequential models were serially adjusted for each subgroup of demographic, socioeconomic, health and behavioural risk factors. Additional models were adjusted for simultaneous psychosocial work stressors exposures. Complete case analyses were conducted.

Effect modification was tested by the change in the log-likelihood ratio after the addition of gender-stressor product terms in the models. The proportional hazard assumption was assessed by examining the relationship between the scaled Schoenfeld residuals and survival time scaled. Estimates were stratified by confounding factors found to have violated the assumption of proportionality. Sensitivity analyses were performed to assess if psychosocial work stressors exerted distal effects on mortality regardless of time since last observed wave. This was done by limiting follow-up to 3 years from the time of the last interview. All analyses were conducted using STATA/SE V.14.2.

RESULTS

The study sample consisted of 18 444 participants with 132 743 person-years. The earliest observed entry into the study was at 15 years of age and the last observed exit was 85 years. During the median follow-up of 6 years, 298 deaths were observed. The mean age at study entry was 33.5 years and half were male (table 1). Most participants were born in Australia, resided in couples households and did not have any long-term health conditions. Of the participants, 25% were cigarette smokers, 13% reported heavy alcohol consumption and 22% participated in almost no physical activity. Most participants were employed in jobs requiring low or medium occupational skill level (71%) and half were permanently employed. Table 2 presents the details of the scales, cut points and the proportion of participants with adverse stressors at baseline.

The HR for mortality was 1.40 (95% CI 1.09 to 1.79) for low versus high job control. On the ordinal scale, for each one-point increase in control, the risk of death fell by 2% (HR=0.98;

Table 1 Descriptive statistics of final analytic sample

Sample	
Number of participants	18 444
Person-years	132 743
Median (IQR) follow-up (years)	6 (2–14)
Median (IQR) wave of first entry into HILDA survey	2003 (2001–2011)
Number of deaths	298
Sociodemographic factors	
Age (years), mean (SD)	33.5 (13.9)
Age group (years), n (%)*	
15–24	6202 (33.6)
25–34	4057 (22.0)
35–44	3727 (20.2)
45–54	2881 (15.6)
55–64	1344 (7.3)
65+	233 (1.3)
Gender, n (%)*	
Male	9205 (49.9)
Female	9239 (50.1)
Country of birth, n (%)*	
Australia	14 747 (80.0)
English-speaking	1717 (9.3)
Other	1976 (10.7)
Education, n (%)*	
Bachelor's degree and above	5118 (27.7)
Year 12/certificate/diploma	9254 (50.2)
School not completed	4072 (22.1)
Household structure, n (%)*	
Couple with no children	4701 (25.5)
Couple with children	8815 (47.8)
Lone parent with children	1828 (9.9)
Lone person	1381 (7.5)
Other	1719 (9.3)
Population quintiles of disposable income, n (%)*	
1 (lowest)	1498 (8.1)
2	2598 (14.1)
3	3878 (21.0)
4	4751 (25.8)
5 (highest)	5719 (31.0)
Occupational skill level, n (%)*	
High	5308 (28.8)
Medium	7017 (38.1)
Low	6102 (33.1)
Employment arrangement, n (%)*	
Permanent	8767 (47.6)
Casual	6074 (33.0)
Fixed term	1348 (7.3)
Self-employed	2223 (12.1)
Health factors	
Long-term health conditions, n (%)*	
No	15 746 (85.4)
Yes	2698 (14.6)
Behavioural factors	
Smoking status, n (%)*	
Never	10 060 (54.6)
Ex-smoker	3835 (20.8)
Current	4532 (24.6)
Alcohol consumption, n (%)*	
Abstinence	6913 (38.9)
Low	2367 (13.3)
Moderate	6202 (34.9)
Heavy	2275 (12.8)
Physical activity, n (%)*	
>3 times per week	6963 (37.8)
1–3 times per week	7345 (39.8)
Less than once a week/never	4126 (22.4)

*Percentages were calculated with the number of available observations used as the denominator.
HILDA, Household, Income and Labour Dynamics in Australia.

95% CI 0.95 to 1.00). The HR was essentially unchanged after further adjustments for socioeconomic, health and behavioural factors on the ordinal or binary scale (table 3). The HR for mortality was 0.76 (95% CI 0.60 to 0.96) for high job demands compared with low job demands controlling for gender and calendar year, but the risk was attenuated after adjustments for risk factors. On the ordinal scale, for each one-point increase in demand, the risk of death fell by 2% (HR=0.98; 95% CI 0.95 to 1.00), but the risk was attenuated after adjusting for risk factors. There did not appear to be an association between higher job security and mortality, nor between higher fairness of pay and mortality. Limiting follow-up of participants to 3 years of the last interview did not significantly change the results for psychosocial work stressors and the risk of mortality (online supplementary table 1).

Table 4 presents the results for simultaneously adjusted psychosocial work stressors and risk of mortality. The relative HRs for mortality and psychosocial work stressors in serially adjusted models for socioeconomic, health and behavioural factors and inclusion of all psychosocial work stressors did not substantially alter the results. Age-adjusted HRs for mortality, controlling for socioeconomic, health and behavioural factors, were 1.36 (95% CI 1.02 to 1.82), 0.82 (95% CI 0.62 to 1.08), 0.99 (95% CI 0.76 to 1.28) and 1.00 (95% CI 0.77 to 1.29) for low control, high demands, low security and low fair pay, respectively. On the ordinal scale, the HRs included the null. Similarly

Table 2 Description of measures of psychosocial work stressors

		Ordinal scale characteristics			
Item/scale	Total	Range	Mean (SD)	Cut point	% adverse
Job demands and complexity*	18 436	3–21	13.4 (4.4)	>14	45.0
Job control†	18 430	3–21	12.2 (4.9)	<12	44.1
Job security‡	18 440	3–21	15.5 (4.0)	<16	47.7
Paid fairly§	18 400	1–7	4.7 (1.8)	<5	41.7

*Job demands and complexity exposure: 'job is complex and difficult', 'job requires learning new skills', 'skills and abilities are used in job' (higher score indicates higher job demands and complexity).
†Job control exposure: 'freedom to decide how to work', 'a lot of say about what happens', 'freedom to decide when to work' (higher score indicates higher job control).
‡Job security exposure: 'secure future in job', 'worry about future of job', 'company will still exist 5 years from now' (higher score indicates higher job security).
§Perception of unfair pay exposure: 'get paid fairly for the things I do in job' (higher score indicates higher fair pay).

Table 3 Age-adjusted HR from survival analysis of serially adjusted association between mutually exclusive psychosocial work stressors exposures, job control*, job demands and complexity†, job security‡ and fairness of pay§, and risk of mortality

	Adjusted for gender and calendar year			Adjusted for gender, calendar year and socioeconomic status¶			Adjusted for gender, calendar year, socioeconomic status and health**			Adjusted for gender, calendar year, socioeconomic status, health and behavioural factors††		
Psychosocial job exposure	**n (total)‡‡**	**n (events)**	**HR (95% CI)**	**n (total)‡‡**	**n (events)**	**HR (95% CI)**	**n (total)‡‡**	**n (events)**	**HR (95% CI)**	**n (total)‡‡**	**n (events)**	**HR (95% CI)**
Job control ordinal scale	18430	297	0.98 (0.95 to 1.00)	18377	295	0.98 (0.95 to 1.00)	18377	295	0.98 (0.95 to 1.00)	17689	264	0.98 (0.95 to 1.01)
Low job control	8134	109	1.40 (1.09 to 1.79)	8113	109	1.39 (1.07 to 1.80)	8113	109	1.37 (1.06 to 1.79)	7853	97	1.39 (1.04 to 1.85)
High job control	10296	188	Reference	10264	186	Reference	10264	186	Reference	9836	167	Reference
Job demands ordinal scale	18436	297	0.98 (0.95 to 1.00)	18383	295	0.99 (0.96 to 1.03)	18383	295	0.99 (0.96 to 1.03)	17695	264	0.99 (0.95 to 1.02)
Low job demands	10133	172	Reference	10103	170	Reference	10103	170	Reference	9751	153	Reference
High job demands	8303	125	0.76 (0.60 to 0.96)	8280	125	0.85 (0.66 to 1.10)	8280	125	0.84 (0.65 to 1.08)	7944	111	0.79 (0.60 to 1.04)
Job security ordinal scale	18440	297	0.99 (0.96 to 1.02)	18388	295	1.00 (0.97 to 1.03)	18388	295	1.00 (0.98 to 1.04)	17700	264	1.01 (0.98 to 1.04)
Low job security	8793	154	1.18 (0.94 to 1.48)	8770	153	1.10 (0.86 to 1.40)	8770	153	1.06 (0.84 to 1.36)	8390	134	1.03 (0.79 to 1.33)
High job security	9647	143	Reference	9618	142	Reference	9618	142	Reference	9310	130	Reference
Fairness of pay ordinal scale	18400	295	1.00 (0.94 to 1.07)	18348	294	1.01 (0.94 to 1.08)	18348	294	1.02 (0.95 to 1.09)	17662	263	1.02 (0.95 to 1.10)
Low fairness of pay	7667	122	1.12 (0.89 to 1.42)	7643	122	1.09 (0.86 to 1.38)	7643	122	1.07 (0.84 to 1.36)	7321	105	1.04 (0.80 to 1.34)
High fairness of pay	10733	173	Reference	10705	172	Reference	10705	172	Reference	10341	158	Reference

*Job control exposure: 'freedom to decide how to work', 'a lot of say about what happens', 'freedom to decide when to work'.
†Job demands and complexity exposure: 'job is complex and difficult', 'job requires learning new skills', 'skills and abilities are used in job'.
‡Job security exposure: 'secure future in job', 'worry about future of job', 'company will still exist 5 years from now'.
§Fairness of pay exposure: 'get paid fairly for the things I do in job'.
¶Model adjusted for gender, calendar year, country of birth, income, occupation level, employment arrangement and household structure, stratified by education.
**Model adjusted for gender, calendar year, country of birth, income, occupation level, employment arrangement, household structure and long-term health conditions, stratified by education.
††Model adjusted for gender, calendar year, country of birth, income, occupation level, employment arrangement, household structure, long-term health condition, alcohol consumption and physical activity, stratified by education and smoking.
‡‡The number of participants varies because of missing data in covariates.

Table 4 Age-adjusted HRs from survival analysis of serially adjusted association between simultaneously adjusted psychosocial work stressors exposures, job control*, job demands and complexity†, job security‡ and perception of unfair pay§, and risk of mortality

Adjustment	n (total)¶	n (events)	Job control on ordinal scale	Low job control compared with high job control	Job demands on ordinal scale	High job demands compared with low job demands	Job security on ordinal scale	Low job security compared with high job security	Fair pay on ordinal scale	Unfair pay compared with fair pay
			HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)
Gender, calendar year	18 384	293	0.98 (0.95 to 1.00)	1.32 (1.03 to 1.70)	0.98 (0.96 to 1.01)	0.80 (0.63 to 1.02)	1.00 (0.97 to 1.03)	1.09 (0.87 to 1.38)	1.01 (0.94 to 1.08)	1.08 (0.85 to 1.36)
Gender, calendar year and socioeconomic status**	18 333	292	0.97 (0.95 to 1.00)	1.34 (1.03 to 1.75)	1.00 (0.97 to 1.03)	0.87 (0.67 to 1.12)	1.01 (0.97 to 1.04)	1.05 (0.83 to 1.34)	1.02 (0.95 to 1.09)	1.05 (0.82 to 1.33)
Gender, calendar year, socioeconomic status and health††	18 333	292	0.97 (0.95 to 1.00)	1.33 (1.02 to 1.74)	1.00 (0.97 to 1.03)	0.86 (0.67 to 1.11)	1.01 (0.98 to 1.04)	1.02 (0.80 to 1.31)	1.02 (0.95 to 1.10)	1.03 (0.81 to 1.32)
Gender, calendar year, socioeconomic status, health and behavioural factors‡‡	17 647	261	0.98 (0.95 to 1.01)	1.36 (1.02 to 1.82)	0.99 (0.96 to 1.03)	0.82 (0.62 to 1.08)	1.01 (0.98 to 1.04)	0.99 (0.76 to 1.28)	1.03 (0.95 to 1.11)	1.00 (0.77 to 1.29)

*Job control exposure: 'freedom to decide how to work', 'a lot of say about what happens', 'freedom to decide when to work'.
†Job demands and complexity exposure: 'job is complex and difficult', 'job requires learning new skills', 'skills and abilities are used in job'.
‡Job security exposure: 'secure future in job', 'worry about future of job', 'company will still exist 5 years from now'.
§Perception of unfair pay exposure: 'get paid fairly for the things I do in job'.
¶The number of participants varies because of missing data in covariates.
**Model adjusted for gender, calendar year, country of birth, income, occupation level, employment arrangement and household structure, stratified by education.
††Model adjusted for gender, calendar year, country of birth, income, occupation level, employment arrangement, household structure and long-term health conditions, stratified by education.
‡‡Model adjusted for gender, calendar year, country of birth, income, occupation level, employment arrangement, household structure, long-term health condition, alcohol consumption and physical activity, stratified by education and smoking.

limiting follow-up to 3 years of the last interview did not change the results (online supplementary table 2).

There was no evidence of effect modification by gender other than for job control (p value for interaction: job control ordinal scale=0.046 and low vs high=0.015; job demands ordinal scale=0.676 and high vs low=0.989; job security ordinal scale=0.209 and low vs high=0.441; fairness of pay ordinal scale=0.902 and low vs high=0.871). Analyses for job control and mortality were stratified by gender. The results for men were similar, although stronger, to the overall results. The risk of mortality for low versus high job control was HR=1.66 (95% CI 1.24 to 2.24) adjusted for calendar year and HR=1.73 (95% CI 1.22 to 2.47) adjusted for all risk factors. On the ordinal scale, the risk of mortality for each additional unit of increasing job control was HR=0.97 (95% CI 0.94 to 0.99) and HR=0.96 (95% CI 0.93 to 1.00) adjusted for all risk factors. The low number of deaths observed among women precluded analyses by female gender.

DISCUSSION

Main findings

Low job control was associated with a 39% increase in the risk of all-cause mortality in this nationally representative Australian working population sample. Adjustment for socioeconomic, health and behavioural factors, other stressor exposures, or limiting deaths to 3 years of last interview did not substantially affect risk estimates. When measured on an ordinal scale, for each additional unit of increase in job control, the risk of death fell by 2% (an increase of 2% per unit on the ordinal scale for an observed range of 18=36%). This suggests that low job control may affect mortality independent of other stressor exposures, or socioeconomic, health and behavioural factors.

A decreased risk of mortality for workers with exposure to high job demands adjusted for gender and calendar year was observed; however, the risk was attenuated after adjustments for socioeconomic, health and behavioural factors. There is no evidence for an association between exposure to job insecurity and mortality, or unfair pay and mortality.

Comparison with other studies

The findings of studies examining job control and mortality have been mixed, with some reporting that low job control increased mortality,[25 41] while others did not.[16 18 23] Many studies observe an elevated risk of mortality and low job control, but the association was not supported following adjustments for relevant covariates.[14 16 22 26] However, these studies were generally small and underpowered to detect associations between mortality and job control, and in some studies exposure status was estimated and assigned with objective data based on occupation using a job exposure matrix. Subjective assessment of work characteristics based on self-reports may be a more accurate representation of the work environment.

Similarly, previous studies investigating job demands and mortality have reported inconsistent results. Some studies reported no association between high job demands and mortality,[17 18] while others found that high job demands increased the risk of coronary heart disease mortality.[15 41] One study reported that high job demands decreased the risk of mortality.[23] Methodological differences, including differences in scale composition, different occupational groups across studies and adjustments for confounders, may have contributed to the inconsistent results. Furthermore, self-reported assessments of job demands may not be a homogeneous measure across different occupations. There

is a such a diversity of demands that responses might represent psychological, physical, emotional, social or other job demands. It may also be that perceptions of job demands have changed over time. This, along with inconsistent harmonisation of scales across studies, may explain the inconsistency of results for job demands and mortality in the literature, and the suggestion that job demands may have a protective effect with respect to mortality observed in this study.

There is relatively little previous research with which to compare our null findings with respect to job insecurity and organisational justice. The few available studies in the literature that examined job insecurity and mortality have reported inconclusive findings.[19 23] Further research on the relationship between these job stressors and mortality is warranted.

There is precedent for our finding of a stronger relationship among men than women, although previous studies are mixed.[13 42 43] One possible explanation is the 'role theory': the notion that work is typically more central to identity among men than among women,[43] which aligns with the proposed mechanism outlined above. In our results, the difference between genders was not substantial, and its meaning in practical terms is unclear, but it is also possible that earlier studies were accurate and gender differences are narrowing over time with increasing rates of female participation in the labour market and changing social roles.

Strengths and limitations of the study

The limitations must be balanced against the strengths of the study, including the long follow-up time, the large representative national sample and the validated measures of psychosocial work stressors. In addition, the considerable sample size and low attrition rate are strengths of the study. Furthermore, the outcome, all-cause mortality, was ascertained objectively.

The study is limited in certain ways. Construction of the psychosocial work stressors was limited to those scales and items available in the HILDA survey. Perceptions of job security based on three items and satisfaction with pay based on a single item rather than other psychological benefits of work may not have high validity, and this may partly explain the lack of association between mortality and perceived job security, and mortality and perception of fair pay. Nevertheless, the measures used have been validated and have demonstrated predictable associations with mental and physical health outcomes.[32 34]

Psychosocial work stressors were measured at baseline, which explicitly asserts the predictability of mortality from baseline exposures. This is limited in that a single measurement may be inadequate to assess the effects of exposure duration and changing psychosocial work stressors. Nonetheless, understanding the association between psychosocial work stressors and mortality at a point in time is useful for targeting interventions to improve work quality. Future research will focus on examining the effect of time-varying stressors on health and mortality.

Measurements of psychosocial work stressors were self-reported and may have been subjected to reporting bias. However, external measurements of work stress, such as organisational downsizing, have associated with an increased risk of cardiovascular mortality.[44] Subjective assessment of work stressors may be preferable to objective assessment because health could also be influenced by perceptions of work in addition to the actual conditions of the work environment. Although the exposure measure preceded outcome determination, reverse causation cannot be ruled out as exposure reporting can also be affected by non-work-related illnesses. For example, negative self-assessments of working conditions may be more likely to occur among workers with mental disorders. However, our results were essentially unchanged after adjusting for baseline health measures.

While dichotomising work stressors as high or low by splitting at the sample median is a common operationalisation of psychosocial work factors, groups might not be that different from each other because most individuals centre around the median rather than in the extreme ends of the distribution, resulting in a low exposure contrast. Alternative operationalisations that create more exposure contrast may result in stronger associations; therefore, it is possible that median-split operationalisation may result in underestimation of the associations.[36] The null results observed for most of the ordinal measures may be due to the small number of deaths within ordinal intervals reducing the statistical power.

The socioeconomic, health and behavioural factors adjusted for in our analyses were limited and restricted to baseline measures. We assessed potential confounders (including some which could also be mediators), identified a priori, by serially adjusting for each subgroup of demographic, socioeconomic, health and behavioural factors. There may be an indirect effect of psychosocial work stressors on mortality mediated by poor health and an unhealthy lifestyle. However, health and behavioural factors are strong predictors of mortality as well as being associated with work stress,[45] and adjustment for these risk factors may control for selection into stressful work environments as a result of early risk factors and adverse environments during childhood and adolescence.[46] We included analyses with these adjustments. These adjustments did not substantially change our results for most of the psychosocial work stressors examined in our study, but there is some uncertainty about job demands. While the effect of job demands, measured on the ordinal scale, on mortality remained similar after adjustment for health and behavioural factors, the effect of high job demands versus low job demands on mortality was affected after adjustments. However, it is not clear if this is due to an indirect effect of job demands on mortality, less precision in the fully adjusted model or loss of information in the process of dichotomisation of the exposure job demands.[47] Nonetheless, it remains uncertain if over time these covariates are more likely to be mediators, confounders or a combination of both. We cannot rule out residual confounding by unmeasured risk factors such as personality traits and life stressors.

Furthermore, any study into the effects of work characteristics must be limited to individuals with jobs; hence, there is a potential for bias through the healthy worker effect. Additionally, HILDA may under-represent people with severe health conditions, and generally has a lower proportion of migrants and people from lower socioeconomic groups.[29] Thus, the protective effect from high job demands could in part be explained by a healthy worker effect as healthy individuals are more likely to have jobs with high job demands. While the majority of deaths were confirmed by linkage to the NDI, there were a small proportion (4%) that could not be linked but were included in the analyses nonetheless. Also, there were deaths incorporated into the data files after the data linkage. However, this is unlikely to have resulted in outcome misclassification measurement error as all deaths were verified and confirmed by HILDA survey fieldwork.

Moreover, although the HILDA survey is a national, population-based study, the 66% initial response rate in the first wave suggests that it may not be wholly representative of working Australians. The greater retention of persons of higher versus lower socioeconomic status may affect generalisation.

The disproportionate loss of participants of lower socioeconomic status and lower occupational status, who are more likely to be exposed to poor psychosocial working conditions and adverse psychosocial work stressors, may have biased our results towards the null.[48] However, loss to follow-up in consecutive HILDA waves was low (<10% for most waves).[31] It is also unclear to what extent our results can be generalised to other countries.

CONCLUSION

In conclusion, the results of this study suggest that low job control is associated with an independent increased risk of all-cause mortality. There is some suggestion of reduced risk of mortality associated with high job demands, but the results are not conclusive. This study adds to a growing body of evidence showing an effect of deleterious psychosocial work stressors on mortality. Further research is recommended to examine mechanisms underlying the observed effects between low job control and increased mortality, and to elucidate the relationship between psychosocial work stressors, potential confounders and mediators, and mortality.

Psychosocial work stressors have been identified as significant emerging risks linked to global changes in the structure, organisation and management of work during the previous decades, presenting new challenges to occupational health and safety.[49] Given the high prevalence of exposed individuals, job control-associated increase in mortality thus may have considerable public health consequences. Policy and practice interventions that reduce the adverse impact of low job control and target job demands in stressful work environments could be implemented to improve health and decrease risk of mortality.

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Ethics approval The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008. The study received ethical approval from the Melbourne School of Population and Global Health's Human Ethics Advisory Group (1750707.1). The HILDA survey is conducted by the Melbourne Institute of Applied Economic and Social Research at the University of Melbourne on behalf of the Australian Commonwealth Government Department of Social Services (DSS). All relevant ethical issues associated with this research project (including consent) were addressed by the Melbourne Institute of Applied Economic and Social Research at the University of Melbourne, which operates within the University's Ethics Guidelines. All processes involving consent have been approved by both the University of Melbourne's Human Research Ethics Committee and the Australian Institute of Health and Welfare's Ethics Committee. All members of the group and researchers involved in the HILDA project must comply with the Privacy Act (1988). The authors were provided de-identified data by the organisational data custodian, the names and addresses of which have been removed.

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SUPPLEMENTARY MATERIAL

PSYCHOSOCIAL WORK STRESSORS AND RISK OF MORTALITY IN AUSTRALIA: ANALYSIS OF DATA FROM THE HOUSEHOLD, INCOME AND LABOUR DYNAMICS IN AUSTRALIA SURVEY

Yamna Taouk, Anthony D. LaMontagne, Matthew J. Spittal, Allison J. Milner

SUPPLEMENTARY FILES

Supplementary Table 1. Age-adjusted Hazard Ratios from survival analysis of serially adjusted association between mutually exclusive psychosocial work stressors exposures **job control[1], job demands and complexity[2], job security[3] and fairness of pay[4]** and risk of mortality censored within 3 years of study end

	Adjusted for gender and calendar year			Adjusted for gender, calendar year and socioeconomic status[5]			Adjusted for gender, calendar year, socioeconomic status and health[6]			Adjusted for gender, calendar year, socioeconomic status, health and behavioural factors[7]		
Psychosocial job exposure	N (Total)*	N (Events)	HR (95% CI)	N (Total)*	N (Events)	HR (95% CI)	N (Total)*	N (Events)	HR (95% CI)	N (Total)*	N (Events)	HR (95% CI)
Job control ordinal scale	18430	264	0.98 (0.96 – 1.01)	18377	262	0.98 (0.96 – 1.01)	18377	262	0.99 (0.96 – 1.01)	17689	244	0.99 (0.96 – 1.02)
Low job control	8134	95	1.37 (1.05 – 1.79)	8113	95	1.35 (1.02 – 1.78)	8113	95	1.34 (1.01 – 1.77)	7853	89	1.34 (0.99 – 1.80)
High job control	10296	169	Reference	10264	167	Reference	10264	167	Reference	9836	155	Reference
Job demands ordinal scale	18436	264	0.97 (0.94 – 1.00)	18383	262	0.99 (0.95 – 1.02)	18383	262	0.99 (0.95 – 1.02)	17695	244	0.99 (0.95 -1.02)
Low job demands	10133	156	Reference	10103	154	Reference	10103	154	Reference	9751	144	Reference
High job demands	8303	108	0.73 (0.57 – 0.93)	8280	108	0.78 (0.60 – 1.02)	8280	108	0.78 (0.60 – 1.02)	7944	100	0.77 (0.58 – 1.03)
Job security ordinal scale	18440	264	0.99 (0.96 – 1.02)	18388	262	1.00 (0.97 – 1.04)	18388	262	1.01 (0.97 – 1.04)	17700	244	1.01 (0.98 – 1.04)
Low job security	8793	135	1.15 (0.90 – 1.46)	8770	134	1.03 (0.80 – 1.33)	8770	134	1.01 (0.78 – 1.31)	8390	125	1.00 (0.76 – 1.31)
High job security	9647	129	Reference	9618	128	Reference	9618	128	Reference	9310	119	Reference
Fairness of pay ordinal scale	18400	262	1.00 (0.93 – 1.08)	18348	261	1.02 (0.95 – 1.09)	18348	261	1.02 (0.95 – 1.10)	17662	243	1.03 (0.95 – 1.11)
Low fairness of pay	7667	109	1.14 (0.89 – 1.46)	7643	109	1.10 (0.85 – 1.43)	7643	109	1.09 (0.84 – 1.41)	7321	98	1.06 (0.80 – 1.39)
High fairness of pay	10733	153	Reference	10705	152	Reference	10705	152	Reference	10341	145	Reference

*The number of participants vary because of missing data in covariates.

HR indicates Hazard ratio; CI, confidence interval; N, number

[1]Job control exposure: “freedom to decide how to work”, “a lot of say about what happens”, “freedom to decide when to work”

[2]Job demands and complexity exposure: “job is complex and difficult”, “job requires learning new skills”, “skills and abilities are used in job”

[3]Job security exposure: “secure future in job”, “worry about future of job”, “company will still exist 5 years from now”

[4]Fairness of pay exposure: “get paid fairly for the things I do in job”

[5]Model adjusted for gender, calendar year, country of birth, income, occupation level, household structure; stratified by education, employment arrangement

[6]Model adjusted for gender, calendar year, country of birth, income, occupation level, household structure, long term health conditions; stratified by education, employment arrangement

[7]Model adjusted for gender, calendar year, country of birth, income, occupation level, household structure, long term health condition, alcohol consumption, physical activity; stratified by education, employment arrangement, smoking

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Supplementary Table 2. Age-adjusted Hazard Ratios from survival analysis of serially adjusted association between simultaneously adjusted psychosocial work stressors exposures **job control[1], job demands and complexity[2], job security[3] and perception of unfair pay[4]** and risk of mortality censored within 3 years of study end

			Job control on ordinal scale	Low job control compared with high job control	Job demands on ordinal scale	High job demands compared with low job demands	Job security on ordinal scale	Low job security compared with high job security	Fair pay on ordinal scale	Unfair pay compared with fair pay
Adjustment	N (Total)*	N (Events)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)	HR (95% CI)
Gender, calendar year	18384	260	0.99 (0.96 – 1.01)	1.28 (0.98 – 1.68)	0.98 (0.95 – 1.01)	0.76 (0.59 – 0.98)	1.00 (0.97 - 1.03)	1.06 (0.83 – 1.35)	1.01 (0.94 – 1.09)	1.10 (0.86 – 1.41)
Gender, calendar year and socioeconomic status[5]	18333	259	0.98 (0.96 – 1.01)	1.30 (0.98 – 1.72)	0.99 (0.96 – 1.03)	0.81 (0.62 – 1.05)	1.01 (0.97 – 1.04)	0.98 (0.76 – 1.27)	1.03 (0.95 – 1.10)	1.07 (0.83 – 1.39)
Gender, calendar year, socioeconomic status and health[6]	18333	259	0.98 (0.96 – 1.01)	1.29 (0.98 – 1.72)	0.99 (0.96 – 1.03)	0.80 (0.61 – 1.05)	1.01 (0.97 – 1.04)	0.97 (0.75 – 1.25)	1.02 (0.95 – 1.10)	1.06 (0.82 – 1.38)
Gender, calendar year, socioeconomic status, health and behavioural factors[7]	17647	241	0.99 (0.96 – 1.02)	1.29 (0.96 – 1.75)	0.99 (0.95 – 1.03)	0.80 (0.60 – 1.07)	1.01 (0.98 – 1.04)	0.97 (0.74 – 1.27)	1.03 (0.95 – 1.11)	1.03 (0.78 – 1.35)

*The number of participants vary because of missing data in covariates.

HR indicates Hazard ratio; CI, confidence interval; N, number

[1]Job control exposure: “freedom to decide how to work”, “a lot of say about what happens”, “freedom to decide when to work”

[2]Job demands and complexity exposure: “job is complex and difficult”, “job requires learning new skills”, “skills and abilities are used in job”

[3]Job security exposure: “secure future in job”, “worry about future of job”, “company will still exist 5 years from now”

[4]Fairness of pay exposure: “get paid fairly for the things I do in job”

[5]Model adjusted for gender, calendar year, country of birth, income, occupation level, household structure; stratified by education, employment arrangement

[6]Model adjusted for gender, calendar year, country of birth, income, occupation level, household structure, long term health conditions; stratified by education, employment arrangement

[7]Model adjusted for gender, calendar year, country of birth, income, occupation level, household structure, long term health condition, alcohol consumption, physical activity; stratified by education, employment arrangement, smoking

Chapter 5 Study III - All-cause mortality and the time-varying effects of psychosocial work stressors: a retrospective cohort study using the HILDA survey

This chapter is presented as the peer-reviewed research article published in the Journal of Social Science and Medicine (2020; 266:113452).

5.1 Overview

This published study which this chapter is based on (with supplementary material) examines the effect of repeated exposure to poor-quality work on mortality. While it is generally accepted that exposure to psychosocial work stressors may impact on health it is not clear whether exposure over time translates into an increased risk of mortality. This study was the first examination of repeated exposure to poor psychosocial work stressors including high job demands, low job control, job insecurity and effort-reward imbalance over time in Australian workers. Low job control and job insecurity were found to be associated with increased risk of mortality, though associations were stronger for men than women.

5.2 Research Questions and Objectives

5.2.1 Research questions

The overall research question was is exposure to adverse psychosocial work stressors over time associated with increased risk of death?

The specific research questions are:

1. What is the effect of repeated exposures to adverse psychosocial work stressors including high job demands, low job control, job insecurity and effort-reward imbalance over time on all-cause mortality?

2. Is the relationship between exposure to adverse psychosocial work stressors over time and mortality modified by gender?
3. What is the effect of long-term exposure to adverse psychosocial work stressors on "on the job deaths"?

5.2.2 Research objectives

1. To examine the effect of time-varying exposures and covariates on mortality in a longitudinal study of Australian workers including the effects of unemployment, not in the workforce and retirement over a period of 15 years.
2. To investigate if gender is an effect-modifier on the relationship between long-term exposure to adverse psychosocial work stressors and mortality.
3. To examine the effect of exposure to adverse psychosocial work stressors over time on deaths occurring in the labour force.

5.3 Publication

5.3.1 Main document and supplementary material

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All-cause mortality and the time-varying effects of psychosocial work stressors: A retrospective cohort study using the HILDA survey

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ABSTRACT

The effects of poor-quality work (high job demands, low job control, job insecurity, and effort-reward imbalance) are harmful to health but it isn't clear whether exposure to these psychosocial work stressors over time translates into increased risk of mortality.

Objective: We investigated the effect of time-varying psychosocial work stressors on mortality using data from a longitudinal cohort of working Australians by examining association between job control, job demands, job insecurity, unfair pay overtime and all-cause mortality. We examined whether gender modified these relationships.

Methods: Over 20,000 participants from the Household Income and Labour Dynamics in Australia survey with self-reported repeated exposure measures were followed for 15 years. Survival analysis models with baseline hazard specified by the Weibull distribution were used to examine the association between psychosocial work stressors over time and mortality.

Results: Low job control (HR=1.39; 95% CI: 1.06-1.83) and job insecurity (1.36; 1.06-1.74) were associated with increased risk of mortality. High job demands (1.01; 0.75-1.34) and effort-reward unfairness (1.20; 0.90-1.59) were not associated with mortality. The effect of job insecurity was attenuated (1.20; 0.93-1.54) after controlling for sociodemographic and health risk factors. Male participants exposed to low job control and job insecurity had an 81% and 39% increase risk of mortality, respectively.

Conclusions: Long-term exposure to low job control and low job security is associated with increased risk of all-cause mortality. Effects were largely restricted to males and persisted after adjustments for sociodemographic and health characteristics. The lack of effects observed for females may have been due to the small number of deaths in females. Awareness of implications of the adverse effects of psychosocial work stressors on health and mortality in workplaces, and interventions to improve job control and job security could contribute to better health and wellbeing, reducing the effect of psychosocial work stressors on mortality.

Authors' contributions

Yamna Taouk designed the study, performed the statistical analyses and wrote the manuscript. Matthew J. Spittal contributed to the study design and reviewd the manuscript. Allison J. Milner provided logistic support at all stages of the study and reviewed the manuscript. Anthony D. LaMontagne contributed to the conception and design of the work, provided support and reviewed the manuscript.

1. Introduction

Work has a powerful influence on health. Not only does it provide the means of earning a source of income sufficient for supporting people's lifestyles, but also it has an important social and psychological role in people's lives. Work offers people opportunities for expansion of their social connections, increases their participation in a community, and provides a sense of purpose and identity (Dooley et al., 1996; Jahoda, 1981). Inclusion of non-working people in the working-age population might be by choice or necessity (not in the labour force), or otherwise (unemployment). Even so, the ill-effects of not working when seeking

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work (i.e., being unemployed) on mental and physical health are well documented (Jin et al., 1995; Paul and Moser, 2009).

An emerging body of research has demonstrated the effects of poor-quality work (characterised by high job demands, low job control, job insecurity, and effort-reward imbalance) on health and well-being (Fishta and Backe, 2015; Nyberg et al., 2014; Stansfeld and Candy, 2006). In fact, some evidence suggests that poor quality work may be as bad (or even worse) for health than unemployment (Butterworth et al., 2013; Chandola and Zhang, 2018; Milner et al., 2015). The effects of high job demands (the need to work quickly and intensely), and low job control (lack of control over skill use, time allocation, and organisational decisions) in the work environment may result in stress-related ill health (Karasek and Theorell, 1990; Karasek, 1979). Recurrent imbalances in the rewards associated with work (e.g. renumeration, recognition, career advancements, and job security) and efforts spent at work (e.g. workload, duration, and intensity of work efforts), resulting in lack of reciprocity in terms of high efforts and low rewards may also give rise to stress-related health outcomes (Siegrist, 1996). Job insecurity (one's perceptions regarding employment continuation and the consequences of job loss) (De Witte, 2005) also appears to influence mental and physical health (Kim and von dem Knesebeck, 2016; Sverke et al., 2002).

There have been numerous studies demonstrating the adverse effects of exposure to psychosocial work stressors on physical health, particularly cardiovascular health (Fishta and Backe, 2015; Huang et al., 2015; Nyberg et al., 2014; Virtanen et al., 2013) and mental health (Stansfeld and Candy, 2006). However, it has not been fully established whether exposure to these psychosocial work stressors translates into an increased risk of mortality. Relatively few studies have examined the relationship between psychosocial work stressors and mortality and results to date, including investigation of gender-specific effects, have been inconsistent (Eaker et al., 2004; Karasek et al., 1981; Kivimaki et al., 2002; Padyab et al., 2014; Shirom et al., 2011; Slopen et al., 2012; Taouk et al., 2020b). This may be due to different study designs and methodological approaches. Critically, very few studies on mortality have assessed time-varying psychosocial work stressors, allowing for changing exposures over time. Typically, studies measure the exposure only once at baseline, which may also explain inconsistent findings between studies (Johnson et al., 1996; Landsbergis et al., 2000; Taouk et al., 2020a).

Using only baseline measurements assumes that all relevant information pertaining to a person's experience of psychosocial work stressors can be accurately summarised at this one-time point and remains constant or represents life course exposure from that point onwards. Nor is it clear how much or how often work stressors vary over time given the changing landscape of contemporary workplaces, where changing occupations and career mobility due to digital disruption are becoming more common. In support of this, there is some evidence that psychosocial work stressors are not static (Bentley et al., 2015; LaMontagne et al., 2019; LaMontagne et al., 2013). Two studies have examined the relationship between mortality and psychosocial work stressors over time and found that consistent low job control over time is strongly associated with an increased risk of death (Amick et al., 2002; Johnson et al., 1996).

In contrast, the benefits of using time-varying measures of work stressors allows for the assessment of changes in psychosocial work stressors, as well as changes in socioeconomic, occupational and well-being characteristics over time. Further advantages to examining changing psychosocial work stressors is the capacity for including transitions from employment in and out of unemployment and retirement to measure their impact on the relationship between mortality and work stressors. It is known that retirement and unemployment are strong predictors of mortality (Kasl and Jones, 2000), yet in most research on exposure to psychosocial work stressors and mortality transitions to unemployment and retirement have not been assessed.

The present study was designed to examine the effect of time-varying psychosocial work stressors on mortality using data from a longitudinal cohort of working Australians with over 15 annual waves of data. This is one of the few studies to examine the relationship time-varying exposure to work stressors and mortality over a substantial period of observation and follow-up in a nationally representative working sample. In the present study we examined the association between job control, job demands, job insecurity and unfair pay over time – all measured as time-varying variables – and all-cause mortality. We also examined whether gender modified these relationships.

2. Method

2.1. Study population

Data from the Household, Income and Labour Dynamics in Australia (HILDA) survey were used in the analyses. Commenced in 2001, HILDA is a nationally representative longitudinal study of Australian households following over 17,000 Australian lives annually (Wilkins, 2017). The HILDA survey contains extensive data on employment and labour-force characteristics of working Australians with over 15 annual waves of data and has been linked to the National Death Index. Households comprise the source of the panel data and are interviewed in each wave of data collection from a combination of face-to-face interviews and self-completion questionnaires. In Wave 1, HILDA had 13,969 respondents from 7,682 households (66% response rate). Retention at Wave 2 was 87% and greater than 90% at subsequent waves. In Wave 11, the sample was topped-up by 4,009 people from 2,153 households to correct for population under-coverage and retain cross-sectional representativeness in the sample to include representation of migrant populations and help alleviate biases from non-random attrition (Watson, 2011).

2.2. Study sample

The analysis is restricted to working-age participants who were employed for one or more waves, responded to interviews and provided the self-reported questionnaires data over a period of 15 waves of data. There were 29,810 participants aged over 15 years during waves 1 to 15 in the HILDA survey, but 7,621 were excluded because they were never employed at any time during the survey. Furthermore, participants who never provided self-reported psychosocial data at any time during the survey were excluded so the final analytical sample comprised 20,423 participants (Fig. 1). Of these, 99.7% of participants had complete information on sociodemographic factors and health status.

2.3. All-cause mortality outcome

The entire HILDA Survey was matched to the National Death Index (NDI) in 2014. Briefly, 1,238 deaths had already been identified through the HILDA survey fieldwork and an additional 333 deaths were identified by the data linkage. Sixty-four deaths (4%) identified via fieldwork could not be matched to the NDI. A further 213 deaths have been identified after the data linkage. A total of 1,784 deaths were confirmed by NDI linkage or identified by fieldwork in HILDA (Watson and Summerfield, 2014).

2.4. Psychosocial work stressors

Self-reported psychosocial work stressors were measured at every wave of the HILDA survey from 2001 to 2015. The items and scales available in all waves of the HILDA survey were based on a variety of economic and social surveys (Summerfield et al., 2016), and three separate measures of the psychosocial work environment, previously identified through factor analysis and structural equation modelling were constructed. These were: job demands and complexity (three items, $\alpha = 0.72$), job control (three items, $\alpha = 0.83$), and perceived job security (three items, $\alpha = 0.66$) (Leach et al., 2010). The effort-reward

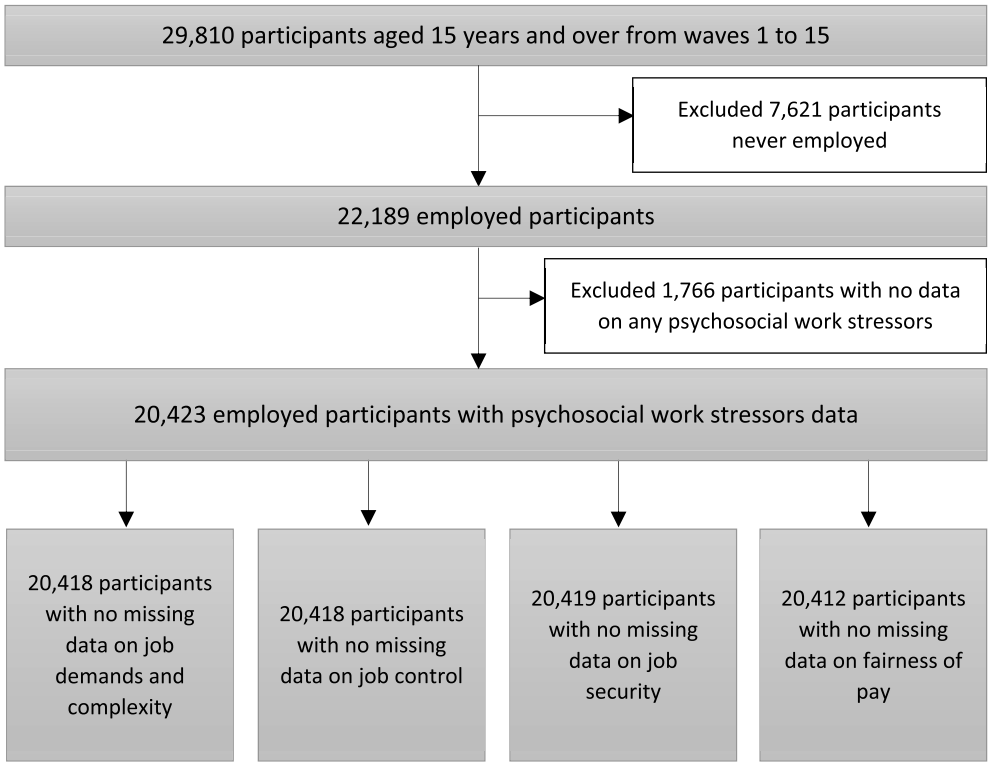

Fig. 1. Flow chart showing selection of HILDA participants for the analyses.

imbalance model maintains that work stressors result from a perceived inequality by workers between their efforts at work and a range of rewards (Siegrist, 1996), but only a single item in HILDA assessed financial rewards relative to effort was available for assessment of any aspects of effort-reward imbalance. These psychosocial work measures have strong associations with more widely used measures including high job demands, low job control, low job security, and effort-reward unfairness (Butterworth et al., 2011; Leach et al., 2010).

Time-varying measures of each of these psychosocial work stressors were constructed by considering each stressor at each time point as a separate observation. All items were re-coded in the same direction, such that high scores represented higher control, greater demand and complexity, greater security, and higher fairness of pay; and scores for each scale were summed. For those participants with missing data for some items (1.6%), scale scores were based on completed items and weighted up to the expected total had all items been answered. As the psychosocial work stressors exposure is a relative measure (Karasek et al., 1981; Stansfeld et al., 1999), the choice of cut points was constructed to identify approximately a third of the participants as experiencing the most adverse levels of each psychosocial work stressor. Scales were therefore divided into tertiles. For each psychosocial stressor exposure measured, participants with scores in the tertile of the distribution corresponding to the most adversity (lowest job control, highest job demands, lowest job security, lowest perceptions of fair pay) were categorised as exposed. The remaining two tertiles for each scale were combined to create the reference category.

Covariates.

Potential confounders were identified a priori, including labour force status, sociodemographic factors and health status. Wave of first entry into HILDA survey was grouped into two levels (2001–2010 and 2011–2015) to control for the top up sampling that occurred in 2011. Age was calculated in ten-year bands (15–24, 25–34, 35–44, 45–54, 55–64, 65+) and fixed at time of study entry. An additional seven sociodemographic factors were assessed. Two were time-invariant (gender and country of birth) and the rest were time-varying (education, household structure, occupation, employment arrangement and income) and assessed at the year preceding the current interview year. Country of birth was classified as Australia, other English-speaking country, or other country. Educational attainment was categorised as no completion of high school, completion of high school or vocational certificate/diploma, or bachelor's degree or above. Household structure was grouped as couple with no children, couple with children, lone parent with children, lone person, or other.

Occupational skill level (low, medium, high) was defined according to Australian and New Zealand Standard Classification of Occupational groupings (ABS, 2013), and employment arrangement was classified as permanent, casual or labour hire, fixed term, or self-employed. Household disposable income, calculated by summing the income components for the previous financial year for all adults in the household, was equivalised using the modified OECD scale (Hagenaars et al., 1994). For each wave of data, nominal household income values were converted to quintiles of the Australian population distribution using percentile statistics for the corresponding financial year from the ABS biennial Survey of Income and Housing (ABS, 2017). The highest quintile of the population distribution of disposable household income was used as the referent group.

Employment status was ascertained yearly from a work-status question in the HILDA survey and participants were classified as employed, unemployed, or not in the labour force (NILF)/retired for the year preceding the current interview year. Finally, health status was ascertained from a question asking about any long-term disability, or mental or physical health conditions lasting 6 months or more that restricted everyday activity and not corrected by medication or medical aids for the year preceding the current interview year.

2.5. Statistical analysis

We constructed a person-wave dataset (multiple observations per person where each observation recorded the value of each time-varying covariate at each wave). Time at risk commenced at wave of first

recorded employment and ceased at time of death, or year of last interview. Participants who transitioned into long-term unemployment or NILF/retirement but continued in the survey were censored five years following their last year of employment. We did this based on the conservative assumption that the effect of psychosocial work stressor exposures may persist for a period of up to five years after a person exited the labour force (Amick et al., 2002; McDonough et al., 1997). For these participants, psychosocial work stressor exposures were fixed at time of last interview with employment data. To capture the effects of unemployment and NILF/retirement, this variable was measured yearly and included in the analyses as a confounder. Participants' occupation and employment arrangement data at time of last employment were carried forward to time of last interview or death for participants who ceased to work during the study.

We used fully parametric survival analysis models to calculate hazard ratios (*HR*) and 95% confidence intervals (*CI*) of the association psychosocial work stressors and mortality. The baseline hazard was specified using the Weibull distribution. Two regression models were fit to the data. In addition to the work stressor exposures, the initial model adjusted for age at entry into study, year of entry into survey, gender, and labour force status. The subsequent model evaluated the robustness of the results with the inclusion of sociodemographic and health covariates. Effect modification was tested by the change in the log-likelihood ratio after the addition of gender-stressor product terms in the models. We conducted our study using complete case analysis. We calculated cluster-adjusted robust standard errors to account for correlations among participants from same households. Additional sensitivity analyses examined the inclusion of all psychosocial work stressors in the models; and censoring individuals at time of last employment. All analyses were conducted using STATA/SE version 14.2.

3. Results

The analytic sample consisted of 20,423 participants and 154,634.2 person-years. During the median follow-up of six years, 247 deaths were observed (mortality rate 16.0 (14.1–18.1) per 10,000 person-years), and 70% of deaths occurred in males. The mean age at study entry was 33.4 (*SD* = 13.9) years. There were 34% of participants aged 15–24 years, 22% 25–34 years, 20% 35–44 years, and 24% aged ≥45 years. Half of the participants were male (50%), and most were born in Australia (80%), resided in couples with or without children households (73%) and did not have long-term health conditions (85%). Twenty-two percent of participants had not completed their high school education and 27% had a tertiary education qualification. Thirty-one percent of participants were in the highest population quintile of disposable income, 29% were employed in high skills occupations and 47% were permanently employed (Table 1). Table 2 presents details of the scales, cut points, the proportion of participants in the most adverse tertile for psychosocial work stressors exposures.

Compared to those with high job control, low job control was associated with a 1.39 times higher risk of mortality (Table 3). Similarly, compared to those with high job security, low job security was associated with increased risk of mortality (*HR* = 1.36; *95% CI*: 1.06, 1.74). High job demands and low fairness of pay were not associated with mortality (*HR* = 1.01; *95% CI*: 0.75, 1.34 and *HR* = 1.20; *95% CI*: 0.90, 1.59 respectively). Results did not change substantially with the inclusion of sociodemographic and health covariates in the models, although the effect of low job security was attenuated to include the null (*HR* = 1.20; *95% CI*: 0.93, 1.54). Male gender, older age, no tertiary qualifications, high skills occupation, living alone, long-term health condition and no longer in the labour force were also associated with increased hazard risks of mortality (data not shown).

There was moderate evidence of effect modification by gender on the associations between job control and mortality ($p = 0.02$ for the primary model and $p = 0.01$ for the model further adjusted for sociodemographic and health factors) and some evidence for job security and mortality ($p = 0.04$ and $p = 0.06$). Results from separate multivariate regression models for each psychosocial work stressor exposure and mortality stratified by gender are therefore presented in Table 4. Increased risk of death were observed for low job control (*HR* = 1.71; 95% *CI*: 1.24, 2.36) and low job security (*HR* = 1.62; 95% *CI*: 1.20, 2.17) for males, and these effects persisted after adjustment for sociodemographic and health covariates (low job control *HR* = 1.81; 95% *CI*: 1.29, 2.53; and low job security *HR* = 1.39; 95% *CI*: 1.02, 1.88). There was no association between high job demands and mortality for males but a suggestion of increased risk for low fairness of pay and mortality for males (*HR* = 1.34; 95% *CI*: 0.96, 1.87); this association was attenuated after adjustment for sociodemographic and health covariates (*HR* = 1.16 95% *CI*: 0.83, 1.64).

Table 1
Descriptive statistics of final analytic sample.

Sample	
No. participants	20,423
Person-years	154,634.2
Median (IQR) follow up (years)	6 (3–14)
Observed transitions	
No. deaths	247
No. participants at least one wave unemployed	1,156
No. participants at least one wave no longer in labour force	5,630
Rate of death per 10,000 person-years	16.0 (14.1–18.1)
Wave of first entry into HILDA	
2001–2010	14,856 (72.7)
2011–2015	5,567 (27.3)
Sociodemographic factors	
	mean (*SD*)
Age (years)	33.4 (13.9)
Age group	***N (%)***
15–24 years	6,996 (34.3)
25–34 years	4,490 (22.0)
35–44 years	4,093 (20.0)
45–54 years	3,118 (15.3)
55–64 years	1,456 (7.1)
65+ years	270 (1.3)
Gender	
Male	10,278 (50.3)
Female	10,145 (49.7)
Country of birth	
Australia	16,266 (79.6)
English Speaking	1,888 (9.2)
Other	2,264 (11.1)
Missing	5 (0.00)
Education	
Bachelor's degree and above	5,595 (27.4)
Year 12/Certificate/Diploma	10,323 (50.5)
School not completed	4,505 (22.1)
Household structure	
Couple with no children	5,148 (25.2)
Couple with children	9,815 (48.1)
Lone parent with children	2,050 (10.0)
Lone person	1,529 (7.5)
Other	1,881 (9.2)
Population quintiles of disposable income	
1 (lowest)	1,727 (8.5)
2	2,951 (14.4)
3	4,321 (21.2)
4	5,183 (25.4)
5 (highest)	6,241 (30.6)
Occupational skill level	
High	5,820 (28.5)
Medium	7,732 (37.9)
Low	6,851 (33.5)
Missing	20 (0.1)
Employment Arrangement	
Permanent	9,536 (46.7)
Casual	6,795 (33.3)
Fixed Term	1,468 (7.2)
Self employed	2,592 (12.7)
Missing	32 (0.2)
Health	
Long-term health conditions	2,993 (14.7)

Note. Sociodemographic and health characteristics at baseline.

Table 2
Description of psychosocial work stressors measures.

Item/scale	Number of observations	Range	Mean (*SD*)	Cut point	% most adverse tertile
Job demands and complexity[a]	118,168	3–21	13.9 (4.1)	>16	29.4
Job control[b]	118,157	3–21	12.8 (4.8)	<11	32.6
Job security[c]	118,187	3–21	15.9 (3.8)	<14	33.3
Effort-reward unfairness[d]	117,988	1–7	4.7 (1.7)	<4	24.7

[a] Job demands and complexity exposure: "job is complex and difficult", "job requires learning new skills", "skills and abilities are used in job."
[b] Job control exposure: "freedom to decide how to work", "a lot of say about what happens", "freedom to decide when to work."
[c] Job security exposure: "secure future in job", "worry about future of job", "company will still exist 5 years from now."
[d] Perception of unfair pay exposure: "get paid fairly for the things I do in job."

There was no evidence for associations between any psychosocial work stressor and mortality for females.

Results of additional sensitivity analyses including all psychosocial work stressors in the same model are presented in Supplementary Table 1. When all psychosocial work stressors were simultaneously adjusted for, low job control and low job security were observed to be independently associated with mortality (*HR* = 1.37; 95% *CI*: 1.04, 1.80 and *HR* = 1.31; 95% *CI*: 1.02, 1.69 respectively) and there was no evidence that high job demands or low fairness of pay was associated with mortality. The effect of low job control on mortality persisted after adjusting for sociodemographic and health covariates but the risk estimate for low job security was attenuated to include the null (*HR* = 1.37; 95% *CI*: 1.03, 1.83 and *HR* = 1.16; 95% *CI*: 0.90, 1.50 respectively). As observed in the separate multivariate regression models, increased hazards of death were observed for low job control (*HR* = 1.66; 95% *CI*: 1.20, 2.29) and low job security (*HR* = *1.52*; 95% *CI*: 1.13, 2.04) for males, but these effects were attenuated to include the null for low job security (*HR* = 1.30; 95% *CI*: 0.95, 1.76) after adjustment for sociodemographic and health covariates.

Results of a further sensitivity analysis examining the effect of psychosocial work stressors on mortality in workers who did not retire or permanently exit the labour force over the study period are presented in Supplementary Table 2. There were 145 deaths observed when individuals were censored at time of last employment. Low job control was associated with increased mortality controlling for other work stressors (*HR* = 1.54; 95% *CI*: 1.07, 2.20) and further adjusting for sociodemographic and health factors (*HR* = 1.46; 95% *CI*: 1.01, 2.13). There was no evidence that high job demands, low job security or low fairness of pay was associated with mortality in workers who were in the labour force for the duration of the study period.

4. Discussion

Our results suggest that when a person has a lack of control over their job, they are at an increased risk of death. Participants exposed to low job control had a 39% increased risk of all-cause mortality relative to participants with high job control, even when controlling for periods of unemployment and the effect of NILF/retirement on mortality. The excess mortality risk did not change appreciably after adjustment for sociodemographic information, which included measures of socioeconomic status, and health-related risk factors. The association was robust to adjustment for various potential confounders, including some that may also be mediators.

We also found that participants exposed to low job security over time had a 36% increase risk of all-cause mortality relative to participants with high job security controlling for the effect of unemployment and retirement on mortality though the risk was attenuated after adjustment for sociodemographic and health characteristics. There were significant differences by gender. Male participants exposed to low job control and low job security had an 81% and 39% increase risk of mortality, respectively, after adjustment for sociodemographic and health risk factors. The observed effects of low job control and low job security on mortality in models with simultaneously adjusted psychosocial work stressors were similar to the effects observed for the separate psychosocial work stressor and mortality. Restricting the analyses to workers who did not retire or permanently exit the labour force over the study period strengthened the association between low job control and mortality.

The findings for job demands and fairness of pay were inconclusive. There was no evidence that exposure to high job demands over time was associated with mortality. A weak effect of fairness of pay, a measure of effort-reward imbalance, over time on mortality was observed but its influence was diminished after adjustment for sociodemographic and health covariates.

To date, only a small number of prospective studies have examined how job control relates to mortality. The results of these studies have been largely inconsistent, with some reporting that low job control increased mortality (Kivimaki et al., 2006; Toivanen and Hemstrom, 2006), whilst others did not find an association (Eaker et al., 2004; Padyab et al., 2014). Yet, in these studies, psychosocial work stressors were measured once at baseline to represent working life course exposure, whereas this study examined the effect of changing psychosocial work stressors exposures over time. Two previous studies have examined the effect of long term exposure to low job control on prospectively measured mortality risk (Amick et al., 2002; Johnson et al., 1996). Johnson et al. reported that workers with low control over their job had

Table 3
Hazard Ratios (and standard errors) of separate time-varying psychosocial work stressors exposures job control, job demands and complexity, job security and fairness of pay and risk of mortality multivariate regression models.

Psychosocial work stressor exposure	Primary model[a]				Model further adjusted for sociodemographic and health factors[b]			
	Person-years	*N* (Events)	*HR* (95% *CI*)	*p*	Person-years	*N* (Events)	*HR* (95% *CI*)	*p*
Low job control[c]	151152.5	247	1.39 (1.06, 1.83)	0.019	151003.2	246	1.39 (1.04, 1.86)	0.026
High job demands[d]	151149.5	247	1.01 (0.75, 1.34)	0.969	151000.2	246	1.06 (0.79, 1.43)	0.700
Low job security[e]	151147.2	246	1.36 (1.06, 1.74)	0.015	151003.9	245	1.20 (0.93, 1.54)	0.170
Low fairness of pay[f]	151077.8	245	1.20 (0.90, 1.59)	0.215	150932.8	245	1.07 (0.80, 1.43)	0.649

[a] Model adjusted for age at entry into study, year of entry into survey, gender, and time-varying labour status.
[b] Model adjusted for age at entry into study, year of entry into survey, gender, time-varying labour status, time-varying sociodemographic factors, and time-varying health status.
[c] Low job control exposure compared with the combination of intermediate and high job control exposure tertiles.
[d] High job demands exposure compared with the combination of low and intermediate job demands exposure tertiles.
[e] Low job security exposure compared with the combination of intermediate and high job security exposure tertiles.
[f] Low fairness of pay exposure compared with the combination of intermediate and high fairness of pay exposure tertiles.

Table 4
Separate multivariate regression models for each psychosocial work stressor exposure job control, job demands and complexity, job security and fairness of pay and the hazard of death for males and females.

Psychosocial work stressor exposure	Males				Females				p for interaction by gender	
	Primary model[a]		Model further adjusted for sociodemographic and health factors[b]		Primary model[c]		Model further adjusted for sociodemographic and health factors[d]			
	HR (95% *CI*)	*p*	HR (95% CI)	p	HR (95% CI)	p	HR (95% CI)	p	Primary model	Adjusted model
Low job control[e]	1.71 (1.24, 2.36)	0.001	1.81 (1.29, 2.53)	0.001	0.88 (0.53, 1.46)	0.612	0.78 (0.46, 1.32)	0.349	0.02	0.01
High job demands[f]	0.98 (0.69, 1.38)	0.901	1.04 (0.73, 1.47)	0.844	1.06 (0.62, 1.81)	0.819	1.07 (0.62, 1.84)	0.811	0.78	0.71
Low job security[g]	1.62 (1.20, 2.17)	0.001	1.39 (1.02, 1.88)	0.035	0.87 (0.53, 1.44)	0.595	0.83 (0.49, 1.40)	0.483	0.04	0.06
Low fairness of pay[h]	1.34 (0.96, 1.87)	0.084	1.16 (0.83, 1.64)	0.380	0.92 (0.54, 1.57)	0.764	0.85 (0.50, 1.45)	0.541	0.25	0.31

[a] Model adjusted for age at entry into study, year of entry into survey, and time-varying labour status for male participants.
[b] Model adjusted for age at entry into study, year of entry into survey, time-varying labour status, time-varying sociodemographic factors and time-varying health status for male participants.
[c] Model adjusted for age at entry into study, year of entry into survey, and time-varying labour status for female participants.
[d] Model adjusted for age at entry into study, year of entry into survey, time-varying labour status, time-varying sociodemographic factors and time-varying health status for female participants.
[e] Low job control exposure compared with the combination of intermediate and high job control exposure tertiles.
[f] High job demands exposure compared with the combination of low and intermediate job demands exposure tertiles.
[g] Low job security exposure compared with the combination of intermediate and high job security exposure tertiles.
[h] Low fairness of pay exposure compared with the combination of intermediate and high fairness of pay exposure tertiles.

a relative risk of 1.83 (95% *CI*: 1.19, 2.82) for cardiovascular disease mortality and Amick et al. found that typically working in low control jobs over the working life course was associated with a 43% (95% *CI*: 1.13, 1.81) increase in the chance of death. These results are largely consistent with ours, despite both being from different countries and earlier periods. A previous study by the authors examining baseline exposures of psychosocial work stressors and mortality reported almost identical results for low job control and mortality (*HR* 1.39; 95% *CI*: 1.04 to 1.85) (Taouk et al., 2020a). In our study, this suggests that baseline exposure is a reasonable proxy for our time-varying measure of current exposure, which can be simplistically understood as longer-term time--weighted average exposure.

The findings from previous studies investigating job demands and mortality with baseline psychosocial work stressors exposure measures have been inconsistent. Some studies found no associations (Padyab et al., 2014; Shirom et al., 2011), while others found that high job demands increased deaths due to cardiovascular disease (Karasek et al., 1981; Kivimaki et al., 2006). One study that examined 'cumulative exposure' to psychological job demands and cardiovascular disease mortality found that higher levels of psychological job demands were inversely associated with cardiovascular disease mortality risk (Johnson et al., 1996). However psychological job demands exposure scores were assigned in this study with a job exposure matrix based on occupational history. This different approach to the measurement of exposure means that the results of this previous study aren't directly comparable to ours (Landsbergis et al., 2000). Another study's findings were more consistent with the results from this study for job demands over time and mortality, in that it reported no association for long term exposure to high job demands and risk of death (Amick et al., 2002).

Our findings for low job security were not consistent with results from the few available studies in the literature. A study examining job insecurity and deaths from cardiovascular disease over a ten-year period in women reported that there was no evidence of an association (*HR* = 1.41; 95% *CI*: 0.75, 2.65) although the direction of the risk indicated excess risk of cardiovascular death for women reporting job insecurity (Slopen et al., 2012). However, their analysis was limited by low power due to the small number of observed cardiovascular deaths ($n = 52$) in their sample and their job-related stressors exposures were measured once at baseline. A second study investigated associations between job insecurity and mortality for working-age respondents using 8 rounds of data from the Russia Longitudinal Monitoring Survey (Perlman and Bobak, 2009). They reported hazards risk of 1.00 (95% *CI*: 0.77, 1.31) for men and 1.20 (95% *CI*: 0.60, 2.37) for models adjusted for age, district, household cluster, education and occupation. However, reliability of their mortality data was hampered by the relatively few deaths observed among the respondents especially among women, and job insecurity was measured once at baseline at the time when respondents entered the survey.

There is relatively little previous research with which to compare our findings with respect to effort-reward imbalance. One study found increased hazards risks of 2.54 (95% *CI*: 1.1, 5.9) for cardiovascular deaths, and 1.08 (95% *CI*: 0.7, 1.8) for all-cause mortality, for high effort reward imbalance compared with low effort-reward imbalance controlling for socioeconomic position (Brunner et al., 2004). The difference in results may be explained by a combination of cultural differences in the way effort-reward imbalance questions are interpreted and differences in methods of measurements. Further research on the relationship between effort-reward imbalance and mortality is warranted given the lack of available studies.

There is precedent for our finding of a stronger relationship among men than women, although previous studies are mixed (De Witte, 2005; Kim & von dem Knesebeck, 2016; Watson and Osberg, 2017). One possible explanation is "role theory": the notion that work is typically more central to identity among males than among females (Watson and Osberg, 2017). However, there may have been insufficient power to detect associations between mortality and work stressors among females given that the majority of deaths (70%) occurred in males.

4.1. Strengths and limitations

The strengths of the study include the examination of the changing relationship between psychosocial work stressors and mortality, with long follow-up time and low attrition rates, using a large national sample which has a strong focus on employment and labour-market conditions. The study had access to individual-level measures of job exposure and used validated measures of psychosocial work stressors. Furthermore, we had an objective measure of outcome that was independent of the exposure data source, thereby avoiding dependent misclassification bias.

The study adds to the small body of evidence showing an association between work stressor exposure and mortality (Amick et al., 2002; Karasek et al., 1981; Kivimaki et al., 2002; Toivanen and Hemstrom, 2006). However, whilst repeated measures of time-varying exposures were used to assess the effect of changing psychosocial work stressors over time on mortality, we cannot be certain that exposure misclassification did not influence our results. Measurements of psychosocial work stressors were self-reported and may have been subjected to reporting bias. Subjective assessment of work stressors may be preferable to objective assessment because health could also be influenced by perceptions of work in addition to the actual conditions of the work environment.

Although the exposure measure preceded the outcome, reverse causation cannot be ruled out, in the case where workers are affected by non-work-related illness resulting in negative self-assessments of working conditions. However, this is unlikely to have occurred because our results were essentially unchanged after adjusting for health of participants as a time-varying covariate for the duration of their involvement in the study. Additionally, we adjusted for changing employment status by the inclusion of periods of unemployment and NILF/retirement in our models to capture possible alternative psychosocial causes of increased mortality risk. The absence of lifestyle risk factors strongly associated with mortality such as smoking, alcohol and physical activity may limit the study's conclusions but these risk factors are more likely to be on the pathway between, rather than confounders of, the association between psychosocial work stressors and mortality and controlling for these mediators would represent overcontrol and an underestimation of risk.

Our study is limited with respect to causal inference, however, the observed associations are biologically plausible. Changes in brain stress-responsive neurocircuitry stimulate peripheral physiological responses in the sympathetic autonomic nervous system and hypothalamus–pituitary–adrenal (HPA) through the release and control of adrenaline and stress hormone cortisol throughout the body increasing inflammatory protein levels. To cope with stress, psychological and physiological processes are altered, resulting in chronic over- or under-activity of allostatic systems setting new baselines levels, causing deterioration of body systems from repeated and unresolved stressors (Kivimaki and Steptoe, 2018; McEwen, 1998). Further research is required to examine mechanisms underlying the observed effect of work stressors on mortality to improve causal inference. As such we plan on further investigating effects of mediating factors on the pathway between psychosocial work stressors and health.

Our study was limited by lack of power due to the small number of mortality events observed in our data. The number of deaths included in the sample was small, involving 247 participants and a mortality rate of 16.0 per 10,000 person-years. Less than 1/7th of HILDA deaths were included in the main analyses, as most people who died were long-term retirees, and fewer than 1/12th of HILDA deaths were included in additional sensitivity analyses of on-the-job mortality. This was more of a concern for female participants as most deaths were observed in males, hence we cannot be certain that the lack of effects observed for females was in fact due to the lack of power in our study to detect associations between psychosocial work stressors and mortality for females. This limitation may have resulted in an underestimation of our risk estimates. Though previous studies have shown evidence of associations between psychosocial work characteristics and cardiovascular morbidity and mortality (Kivimaki and Steptoe, 2018; Taouk et al., 2020b), we could not examine deaths due to cardiovascular heart disease. There was insufficient power to examine cause-specific mortality as a substantial number of deaths (~50%) in HILDA did not have cause of death information available (Watson and Summerfield, 2014).

Furthermore, any study into the effects of work characteristics must be limited to individuals with jobs, hence there is a potential for bias through the healthy worker effect. Additionally, HILDA may underrepresent people with severe health conditions, and generally has a lower proportion of migrants and people from lower socioeconomic groups (Watson, 2011). Whilst the majority of deaths were confirmed by linkage to the NDI, there were a small proportion (4%) that could not be linked but were included in the analyses nonetheless. Additionally, there were deaths incorporated into the data files after the data linkage. However, this is unlikely to have resulted in outcome misclassification measurement error as all deaths were verified and confirmed by HILDA survey fieldwork.

Finally, although the HILDA survey is a national, population-based study, the 66% initial response rate in the first wave suggests that it may not be wholly representative of working Australians. The greater retention of persons of higher versus lower socioeconomic status may affect generalisation. The disproportionate loss of lower SES and lower occupational status participants, who are more likely to be exposed to poor psychosocial working conditions and adverse psychosocial work stressors, would have biased our results towards the null (LaMontagne et al., 2013). However, loss to follow-up in consecutive HILDA waves was low; less than 10% for most waves(Summerfield et al., 2016). It is also unclear to what extent our results can be generalised to other countries.

5. Conclusions

The current study suggests that long-term exposure to low job control and low job security is associated with increased risk of all-cause mortality. Effects were largely restricted to males and persisted after adjustments for sociodemographic and health characteristics, as well as simultaneous adjustment for all psychosocial work stressors exposures. The results strongly suggest that, over time, exposure to low job control and low job security have independent effects on mortality. Due to the small number of deaths observed in females, the absence of evidence in females is not evidence of absence. Psychosocial work stressors have been identified as significant risks linked to global changes in the structure, organisation, and management of work during the previous decades, presenting new challenges to occupational health and safety (Landsbergis et al., 2014; Leka et al., 2010). Although the estimates for the single measures suggest modest increases in the risk of death, this translates to large population impacts because the exposures are common across the working population. Policy and practice interventions to improve job control and security should be implemented to improve health and decrease risk of mortality.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.socscimed.2020.113452.

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ALL-CAUSE MORTALITY AND THE TIME-VARYING EFFECTS OF PSYCHOSOCIAL WORK STRESSORS: A RETROSPECTIVE COHORT STUDY USING THE HILDA SURVEY

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SUPPLEMENTARY MATERIAL

TABLE S1 Hazard Ratios (and standard errors) of time-varying psychosocial work stressors exposures **job control, job demands and complexity, job security and fairness of pay** and risk of mortality multivariate regression models

	All participants				**Male participants**			
	Primary model [a]		**Model further adjusted for sociodemographic and health factors [b]**		**Primary model [c]**		**Model further adjusted for sociodemographic and health factors [d]**	
Psychosocial work stressor exposure	**HR (95% CI)**	**p-value**	**HR (95% CI)**	**p-value**	**HR (95% CI)**	**p-value**	**HR (95% CI)**	**p-value**
Low job control [e]	1.37 (1.04, 1.80)	0.026	1.37 (1.03, 1.83)	0.032	1.66 (1.20, 2.29)	0.002	1.74 (1.24, 2.44)	0.001
High job demands [f]	1.04 (0.78, 1.40)	0.772	1.07 (0.80, 1.45)	0.639	1.05 (0.74, 1.49)	0.777	1.07 (0.75, 1.53)	0.704
Low job security [g]	1.31 (1.02, 1.69)	0.032	1.16 (0.90, 1.50)	0.259	1.52 (1.13, 2.04)	0.006	1.30 (0.95, 1.76)	0.096
Low fairness of pay [h]	1.10 (0.83, 1.46)	0.494	1.01 (0.76, 1.34)	0.966	1.17 (0.84, 1.64)	0.340	1.04 (0.74, 1.47)	0.809
Labour status								
Employed	1.00	-	1.00	-	1.00	-	1.00	-
Unemployed	1.34 (0.55, 3.24)	0.520	1.17 (0.48, 2.86)	0.723	1.06 (0.34, 3.34)	0.920	0.91 (0.29, 2.88)	0.878
not in labour force/retired	1.65 (1.19, 2.29)	0.003	1.39 (1.00, 1.94)	0.053	1.68 (1.15, 2.46)	0.008	1.37 (0.92, 2.03)	0.118
Year of entry into HILDA survey								
2001-2010	1.00	-	1.00	-	1.00	-	1.00	-
2011-2015	0.42 (0.20, 0.86)	0.018	0.46 (0.22, 0.97)	0.040	0.64 (0.31, 1.34)	0.237	0.72 (0.34, 1.52)	0.384
Age								
15-24 years	1.00	-	1.00	-	1.00	-	1.00	-
25-34 years	2.06 (1.07, 3.97)	0.031	1.84 (0.96, 3.54)	0.067	1.32 (0.61, 2.86)	0.479	1.22 (0.56, 2.65)	0.617
35-44 years	3.32 (1.81, 6.09)	<0.001	2.62 (1.43, 4.81)	0.002	2.44 (1.24, 4.81)	0.010	2.04 (1.03, 4.07)	0.042
45-54 years	5.82 (3.22, 10.5)	<0.001	4.30 (2.36, 7.85)	<0.001	4.81 (2.50, 9.26)	<0.001	4.18 (2.12, 8.24)	<0.001

55-64 years	16.9 (9.26, 30.9)	<0.001	11.7 (6.24, 22.1)	<0.001	13.0 (6.65, 25.2)	<0.001	11.2 (5.50, 22.9)	<0.001
65+ years	49.1 (25.5, 94.4)	<0.001	27.0 (13.4, 54.2)	<0.001	35.9 (17.4, 74.4)	<0.001	23.8 (10.7, 52.8)	<0.001
Gender								
Female	1.00	-	1.00	-	-	-	-	-
Male	2.00 (1.51, 2.65)	<0.001	2.02 (1.50, 2.74)	<0.001				
Country of birth								
Australia			1.00	-			1.00	-
English Speaking			1.09 (0.73, 1.61)	0.681			1.04 (0.65, 1.67)	0.869
Other			0.98 (0.64, 1.52)	0.943			0.98 (0.60, 1.62)	0.944
Education								
Bachelor's degree and above			1.00	-			1.00	-
Year 12/Certificate/Diploma			1.53 (1.05, 2.23)	0.025			1.34 (0.84, 2.13)	0.223
School not completed			2.27 (1.49, 3.44)	<0.001			1.96 (1.17, 3.28)	0.010
Household structure								
Couple with no children			1.00	-			1.00	
Couple with children			1.30 (0.89, 1.90)	0.181			1.54 (0.98, 2.42)	0.059
Lone parent with children			1.38 (0.74, 2.57)	0.312			1.58 (0.66, 3.79)	0.310
Lone person			1.88 (1.34, 2.64)	<0.001			2.28 (1.51, 3.46)	<0.001
Other			1.44 (0.70, 2.95)	0.319			2.12 (0.98, 4.58)	0.055
Population quintiles of disposable income								
1 (lowest)			1.04 (0.67, 1.60)	0.869			1.37 (0.81, 2.33)	0.238
2			1.00 (0.65, 1.54)	0.995			1.47 (0.88, 2.46)	0.144
3			0.89 (0.59, 1.35)	0.587			1.18 (0.71, 1.96)	0.528
4			1.05 (0.72, 1.54)	0.791			1.26 (0.78, 2.03)	0.341
5 (highest)			1.00	-			1.00	-
Occupational skill level								
High			1.00	-			1.00	-
Medium			0.66 (0.48, 0.92)	0.014			0.84 (0.57, 1.24)	0.384
Low			0.65 (0.45, 0.94)	0.022			0.59 (0.38, 0.92)	0.019
Employment Arrangement								
Permanent			1.00	-			1.00	-
Casual			0.91 (0.62, 1.33)	0.622			0.96 (0.60, 1.54)	0.868
Fixed Term			0.96 (0.55, 1.69)	0.895			1.21 (0.63, 2.33)	0.559
Self employed			0.99 (0.71, 1.38)	0.971			1.15 (0.78, 1.69)	0.492
Health								

No long-term health conditions			1.00	-			1.00	-
Long-term health conditions			2.95 (2.25, 3.88)	<0.001			2.61 (1.89, 3.59)	<0.001

[a] model included all time-varying psychosocial work stressors and adjusted for age at entry into study, year of entry into survey, gender, and time-varying labour status

[b] model included all time-varying psychosocial work stressors and adjusted for age at entry into study, year of entry into survey, gender, time-varying labour status, time-varying sociodemographic factors and time-varying health status

[c] model included all time-varying psychosocial work stressors and adjusted for age at entry into study, year of entry into survey, and time-varying labour status for male participants

[d] model included all time-varying psychosocial work stressors and adjusted for age at entry into study, year of entry into survey, time-varying labour status, time-varying sociodemographic factors and time-varying health status for male participants

[e] low job control exposure compared with the combination of intermediate and high job control exposure tertiles

[f] high job demands exposure compared with the combination of low and intermediate job demands exposure tertiles

[g] low job security exposure compared with the combination of intermediate and high job security exposure tertiles

[h] low fairness of pay exposure compared with the combination of intermediate and high fairness of pay exposure tertiles

TABLE S2 Hazard Ratios (and standard errors) of time-varying psychosocial work stressors exposures **job control, job demands and complexity, job security and fairness of pay** and risk of mortality multivariate regression models in participants who did not retire or permanently exit the labour force over the study period (n=143,759 observations; n=145 deaths)

	Primary model [a]		**Model further adjusted for sociodemographic and health factors [b]**	
Psychosocial work stressor exposure	**HR (95% CI)**	**p-value**	**HR (95% CI)**	**p-value**
Low job control [c]	1.54 (1.07, 2.20)	0.019	1.46 (1.01, 2.13)	0.047
High job demands [d]	1.02 (0.71, 1.47)	0.913	1.06 (0.73, 1.55)	0.759
Low job security [e]	0.94 (0.67, 1.31)	0.708	0.85 (0.60, 1.19)	0.342
Low fairness of pay [f]	1.11 (0.77, 1.61)	0.576	1.04 (0.71, 1.51)	0.850
Labour status				
Employed	1.00	-	1.00	-
Unemployed	0.46 (0.06, 3.28)	0.437	0.40 (0.06, 2.91)	0.367
not in labour force	0.48 (0.23, 0.99)	0.046	0.42 (0.20, 0.87)	0.020
Year of entry into HILDA survey				
2001-2010	1.00	-	1.00	-
2011-2015	0.36 (0.14, 0.88)	0.026	0.40 (0.16, 0.99)	0.049
Age				
15-24 years	1.00	-	1.00	-
25-34 years	1.56 (0.71, 3.40)	0.265	1.52 (0.70, 3.30)	0.295
35-44 years	2.39 (1.17, 4.87)	0.017	2.09 (1.02, 4.29)	0.044
45-54 years	6.38 (3.27, 12.5)	<0.001	4.77 (2.41, 9.45)	<0.001
55-64 years	19.5 (9.87, 38.7)	<0.001	12.5 (6.02, 25.9)	<0.001
65+ years	52.6 (23.9, 115.9)	<0.001	25.9 (11.3, 59.1)	<0.001
Gender				
Female	1.00	-	1.00	-
Male	1.91 (1.33, 2.75)	<0.001	1.95 (1.31, 2.89)	0.001
Country of birth				
Australia			1.00	-
English Speaking			1.01 (0.60, 1.70)	0.972
Other			0.65 (0.33, 1.29)	0.221
Education				

Bachelor's degree and above			1.00	-
Year 12/Certificate/Diploma			1.39 (0.86, 2.24)	0.176
School not completed			1.92 (1.10, 3.36)	0.022
Household structure				
Couple with no children			1.00	-
Couple with children			0.90 (0.56, 1.45)	0.680
Lone parent with children			0.85 (0.35, 2.04)	0.710
Lone person			2.02 (1.33, 3.09)	0.001
Other			0.81 (0.26, 2.53)	0.711
Population quintiles of disposable income				
1 (lowest)			0.99 (0.53, 1.84)	0.977
2			0.94 (0.52, 1.70)	0.832
3			0.93 (0.56, 1.54)	0.773
4			1.12 (0.71, 1.75)	0.625
5 (highest)			1.00	-
Occupational skill level				
High			1.00	-
Medium			0.76 (0.50, 1.16)	0.207
Low			0.81 (0.50, 1.32)	0.404
Employment Arrangement				
Permanent			1.00	-
Casual			0.98 (0.59, 1.62)	0.941
Fixed Term			1.15 (0.59, 2.27)	0.678
Self employed			0.95 (0.60, 1.50)	0.818
Health				
No long-term health conditions			1.00	-
Long-term health conditions			3.18 (2.25, 4.49)	<0.001

[a] *model included all time-varying psychosocial work stressors and adjusted for age at entry into study, year of entry into survey, gender, and time-varying labour status*

[b] *model included all time-varying psychosocial work stressors and adjusted for age at entry into study, year of entry into survey, gender, time-varying labour status, time-varying sociodemographic factors and time-varying health status*

[c] *low job control exposure compared with the combination of intermediate and high job control exposure tertiles*

[d] *high job demands exposure compared with the combination of low and intermediate job demands exposure tertiles*

[e] *low job security exposure compared with the combination of intermediate and high job security exposure tertiles*

[f] *low fairness of pay exposure compared with the combination of intermediate and high fairness of pay exposure tertiles*

Chapter 6 Study IV - Changes in job control and perceptions of general health: A longitudinal analysis of Australian workers, 2005-2017

This chapter is presented as the research article submitted to the Journal of Occupational and Environmental Medicine.

6.1 Overview

This submitted paper which this chapter is based on (with supplementary material) reports the investigation into changes of job control and perceptions of general health. There has been much research into the effect of poor psychosocial working conditions including job control on physical and mental health, yet barriers to making causal interpretations into the effect of job control on physical and mental health persist mostly due to methodological biases. In addition, the effect of cumulative exposure to low job control on health is not well known. Causal inference from observational studies can be strengthened by triangulating results from different approaches with differing strengths and limitations to strengthen the confidence in results. I explored the causal relationship between job control and general health in a nationally representative longitudinal study of Australian households through triangulation, using three complementary longitudinal modelling approaches. I found a strong stepwise relationship between increasing job control and general health and that this relationship was mostly exposure to outcome. Cumulative exposure to low job control resulted in increasingly worse general health. This study with improved causal inference over previous research showed that increased job control is strongly associated with increasing general health.

6.2 Research Questions and Objectives

6.2.1 Research questions

The overall research question was are changes in job control over time associated with changes in perceptions of general health?

The specific research questions are:

1. Does prior exposure to low job control have continuing long-term effect on health?
2. Is there an indirect effect of general health, whereby health in previous years may affect current health?
3. What are the potential impacts of recurrent or persistent exposure to lower or higher job control on current health?

6.2.2 Research objectives

1. To examine the impact of within-person changes in job control on subsequent general health.
2. To examine the indirect effect of general health in previous years on current health.
3. To analyse the effect of cumulative exposure to low job control on future health.

6.3 Submitted manuscript for publication

6.3.1 Main document and supplementary material

Taouk Y., Spittal M. J., Disney G., LaMontagne A. D. Changes in job control and perceptions of general health: A longitudinal analysis of Australian workers, 2005-2017 (submitted to Journal of Occupational and Environmental Medicine).

Changes in job control and perceptions of general health: A longitudinal analysis of Australian workers, 2005-2017

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Authors Contributions

All authors contributed to the conception and design of the work. Yamna Taouk and Anthony Daniel LaMontagne conceived and designed the study. Yamna Taouk performed the statistical analyses and drafted the manuscript. Matthew John Spittal and George Disney contributed to the writing of the manuscript. Anthony Daniel LaMontagne provided logistic support at all stages of the study and contributed to the writing of the manuscript.

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Conflict of Interest

NONE DECLARED

Ethical Approval

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008. The study received ethical approval from the Melbourne School of Population and Global Health's Human Ethics Advisory Group (1750707.2).

Informed Consent

The HILDA Survey is conducted by the Melbourne Institute of Applied Economic and Social Research at the University of Melbourne on behalf of the Australian Commonwealth Government Department of Social Services (DSS). All relevant ethical issues associated with this research project (including consent) were addressed by the Melbourne Institute of Applied Economic and Social Research at the University of Melbourne which operates within the University's Ethics Guidelines. All processes involving consent have been approved by both the University of Melbourne's Human Research Ethics Committee and the Australian Institute of Health and Welfare's Ethics Committee. All members of the group and researchers involved in the HILDA project must comply with the Privacy Act (1988). The authors were provided de-identified data by the organisational data custodian which the names and addresses have been removed.

Data Availability Statement

This paper uses unit record data from the Household, Income and Labour Dynamics in Australia (HILDA) Survey. The authors are not permitted to copy, reproduce or provide the data files to unauthorised persons. The HILDA Project was initiated and is funded by the Australian Government Department of Social Services (DSS) and is managed by the Melbourne Institute of Applied Economic and Social Research (Melbourne Institute).

Disclaimer

The HILDA Project was initiated and is funded by the Australian Government Department of Social Services (DSS) and is managed by the Melbourne Institute of Applied Economic and Social Research (Melbourne Institute). However, the findings and views reported in this

paper are those of the authors and should not be attributed to either DSS or the Melbourne Institute.

We thank Australian workers for their participation in the Household, Income and Labour Dynamics in Australia Survey. The data used in this paper were extracted using the Add-On Package PanelWhiz for Stata. PanelWhiz (http://www.PanelWhiz.eu) was written by Dr John P. Haisken-DeNew (john@PanelWhiz.eu).

Running Head Title

Changes in job control and perceptions of general health

Clinical Significance

The validation of job control as a potentially modifiable causal factor of general health has considerable public health implications, and substantial economic consequences. Awareness of the implications of the adverse effects of low job control on general health and appropriate work stress interventions might contribute to better health and well-being.

ABSTRACT

Objective

This longitudinal study of Australian workers explores a possible causal relationship between job control and general health.

Methods

Our sample included 105,017 observations (18,574 persons) over 13 annual waves from working age participants with information on job control, general health, and other sociodemographic and health factors. Three complementary longitudinal modelling approaches were used to explore the causal relationship.

Results

There was a strong stepwise, mostly exposure to outcome, relationship between increasing job control and general health. Cumulative exposure to low job control resulted in increasingly worse general health. Taken together, these findings provide good evidence of a causal relationship between low job control and general health.

Conclusion

This analysis with improved causal inference over previous research showed that change in job control is strongly associated with change in general health.

KEYWORDS

job control; general health; fixed-effects analyses; longitudinal study; causal inference

BACKGROUND

Workers' control over their work, that is their ability to make decisions about the way they work (decision authority) and how they use their skills (skill discretion), is a key construct in the relationship between the psychosocial work environment and health.[1,2] A substantial body of research has established associations between low job control and coronary heart disease,[3,4] diabetes,[5,6] poor mental health,[7-10] and mortality.[11] Despite these well-established associations, barriers to making causal interpretations persist.

The first concern is residual confounding. Observed association between job control and health may be confounded by a range of non-workplace health risk factors such as personality traits, childhood experiences, pre-employment factors and stressful life events.[12-14] The second concern is reverse causation. Workers with poor health may perceive their working conditions more negatively than they would had they not been in poor health. Thus associations found could be explained by reverse causality.[15,16] Related to this, the observed association between job control and health may be reciprocal, that is, poor health status may predispose selection into employment with low job control.[17] Third, job control is typically measured at baseline, and any changes in job control and its cumulative effect over time are not usually accounted for, resulting in imprecise measurement and the potential for exposure misclassification.[18] Finally, the literature to date has largely focused on the harmful effects of low control and there has been little investigation of whether improving job control could improve health, which is essential for informing policy and practice.

Causal inference from observational studies can be strengthened by triangulating results from different approaches with differing strengths and limitations.[19] If results of differing approaches are consistent, confidence in the findings is strengthened. To address the first challenge outlined above (time invariant confounding from individual traits) a causally robust fixed-effects analysis that examines the impact of within-person changes in job control on

perceptions of general health can be employed.[20] Fixed-effects models, used widely in economics, remove the influence of time invariant characteristics, such as earlier life experiences, that could predict both the focal exposure of perceived job control and subsequent health. This enhances exchangeability between the exposed and unexposed groups improving causal inference.[21]

However, contemporaneous fixed-effects associations cannot establish directionality, and thus do not account for reverse causation. To address this second challenge, a lagged exposure variable can be included in the model to control for the direct effect of lagged job control on current health to investigate whether prior (i.e. time lagged) exposure to low job control has continuing long-term effect on health.[22,23] Additionally, there may be an indirect effect of general health, whereby health in previous years may affect current health. Dynamic fixed-effects Arellano-Bond regression models allow for the inclusion of lagged outcome variables into the model.[24] Finally, to account for potential impacts of recurrent or persistent exposure to lower or higher job control, and to confidently exclude reverse causality, cumulative job control can be examined. A cumulative exposure approach assessing the potential for both exposure duration and intensity to predict future outcome may improve causal inference by establishing temporal precedence and allowing for assessment of dose-response.[25]

Our study aimed to improve causal inference using the three analytical approaches outlined above to examine the relationship between job control and general health (Figure 1). We used data collected in the Household, Income and Labour Dynamics in Australia (HILDA) survey, a large nationally representative Australian longitudinal study including men and women from across the occupational spectrum.[26]

METHODS

Data Source

We use data from the HILDA survey. Commenced in 2001, HILDA is a nationally representative longitudinal study of Australian households following over 17,000 people living in Australia annually.[26] The HILDA survey collects detailed information about economic and personal well-being, labour market dynamics and family life from household members aged 15 years or older during each survey wave. Information on survey recruitment, retainment and resampling is contained elsewhere.[27,28] Five items concerning skill discretion and decision authority were available from wave 1 and a further 6 items were added to the survey in wave 5. Hence data from wave 5 onwards were included in this study.

Analytic Sample

The analysis is restricted to working age participants employed for one or more waves. There were 27,235 participants (201,036 observations) from 2005 to 2017. The inclusion criteria were: i) aged 15 - 64 years; ii) employed; iii) had job control exposure data; iv) had general health outcome data and v) and non-missing covariate data. Around 8% of participants had no data on job control (7.5%), general health (0.2%) and/or other covariates (0.2%). The final analytic sample consisted of 18,574 participants with 105,017 observations (Figure 2).

Ethics Approval

Ethical approval for this study was granted by the University of Melbourne School of Population and Global Health's Human Ethics Sub-Committee.

Outcome Variable

Our primary outcome was the general health subscale of the 36-item Short Form (SF-36).[29,30] This five-item subscale measures individual perceptions of general health with evaluations

for rating own health, comparison with others' health and with others' proneness to illness. Items are rated on a five-point Likert scale (Cronbach's $\alpha = 0.81$, pooled over 13 waves). This measure of self-related health is a strong predictor of future morbidity and mortality,[31-33] and has been used in health research and large-scale surveys.[34] We used the continuous score, with higher scores representing better health. There is no universally accepted translation of the general health score difference to clinical meaningfulness, however, it has been suggested that a difference of three points on the norm-based scale reflects a minimal clinically important difference[35] and a difference of four or more on the unstandardized scale represents a moderate clinically-significant effect.[36]

Job Control

Measures of psychosocial job characteristics, based on the Karasek job control demand model,[1] were assessed in each wave of the HILDA, to which respondents rated their level of agreement or disagreement using a seven-point scale ranging from 1 = strongly disagree to 7 = strongly agree. Initially 3 items assessing decision authority and 2 items assessing skill discretion were included, however, from wave 5 onwards, an additional 4 decision authority items and 2 skill discretion items were added. Hence 11 items were used to assess aspects of job control (Supplemental Appendix 1, Supplemental Digital Content 1). Summary scales for decision authority and skill discretion were combined into an equally weighted scale of job control. Quintiles of job control were defined using the data from wave 5, with the same cut-off scores then applied to each subsequent wave.

Confounders

We controlled for possible prior common causes of job control and self-rated health. Relevant confounders included gender, age group and survey year. The sociodemographic factors included education, household structure, occupational skill level,[37] employment arrangement

and household disposable income (calculated by summing the income components for the previous financial year for all adults in the household and equivalised using the modified OECD scale[38]). At each wave, nominal household income values were converted to quintiles of the Australian population distribution using percentile statistics for the corresponding financial year.[39]

A further two health covariates, long-term disability and/or health conditions and number of significant life events in past 12 months, were included in models. Health status was ascertained from one question asking respondents if they experienced a disability or health condition that had lasted, or was likely to last 6 months or more, restricted their everyday activities, and was not corrected by medication or medical aids. The significant life events included the following events: separated from spouse or long-term partner; serious personal injury/illness; serious injury/illness to a close relative/family member; death of spouse or child; death of close relative/family member; victim of physical violence; and major worsening in finances.[40,41] We classified participants as having 0, 1, 2 or ≥ 3 significant life events in the previous 12 months.

It is uncertain if over time an unhealthy lifestyle is a confounder, mediator or a combination of both for job control and general health. Hence, health behaviour covariates including cigarette smoking, physical activity and alcohol consumption[42] were included in additional sensitivity analyses (see below).

Statistical Analyses

Descriptive analyses included assessing the frequencies of job control and the mean of self-rated health by each of the variables of interest, plotting its year-to-year changes, and summarising the numbers of contributed survey waves per participant. Longitudinal linear fixed-effects regression models were used to estimate the association between job control and

general health score within individuals. Time-varying factors were controlled for by the inclusion of relevant confounders in the models to account for individual change in these variables that might influence the relationship between job control and general health.

To further investigate the causal association of exposure with outcome beyond a time lag of one-year, we assessed the potential for both exposure duration and intensity to predict subsequent general health. We extracted a set of observations with equal numbers of continuous waves of employment to control for exposure duration while assessing the relationship between cumulative exposure to job control and subsequent general health. HILDA participants with multiple consecutive waves of employment with job control exposure data were identified. In the total 13 waves of job control exposure data, participants contributed on average 4 consecutive waves. Participants could contribute their consecutive waves over any of the observed calendar years (2005-2017) and cumulative exposure was calculated as: job control quintile (first wave + second wave + third wave + fourth wave) where the value of job control quintile each wave is 1 – 5, and cumulative exposure over 4 consecutive waves can take on values of 4 – 20. The observed range for cumulative job control was 4 – 20, with a mean (SD) of 12.0 (4.7) for 8,117 persons/observations.

General health outcome was measured in the subsequent wave to the last observed job control exposure, that is in the fifth consecutive wave for each contributing participant except for 5.3% of participants with missing general health outcome data in the year immediately following the last observed job control exposure, where it was measured in the 6^{th} consecutive wave. The relationship between cumulative job control and general health was modelled using ordinary least squares multiple linear regression (between persons) and controlled for potential confounders including gender, age group, final calendar year of 4 years contributed by each participant, education, household structure, income, occupational

skill level, employment arrangement, long term disability or health condition and significant life events.

Gender was assessed as an effect modifier by the inclusion of interaction terms in the regression models, examination of interaction term coefficients and Wald tests of significance. All analyses were performed using Stata 16.

Sensitivity Analyses

We varied our assumptions in a number of ways to test the robustness of our results. First, we assumed that health behaviours were confounders and included them as covariates. We also considered that there may be a persistent effect of general health, whereby participants' general health in previous years may affect current health, and whether there was a lagged effect of job control and perception of general health. Dynamic fixed-effects Arellano-Bond regression models with three lags of general health, current job control and a 1-year lag of job control were estimated.[24] Three lags of general health were included based on a preliminary analysis of the Arellano-Bond test for serial correlation.[24]

RESULTS

Participants contributed on average 6 years of observations (mean ± SD: 6.6 ± 4.4), ranging from 1 to 13 years. The mean (SD) general health score was 71.8 (18.4) across the whole sample (Table 1). There were approximately equal numbers of observations from males and females, with the largest number of observations contributed by participants aged 25 to 54 years, residing in couple with children households, permanently employed, with no long-term disability and/or health condition and no significant life events. General health scores were lowest in participants who were older, had the lowest disposable income, employed in low skill level work, with long-term disability and/or health conditions. There was a stepwise

decrease in general health score with increasing number of significant life events experienced in the past 12 months. General health scores increased with increasing quintile of job control.

Job control from year-to-year did not change for almost half of all observations and the changes were approximately equal between increases and decreases in job control. From any given year to the next, 37,568 (47.7%) of observations remained in the same quintile of job control, 30,250 (38.4%) shifted one quintile, 8,185 (10.4%) shifted two quintiles, 2,330 (3.0%) shifted three quintiles and 519 (0.7%) shifted four quintiles. Transitions between quintiles of job control in consecutive pairs of years is shown in Figure 3. Most participants who started at the highest and lowest job control quintiles were most likely to remain in that quintile at the subsequent wave. Those starting in the second quintile were likely to improve and those starting in the fourth quintile were more likely to report worse job control at the subsequent wave.

Results of regression analyses are shown in Table 2. Model 1 shows the results of the adjusted within-person fixed-effects model. There was a strong stepped relationship between increasing job control and general health. On average, there was a 2.4-point increase in general health (95% CI 2.0-2.8) for participants in the highest job control quintile compared to those in the lowest quintile. The inclusion of a lag effect of job control supported a small prospective relationship (β=0.17; 95% CI 0.07-0.27) but the contemporaneous association was essentially unchanged (Model 2). In Model 3, the cumulative exposure analysis showed a strong stepwise association between job control and general health (β=0.47; 95% CI 0.38-0.57). Given the observed values of cumulative job control range from 4 – 20 than going from the lowest to the highest observed value of cumulative job control predicted an 8-point higher general health score over a period of 4 years. The inclusion of health behaviour risk factors in the above models did not substantially change these results (Supplemental Table 1, Supplemental Digital Content 1). There was weak evidence of an interaction between job

control and general health by gender in Model 1 (p=0.06) but no evidence of a gender interaction in Model 2 (p=0.66) or Model 3 (p=0.61).

Results for the Arellano-Bond models (Supplemental Table 2, Supplemental Digital Content 1) show that most of the relationship between job control and general health appears to be contemporaneous; the lagged coefficients are small relative to contemporaneous effects (β_{t-1} = 0.07; 95% CI -0.10-0.24, β_t = 0.63; 95% CI 0.46-0.81). The Arellano-Bond models also demonstrate a lagged persistent effect of general health from previous years on current general health (β_{t-1} = 0.23; 95% CI 0.19-0.28), β_{t-2} = 0.10; 95% CI 0.08-0.13, β_{t-3} = 0.03; 95% CI 0.02-0.05). Results were essentially unchanged after the inclusion of health behaviour risk factors in the Arellano-Bond model.

DISCUSSION

Results from the fixed-effect analyses suggest a strong, within-person, stepwise relationship between job control and general health. However, these findings alone do not rule out reverse causation as an explanation. Results from the lagged analyses which controlled for a time lag of one-year of exposure preceding outcome, and the dynamic fixed-effects Arellano-Bond models which identified and controlled for three years of persistence in general health thus minimising effect of general health on job control, supported a mostly exposure to outcome relationship. The cumulative exposure analysis showed a very strong stepwise relationship over four years, with temporal precedence of exposure and accounting for both exposure intensity and duration. The magnitude of associations suggests clinically meaningful impacts, particularly for the cumulative job control exposure. Taken together, these findings provide good evidence of a causal relationship between low job control and general health.

This study advances knowledge about the extent to which the relationship between job control and general health is contemporaneous or related to prior exposures. Our analysis

controlling for time-invariant characteristics is in accordance with several previous studies that have found prospective associations between job control and health.[4,7,10] However lagged associations did not support previous results as coefficients relating to lagged exposure were attenuated and contemporaneous associations essentially unchanged, suggesting that previous studies may have been susceptible to residual time-invariant confounding.[22,23]

In a similar fixed-effects analyses of the same HILDA cohort, LaMontagne et al,[43] examined whether job security improvements were associated with improvements in mental health, and showed improvements in mental health were associated with improving job security consistent with a causal relationship. Another study which used survival analysis to investigate the effect of cumulative exposures to psychosocial work stressors on mortality found that low job control was associated with a 43% increase in mortality.[25] Comparison with our study is problematic due in part to differences in analytic strategy and outcome measurement. Nonetheless, the cumulative exposure results in this study suggest higher impacts, for better and worse, of persistently higher or lower job control; the 8-point difference from lowest to highest job control is much greater than the contemporaneous association by a factor of 3. This difference would be clinically significant given that a difference of three points on the norm-based scale reflects a minimal clinically important difference.[35]

The strengths of our study included the examination of the relationship between changes in job control and perceptions of general health, with long follow-up time and low attrition rates, using a large national sample, with individual level measures of exposure and validated measures of job control. Measured time-invariant characteristics such as gender as well as unmeasured stable factors like personality and temperament, which may increase common method bias, were controlled for in our within-person analyses. Controlling for all within-person unmeasured time-invariant characteristics, including non-workplace health risk factors

such as stable personality traits, genetic factors, childhood experiences and pre-employment factors which may influence the association between job control and health, reduces the likelihood that our effect estimates are subject to bias and are more likely to represent causal associations than previous analyses of observational data.

The main limitation of our study is that results of the cumulative exposure analyses are generalisable only to those individuals who contributed 4 or more consecutive waves of employment (44% of analytic sample) and hence may limit external validity. This condition was imposed on the analysis in order to control for confounding by duration of exposure and to maximise the number of individuals and period of observations included in the analysis. Typically, exposure to job control and health outcomes are measured using self-reported measures so estimated effects may be vulnerable to common method bias resulting in spurious or inflated associations between the exposure and outcome.[44] Some individuals may systematically overstate both their perceptions of job control and health due to negative affectivity or other propensity for overly pessimistic evaluation rather than an objective representation of the actual environment. However, fixed-effects (controls for time-invariant individual traits) and cumulative exposure models would have mitigated against this as a major influence.

Another limitation is that any study into the effects of work characteristics must be limited to individuals with jobs, hence there is a potential for biasing toward the null through the healthy worker effect. Additionally, HILDA may underrepresent people with severe health conditions, and generally has a lower proportion of migrants and people from lower socioeconomic groups.[27] Finally, although the HILDA survey is a national, population-based study, the 66% initial response rate in the first wave suggests that it may not be wholly representative of working Australians. The greater retention of persons of higher versus lower socioeconomic status may affect generalisation. The disproportionate loss of lower SES and

lower occupational status participants, who are more likely to be exposed to poor psychosocial working conditions and adverse psychosocial work stressors, most likely would have biased our results towards the null.[45]

CONCLUSION

Our results provide the best observational evidence to date that changes in job control are causally associated with corresponding changes in perceptions of general health—a well validated predictor of objective health outcomes as well as mortality. The triangulation of our results including the strong stepwise observations in our fixed-effects analyses along with the evidence of a prospective exposure to outcome association for the relationship between job control and health depicted in the cumulative exposure analysis strengthen causal inference over previous observational studies. The validation of job control as a potentially modifiable causal factor of general health has considerable public health implications, with potentially substantial economic consequences for employers and society. Increasing employee control should be integrated into workplace strategies to improve general health and well-being.

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FIGURE LEGENDS

FIGURE 1. Directed Acyclic Graphs (DAGs) for job control and general health (GH) models.

FIGURE 2. Flowchart of participants from HILDA survey into the study sample.

FIGURE 3. Transitions between quintiles of job control in consecutive pairs of years from 2005 to 2017, expressed as proportions of the observations in quintile categories at time T (the first in each pair of years for that person).

LIST OF SUPPLEMENTAL DIGITAL CONTENT

Supplemental Digital Content 1.docx

TABLE 1. General Health scores by sociodemographic, health and significant life events characteristics in working participants of the Hilda survey, 2005-2017 (13 annual waves, pooled N = 18,574 persons; n = 105,017 observations)

Characteristics	Number (%)	GHI-5 score Mean (SD)
Overall	105,017	71.81 (18.44)
Quintile of job control		
1 (Lowest)	21,496 (20.47)	69.00 (19.63)
2	20,128 (19.17)	70.57 (18.28)
3	22,169 (21.11)	71.52 (17.97)
4	22,403 (21.33)	72.79 (17.77)
5 (Highest)	18,821 (17.92)	75.51 (17.81)
Sociodemographic factors		
Age group		
15-24 years	19,341 (18.42)	73.12 (18.32)
25-34 years	22,704 (21.62)	73.28 (17.91)
35-44 years	23,586 (22.46)	72.28 (18.01)
45-54 years	24,677 (23.50)	70.37 (18.75)
55-64 years	14,709 (14.01)	69.46 (19.11)
Gender		
Male	53,174 (50.63)	71.72 (17.88)
Female	51,843 (49.37)	71.90 (18.99)
Education		
School not completed	20,700 (19.71)	70.05 (19.03)
High school completion	17,915 (17.06)	72.98 (18.15)
Certificate/Diploma	34,840 (33.18)	70.84 (18.33)
Bachelor's degree and above	31,562 (30.05)	73.37 (18.14)
Household structure		
Couple with no children	27,389 (26.08)	72.09 (18.49)
Couple with children	51,386 (48.93)	72.28 (17.89)
Lone parent with children	8,362 (7.96)	70.08 (19.32)
Lone person	13,025 (12.40)	70.94 (19.37)
Other	4,855 (4.62)	70.55 (19.31)
Population quintiles of disposable income		
1 (lowest)	5,944 (5.66)	68.59 (19.96)

2	14,734 (14.03)	70.29 (18.81)
3	23,328 (22.21)	71.09 (18.42)
4	29,782 (28.36)	71.97 (18.25)
5 (highest)	31,229 (29.74)	73.52 (17.96)
Occupational skill level		
Low	24,554 (23.38)	70.48 (18.62)
Medium	41,235 (39.27)	71.50 (18.56)
High	39,228 (37.35)	72.97 (18.12)
Employment Arrangement		
Permanent	62,812 (59.81)	71.85 (18.26)
Casual	20,146 (19.18)	71.08 (19.07)
Fixed Term	8,467 (8.06)	72.34 (18.44)
Self employed	13,592 (12.94)	72.33 (18.26)
Long-term disability and/or health conditions		
No	88,112 (83.90)	74.48 (16.60)
Yes	16,905 (16.10)	57.87 (21.05)
No. significant life events in past 12 months		
0	72,823 (69.34)	73.25 (17.60)
1	24,514 (23.34)	69.69 (19.25)
2	6,451 (6.14)	65.91 (20.66)
≥3	1,229 (1.17)	59.40 (22.26)

TABLE 2. Regression models of job control and general health

	Model 1 - fixed-effects regression n=18,574 persons, N=105,017 observations		**Model 2 – fixed-effects regression with 1-year lag of exposure n=14,395 persons, N=77,490 observations**		**Model 3 – ordinary least squares regression with cumulative job control exposure over 4 years N=8,117 persons/observations**	
	Beta Coefficient (95% CI)	**p-value**	**Beta Coefficient (95% CI)**	**p-value**	**Beta Coefficient (95% CI)**	**p-value**
Quintile of job control						
1 (Lowest)	0.00 (Reference)		0.00 (Reference)		-	-
2	0.50 (0.23, 0.78)	<0.001	0.46 (0.14, 0.78)	0.005	-	-
3	0.83 (0.54, 1.13)	<0.001	0.88 (0.54, 1.23)	<0.001	-	-
4	1.37 (1.04, 1.70)	<0.001	1.31 (0.92, 1.69)	<0.001	-	-
5 (Highest)	2.39 (2.00, 2.79)	<0.001	2.31 (1.86, 2.76)	<0.001	-	-
Quintile of job control (t_{-1})	-		0.17 (0.07, 0.27)	0.001	-	-
Cumulative job control	-		-	-	0.47 (0.38, 0.57)	<0.001
Gender						
Male	-		-	-	0.00 (Reference)	-
Female	-		-	-	1.77 (0.98, 2.55)	<0.001
Age group						
15-24 years	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
25-34 years	1.22 (0.72, 1.73)	<0.001	1.13 (0.51, 1.75)	<0.001	-1.47 (-2.89, -0.04)	0.043
35-44 years	1.31 (0.60, 2.03)	<0.001	0.97 (0.12, 1.82)	0.025	-1.69 (-3.09, -0.29)	0.018
45-54 years	1.02 (0.15, 1.90)	0.022	0.79 (-0.23, 1.82)	0.129	-3.05 (-4.46, -1.65)	<0.001
55-64 years	0.56 (-0.50, 1.61)	0.302	0.48 (-0.73, 1.69)	0.432	-2.93 (-4.60, -1.26)	0.001

Education						
School not completed	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
High school completion	-0.53 (-1.23, 0.16)	0.134	-0.57 (-1.52, 0.38)	0.238	1.77 (0.37, 3.17)	0.013
Certificate/Diploma	-0.07 (-0.85, 0.71)	0.862	-0.15 (-1.13, 0.83)	0.767	0.60 (-0.60, 1.81)	0.324
Bachelor's degree and above	0.44 (-0.52, 1.40)	0.367	0.03 (-1.20, 1.26)	0.961	1.56 (0.22, 2.91)	0.023
Household structure						
Couple with no children	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
Couple with children	-1.01 (-1.34, -0.68)	<0.001	-1.09 (-1.47, -0.71)	<0.001	-0.62 (-1.62, 0.39)	0.228
Lone parent with children	0.07 (-0.53, 0.67)	0.891	-0.01 (-0.73, 0.71)	0.985	-1.02 (-2.75, 0.70)	0.246
Lone person	0.06 (-0.40, 0.51)	0.808	0.10 (-0.44, 0.63)	0.725	-0.60 (-1.97, 0.78)	0.395
Other	-0.68 (-1.24, -0.12)	0.017	-0.96 (-1.63, -0.29)	0.005	0.07 (-2.02, 2.16)	0.949
Population quintiles of disposable income						
1 (lowest)	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
2	0.28 (-0.15, 0.71)	0.200	0.40 (-0.13, 0.93)	0.142	-0.06 (-2.24, 2.12)	0.955
3	0.21 (-0.23, 0.66)	0.347	0.30 (-0.26, 0.86)	0.288	-0.74 (-2.83, 1.36)	0.491
4	0.30 (-0.15, 0.76)	0.191	0.44 (-0.12, 1.01)	0.126	0.48 (-1.58, 2.55)	0.646
5 (highest)	0.54 (0.05, 1.03)	0.030	0.60 (0.002, 1.20)	0.049	0.02 (-2.09, 2.13)	0.986
Occupational skill level						
Low	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
Medium	-0.50 (-0.84, -0.16)	0.004	-0.35 (-0.76, 0.06)	0.096	0.44 (-0.65, 1.52)	0.430
High	-0.46 (-0.84, -0.07)	0.020	-0.23 (-0.69, 0.22)	0.314	0.15 (-1.09, 1.40)	0.809
Employment Arrangement						
Permanent	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-

Casual	-0.07 (-0.38, 0.25)	0.674	-0.09 (-0.48, 0.30)	0.649	0.68 (-0.52, 1.89)	0.267
Fixed Term	-0.15 (-0.48, 0.17)	0.358	-0.11 (-0.48, 0.26)	0.545	0.27 (-1.17, 1.70)	0.716
Self employed	-0.19 (-0.65, -0.27)	0.427	-0.37 (-0.91, -0.16)	0.169	0.48 (-0.72, 1.67)	0.434
Long-term disability and/or health conditions						
No	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
Yes	-5.06 (-5.40, -4.72)	<0.001	-4.87 (-5.25, -4.48)	<0.001	-14.2 (-15.5, -12.9)	<0.001
Number of significant life events in past 12 months						
0	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
1	-1.34 (-1.52, -1.16)	<0.001	-1.27 (-1.47, -1.06)	<0.001	-1.72 (-2.66, -0.79)	<0.001
2	-2.57 (-2.91, -2.22)	<0.001	-2.44 (-2.84, -2.04)	<0.001	-4.85 (-6.59, -3.11)	<0.001
≥3	-3.97 (-4.87, -3.07)	<0.001	-4.70 (-5.83, -3.58)	<0.001	-4.40 (-9.07, 0.26)	0.064

Model 1 adjusted for age, year, education, income, occupational level, employment arrangement, significant life events, long-term disability or health condition

Model 2 adjusted for 1-year lag of job control, age, year, education, income, occupational level, employment arrangement, significant life events, long-term disability or health condition

Model 3 ordinary least squares multiple linear regression (between persons) for cumulative job control exposure calculated as job control quintile ($t_1 + t_2 + t_3 + t_4$) and outcome GHI-5 measured in the subsequent year to last observed job control exposure (t_5), adjusted for gender, age, year, education, income, occupational level, employment arrangement, significant life events, long-term disability or health condition in the final year of 4 years contributed by each participant (t_4)

FIGURE 1. Directed Acyclic Graphs (DAGs) for job control and general health (GH) models

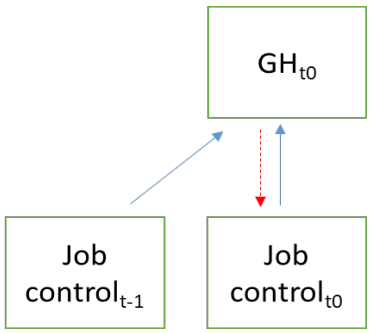


Figure 1a. Fixed-effects with 1-year lag of exposure

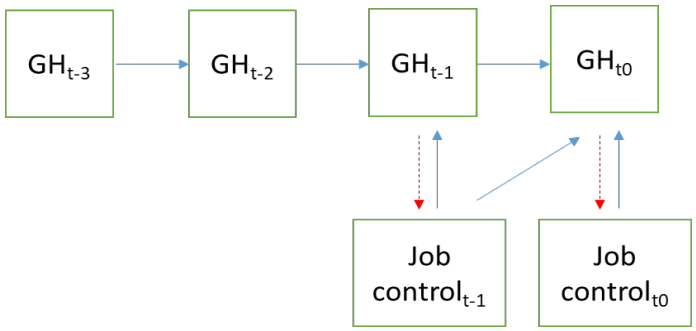


Figure 1b. Arellano-Bond model

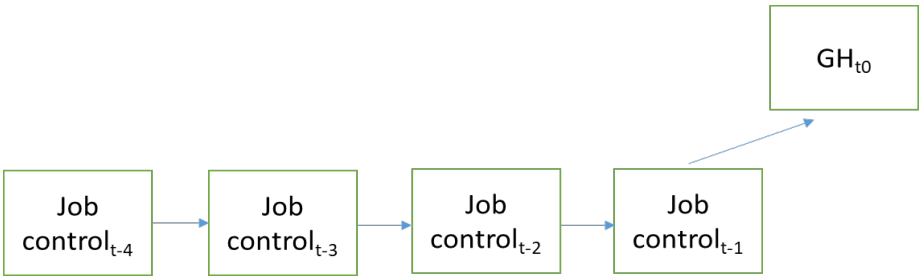


Figure 1c. Cumulative job control exposure over 4 years

FIGURE 2. Flowchart of participants from HILDA survey into the study sample

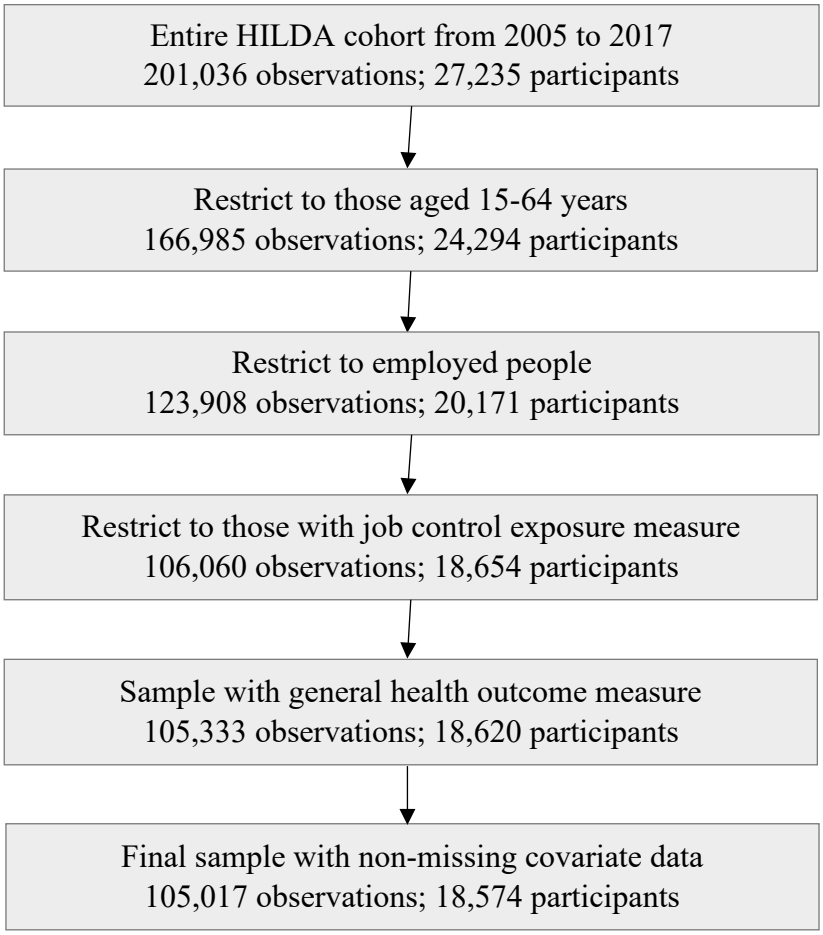
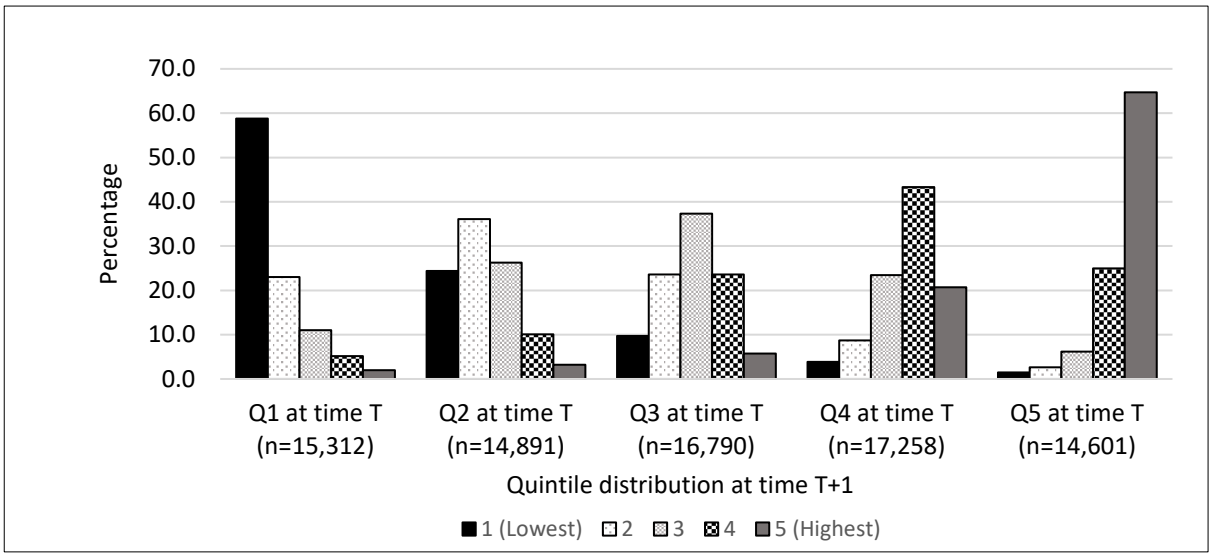


FIGURE 3. Transitions between quintiles of job control in consecutive pairs of years from 2005 to 2017, expressed as proportions of the observations in quintile categories at time T (the first in each pair of years for that person)



SUPPLEMENTARY MATERIAL

CHANGES IN JOB CONTROL AND PERCEPTIONS OF GENERAL HEALTH: A LONGITUDINAL ANALYSIS OF AUSTRALIAN WORKERS, 2005-2017

Yamna Taouk, Matthew J. Spittal, George Disney, Anthony D. LaMontagne

Supplemental Digital Content

Appendix S1. Construction of job control exposure assessment

Table S1. Job control and general health regression models further adjusted for health behaviours; Household, Income and Labour Dynamics in Australia (HILDA), 2005-2017.

Table S2. Job control and perception of general health Arellano-Bond regression models; Household, Income and Labour Dynamics in Australia (HILDA), 2005-2017.

Appendix S1. Construction of job control exposure assessment

Construct name	Items in the construct (from 1=strongly disagree to 7=strongly agree)	Cronbach's alpha
Decision authority	I have a lot of freedom to decide how I do my own work	0.87
	I have a lot of say about what happens on my job	
	I have a lot of freedom to decide when I do my work	
	I have a lot of choice in deciding what I do at work	
	My job requires me to take initiative	
	My working times can be flexible	
	I can decide when to take a break	
Skill discretion	My job often requires me to learn new skills	0.70
	I use many of my skills and abilities in my current job	
	My job provides me with a variety of interesting things to do	
	My job requires me to do the same things over and over again – reverse coded so that 1= strongly agree to 7= strongly disagree	
Job control	Combined decision authority and skill discretion scales (range 11 – 77)	0.85

The skill discretion item "my job requires me to do the same things over and over again" was reverse-coded so that high scores represented higher control for all items. Scores for decision authority and skill discretion scales were calculated as the sum of the item scores. For those few participants with missing data for some items (decision authority 1.0% and skill discretion 0.6%), scale scores were based on completed items and weighted up to the expected total had all items been answered. 92% of partially missing data for skill discretion involved only one item missing, 7% two items missing and 1% 3 items missing. 89% of partially missing data for decision authority involved one item missing, 8% two items missing, 1% three items missing and 2% more than three items missing.

Table S1. Job control and general health regression models further adjusted for health behaviours; Household, Income and Labour Dynamics in Australia (HILDA), 2005-2017

	Model 1 - fixed-effects regression n=18,523 persons, N=104,103 observations		**Model 2 – fixed-effects regression with 1-year lag of exposure n=14,372 persons, N=76,839 observations**		**Model 3 – ordinary least squares regression with cumulative job control exposure over 4 years N=8,021 persons/observations**	
	Beta Coefficient (95% CI)	**p-value**	**Beta Coefficient (95% CI)**	**p-value**	**Beta Coefficient (95% CI)**	**p-value**
Quintile of job control						
1 (Lowest)	0.00 (Reference)		0.00 (Reference)		-	-
2	0.48 (0.21, 0.75)	0.001	0.43 (0.11, 0.75)	0.009	-	-
3	0.79 (0.50, 1.08)	<0.001	0.84 (0.50, 1.18)	<0.001	-	-
4	1.30 (0.97, 1.62)	<0.001	1.23 (0.85, 1.61)	<0.001	-	-
5 (Highest)	2.26 (1.88, 2.65)	<0.001	2.18 (1.74, 2.63)	<0.001	-	-
Quintile of job control (t_{-1})	-	-	0.18 (0.08, 0.27)	<0.001	-	-
Cumulative job control	-	-	-	-	0.43 (0.34, 0.52)	<0.001
Gender						
Male	-	-	-		0.00 (Reference)	-
Female	-	-	-		2.35 (1.57, 3.13)	<0.001
Age group						
15-24 years	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
25-34 years	1.29 (0.79, 1.79)	<0.001	1.19 (0.57, 1.80)	<0.001	-0.30 (-1.71, 1.11)	0.674
35-44 years	1.37 (0.66, 2.07)	<0.001	1.03 (0.19, 1.86)	0.016	-0.06 (-1.46, 1.34)	0.018
45-54 years	0.99 (0.12, 1.85)	0.025	0.79 (-0.22, 1.80)	0.126	-1.67 (-3.09, -0.26)	0.021
55-64 years	0.49 (-0.55, 1.53)	0.360	0.50 (-0.70, 1.69)	0.413	-1.60 (-3.27, 0.07)	0.060

Education						
School not completed	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
High school completion	-0.20 (-0.90, 0.50)	0.573	-0.41 (-1.36, 0.53)	0.390	1.11 (-0.27, 2.48)	0.115
Certificate/Diploma	0.20 (-0.57, 0.97)	0.609	0.01 (-0.94, 0.97)	0.977	-0.002 (-1.19, 1.18)	0.997
Bachelor's degree and above	0.65 (-0.30, 1.60)	0.179	0.11 (-1.10, 1.32)	0.860	0.39 (-0.95, 1.72)	0.570
Household structure						
Couple with no children	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
Couple with children	-0.79 (-1.12, -0.47)	<0.001	-0.84 (-1.21, -0.47)	<0.001	-0.47 (-1.45, 0.52)	0.355
Lone parent with children	0.14 (-0.46, 0.73)	0.648	0.08 (-0.63, 0.79)	0.833	-0.43 (-2.13, 1.27)	0.619
Lone person	0.05 (-0.40, 0.50)	0.840	0.04 (-0.48, -0.57)	0.878	-0.62 (-1.97, 0.73)	0.365
Other	-0.55 (-1.09, -0.001)	0.049	-0.84 (-1.49, -0.19)	0.011	0.45 (-1.59, 2.50)	0.664
Population quintiles of disposable income						
1 (lowest)	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
2	0.20 (-0.22, 0.63)	0.341	0.31 (-0.21, 0.84)	0.242	0.28 (-1.87, 2.44)	0.796
3	0.18 (-0.26, 0.62)	0.421	0.24 (-0.31, 0.79)	0.390	-0.48 (-2.55, 1.59)	0.649
4	0.24 (-0.21, 0.69)	0.300	0.36 (-0.20, 0.92)	0.212	0.38 (-1.67, 2.43)	0.715
5 (highest)	0.45 (-0.03, 0.93)	0.066	0.50 (-0.09, 1.09)	0.094	-0.50 (-2.59, 1.59)	0.638
Occupational skill level						
Low	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
Medium	-0.39 (-0.73, -0.06)	0.022	-0.25 (-0.67, 0.16)	0.228	0.36 (-0.71, 1.43)	0.506
High	-0.32 (-0.70, 0.06)	0.095	-0.11 (-0.56, 0.34)	0.625	-0.02 (-1.25, 1.21)	0.980
Employment Arrangement						
Permanent	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-

Casual	-0.08 (-0.39, 0.23)	0.622	-0.07 (-0.45, 0.32)	0.737	0.46 (-0.72, 1.64)	0.449
Fixed Term	-0.15 (-0.47, 0.17)	0.368	-0.12 (-0.48, 0.25)	0.531	0.29 (-1.12, 1.70)	0.690
Self employed	-0.20 (-0.65, 0.25)	0.383	-0.40 (-0.92, 0.13)	0.139	0.23 (-0.94, 1.40)	0.703
Long-term disability and/or health conditions						
No	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
Yes	-4.91 (-5.25, -4.57)	<0.001	-4.71 (-5.08, -4.33)	<0.001	-13.5 (-14.8, -12.3)	<0.001
Number of significant life events in past 12 months						
0	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
1	-1.30 (-1.48, -1.12)	<0.001	-1.23 (-1.43, -1.02)	<0.001	-1.53 (-2.45, -0.61)	0.001
2	-2.49 (-2.83, -2.15)	<0.001	-2.36 (-2.75, -1.96)	<0.001	-4.52 (-6.23, -2.81)	<0.001
≥3	-3.99 (-4.87, -3.10)	<0.001	-4.63 (-5.73, -3.53)	<0.001	-4.22 (-9.00, 0.56)	0.084
Smoking status						
Never	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
Ex-smoker	-0.84 (-1.40, -0.33)	0.001	-0.54 (-1.15, 0.06)	0.080	-1.72 (-2.64, -0.79)	<0.001
Current	-2.30 (-2.91, -1.68)	<0.001	-1.79 (-2.52, -1.06)	<0.001	-5.47 (-6.53, -4.41)	<0.001
Alcohol consumption						
Abstinence	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-
Low	-0.10 (-0.40, 0.21)	0.544	0.03 (-0.33, 0.40)	0.851	2.10 (0.94, 3.27)	<0.001
Moderate	0.003 (-0.34, 0.34)	0.985	0.15 (-0.25, 0.54)	0.476	2.52 (1.58, 3.46)	<0.001
Heavy	-0.84 (-1.32, -0.35)	0.001	-0.61 (-1.17, -0.05)	0.034	1.56 (0.33, 2.78)	0.013
Physical activity						
>3 times per week	0.00 (Reference)	-	0.00 (Reference)	-	0.00 (Reference)	-

1-3 times per week	-2.67 (-2.88, -2.46)	<0.001	-2.49 (-2.73, -2.24)	<0.001	-4.13 (-4.97, -3.29)	<0.001
Less than once a week/never	-5.72 (-6.01, -5.42)	<0.001	-5.56 (-5.90, -5.22)	<0.001	-8.41 (-9.44, -7.38)	<0.001

Model 1 adjusted for age, year, education, income, occupational level, employment arrangement, significant life events, long-term disability or health condition, smoking status, alcohol use, physical activity

Model 2 adjusted for 1-year lag of job control, age, year, education, income, occupational level, employment arrangement, significant life events, long-term disability or health condition, smoking status, alcohol use, physical activity

Model 3 ordinary least squares multiple linear regression (between persons) for cumulative job control exposure calculated as job control quintile ($t_1 + t_2 + t_3 + t_4$) and outcome GHI-5 measured in the subsequent year to last observed job control exposure (t_5), adjusted for gender, age, year, education, income, occupational level, employment arrangement, significant life events, long-term disability or health condition, smoking status, alcohol use, physical activity in the final year of 4 years contributed by each participant (t_4)

Table S2. Job control and perception of general health Arellano-Bond regression models; Household, Income and Labour Dynamics in Australia (HILDA), 2005-2017

	Model 1* **n=9,498 persons, N=45,435 observations**		**Model 2^** **n=9,480 persons, N=45,059 observations**	
	Beta Coefficient (95% CI)	**p-value**	**Beta Coefficient (95% CI)**	**p-value**
Outcome				
General Health (t_{-1})	0.23 (0.19, 0.28)	<0.001	0.22 (0.18, 0.26)	<0.001
General Health (t_{-2})	0.10 (0.08, 0.13)	<0.001	0.10 (0.07, 0.12)	<0.001
General Health (t_{-3})	0.03 (0.02, 0.05)	<0.001	0.04 (0.02, 0.05)	<0.001
Quintile of job control (continuous)	0.63 (0.46, 0.81)	<0.001	0.63 (0.46, 0.81)	<0.001
Quintile of job control$_{t-1}$ (continuous)	0.07 (-0.10, 0.24)	0.401	0.10 (-0.07, 0.27)	0.234
Age group				
15-24 years	0.00 (Reference)	-	0.00 (Reference)	-
25-34 years	0.36 (-0.88, 1.60)	0.571	0.26 (-0.97, 1.48)	0.678
35-44 years	-0.10 (-1.75, 1.56)	0.909	-0.17 (-1.81, 1.46)	0.836
45-54 years	0.29 (-1.63, 2.21)	0.769	0.01 (-1.89, 1.92)	0.989
55-64 years	0.47 (-1.68, 2.63)	0.667	0.34 (-1.80, 2.47)	0.759
Education				
School not completed	0.00 (Reference)	-	0.00 (Reference)	-
High school completion	0.69 (-2.37, 3.75)	0.658	0.93 (-2.14, 4.00)	0.552
Certificate/Diploma	-0.69 (-3.55, 2.17)	0.635	-0.39 (-3.24, 2.46)	0.788
Bachelor's degree and above	1.33 (-1.98, 4.64)	0.431	1.48 (-1.81, 4.77)	0.377
Household structure				

Couple with no children	0.00 (Reference)	-	0.00 (Reference)	-
Couple with children	0.14 (-0.55, 0.84)	0.685	0.19 (-0.50, 0.89)	0.584
Lone parent with children	0.75 (-0.57, 2.07)	0.266	0.78 (-0.52, 2.08)	0.238
Lone person	0.91 (-0.07, 1.90)	0.069	0.93 (-0.05, 1.90)	0.064
Other	-0.48 (-1.63, 0.67)	0.416	-0.60 (-1.75, 0.55)	0.308
Population quintiles of disposable income				
1 (lowest)	0.00 (Reference)	-	0.00 (Reference)	-
2	0.30 (-0.56, 1.16)	0.498	0.34 (-0.51, 1.19)	0.437
3	0.45 (-0.45, 1.35)	0.330	0.50 (-0.39, 1.40)	0.270
4	0.74 (-0.20, 1.68)	0.122	0.77 (-0.16, 1.70)	0.105
5 (highest)	0.62 (-0.38, 1.62)	0.227	0.68 (-0.31, 1.68)	0.177
Occupational skill level				
Low	0.00 (Reference)	-	0.00 (Reference)	-
Medium	-0.54 (-1.27, 0.18)	0.141	-0.43 (-1.15, 0.28)	0.235
High	-0.80 (-1.57, -0.03)	0.041	-0.56 (-1.31, 0.20)	0.147
Employment Arrangement				
Permanent	0.00 (Reference)	-	0.00 (Reference)	-
Casual	-0.27 (-0.95, 0.40)	0.425	-0.19 (-0.86, 0.48)	0.579
Fixed Term	-0.19 (-0.79, 0.42)	0.545	-0.16 (-0.75, 0.44)	0.607
Self employed	-0.42 (-1.36, 0.52)	0.377	-0.41 (-1.34, 0.53)	0.392
Long-term disability and/or health conditions				
No	0.00 (Reference)	-	0.00 (Reference)	-
Yes	-3.30 (-3.84, -2.76)	<0.001	-3.16 (-3.70, -2.62)	<0.001
No. significant life events in past 12 months				

0	0.00 (Reference)	-	0.00 (Reference)	-
1	-1.17 (-1.48, -0.86)	<0.001	-1.16 (-1.47, -0.85)	<0.001
2	-2.64 (-3.25, -2.03)	<0.001	-2.49 (-3.10, -1.88)	<0.001
≥3	-4.36 (-6.03, -2.69)	<0.001	-4.42 (-6.06, -2.78)	<0.001
Smoking status				
Never	-	-	0.00 (Reference)	-
Ex-smoker	-	-	-0.15 (-1.13, 0.83)	0.760
Current	-	-	-0.77 (-1.98, 0.43)	0.209
Alcohol consumption				
Abstinence	-	-	0.00 (Reference)	-
Low	-	-	0.04 (-0.55, 0.62)	0.905
Moderate	-	-	0.03 (-0.63, 0.69)	0.920
Heavy	-	-	-1.05 (-1.93, -0.18)	0.018
Physical activity				
>3 times per week	-	-	0.00 (Reference)	-
1-3 times per week	-	-	-2.19 (-2.56, -1.81)	<0.001
Less than once a week/never	-	-	-4.55 (-5.09, -4.02)	<0.001

*** Model 1** included 3 lags of general health, current job control and a 1-year lag of job control; and adjusted for age, year, education, income, occupational level, employment arrangement, significant life events, long-term disability or health condition (test for autocorrelation in first-difference d errors – order=1: z=-30.2, p<0.001; order=2: z=-0.28, p=0.779; order=3: z=0.62, p=0.535)

^ **Model 2** included 3 lags of general health, current job control and a 1-year lag of job control; and adjusted for age, year, education, income, occupational level, employment arrangement, significant life events, long-term disability or health condition, smoking status, alcohol use, physical activity (test for autocorrelation in first-difference d errors – order=1: z=-30.0, p<0.001; order=2: z=0.05, p=0.963; order=3: z=1.08, p=0.279)

Chapter 7 Discussion

7.1 Summary of the Main Findings

Psychosocial work stressors influence a range of physical and psychological health outcomes and contribute to the social gradient in ill health. The benefits and harms of working life are unequally distributed among the population. There are social inequalities in the quality of work and a social gradient of health with respect to occupation. This thesis sought to examine the effect of psychosocial work stressors, commonly experienced by workers, on their health and mortality risk, in response to the literature gap concerning this significant issue, and to contribute to a better understanding of the underlying causal relationships.

The purpose of this PhD was to comprehensively review the evidence regarding the effect of exposure to psychosocial work stressors in the working environment such as job control, job demands, job strain, long working hours, job insecurity and shift work on health and mortality. The thesis also looked at quantifying the risks associated with adverse psychosocial work stressors, which workers are commonly exposed to in the workplace, on health and mortality. The PhD research exploited a repeated measures panel study to capture natural experiments of changes in psychosocial work stressors associated with changes in health, well-being and mortality to investigate whether and how the effect of psychosocial job stressors on mortality differs in relation to demographic and socioeconomic characteristics.

7.1.1 Study I – Psychosocial work stressors and risk of all-cause and CHD mortalities: A systematic review and meta-analysis

Psychosocial working conditions, or job stressors, are important and modifiable determinants of health behaviours and health. Most of the literature in this area assesses disease or other morbidity outcomes, with a growing number of studies on mortality. This thesis provided the first detailed and comprehensive systematic review, including quality assessment and meta-

analyses, of eligible observational studies on psychosocial work stressors, including low job control, high job demands, high job strain, low support at work, job insecurity, organisation injustice, ERI, long working hours, shift work and mortality. The review focused on all-cause and CHD mortalities to clarify the underlying relationship between psychosocial work stressors and mortality. The evidence to date showed that workers with low job control were at a higher risk of all-cause and CHD mortalities than workers with high job control. The strongest effect observed was for low job control and deaths due to CHD, even after controlling for relevant confounders and excluding low-quality studies. There was also some weak evidence of a decreased risk of mortality for workers with exposure to high job demands across all subgroup analyses and an elevated risk of mortality for job strain and insecurity.

Further research is required to examine the mechanisms underlying the observed effect between low job control and mortality and elucidate the relationship between psychosocial work stressors and mortality. Improving the psychosocial quality of work may contribute to better health and well-being, and thus reduce the effect of psychosocial work stressors on mortality. If the observed association is causal, then policy and practice interventions to improve job control could reduce all-cause and CHD mortalities across the working population.

7.1.2 Study II – Psychosocial work stressors and risk of mortality in Australia: Analysis of data from the HILDA survey

Psychosocial working conditions, or job stressors, are important and modifiable determinants of health and health behaviours. Most of the literature in this area assesses disease or other morbidity outcomes, with a limited number of studies on mortality. There is some evidence that the effects of psychosocial work stressors on health are stronger among men than women. Whether exposure to these psychosocial work stressors and associated adverse health outcomes translates into increased mortality is unclear. To address these inconsistencies, data collected from over 18,000 working participants from the HILDA survey with self-reported job

demands, job control, job security and fair pay psychosocial work stressors exposures at the baseline and followed over time for up to 15 years were analysed. Due to the rare outcome in the study, mortality, and according to the most common operationalisation of psychosocial work factors, the main analyses were based on dichotomous measures to optimise statistical power and accuracy. Psychosocial work stressors were measured at the baseline, which implicitly asserts the predictability of mortality from baseline exposure. This is limited in that a single measurement may be inadequate for assessing the effects of exposure dynamics over time and changing psychosocial work stressors. Nonetheless, understanding the association between psychosocial work stressors and mortality at a point in time is useful for targeting interventions to improve work quality.

Low job control was associated with a 39% increased risk of all-cause mortality after accounting for demographic, socioeconomic, health and behavioural factors. When measured on an ordinal scale, for each additional unit of increase in job control, the risk of death fell by 2% (an increase of 2% per unit on the ordinal scale for an observed range of 3 to 21 = 36%). The results of the present study suggested that low job control was associated with an independent increased risk of all-cause mortality. There was some evidence of a reduced risk of mortality for workers with exposure to high job demands; however, the results were not conclusive. The difference between sexes was not substantial, and its meaning in practical terms is unclear; however, it is possible that previous studies were accurate and sex differences are narrowing over time, with increasing rates of female participation in the labour market and changing social roles.

Psychosocial work stressors have been identified as significant emerging risks linked to global changes in the structure, organisation and management of work during the previous decades, presenting pressing challenges to occupational health and safety and workplace health, in general. Given the high prevalence of exposed individuals across the working population, job

control-associated increases in mortality could have considerable public health consequences. Awareness of the implications of the adverse effects of psychosocial work stressors on health and mortality in workplaces, and the implementation of interventions to improve job control could contribute to better health and well-being, thus reducing the effect of psychosocial work stressors on mortality to the benefit of workers, workplaces and society.

7.1.3 Study III – All-cause mortality and the time-varying effects of psychosocial work stressors: A retrospective cohort study using the HILDA survey

Very few studies on mortality have assessed time-varying psychosocial work stressors, allowing for changing exposures over time. Rather, most studies have measured the exposure only once at the baseline and the results to date, including investigation of sex-specific effects, have been inconsistent. Time-varying measures of work stressors allow for the assessment of changes in psychosocial work stressors over time and temporal changes in socioeconomic, occupational and well-being characteristics. Further advantages in examining changing psychosocial work stressors are the capacity to control for transitions from employment in and out of unemployment and retirement, which are strong predictors of mortality, to measure their impact on the relationship between mortality and work stressors.

To address these limitations, the effect of time-varying psychosocial work stressors on mortality was investigated using data from a longitudinal cohort of working Australians by examining the associations among job control, job demands, job insecurity, unfair pay and all-cause mortality, and whether sex modified these relationships. This is one of the few studies to have examined the relationship between time-varying exposure to work stressors and mortality over a substantial observation period and follow-up in a nationally representative working sample. Long-term exposure to low job control and job security was associated with a 39% and 36% increased risk of all-cause mortality, respectively, even when controlling for unemployment periods and the effect of not participating in the labour force/retirement on

mortality. The effects were largely restricted to males and persisted after adjustments for sociodemographic and health characteristics, and simultaneous adjustment for all psychosocial work stressors exposures. Restricting the analyses to workers who did not retire or permanently exit the labour force over the study period strengthened the association between low job control and mortality. The results strongly suggest that, over time, exposure to low job control and job security has independent effects on mortality. The lack of effects observed for females may have been due to the small number of deaths in females; thus, it is not certain that the absence of evidence in females is evidence of absence.

Psychosocial work stressors have been identified as significant risks linked to global changes in the structure, organisation and management of work during the previous decades, presenting new challenges to occupational health and safety. Although the estimates for the single measures suggest modest increases in the risk of death, this translates to large population impacts because the exposures are common across the working population. Policy and practice interventions to improve job control and security should be implemented to improve health and decrease the mortality risk.

7.1.4 Study IV – Changes in job control and perceptions of general health: A longitudinal analysis of Australian workers

There has been much research into the effect of poor psychosocial working conditions, including job control, on physical and mental health such as increased CHD, diabetes, depression and anxiety and mortality. Despite these well-established associations, barriers to making causal interpretations persist, mostly due to methodological limitations, including confounding and reverse causation. In addition, most studies have used a baseline measurement of job control exposure to assess the association between health and low job control. Hence, the effect of cumulative exposure to low job control on health is not well understood. Alternative methods are required to improve the causal inference and our understanding of the

precise relationship between job factors and mortality. Causal inference from observational studies can be improved by triangulating results from different approaches with differing strengths and limitations to strengthen the confidence in results.

The relationship dynamics between the commonly examined psychosocial work stressor perceived job control, a valid appraisal of the objective stressor, and general health, a strong predictor of future morbidity, was investigated to provide evidence of a causal relationship between job control and general health (as a proxy measurement for mortality) to complement mortality studies. The causal relationship between job control and general health was explored using a nationally representative longitudinal study of Australian households via triangulation using three complementary longitudinal modelling approaches. Over 18,500 persons and 13 annual waves from working-age participants with information on job control, general health and other sociodemographic and health factors were analysed. Dynamic fixed-effects regressions to control for time-invariant confounding and ordinary least squares regressions with cumulative job control exposure to analyse cumulative exposure to low job control and subsequent general health to address reverse causation were performed.

Results from the fixed-effect analyses suggested a strong, within-person, stepwise relationship between job control and general health. However, these findings alone do not rule out reverse causation as an explanation. Results from the lagged analyses, which controlled for a time lag of 1-year of exposure preceding outcome, and the dynamic fixed-effects Arellano-Bond models, which identified and controlled for 3 years of persistence in general health, thus minimising the effect of general health on job control, supported an exposure to outcome relationship. The cumulative exposure analysis showed a very strong stepwise relationship over 4 years, with a temporal precedence of exposure and accounting for both exposure intensity and duration. The magnitude of associations suggests clinically meaningful effects, particularly for the cumulative job control exposure. Triangulation of the results provided the best

observational evidence to date that changes in job control are causally associated with corresponding changes in the perceptions of general health—a well-validated predictor of objective health outcomes and mortality. This study advances our knowledge about the extent to which the relationship between job control and general health is contemporaneous or related to prior exposures.

The validation of job control as a potentially modifiable causal factor of general health has considerable public health implications with potentially substantial economic consequences for employers and society. The results of the present study support integrating increasing employee control into workplace strategies to improve general health and well-being.

7.2 Methodological Considerations

There is always the potential for error sources in epidemiology that warrant consideration in any given research study. Every study included in this thesis was conducted in the most appropriate manner to objectively address the specific research questions given the available data. Nonetheless, there is always a measurement error. Inherent in the construct, process and result of measurements is the role of variability. There are two broad groups of errors typically observed in epidemiology – precision (lack of random error) and validity (lack of systematic error). Issues of precision and validity related to the studies included in this thesis will be discussed in this section. Systematic errors, or biases, are related to the validity of the methodology in making inferences about the source population (internal validity) and the validity of inferences to the general population (external validity or generalisability) (Rothman, 2012). Most errors of internal validity can be grouped into three general categories: selection bias, confounding and information bias (Rothman, 2012).

7.2.1 Selection bias

Observational studies are commonly prone to problems related to the selection of study participants. If randomly selected participants refuse to participate or drop out of the study, or if there is missing data on particular items or measures, then selection bias may be likely if the association between exposure and outcome varies between responders and non-responders (Rothman, 2012). Any study investigating the effects of work stressors has some degree of potential selection bias because they must be limited to people with jobs. The individual studies included in the review presented in Study I were affected by selective attrition due to cessation of employment because of work stressor exposure during the follow-up period, and healthy worker effect due to healthier participants being more willing to participate in health surveys and less likely to be in low-status jobs with high stress. In shift work studies, survivor bias might have influenced mortality risk estimates as healthier workers are more likely to take up shift work than less healthy workers, and remain in shift work employment compared to those who develop debilitating conditions and cease shift work or leave the workforce (due to retirement or permanent disability) altogether. Studies that did not exclude participants based on baseline health status or adjust for unhealthy participants were excluded in additional sensitivity analyses to reduce the effects of health on selection bias.

Data from HILDA were used in the mortality studies included in the thesis and the analyses for changes in job control and general health. HILDA is a large, nationally representative Australian longitudinal study with men and women from across the occupational spectrum (Wilkins et al., 2019). Initially, HILDA had a 66% response rate in the first wave, but with each successive wave, the participant response rate has improved. In later waves, 95%–97% response rates have been achieved, which is of particular significance as HILDA is a longitudinal study and participants who withdraw from the study are generally not replaced except for the top-up sample during Wave 11 (Summerfield et al., 2019). The re-interview rate

in the HILDA survey was lowest among young adults, migrants, indigenous people, workers in low-skilled occupations, those unmarried and the unemployed. The difference in attrition over time was most noticeable concerning age, country of birth, labour force and occupation, but groups with low re-interview rates remained engaged with the study over the follow-up period, albeit irregularly. Even though the HILDA survey data came from a random representative sample of Australian households with high response rates, selection bias cannot fully disregard selection bias due to non-response, given that those who participate in studies are generally healthier than the population as a whole. Healthy worker effect selection bias might have influenced the results of Studies II, III and IV. People with poor health may avoid jobs that are demanding, or ill health may influence the length of employment in a job, which is of particular concern for the survival analysis with baseline measures of work stressors and covariates. If health preselects entry into employment or survival in the job, the healthy worker effect may have attenuated results. Health status was adjusted for during the analyses to mitigate selection bias; however, it cannot be totally disregarded due to its influence on the recruitment and drop out of HILDA survey participants. This adjustment may have been overly conservative, as poor health might have been produced by previous exposure to poor working conditions.

7.2.2 Confounding

A major source of systematic error in observational studies is confounding because the exposure of interest is seldom the only factor that differs between exposed and unexposed groups (Rothman, 2012). Confounding may result in an over- or underestimate of the observed association between the exposure and outcome and could even change the apparent direction of an effect. It is often impossible in observational studies to completely identify and accurately measure all potential confounders present, thus some residual confounding may remain even after adjustments for confounders (Pourhoseingholi, Baghestani, & Vahedi, 2012).

Meta-analyses can be invaluable for appraising the overall strength of evidence and estimating true effect sizes via the aggregation of multiple estimates. An important underlying assumption of the random-effects meta-analyses is that the between-study variation affects the effect estimates. However, random-effects meta-analyses of observational studies can yield biased estimates if unmeasured confounding is present in the synthesised studies with the potential to attenuate estimated effects or reverse the direction of estimates (Aune et al., 2011; Mathur & VanderWeele, 2020). Many, but not all, of the individual studies included in the quantitative synthesis examining psychosocial work stressors and mortality in Study I adjusted for potential confounding factors, although not every potential confounder was adjusted for in every study. As different studies adjusted for different sets of confounders, in addition to the confounder-adjusted estimates, unadjusted estimates were extracted, and meta-analyses of multivariate- and minimally adjusted were performed in Study I. Comparisons of adjusted and crude estimates allow for some assessment of the extent of confounding bias (Dekkers et al., 2019). Heterogeneity across studies may mitigate against residual confounding to some extent. For example, lifestyle factors such as cigarette smoking, alcohol consumption and physical activity are included in many observational studies and these factors tend to be highly correlated in many countries. However, the extent of their effect on mortality is difficult to determine and extricate. The inclusion of studies with diverse populations and different lifestyle factors may, therefore, alleviate residual confounding (Dekkers et al., 2019). In sensitivity analyses stratified by adjustment for study quality, measured by the extent of unmeasured confounding as the main criterion for acceptable quality, results were attenuated, nonetheless still excluding the null, with estimates remaining consistent even when omitting low-quality studies.

Due to the availability of rich prospective data in HILDA, several potential confounders were controlled for in the mortality analyses and in analyses examining changes in job control over time and general health. However, in the mortality studies, the possibility of residual

confounding by factors associated with early life and unmeasured confounding cannot be excluded (Rothman, 2012). Over time, the ageing process accelerates and accumulates cell mutations and tissue damage, leading to impaired health and diseases. Thus, age is singularly the most powerful predictor of mortality. From the time of birth, death as a foregone conclusion is a certainty. Including age as the underlying time scale and adjustment for age in mortality analyses mitigates this major confounding bias. Analyses are also adjusted for a variety of sociodemographic, health and behavioural factors in multivariate models. However, such variables are likely to be in the causal chain. Thus, there is a risk that over adjustment may have attenuated the association between work stress and the outcomes (mortality and general health). There is also a risk that residual confounding may influence the observed associations.

Socioeconomic conditions are related to psychosocial working conditions, and health and mortality, and therefore potentially confounding the relationship between psychosocial work stressors, and health and mortality (Macleod et al., 2001; Marmot et al., 1997; Niedhammer, Chastang, David, & Kelleher, 2008; Smith, Hart, Blane, Gillis, & Hawthorne, 1997). Socioeconomic inequalities in health, and consequently mortality, are pervasive and undoubtedly have a flow-on effect in the work environment (Marmot, 2004). For the most part, adverse psychosocial work stressors are more prevalent among lower SES groups and the lower the SES, the greater the risk of exposure to adverse psychosocial work stressors internationally and in Australia (LaMontagne et al., 2013; Siegrist, 2002b). SES may contribute to selection into poor-quality jobs; thus, psychosocial work stressors could potentially mediate the relationship between SES and health. Low job control in the workplace may partly explain the inverse relationship between SES and CHD (Marmot et al., 1997) and the effect of socioeconomic position on the risk of myocardial infarction may be partly explained by the mediating effect of skill discretion (Andersen et al., 2004). Alternatively, SES can modify the association between psychosocial work stressors and health with stronger effects observed

among workers from lower SES groups (Hoven & Siegrist, 2013; Marmot, Siegrist, & Theorell, 2006). A systematic review of mediation and moderation effects in work characteristics revealed that SES and health supported stronger mediating effects of psychosocial work stressors on the association between SES and health than modifying effects of SES on psychosocial work stressors and health. However, due to the very small numbers of studies included in the review, a high degree of heterogeneity was observed (Hoven & Siegrist, 2013).

The complexities and interrelations of socioeconomic conditions, psychosocial working conditions and health made it difficult to model the effect of SES in the analyses with a high degree of certainty. The factors chosen to measure SES in all the analyses included education, occupation and income. These three factors are commonly used to encapsulate the resource and prestige domains of SES in studies of health inequalities, with higher levels generally being more sought after by society (Gallo & Matthews, 2003). Each factor uniquely contributes to the study of SES-based health distinctions. Income and occupation may be most closely related to earned social prestige in adulthood; however, the relationship between education and health and subsequent mortality from major health problems that have their onset after education is less likely to reflect reverse causation. Thus, relating education to health can help isolate the directional effects of SES on health as inequalities in health in adulthood often have their origins in early life (Christie & Barling, 2009; Marmot, 2004). Including all three factors as SES measures in the models may have resulted in over adjustment, thus underestimating the effect of psychosocial work stressors on health and mortality. However, using a stepwise model to adjust for SES did not substantially change the associations between psychosocial work stressors and mortality. Additional analyses examining the interaction effects between SES and psychosocial work stressors on mortality did not support evidence of effect moderation.

Health and health behaviour factors may be associated with the working environment. Individuals with poor health or engaged in health risk behaviour may be more likely to be

involved in employment in workplaces with high exposure to adverse psychosocial work stressors (Chandola et al., 2006; Siegrist & Rodel, 2006). As well as being confounders and mediators, health and health behaviour factors are also strong predictors of mortality and are associated with work stress. Adjusting for these risk factors may control for selection into stressful work environments due to early risk factors and adverse environments during childhood and adolescence (Hughes et al., 2017). Hence, long-term health conditions and health behaviour risk factors were measured and sequentially adjusted for in the models included in the thesis and their effect on results from various sets of inclusions and exclusions examined. It is unlikely that there is a strong indirect effect of psychosocial work stressors on mortality mediated by poor health and an unhealthy lifestyle as effect estimates did not substantially change after adjustment for health and behavioural factors in the models. Nonetheless, the selection of occupations and perceived work stress might be affected by the individual's physical and mental state; thus, controlling for long-term health conditions in the analyses mitigated against this issue greatly influencing results. In Study IV, the number of significant life events was adjusted for job control and general health analyses. Significant life events strongly predict declines in well-being and including this variable in the analyses controlled for the influence of non-work sources of physical and mental impairment on workers' perceptions of general health.

7.2.3 Information bias

7.2.3.1 Differential versus nondifferential misclassification

Another type of bias is information bias, such that systematic differences in the accuracy of exposure or outcome measurements results in erroneous comparisons. If the probability of exposure or outcome misclassification is not related to other variables in the study, nondifferential misclassification may arise, generally biasing estimates of the observed associations towards the null. If the probability of misclassifying the exposure or outcome is

not independent of other variables in the study, differential misclassification may occur, resulting in the under- or overestimation of the observed associations (Porta, 2014; Rothman, 2012). Misclassification of the outcome in the mortality studies is unlikely given the outcome status is alive or dead. The outcomes, all-cause mortality and mortality due to CHD, were ascertained via record linkage and ICD-10 codes for most studies included in the review, subsequent to the evaluation of exposure to psychosocial work stressors in Study I. In Studies II and III, the majority of deaths were confirmed by linkage to the NDI; however, there were a small proportion of HILDA deaths (4%) that could not be linked to the NDI but were still included in the analyses. Additionally, there were extra deaths recorded in HILDA, and consequently the analyses, which had occurred after the data linkage. However, this is unlikely to have resulted in outcome misclassification measurement error as all deaths were verified and confirmed by survey fieldwork.

Misclassification of psychosocial work stressors exposures cannot be excluded. The few studies included in the review in Study I that used occupation-based measures of psychosocial work stressors based on a job exposure matrix may have been susceptible to nondifferential exposure misclassification bias, biasing estimates towards the null. In particular, the psychosocial work stressor job demands are a subjective measure of worker perceptions regarding the pressures of their workload and tasks, and may not be accurately measured in an exposure imputation system (Alterman et al., 1994; Johnson et al., 1996; Karasek et al., 1981). Job demands can vary in any job, although it is not entirely clear whether psychological job demands vary by occupational skill level. If exposure does not vary by occupation, then using a job exposure matrix, which is based on occupation, may not accurately reflect environmental differences. The exposure measurements in Studies II, III and IV were self-reported; however, non-work-related factors such as personality or health status might affect how workers perceive their psychosocial working environment characteristics. Thus, it is not inconceivable that

results might have been affected by nondifferential or differential misclassification of the exposure, resulting in under- or overestimation of effect estimates (Rugulies, 2012).

7.2.3.2 Definition of exposure and outcome

The construction and operationalisation of the exposures, outcomes and covariates may have contributed to potential misclassification. Constructs of psychosocial work stressor exposures were undertaken using several items from the HILDA survey based on the work stress models, including the Karasek job demand-control and ERI models. The benefit of using multiple items to form a scale is the stronger agreement with the measure of interest as shown by the high Cronbach's alpha in our studies.

The psychosocial work stressor exposures were dichotomised as high or low using median splits in Study II. While dichotomising work stressors as high or low by splitting at the sample median is a common operationalisation of psychosocial work factors, groups might not be that different from each other because most individuals centre around the median rather than in the extreme ends of the distribution, resulting in a low exposure contrast. Alternative operationalisations that create more exposure contrast may result in stronger associations; therefore, it is possible that median-split operationalisation may underestimate associations. However, construct of the exposures was hindered by the small number of deaths observed in the study limiting the statistical power (Landsbergis, Schnall, Warren, Pickering, & Schwartz, 1994). Less crude measures of the exposures were constructed with the inclusion of more observations in Study III. The choice of cut points was constructed to identify approximately one-third of those experiencing the most adverse levels of each psychosocial work stressor; therefore, scales were divided into tertiles. The tertile of the distribution corresponding to the most adversity (lowest job control, highest job demands, lowest job security and lowest perceptions of fair pay) was defined as exposed and the remaining two tertiles for each scale were combined to create the reference category. In Study IV, job control quintiles were

calculated based on baseline continuous distributions with the same cut-off scores and then applied to each subsequent wave, allowing for a more detailed exposure measurement.

The outcome general health used in Study IV was calculated by summing across the relevant items in the same scale and transforming the scores to a 0–100 scale with higher scores representing better general health. The continuous scale was used in the analyses to measure with optimal precision the underlying information. This measure of self-related health is a strong predictor of future morbidity and mortality (DeSalvo et al., 2006; Idler & Benyamini, 1997; Larsson et al., 2002) and has been used in health research and large-scale surveys (Subramanian et al., 2010); however, the presence of nondifferential or differential misclassification bias cannot be confidently excluded.

7.2.3.3 Common method bias

In Study IV, both the exposure and outcome measurements were based on self-reported items; therefore, estimated effects may have been vulnerable to common method bias resulting in spurious or inflated associations (Bonde, 2008). The measurement of the variables rather than the construct of the measurements can influence the observed association between the main exposure of interest and the outcome (Podsakoff, MacKenzie, Lee, & Podsakoff, 2003). Some individuals may systematically overstate their perceptions of job control and health due to negative affectivity or other propensities for overly pessimistic evaluation rather than an objective representation of the actual environment. In such cases, the observed associations do not reflect the true effect that exposure to adverse psychosocial work stressors has on health, rather it reflects the pessimistic evaluation. To the extent that the same pessimistic evaluation affects self-reported health, this may result in an overestimation of the associations. However, the fixed-effects (controls for time-invariant individual traits) and cumulative exposure models undertaken in Study IV mitigated against this as a major influence because the measurements of exposure and outcome were taken over separate times using lagged effects, including the

persistent effect of previous health in the dynamic models (Podsakoff et al., 2003). Moreover, it is not typically thought that common method bias is a concern in research studies that involve psychosocial work stressors and health (Bosma, Peter, Siegrist, & Marmot, 1998; Stansfeld et al., 1999).

7.2.4 External validity – generalisability

External validity, or generalisability, includes the representativeness of the study sample and the extent of the generalisation of study findings across wider populations. The HILDA survey is a nationally representative cohort of the Australian population, although it may underrepresent people with severe health conditions and generally contains a lower proportion of migrants and people from lower socioeconomic groups (Watson, 2011). The 66% initial response rate in the first wave suggests that it may not be wholly representative of working Australians and the greater retention of persons of higher versus lower SES across waves may affect generalisation. The disproportionate loss of lower SES and occupational status participants, who are more likely to be exposed to poor psychosocial working conditions and adverse psychosocial work stressors, may have biased results towards the null (LaMontagne et al., 2013). However, in Wave 11, the sample was topped-up to correct for population under-coverage and retain cross-sectional representativeness in the sample to include representation of migrant populations and help alleviate biases from non-random attrition (Watson, 2011). Retention across the characteristics of the top-up sample was considerably higher than that reported for previous waves (Summerfield et al., 2019).

The main work stress model used in the construct of the psychosocial work stressors exposure measurements was the job demand-control-support model. There has been some criticism that the job demand-control-support model is too task-focused and does not take into account the context of tasks (Mark & Smith, 2008). The overarching concern expressed includes its redundancy and irrelevancy to modern working environments. The model was developed over

half a century ago, when the workplace was dominated by industrial occupations and male workers (Berkman, Kawachi, & Theorell, 2014). Over the decades, the workplace has changed drastically, including the nature of work itself and the increasing inclusion of women in the workplace. The constructs of job demands and job control, based on the work habits in the middle of the 20th century, may no longer accurately reflect the contemporary workplace. Some items originally included in the job demand-control model might need to be excluded from the construct of job stressors. A study that examined the factor structure and evaluated the longitudinal measurement invariance of the job demand-control-support model found that excluding the repetitive work item from the construct of job control together with either work fast or work intensively from the construct of job demands improved the model fit (Chungkham, Ingre, Karasek, Westerlund, & Theorell, 2013).

Furthermore, while the demand for flexibility in the workplace has been growing over the last decade, the recent COVID-19 pandemic has exacerbated the demand for flexibility. Many workers have been forced to work from home, yet the boundaries between home and work are not clear. This has ramifications as the development of new problems of decision latitude and skill discretion may render the existing work stress model less relevant. The changing nature of work exacerbated by the pandemic has contributed to increasing job insecurity and a distinct lack of control-associated issues. So-called essential workers who are overwhelmingly employed in low paid and highly casualised occupations such as non-professional healthcare workers, warehouse distribution workers, delivery drivers, and meat processing and supermarket workers are faced with new decision latitude items and even less control over their work to the detriment of their health and well-being. Thus, the job demand-control model needs to be redeveloped with respect to current issues pertaining to job demands and job control in today's world. However, for the period relevant to the research presented in this thesis, which

precedes COVID-19, the job demand-control model is generally suitable for studying occupation-related health (Ahlin, Westerlund, Griep, & Magnusson Hanson, 2018).

7.2.5 Precision – Random error

In contrast with internal validity, precision relates to the lack of random error. Normally in epidemiological research, internal validity is considered to have priority over precision; however, a slightly biased, highly precise estimate may be more desirable than an unbiased but highly imprecise estimate (Porta, 2014). Random error may be of some concern when the study sample size is small. The smaller the study, the greater the chance of not detecting true associations due to the lack of power. A measure of precision is CIs or margin of error. Generally, the smaller the sample size, the wider the CIs and the higher the margin of error limiting our ability to precisely estimate associations. In Study I, the statistical combination of results from multiple studies using a meta-analysis approach increased power (over individual studies), improving the estimates of the effect size and decreasing uncertainty. The survival analyses conducted in Studies II and III were limited by the lack of power due to the small number of mortality events observed in the HILDA study population. The number of deaths included in the sample was small. There were 298 observed deaths with a mortality rate of 22.4 per 10,000 person-years in Study II and 247 observed deaths and a mortality rate of 16.0 per 10,000 person-years in Study III. The small number of outcome events observed in these studies may have affected the precision. This was more of a concern for female participants as the majority of deaths were observed in males; hence, I cannot be certain that the lack of effects observed for females was due to the lack of power in the study to detect associations between psychosocial work stressors and mortality for females. This limitation may have resulted in an underestimation of our risk estimates.

7.3 Association and Causality

As discussed in the introduction, evidence on mortality and psychosocial work stressors is weak and inconsistent, and research is lacking on the causal mechanisms through which job stressors affect health. It is not yet fully understood the extent that exposure to adverse psychosocial work stressors causes ill health due to methodological issues, including confounding, reverse causation and common method bias survey-based observational studies. The strongest evidence for causation is commonly derived from intervention studies, yet observational studies have provided almost all the evidence on psychosocial work stressors and health and mortality, thus limiting causal inference. To strengthen the causal inference, a thorough systematic review and meta-analysis was undertaken in Study I. Longitudinal designs were applied in Studies II and III, ensuring the temporal precedence of exposure in relation to outcome, controlling for sociodemographic and health characteristics at the baseline and over time. Causal inference from observational studies can be strengthened by triangulating results from different approaches with differing strengths and limitations to strengthen the confidence in results (Lawlor, Tilling, & Davey Smith, 2016). The causal relationship between job control and general health was explored via the integration of different analytical and epidemiological designs, using three complementary longitudinal modelling approaches, including dynamic fixed-effects regression to control for time-invariant confounding and ordinary least squares regression with cumulative job control exposure to preclude reverse causation.

Mechanistic evidence strengthens the case for causal inference. The observed associations presented in the thesis are biologically plausible. Changes in brain stress-responsive neurocircuitry stimulate peripheral physiological responses in the sympathetic autonomic nervous system and HPA via the release and control of adrenaline and the stress hormone cortisol in the body, resulting in increased inflammatory protein levels (Kivimaki & Steptoe, 2018). To cope with stress, psychological and physiological processes are altered, resulting in

chronic over- or under-activity of allostatic systems setting new baseline levels that can lead to the deterioration of body systems from repeated and unresolved stressors (McEwen, 1998). There is evidence that work stress is related to elevated stress responses in terms of sympatho-adrenal and HPA axis biomarkers (Chandola, Heraclides, & Kumari, 2010). Previous cross-sectional analysis from the Whitehall II study has shown low job control is associated with poor autonomic function (Hemingway et al., 2005) and neuroendocrine activation during the working day (Kunz-Ebrecht, Kirschbaum, & Steptoe, 2004). Further longitudinal analyses have shown that work stress is related to CHD (Kuper & Marmot, 2003) and the metabolic syndrome (Chandola et al., 2006). The cardiovascular system is particularly affected by stress. The combined effects of dysfunction of the autonomic nervous system and HPA responses over time can result in increases in platelet activation, blood fibrinogen levels and blood pressure levels, accelerating the atherosclerotic process and increasing the likelihood of fatal and non-fatal cardiovascular or cerebrovascular events (Kivimaki & Steptoe, 2018; McEwen, 1998). Analyses of cumulative work stress found chronic stress can lead to CHD via direct activation of neuroendocrine stress pathways and indirectly via health behaviours, especially among the younger, working-age population (Chandola et al., 2008). Approximately 32% of the effect of work stress on CHD was via indirect effects of health behaviours and direct effects on the metabolic syndrome (Chandola et al., 2008).

The evidence of an association for a causal relationship in observational studies can be assessed using the Bradford Hill criteria (Hill, 1965; Rothman, 2012). Although observational studies cannot unequivocally prove causation, the greater their accordance with the nine Bradford Hill criteria, the stronger the causal inference. The main associations in this thesis can be assessed for possible causality according to the Bradford Hill criteria. However, although they are presented separately, it is important to consider the nine factors collectively when assessing causality. The first criterion involves the strength of the association. A strong association is

more likely to be causal than a weak association and less likely to be explained by unrelated biases, although causality should not be readily dismissed in the absence of an observed association. This condition was met in all studies in the thesis. A persistent and strong association was observed for job control and mortality and health across all the studies. The second criterion, consistency such that repeated observations of an association in different populations and settings provide additional support for a causal association, was met in Study I. Meta-analyses of all observational studies to date have examined psychosocial work stressors and mortality using different populations, occupations and exposure definitions and showed strong associations between low job control and increased mortality. The third criterion is specificity, whereby an exposure leads to a single outcome, although this was later qualified to acknowledge that an outcome may be due to multiple causes rather than one single cause. This is a weak criterion because exposure to adverse psychosocial work stressors is likely to have multiple effects, but causality can be strengthened if an association is restricted to particular groups with a specific environmental exposure or magnified in these groups. Specificity of exposure was demonstrated by the results found for workers exposed to low job control and health, and mortality in all studies. Bradford Hill and Rothman both maintained that the next criterion, temporality, which is the cause, must precede the effect and is a necessary and inviolable criterion in assessing causality. This criterion was met in all studies in the thesis. The exposure measurements preceded the outcome of interest, mortality, in the meta- and survival analyses. The dynamic fixed-effects regression modelling and cumulative exposure analysis performed in Study IV allowed for the temporal precedence of exposure and accounted for both exposure intensity and duration. A further criterion, biological gradient as demonstrated by a dose-response association, was put forward by Bradford Hill, although Rothman disagreed believing that monotonic, dose-response relationships are not causal and are sometimes the result of confounding, in that the confounder itself is the cause of

monotonicity (maybe because the confounder is a biological gradient). He argued that the existence of a monotonic association was neither necessary nor sufficient for a causal relationship. Nonetheless, a strong stepwise relationship between increasing job control and general health was observed for participants in the highest job control quintile compared to those in the lowest quintile in Study IV.

The next Bradford Hill criterion, plausibility, refers to the scientific plausibility of an association. A known mechanism linking the cause to the effect supports a causal interpretation; however, absence of a plausible mechanism is not evidence of no causality. Plausibility is based on prior knowledge and can change with time as new knowledge is gained; hence, biological plausibility depends on the biological knowledge of the day. Nonetheless, it should not be discounted and, as described in the introduction and above discussion, the observed associations are biologically plausible. The seventh criterion, coherence, requires that the association is explainable by well-known facts and existing theory. The absence of coherent information does not rule out a causal relationship. Similar to the plausibility criterion, the coherence criterion was met. The eighth criterion required in support of causal inference involves experimental evidence. The most persuasive evidence for causation is observing changes in outcome directly related to modifications in the exposure. Although difficult and challenging, experimental manipulation of psychosocial work stressors and assessing the associated effects on health and mortality is still possible. Ethical interventions aimed at reducing exposure to adverse psychosocial work stressors such as low job control and job insecurity and increasing social support at work have shown improved health from pre-intervention conditions (Marmot et al., 2006). Data from some natural experiments have shown that organisational changes such as downsizing and mergers used as proxy measures for work stressors are associated with an increased risk of cardiovascular mortality and ill health among the remaining employees in the organisations (Kivimaki et al., 2000; Vahtera et al., 2005;

Vahtera et al., 2004; Westerlund et al., 2004). The final criterion, analogy, is considered a weak criterion of causal inference. The transferring of knowledge across contexts may be distorted by some researchers to support the hypothesis about an association in question. It is not difficult for inventive researchers to put forward an analogy in support of causality.

The collective assessment of the nine Bradford Hill criteria regarding the results of the four studies in the thesis supports a possible causal relationship between psychosocial work stressors (particularly job control) and health and mortality. Nonetheless, it is difficult to infer causality with total confidence owing to methodological biases as discussed above and several limitations in the studies, which will be discussed further below.

7.4 Strengths and Limitations of the Research

The strengths and limitations of this thesis, which have been touched on in the discussion regarding methodological considerations, will be reviewed below, focusing on the analyses and data included in the studies.

7.4.1 Strengths

To the best of my knowledge, this thesis includes the first review to systematically and quantitatively synthesise the respective epidemiological evidence for psychosocial work stressors exposures and CHD mortality or all-cause mortality. The findings of the detailed and comprehensive systematic review, including quality assessment, and meta-analyses of the available literature further clarified the relationship between psychosocial work stressors and mortality. While many individual studies observed an elevated risk of mortality and low job control, the association was not supported following adjustments for related factors typically due to lack of statistical power as most studies contained small sample sizes with fewer than several hundred deaths. Thus, combining studies in a meta-analysis greatly increased the statistical power and allowed the association to be examined with confidence. The observed

association detected between low job control and increased all-cause mortality, in addition to CHD mortality, in minimally- and multivariable-adjusted meta-analyses completed in this thesis strengthens the argument that job control is predictive of mortality.

The thesis presents results of the first Australian studies to rigorously examine the relationship between psychosocial work stressors and mortality from a nationally representative working population sample. Psychosocial work stressors were measured at the baseline, which explicitly asserts the predictability of mortality from baseline exposures in the initial study followed by examining the effect of time-varying psychosocial work stressors on health and mortality. This allowed for a robust examination of the association using a single measurement in time for the exposure versus multiple repeated measures over time, allowing for assessment of the predictability of mortality based on single and multiple measures of psychosocial work stressors. Significantly, low job control was associated with a 39% increased risk of all-cause mortality regardless of whether the exposure was based on a single or multiple repeated measures of psychosocial work stressors. This suggests that for this cohort and this historical period, a single baseline exposure measurement served as a reasonable proxy for exposure over time from that point forward for the assessment of job control and mortality. Although causal inference is limited in longitudinal studies, they do allow for the temporal precedence of the exposure, which reduces the effects of reverse causation and confounding, and generates new insights into the relationship between psychosocial work stressors and mortality (Taris & Kompier, 2003). The study quality was increased using time-varying measures of psychosocial work stressors that allowed for assessing changes in psychosocial work stressors, and changes in other relevant factors over time (de Lange et al., 2003). Further advantages of examining changing psychosocial work stressors are the capacity to include transitions from employment in and out of unemployment and retirement to measure their effect on the relationship between mortality and work stressors. In Study IV, examining the causal relationship between job

control and general health via triangulation, using three complementary longitudinal modelling approaches, permitted stronger conclusions regarding the potential causal relationship between job control and general health. Dynamics of the connexions between the exposure and outcome accounting for exposure intensity, duration and change were examined controlling for time-invariant confounding and reverse causation. These analyses were made possible by the availability of annual waves of the HILDA survey data.

The HILDA survey is a nationally representative longitudinal study of Australian households that has been collecting detailed information about economic and personal well-being, labour market dynamics and family life annually since 2001. It has a large sample size with long follow-up duration, following over 17,000 Australian lives annually. The panel data allows for the assessment of cumulative psychosocial job stressors and changes in socioeconomic, occupational and well-being characteristics over time. Health-related questions enable the scrutiny of changes in self-reported health status, long-term disabilities and impairments and health behaviours, allowing for the analyses of changes in general health regarding changes in job control over time. The HILDA survey is linked to the NDI, which allowed for an objective measure of outcome that was independent of the exposure data source, thereby avoiding dependent misclassification bias. The use of HILDA survey data in the analyses enhanced the statistical power and precision, and the generalisability of the conclusions.

7.4.2 Limitations

Several limitations need to be considered, most of which have been alluded to in the methodological considerations. There is a concern regarding insufficient variance for psychosocial work stressor exposures when using generic work strain instruments in single occupation studies that may have biased results of the meta-analyses towards the null. These generic measures, although well-validated, may not have measured psychosocial work stressors appropriate to some occupations such as exposure to violence in healthcare workers.

The lack of high-quality individual studies in the meta-analyses may have potentially biased the results towards the null. The main limitation concerning the meta-analyses was the inability to meaningfully examine the effect of sex, an important potential effect-modifier, on the association between psychosocial work stressors and mortality owing to the insufficient number of available studies. Nonetheless, results for the sex-stratified random-effects meta-analyses were included as supplementary analyses. Similarly, it was not possible to perform meta-regression analyses to evaluate the effect of other potential sociodemographic and health modifying factors on the association between psychosocial work stressors and mortality due to the small number of studies in the meta-analyses.

Psychosocial work stressors exposures were measured at the baseline in Study II; however, a single exposure measurement may not be adequate for assessing the effects of exposure duration and changing psychosocial work stressors. Nonetheless, understanding the association between psychosocial work stressors and mortality at a point in time is useful for comparisons with previous research, which are mostly based on the baseline measurements of exposures, and the targeting of interventions to improve work quality. The construction of psychosocial work stressors was limited to those scales and items available in the HILDA survey. Perceptions of satisfaction with pay based on a single item rather than other psychological benefits of work may not have high validity, which may explain the inconclusive results. Constructs of the psychosocial work stressors were based on work stress models developed last century. It may be that exposure measurements derived from potentially outdated theoretical models do not accurately reflect psychosocial work stressors in contemporary working environments. This may be more of a concern for the measurements of job security and job demands as their perceptions may have changed over the decades due to a shift in community acceptability of increasing job demands and high job insecurity in modern work. Nevertheless,

the measures used have been validated and demonstrated predictable associations with mental and physical health outcomes (Butterworth et al., 2011; Leach et al., 2010).

Measurements of psychosocial work stressors were self-reported and may have been subjected to reporting bias; hence exposure misclassification cannot be totally excluded. However, the subjective assessment of work stressors may be preferable to objective assessment because health could also be influenced by perceptions of work and the actual conditions of the work environment (Frese & Zapf, 1988). Common method bias may have been a limitation in Study IV. Both the exposure and outcome measures were based on self-reported measurements; hence the possibility that perceptions of job control and health could have been systematically exaggerated. However, in studies of work characteristics, self-reports may be a more accurate assessment as characteristics of the working environment are best represented by the workers themselves (Rugulies, 2012).

There may be an indirect effect of psychosocial work stressors on mortality mediated by poor health and an unhealthy lifestyle; however, it is not clear whether over time these covariates are more likely to be mediators, confounders or a combination of both. However, health and behavioural factors are strong predictors of mortality and are associated with work stress (Chandola et al., 2006). Adjustment for these risk factors may control for selection into stressful work environments because of early risk factors and adverse environments during childhood and adolescence (Hughes et al., 2017). However, controlling for these mediators may have resulted in over adjustment and an underestimation of risk. Additionally, residual confounding by unmeasured risk factors such as personality traits and life stressors cannot be ruled out.

Longitudinal studies are prone to selection bias owing to various attrition issues, including non-response, loss to follow-up and missing observations, as previously discussed in the methodological considerations. Missing data has the potential to increase the risk of systematic

and random errors. Although the HILDA survey is fairly representative of the Australian workforce, healthy worker selection bias cannot be precluded. The underrepresentation of people with poor health and from lower socioeconomic groups and the disproportionate loss of respondents from lower socioeconomic and occupational groups who are more likely to be exposed to poor health and psychosocial working conditions limit generalisation of the studies using HILDA data. However, loss to follow-up in consecutive HILDA waves was low (< 10% for most waves) and the effect of missing data across waves was minimised using unbalanced time-varying survival models and dynamic panel models with fixed-effects. It is also unclear to what extent our results can be generalised to other countries.

7.5 Conclusions

In this thesis, the effect of exposures to psychosocial work stressors in the working environment such as job control, job demands, job strain, long working hours, job insecurity and shift work on health and mortality were investigated. A comprehensive systematic review including meta-analyses of the epidemiological research was conducted. Panel data from the HILDA survey was used to capture natural experiments of changes in psychosocial work stressors associated changes in health and well-being, and mortality to investigate whether and how the effect of psychosocial work stressors on mortality differs in relation to demographic and socioeconomic characteristics.

Psychosocial working conditions, or work stressors, are important and modifiable determinants of health and health behaviours and have been identified as significant risks related to global changes in the structure, organisation and management of work. Most of the literature to date has assessed disease or other morbidity outcomes, with a growing number of studies on mortality. The first study represented the first systematic review and meta-analysis on the relationship between psychosocial work stressors and mortality. Pooling results from individual studies together, it was found that workers with low job control are at an increased

risk of all-cause and CHD mortalities compared to workers with high job control, even after controlling for other risk factors. This is an important finding, because if we can improve how people work and contribute to better health and well-being, reducing sickness absence and presenteeism, there is the potential to reduce those workers risk of premature mortality.

The second study quantified the effect of adverse psychosocial work stressors in working environments, such as high job demands, low job control, job insecurity and low fairness of pay, on all-cause mortality and is the first Australian study to examine these relationships. This study adds to a growing body of evidence showing an effect of deleterious psychosocial work stressors on mortality. Low job control was associated with an increased risk of all-cause mortality independent of demographic, socioeconomic, health and behavioural factors. Given the high prevalence of exposed individuals across the working population, job control-associated increases in mortality could have considerable public health consequences.

Building on the results from the second study, the third study was the first Australian study to quantify the effect of repeated exposures to adverse psychosocial work stressors including high job demands, low job control, job insecurity and ERI over time on all-cause mortality, controlling for unemployment periods, and the effect of non-participation in the labour force or retirement on mortality. The findings in this study have implications for workers exposed to low job control and job security over time, which were associated with a 39% and 36% increased risk of all-cause mortality, respectively. Although the estimates for the single measures suggest modest increases in the risk of death, this translates to large population impacts because the exposures are common across the working population.

The final study examined whether changes in job control over time were associated with changes in general health perceptions through triangulation, using three complementary longitudinal modelling approaches, in a nationally representative longitudinal study of

Australian households. Using within- and between-person dynamic modelling methods, a strong stepwise mostly exposure to outcome relationship between increasing job control and improving general health was observed. Cumulative exposure to low job control results in increasingly worse general health over the years. The findings of this study provide the best observational evidence to date that changes in job control are causally associated with corresponding changes in perceptions of general health—a well-validated predictor of objective health outcomes and mortality.

All four studies are emblematic of the work stress process depicted in presented framework (Figure 1.1) as described by Israel (Israel et al., 1996), illustrating the complexity and diversity associated with the work environment and health. The findings of this thesis support the construct of the work stress process as a multifaceted and dynamic process. Irrespective whether the relationship between psychosocial work stressors and health, and mortality is proximate or through perceived stress and short-term responses to stress, or influenced by persistent long-term health outcomes, previous psychosocial work stressors, or socio-demographic, health and health behaviour risk factors, the deleterious effects of exposure to adverse psychosocial work stressors is demonstrated in this thesis.

7.6 Relevance and Implications for Public Health

Scientific evidence is important; however, absolute proof is not always necessary before introducing recommendations for interventions and change. Sometimes it is necessary to act when there is evidence beyond reasonable doubt that supports an action. There is now an accumulation of evidence beyond reasonable doubt that acting to reduce exposures to adverse psychosocial work stressors is better for workers' health and well-being than the risks associated with the status quo. Action is justified when enough is known to justify it. Waiting for all the facts and being overly cautious will result in preventable harm to workers. This was exemplified by John Snow in 1855 during the cholera epidemic in London. He did not wait

until being absolutely certain of the cause of the outbreak before strongly advocating for the removal of the Broad Street water pump. He was certain beyond reasonable doubt, although not absolutely certain, that the outbreak was linked to the water pump in Broad Street.

> The most terrible outbreak of cholera which ever occurred in this kingdom, is probably that which took place in Broad Street, …. I found that nearly all the deaths had taken place within a short distance of the pump. I had an interview with the Board of Guardians of St. James' parish, … In consequence…, the handle of the pump was removed on the following day. (Snow, 1855)

Snow felt that although the cause of the outbreak was not yet known, enough was known to justify the action subsequently bringing the cholera epidemic in London to an end.

In the present era, 165 years after the London cholera epidemic, we are now in the midst of a worldwide severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) pandemic. There is enough knowledge about the benefits of using masks during the pandemic to justify making wearing masks mandatory, yet inaction by governments in many countries worldwide persists. Although the full extent to which asymptomatic and pre-symptomatic people with coronavirus disease (COVID-19) could spread the virus was not understood, there was considerable consensus in the scientific community that enough was known to justify the mandatory use of masks. However, masks became a source of contention and, rather than acting on what was known and instigating mandatory masks earlier in the pandemic, the virus was allowed to spread rapidly throughout many countries (Peeples, 2020). The small number of countries including Australia and New Zealand that acted early during the pandemic without waiting for absolute knowledge mitigated risks associated with COVID-19, reducing harm to their citizens. In contrast, countries that resisted action are now in the midst of the uncontrolled virus with rates of new COVID-19 cases and deaths continuing to increase (World Health Organization,

2020). In public health, adequate knowledge is sufficient for action as no action while waiting for absolute knowledge can be harmful to the individual and society at large.

It is now known that poor-quality work is a major driver of inequalities in physical and mental health. The conditions in which we work impact on our health. While good-quality work may be protective of health, exposure to psychosocial work stressors is associated with adverse health outcomes. Furthermore, poor-quality work is unequally distributed with those workers at the lower end of the social gradient disproportionately affected (Durcan, 2015). Despite this knowledge and the recommendations for improving the quality of work across the social gradient made by researchers to governments over the last decade, there is considerable reluctance and a lack of will for change due to vested interests from stakeholders (Marmot, Allen, Boyce, Goldblatt, & Morrison, 2020). Yet the solutions are obvious and include the development of better-quality jobs and the improvement of the quality of existing low-skilled jobs using our existing knowledge about what comprises good- and poor-quality work (Durcan, 2015). Industry in partnership with government can implement strategies that reduce exposure to adverse psychosocial work stressors and promote conditions for a healthy workplace.

The findings in this thesis in addition to the existing knowledge about the detrimental effects of adverse psychosocial work stressors on health and mortality indicate that there exists sufficient knowledge warranting public health action. This thesis suggest that it is important to reduce exposure to psychosocial work stressors, in particular exposure to low job control. A lack of control, whether it be in humans or animals, is known to cause stress, regardless of the social status of the organism (Marmot et al., 2006). Chronic stress signals the brain to mobilise cortisol, adrenaline and noradrenaline hormones causing profound biological changes in metabolic, cardiovascular and immune systems, resulting in diseases (Ganster & Rosen, 2013). There is a social gradient for job control; the lower the social position, the more common it is to lack control over work (Marmot et al., 1997). Hence, it is vital that workers are abetted and

enabled to increase skill discretion and decision authority over their work. Providing opportunities to employees for progression and development, autonomy and independence, and involvement in the decision-making process limit exposure to adverse psychosocial work stressors, improving working environments. Factors that contribute to the development of healthy workplaces include accessibility to management, transparent and fair decision-making processes, effective communication across all management levels, safe and collaborative working environments, and an adequate number of employees (Lindberg & Vingard, 2012).

Where organisational changes such as downsizing and mergers have been used as proxy measures for work stressors, it has been shown that work stress reduction programs are effective. A study conducted during a period of structural organisational changes in Sweden found that after 1 year, serum cortisol levels had decreased and perceptions of decision authority were higher in the intervention group due to decreased uncertainty among the workers following implementation of the program (Theorell, Emdad, Arnetz, & Weingarten, 2001). Other researchers have shown improved health outcomes associated with interventions targeting psychosocial work stressors in the workplace (Wahlstedt, Håkan Nygård, Kemmlert, Torgén, & Björkstén, 2000). It is difficult to ascertain the effectiveness of such interventions. The unpredictability and instability of labour markets may limit evaluation due to interruptions caused to the implementations from mergers, downsizing and outsourcing. Furthermore, it is usually not meaningful to randomise individual workers in the workplace owing to the strong possibility of contamination between the intervention and control groups, although cluster randomised controlled trials may mitigate somewhat against this risk. In general, interventions are costly, disruptive and time-consuming to the workplace; all of which make experimental manipulation of psychosocial work stressors very difficult. Hence, there is uncertainty regarding the effects of intervening on psychosocial work stressors on health and well-being. However, this is not to say that we should endorse not acting. On the contrary, the level of

knowledge regarding the effects of adverse psychosocial work stressors on health and mortality dictates that we actively establish public health policies for societal change at national and organisational levels.

There are efforts underway to improve and promote mentally healthy workplaces by the Australian Federal Government. An investment of $11.5 million over 4 years for the National Workplace Initiative was announced in the 2019–20 federal budget (Australian Government National Mental Health Commission, 2020). The National Workplace Initiative was initiated by an alliance between business, union, community and government sectors to improve workplace mental health outcomes. It is hoped that an evidence-based framework for workplace mental health strategies in Australia will be developed and implemented. The World Health Organization has initiated the Global Plan of Action on Worker's Health (2008–2017) and the Mental Health Action Plan (2013–2030) to address policy globally. They outline appropriate principles, objectives and implementation strategies for the development of mentally healthy workplaces. Working conditions that contribute to the social determinants of mental health are addressed and health promotion programs recommended.

Public health encompasses an assembly of cohesive and interconnected multi-systems entrenched in cultural and societal conventions. Knowledge and expertise in public health are determined by science, governance and implementation of social policy in agreement with science must be undertaken by governments and there must be trust in science and the government by the people and individual responsibility to comply with the public health measures. Reducing adverse psychosocial work stressors entails public health action at three levels: individual, interpersonal and structural, requiring commitment from governments and industry involving compulsory work policies and regulatory conditions (Marmot et al., 2006). The findings in this thesis showed that the psychosocial work environment is an important determinant of health and contributor to the social gradient in ill health and mortality. Ideally,

there should not be a trade-off between work productivity and health if employers were to take a long-term view and honour the social contract of work rather than consider their employees as disposable. Reducing exposure to adverse work stressors is advantageous to employers as it will lead to happier, healthier and more productive workers and hence a more productive workplace. Interventions targeting those psychosocial factors that can be improved and are practical for modification such as the dynamic involvement of workers in the decision-making process at every level in an organisation are beneficial to the employee and the organisation.

Internal procedures and processes in organisations that provide workers with increased control, diversity and employment prospects should be developed and incorporated by employers. Although improving the quality of the psychosocial work environment may be perceived negatively by many employers, improvements are in reality beneficial to the prosperity of the organisation. The employer has a responsibility to minimise the exposure risk to adverse work stressors in the workplace to protect and promote the health of workers. In the long-term, the implementation of interventions to improve the health of workers and maintain healthy workplaces is financially advantageous for organisations and society at large, through the reductions in healthcare and welfare costs. Employers can expect a return on investment via substantial medium-term cost savings of such interventions in the workplace (Marmot et al., 2006). Joint efforts are required from stakeholders, governments and organisations to improve the working environment and social inequalities in health. Appropriate remuneration and rewards should be provided for stressful work conditions. Workers should be provided with the opportunities for employment training, developing and learning new skills, and increased job security. It should be a mandatory legal requirement that organisations deliver effective workplace strategies and measures with provisions for workplace inspections and evaluations. Ideally, workplace health services are incorporated into organisations whereby recognition and management of work-related stress health problems is promptly implemented.

These recommendations are not new. Researchers including Marmot and Theorell have been advocating for these changes in occupational policy for over a decade. Efforts to improve working conditions have led to changes in work health and safety in Australia. Employers are now legally responsible for the physical and psychological health and safety of their workers in the workplace. The lack of suitable action by employers to maintain a healthy and safe work environment can result in criminal prosecution or fines under the Work Health and Safety Act (Work Health and Safety Act, 2011). Safe Work Australia (2020) sets out the various regulations, codes of practice and guidance material to assist with managing risks to physical and psychological health as required by the Work Health and Safety Act. However, strengthening the protection and promotion of psychological health in workplaces remains a challenge for all jurisdictions (Potter et al., 2017).

The consequences of not protecting workers from exposures to adverse psychosocial work stressors have been demonstrated during the COVID-19 pandemic. The pandemic has demonstrated that the inequity in society is a risk for the spread of infection (Marmot & Allen, 2020). Contrary to popular belief, COVID-19 does discriminate. We may all be faced with the possibility of contracting the virus, but we do not all have the same chance of infection. While COVID-19 is caused by a virus, the same social determinants of health that are responsible for the social gradient in health are the drivers for the distribution of the virus. There are specific causes that magnify this inequity, including employment in frontline essential occupations. Most essential work is insecure work in low-skilled occupations where workers are poorly paid and have little control over their work. COVID-19 has exposed the reliance on essential workplaces, including meat processing plants, aged care, hospitality and supermarkets, on a highly casualised workforce and exacerbated the effects of psychosocial work stressors on health and mortality (Windsor-Shellard & Butt, 2020). The pervasiveness of low job control, high job demands and high

job insecurity in these essential workplaces contribute to the high risk for infection transmission. Most of the excess mortality in vulnerable populations from COVID-19 can be attributed to essential employment and crowded conditions in multi-generational households (Brandily, Brebion, Briole, & Khoury, 2020; Selden & Berdahl, 2020).

The findings of this thesis have been highlighted by COVID-19 on a global stage. Characteristics of the working environment have become important drivers of the spread of the disease and our governments are beginning to take notice. Only recently, the Premier of Victoria, Daniel Andrews, announced a 2-year trial of a minimum sick pay and carers pay scheme for workers in insecure work, focusing on industries where insecure work is known to be widespread such as hospitality workers, aged care workers, cleaners, security guards and supermarket staff (Masige, 2020). This was in direct response to a large-scale outbreak of COVID-19 in the community resulting in devastating social and economic consequences for the state because of outsourcing hotel quarantine security to casual workers (Coate, 2020). The research presented in this thesis, while predominantly academic, is also highly relevant to all aspects of life, including work and health, and reaching across all levels of society, including the individual, organisations and governments.

7.7 Recommendations for Future Research

This thesis has contributed to the knowledge of the relationship between psychosocial work stressors, and health and mortality by comprehensively reviewing the evidence in the literature and applying longitudinal and statistical methods to data from a large national representative sample of the Australian population. Nonetheless, there is scope for further work. There is a lack of research into the cumulative effect of psychosocial work stressors, and health and mortality. It would be of interest to examine the effect of changes in work stressors over time on general health and mortality in large longitudinal studies to consolidate our understanding and determine whether changes in psychosocial work stressors over time translate to changes

in health and mortality. This understanding is vital for effective interventions in the workplace to reduce work stress-related health problems as interventions can be targeted at specific changes to achieve optimal effect. While the causal relationship between job control and health was examined in this thesis, further research is required to investigate the direct and indirect pathways between psychosocial work stressors, health and mortality to disentangle the subtleties of the relationship among exposure, mediator and outcome factors and strengthen causal inference. It is also important to further investigate the role of gender in the relationship between psychosocial work stressors and health and mortality. Large-scale longitudinal studies with sufficient power are required to accurately assess whether the relationship is modified by gender. Understanding the role of effect modifiers and mediators will better inform potential intervention opportunities.

There is also a need for natural experiments, or quasi-experimental designs, which are particular useful when effects are modest or occur over a long period (Craig et al., 2012). Changes in the work policies and regulatory conditions in workplaces implemented by organisations, such as those mentioned above, can be assessed using instrumental variable analyses to estimate the causal relationship between psychosocial work stressors and health, and strengthen causal inference. While the data on adverse psychosocial work stressors and health is considerable, it is sparse for the effect of good-quality work on health and mortality. There is little detailed information on which physical and psychosocial characteristics of work are protective of health across different occupations. There is a need for further research into the longitudinal monitoring of workplaces that have implemented programs improving psychosocial work stressors detrimental to health in the workplace. It is difficult to access improvements in working conditions and workers health if no baseline data or monitoring of progress is available (Durcan, 2015).

It is clear that the nature of work is changing drastically, and we may have to re-evaluate the way exposure measurements are constructed. The methods used to date, including in this thesis, to measure psychosocial work stressors may need reviewing in the future. The continuation of use of the standard job demand-control-support model and the ERI model in occupational research requires critical appraisal (Theorell, 2006). The relevance of job demands, job control, job security and fairness of pay constructed using these work stress models from last century to the modern psychosocial working environment requires evaluation. Other items reflecting common psychosocial work stressor exposures in the workplace today, for example exposure to violence and bullying, should also be considered. Updated work stress models reflecting characteristics of contemporary psychosocial working environment will allow for more accurate exposure measurements, enhancing the validity of research of psychosocial work stressors, and health and mortality.

7.8 Final Word

Psychosocial work stressors have been identified as significant emerging risks linked to global changes in the structure, organisation and management of work during previous decades, presenting great challenges to occupational health and safety and workplace health in general. I have attempted to clarify the relationship between psychosocial work stressors, and health and mortality using various statistical and epidemiological approaches. The findings of the thesis add to the evidence that exposure to adverse psychosocial work stressors is detrimental to health and well-being and strengthens the evidence for a causal relationship. The impact of low job control on worker health and mortality was consistently demonstrated across all studies. Given the high prevalence of exposed individuals across the working population, job control-associated increases in poor health and mortality have considerable public health consequences.

The validation of job control as a potentially modifiable causal factor of general health has considerable public health implications with potentially substantial economic consequences for employers and society. Awareness of the implications of the adverse effects of psychosocial work stressors on health and mortality in workplaces and appropriate work stress interventions to improve job control may contribute to better health and well-being, reducing sickness absence and presenteeism, and decreasing the effect of psychosocial work stressors on mortality benefiting the individual, organisations and society at large.

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