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Title:
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Date:
2019

Persistent Link:
https://hdl.handle.net/11343/235776

How do socio-economic characteristics influence the effect of disability acquisition on mental health? An analysis of effect modification and mediation

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Submitted in total fulfilment of the requirements of the degree of

Doctor of Philosophy

Centre for Health Equity

Melbourne School of Population and Global Health

Faculty of Medicine, Dentistry and Health Sciences

THE UNIVERSITY OF MELBOURNE

November 2019

Abstract

Background

People with disabilities in Australia experience poorer mental health than people without disability. However, the mechanisms by which disability leads to poor mental health are inadequately understood. This PhD thesis aims to form a better understanding of how people's socio-economic circumstances influence the effect of disability acquisition on mental health. Elucidating the causal mechanisms underpinning this relationship will inform the development of effective public health and social policies to improve the mental health of people with disabilities.

Methods

I used data from the Household, Income and Labour Dynamics in Australia Survey – a large nationally representative longitudinal dataset – to quantify the effect of disability on mental health and to examine the socio-economic mechanisms leading from disability acquisition to poor mental health. I identified adults who acquired a disability during their participation in the survey and used data both before and after they acquired the disability to estimate the causal effect of disability on mental health. Firstly, I conducted analyses of effect modification, examining a wide range of different socio-economic factors to determine whether they influenced the magnitude of the effect of disability acquisition on mental health, using inverse probability weighting and fixed effects models to better control for confounding. Secondly, I conducted causal mediation analyses to further examine the socio-economic mechanisms leading from disability to poor mental health, using sequential mediation analysis to examine a broad range of socio-economic characteristics and an interventional mediation approach to quantify indirect effects operating through two distinct socio-economic characteristics: employment and income. Finally, I examined the indirect effect mediated by employment in greater detail, further decomposing the natural indirect effect through employment to estimate the proportion attributable to interaction, mediation and their joint effects.

Results

There is a clinically significant and large effect of disability acquisition on mental health. The analyses of effect modification provided evidence that the magnitude of the effect differed according to people's socio-economic characteristics, with greater effects observed for more disadvantaged groups. The mediation analyses provided additional evidence that socio-economic characteristics contribute to the effect of disability acquisition on mental health. A third of the effect was found to be mediated by material socio-economic factors such as employment, income, wealth, financial hardship and housing characteristics, and a further investigation of the indirect effect through employment and income highlighted employment (but not income) as an important contributing factor, explaining 11% of the effect alone. Finally, further decomposition of the indirect effect through employment suggested that the mediated effect was due to interaction between disability and employment, rather than pure mediation.

Conclusion

The findings of this thesis highlight the importance of the social determinants of health in generating mental health inequalities. Interventions should prioritise addressing the social determinants of health to improve the mental health of people with disabilities and reduce disability-related mental health inequalities. Furthermore, the evidence that employment is a key mediator of the effect of disability acquisition on mental health indicates that policy strategies are needed to target the causes of low employment rates for people with disabilities.

Declaration

This is to certify that:

1. the thesis comprises only my original work towards the PhD except where indicated in the preface,
2. due acknowledgement has been made in the text to all other material used,
3. the thesis is less than 100,000 words in length, exclusive of tables, bibliographies and appendices.

Zoe Aitken, November 2019

Preface

Sources of funding

I am a grateful recipient of an Australian Government Research Training Program Scholarship and of a National Health and Medical Research Council Postgraduate Scholarship (1093740).

This paper uses unit record data from the Household, Income and Labour Dynamics in Australia Survey. The HILDA Project was initiated and is funded by the Australian Government Department of Social Services and is managed by the Melbourne Institute of Applied Economic and Social Research.

Co-author contributions to published papers

Section 4.1.2 of this thesis consists of a reprint of the following paper published in BMJ Open in July 2017:

"Disability acquisition and mental health: effect modification by demographic and socioeconomic characteristics using data from an Australian longitudinal study" [1].

I contributed 90% of the content in the publication, conceiving the research question, choosing the statistical methods, conducting the analysis, interpreting the results, drafting the manuscript and revising the manuscript following feedback from the co-authors and journal reviewers. My co-authors Julie Simpson, Rebecca Bentley and Anne Kavanagh contributed to the study conception, the choice of analytic methods, the interpretation of results and critically reviewed the manuscript.

Section 4.2.2 of this thesis consists of a reprint of the paper published in Social Psychiatry and Psychiatric Epidemiology in October 2019.

"Does the effect of disability acquisition on mental health differ by employment characteristics? A longitudinal fixed-effects analysis" [2].

I contributed 90% of the content in the publication. I designed the research question and chose the statistical methods to best conduct the analysis, carried out the analysis with guidance from Allison Milner, drafted the manuscript and completed the revisions. My co-authors Julie Simpson, Rebecca Bentley, Allison Milner, Anthony LaMontagne and

Anne Kavanagh helped design the research question, interpret the results and commented on the manuscript.

Section 4.3.2 of this thesis consists of a reprint of the paper published in Social Psychiatry and Psychiatric Epidemiology in July 2017.

“Does social support modify the effect of disability acquisition on mental health? A longitudinal study of Australian adults” [3] .

I contributed 80% of the content in the publication, conceiving the research question and choosing the statistical methods in collaboration with Lauren Krnjacki and Allison Milner, conducting the analysis, writing the draft of the manuscript and editing the manuscript in response to feedback from co-authors and reviewers. My co-authors Lauren Krnjacki, Anne Kavanagh, Anthony LaMontagne and Allison Milner assisted with the choice of methods, interpretation of results and reviewed the manuscript.

Section 5.1.2 of this thesis consists of a reprint of the paper published in International Journal of Epidemiology in January 2018.

“Do material, psychosocial and behavioural factors mediate the relationship between disability acquisition and mental health? A sequential causal mediation analysis” [4].

I contributed 90% of the content in the publication, coming up with the research question, choosing the analytic methods, writing the statistical code and conducting the analysis with advice from Lyle Gurrin, writing the paper and completing revisions based on comments from co-authors and reviewers. My co-authors Julie Simpson, Lyle Gurrin, Rebecca Bentley and Anne Kavanagh were involved with the conception of the research question, the interpretation of the results and all co-authors reviewed the manuscript.

Acknowledgements

First and foremost, I would like to thank my amazing team of supervisors: Professor Anne Kavanagh, Professor Julie Simpson and Professor Rebecca Bentley. I could not have felt more supported, especially these last couple of months in the final sprint towards the finish line. Firstly Anne, thank you for encouraging me to *take the plunge* into the PhD world and for providing me with guidance, wisdom and encouragement every step of the way, helping me to navigate the constant juggle of work, PhD and parenthood. Julie, your patience, optimism, confidence in my abilities and unwavering support – as well as your unparalleled statistical expertise and pragmatic advice – were invaluable and made it possible for me to submit in a timely manner. Bec, thank you for helping me to navigate the important and difficult decisions relating to the scope of the thesis in my first year and helping me to devise a programme of research that incorporated both my research and methodological interests.

Thank you to Peter Butterworth, the late Allison Milner and Dallas English for chairing my advisory committee at different times throughout my candidature and for providing excellent feedback, advice and valued support. Thank you also to Belinda Hewitt for being on my advisory committee and for always providing me with the most detailed and comprehensive feedback and valued support, guidance and mentorship. Thank you for the laksa lunches and all the laughs.

Allison, as well as being the chair of my advisory committee, a co-author on two of the publications and a valued mentor, you were most importantly a true friend to me and provided me with unwavering support throughout this PhD journey. You were an absolutely extraordinary researcher, an inspiring collaborator from whom I learned so much and loved working with. I will always fondly remember your brutal honesty, caring nature and unique, quirky sense of humour.

Thank you to my wonderful colleagues in the Disability and Health Unit and the Gender and Women's Health Unit at the Melbourne School of Population and Global Health for providing such a supportive and inspiring work environment. I love coming into work every day because of you all. Thank you particularly to Claudia and Ghazaleh for all the laughs, lunches, and coffees and to Tania, Ali and Lauren for your friendship over the last few years. I have also been very lucky to be surrounded by a talented and supportive group of fellow PhD students – to name a few, Liz, Sarah, Emma, Georgia, Lila, Marie, Ghazaleh, Lauren and Alex.

On a personal note, my deepest thanks go to my family and oldest friends – Annie, Sara, Kat, Wilko and Maz – who have provided unwavering support and encouragement through this PhD journey. My parents, for inspiring my love of learning and providing constant encouragement, support and belief; they have always been my greatest supporters. To my sister Maria – thank you for always being there for me, for your positive energy, your utmost confidence in my ability to see this through, the wonderful holidays together, and your beautiful boys Olby and Max for providing entertainment and distraction when I needed it. To Jimmy, I could not have achieved this without you by my side. Thank you for everything you have done to support me over the last four years, for your patience especially during these last few months, for providing me with encouragement and love when it was needed the most, and for entertaining me with your terrible jokes. Thank you to my beautiful boy Teddy, the light of my life, for seeing joy in everything, keeping me grounded, and making the last two years the happiest of my life. Finally, to my little girl – thank you for giving me the very firm deadline I needed to get this thesis finished. I cannot wait to meet you next week.

Contents

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List of abbreviations

CDE	Controlled direct effect
CI	Confidence interval
CRPD	Convention on the Rights of Persons with Disabilities
HILDA	Household, Income and Labour Dynamics in Australia
ICF	International classification of Functioning, Disability and Health
ICIDH	International classification of Impairments, Disabilities and Handicaps
IDE	Interventional direct effect
IIE	Interventional indirect effect
IPW	Inverse probability weight
MAR	Missing at random
MCAR	Missing completely at random
MHI	Mental health inventory
MI	Multiple imputation
MNAR	Missing not at random
NDE	Natural direct effect
NDIS	National Disability Insurance Scheme
NIE	Natural indirect effect
PM	Proportion mediated
SEIFA	Socio-economic indexes for areas
SF-36	Short form 36
TCE	Total causal effect
WHO	World Health Organization

Chapter 1 Introduction

1.1 Rationale

People with disabilities in Australia experience poor mental health compared to those without disability. A significant component of these mental health inequalities is driven by social and economic disadvantage rather than people's disability. This highlights that in principle this inequality is avoidable and the poor mental health of people with disabilities can be addressed through policy intervention. However, there is sparse evidence about the socio-economic determinants of the disability-related mental health inequalities and consequently a lack of knowledge about how to improve them.

This gap in knowledge is a significant impediment to the development of effective public health and social policies. Without research specifically into the experiences of people with disabilities and the complex interplay between disability and the social determinants of health, policies are unlikely to improve people with disabilities' mental health outcomes. Evidence relating to the social and economic mechanisms driving the mental health inequalities is necessary to understand the complex relationships between disability and poor mental health, which will identify targets for interventions to reduce the current inequalities and improve the health, well-being and lives of people with disabilities.

1.2 Problem statement

To date, there has been little research examining the mechanisms by which disability affects people's mental health. Most of the work that has been done has been conducted in studies of adolescents or older people, limiting the generalisability of the findings, and has been cross-sectional in design, making it hard to infer cause and effect. Furthermore, no study has examined a broad range of socio-economic factors that influence the effect of disability on subsequent mental health to comprehensively understand important determinants and drivers, and their relative importance.

A better understanding of mechanistic pathways will address an important evidence gap, providing vital information about how to reduce current disability-related mental health inequalities. It will identify socio-economic factors that contribute to the effect of disability on mental health, shedding light on modifiable targets for policy interventions to improve

the mental health of people with disabilities at a pivotal point in time given the changing disability policy landscape in Australia.

1.3 Aims, objectives and research questions

This thesis aims to form a better understanding of the mechanisms by which disability leads to a deterioration in people's mental health, specifically how people's socio-economic circumstances influence the effect of acquiring a disability on mental health. The research goal is to progress beyond quantifying the magnitude of the effect of disability acquisition on mental health, to gain insight into why, how and for whom the effect occurs. The thesis investigates how the effect of disability acquisition on mental health differs according to people's socio-economic characteristics to identify *for whom* the effect occurs. It also explores the pathways linking disability and mental health to understand *why* and *how* the effect operates, to understand the causal mechanisms at play.

In order to address my overarching research aim, the public health substantive research questions and objectives of my thesis are:

Research question 1:

Is the effect of disability acquisition on mental health modified by socio-economic characteristics prior to disability acquisition?

The first objective is to identify socio-economic factors that make people who acquire a disability vulnerable to poor mental health outcomes to differentiate subgroups of people with disabilities who are most likely to experience poor mental health and shed light on mechanisms leading from disability acquisition to poor mental health.

Research question 2:

How much do socio-economic characteristics subsequent to disability acquisition mediate the effect of disability acquisition on mental health?

The second objective is to disentangle the causal mechanisms leading from disability acquisition to poor mental health, identifying factors on the causal pathways between disability acquisition and subsequent mental health that may contribute to poor mental health outcomes.

To address these research questions, I conduct an epidemiological analysis estimating the effect of disability acquisition on mental health using a large nationally representative longitudinal dataset that follows a cohort of people over time, with repeated measures

on each participant. I identify people who acquired a disability in adulthood during their participation in the survey and use data both prior to and subsequent to disability to quantify how disability impacts on their mental health. I aim to identify socio-economic factors that modify and mediate the association between disability acquisition and mental health.

The following methodological approaches are employed:

i) to address research question 1, I conduct an analysis of effect modification to quantify the magnitude of the effect of disability acquisition on mental health according to people's socio-economic characteristics prior to disability;

ii) to address research question 2, I will apply innovative causal mediation analysis methods to decompose the total effect of disability acquisition on mental health into the effects operating through different pathways.

1.4 Effect modification

Effect modification occurs when the effect of an exposure on an outcome is different across levels of a third variable, leading to effect heterogeneity. Analyses of effect modification can explain when, for whom, or under what circumstances an exposure affects an outcome (or is of smaller or greater magnitude). Analyses of effect modification group individuals together based on a shared characteristic (the effect modifier) and quantify the effect of the exposure on the outcome in each subgroup to examine whether the characteristic modifies the effect.

Effect modification can be used to identify subgroups of people who are particularly vulnerable to poor outcomes. In situations where we cannot intervene directly on the exposure, such as disability, analyses of effect modification can identify other contributing factors that could be targets for interventions to eliminate a portion of the effect of the exposure of interest on the outcome. Furthermore, by understanding under which circumstances the effect is of a greater (or smaller) magnitude, effect modification can also provide valuable insight into the mechanism by which the exposure affects the outcome. It can tell us about the relative effects of contributing factors on the outcome, shedding light on the complex ways in which disability and contributing factors interact to affect poor mental health, which can be used to identify leverage points for policy intervention.

1.5 Mediation

Mediation occurs when the effect of an exposure on an outcome operates, at least in part, through an intermediary variable. Analyses of mediation seek to quantify how much of the effect is occurring through the intermediary variable - the indirect effect - and how much is occurring through other pathways – the direct effect.

Mediation analyses shed light on the mechanisms and pathways by which an exposure affects an outcome. Understanding the mechanism by which an effect operates can identify targets for policy interventions. It may be possible to eliminate or limit the effect of the exposure on the outcome by intervening instead on an intermediary variable, particularly in situations where it is not possible to intervene directly on the exposure.

Effect modification and mediation are not distinct phenomena - they can happen separately or simultaneously - and it is possible to separate out quantitatively how much of the effect is due to one or the other.

1.6 Significance

While the causal relationship between disability acquisition and poor mental health is well established, the mechanisms and pathways driving the mental health inequalities are poorly understood. Consequently, knowledge about how to reduce disability-related mental health inequalities is currently lacking.

There is a need for research to go beyond descriptive analyses of the disability-related mental health inequalities to understand the mechanisms linking disability and mental health, in order to identify factors that contribute to the inequalities that are amenable to policy interventions such as socio-economic characteristics. This will provide evidence that can be translated into interventions to improve the mental health of this disadvantaged subgroup of the population.

The research in this thesis has the potential to have real policy impact because it addresses key knowledge gaps and aligns with the priorities of the National Disability Strategy, a national framework to guide policy development by all levels of government to meet their obligations under the United Nations Convention on the Rights of Persons with Disabilities (CRPD) [5]. The Strategy identified improving the health and well-being of people with disabilities as one of six priority areas for policy action and specifically listed the lack of an evidence base on the social determinants of health for people with disabilities as a barrier to progress.

The research aims to generate findings that will facilitate evidence-based decision making to close current mental health inequalities. A strong emphasis on causal inference will maximise the likelihood that policies implemented based on the findings will be effective in improving mental health outcomes.

Given the persistent disadvantage and poor mental health experienced by people with disabilities in Australia over the last decades, this research with a focus on actionable policy interventions to improve mental health is timely and needed. Furthermore, given the implementation of the National Disability Insurance Scheme (NDIS) and the changing disability policy landscape, this is a pivotal opportunity to use robust evidence on modifiable social determinants of health to contribute to policy strategies to improve the mental health of people with disabilities and reduce mental health inequalities.

1.7 Outline

This thesis comprises seven chapters. A detailed description of the background to the thesis and the motivation for the research will be given in Chapter 2. This includes a description of disability in Australia including the epidemiology of disability and the policy environment (section 2.1). The different conceptualisations and approaches to measuring disability are explained in section 2.2. Disability and health inequity is discussed in section 2.3, including how disability is viewed in public health. Section 2.4 presents statistics about the mental health of people with disabilities in Australia and reviews the literature relating to the causal effect of disability on mental health and the influence of socio-economic characteristics.

Chapter 3 outlines the methodological challenges of using observational data to answer the research questions of the thesis (section 3.1) and describes the epidemiological and statistical methods used to address these challenges. Throughout the thesis, in order to address the many methodological challenges, a causal inference framework has been adopted to devise the analytic approach and interpret the findings, described in section 3.2. The data sources and variables are described in sections 3.3 and 3.4. The subsequent sections in this chapter provide detailed explanations of the epidemiological and statistical methods used in the thesis including effect modification analysis (section 3.5), causal mediation analysis (section 3.6), propensity score methods (section 3.7), fixed effects analysis (section 3.8), quantitative bias analysis (section 3.9) and multiple imputation (section 3.10).

Chapters 4, 5 and 6 present the results of the empirical analyses contributing to the thesis, each analysis written up as a standalone piece of work prepared for publication.

The aim of Chapter 4 is to discern whether (and to what extent) socio-economic characteristics modify the effect of disability on mental health. Chapter 4 includes three published articles examining this question. In the first, I examine effect modification by a broad range of demographic and socio-economic characteristics to determine whether they influence the effect of disability acquisition on mental health (section 4.1) [1]. The second paper examines effect modification by employment characteristics in more detail (section 4.2) [2] and the final article in this chapter presents the findings of a study of effect modification by social support, measured at two different time points to understand both how prior social support and changes to social support as a result of disability modify the effect of disability acquisition on mental health (section 4.3) [3].

The second chapter of results presents the findings of the mediation analyses (Chapter 5), including one published article and one unpublished analysis. The first section presents the results of an analysis quantifying the indirect effect of disability acquisition on mental health operating through three broad categories of socio-economic characteristics including material, psychosocial and behavioural factors examined sequentially (section 5.1) [4]. The second section presents the results of an analysis that quantifies mediation through two distinct socio-economic characteristics, employment and income, estimating interventional indirect effects through each pathway (section 5.2).

The final results chapter brings together the concepts of effect modification and mediation quantitatively (Chapter 6). It presents the results of an analysis examining employment as a mediator, further decomposing the indirect effect of disability acquisition on mental health operating through employment into the components due to interaction only, mediation only, to both interaction and mediation, and to neither.

The final chapter summarises the key findings of the thesis, reviews the strengths and limitations of the research, discusses the policy implication of the results, and provides suggestions for directions for future research (Chapter 7).

Chapter 2 Background

This chapter presents a comprehensive description of the background to this thesis, providing the rationale and motivation for the research and an understanding of the setting in which the analyses have been conducted. It begins with a summary of the epidemiology of disability in Australia and the policy context. It then moves on to describing the different ways in which disability has been conceptualised historically and how this has been operationalised into measurement of disability in research, policy and practice, comparing the different measures of disability which are used in Australia, and the implications for research.

Next, the chapter presents descriptive statistics on the overall health of people with disabilities, describes the extent of the health inequalities they experience, hypothesises about the mechanisms by which they arise, and discusses how to address them. The following section focuses specifically on the mental health of people with disabilities, presenting statistics describing disability-related mental health inequalities in Australia, describing the epidemiological evidence relating to the presence of a causal effect of disability on mental health, and reviewing and critiquing the literature examining the influence of socio-economic factors including both studies of effect modification and mediation. The chapter concludes with a section highlighting the rationale for the research in this thesis, including the gap in the evidence and the urgent need for the research.

2.1 Disability in Australia

2.1.1 Epidemiology

In 2018, 17.7% of the Australian population, approximately 4.4 million people, reported that they had a disability - a long-term health condition or impairment that, in interaction with barriers in their environment, restricted their everyday lives [6]. People with disabilities are a population group who experience systemic and systematic disadvantage [7]. As a consequence of the physical, attitudinal and social barriers that prevent them from meaningfully participating in the economic and social life of the community, they have high levels of poverty [8], worse educational and employment outcomes than people without disability [8, 9], live in insecure and inappropriate housing [10], and are likely to experience social exclusion [11], discrimination [12] and violence [13]. They also have poorer health outcomes compared to people without disability, of

which a substantial proportion is explained by the socio-economic disadvantage they face. The Australian Government have recognised this widespread disadvantage and have made a commitment to improving the lives of people with disabilities through extensive policy reforms over the last decades.

2.1.2 The policy environment

Since the 1980s, coinciding with the disability rights movement, there have been major changes in disability policies in Australia at all levels of government [5]. The Commonwealth, State and Territory Governments have progressively expressed a long-term commitment to improving the lives of people with disabilities. This was first demonstrated through the implementation of the *Disability Services Act 1986*, followed by the signing of the first Commonwealth State Disability Agreement in 1991, the *Disability Discrimination Act 1992*, and the Commonwealth Disability Strategy in 1994. More recently, Australia ratified the United Nations CRPD in 2008, developed and implemented the National Disability Strategy 2010-2020 in 2011 and introduced the NDIS in 2013. The most significant policy reforms are described in greater detail below.

The *Disability Discrimination Act 1992* is a federal law that was passed to promote equal rights, opportunities and access for people with disabilities. It provides protection against discrimination on the basis of disability, making it unlawful in Australia to discriminate against a person in different areas of life including employment, education, accommodation, buying land, leisure and sport, accessing public places and provision of goods, services, and facilities because of their disability [14]. It also aimed to promote community acceptance of the equal rights of people with disabilities.

In 1987, the United Nations recommended the development of a convention to specifically promote and protect the rights of people with disabilities and eliminate discrimination. Twenty years of advocacy, development and negotiations later, the CRPD entered into force in 2008. The CRPD is an international human rights treaty intended “to promote, protect and ensure the full and equal enjoyment of all human rights and fundamental freedoms by all persons with disabilities, and to promote respect for their inherent dignity” [15]. The eight key principles underlying the CRPD include respect for human dignity; respect for difference and acceptance of diversity; equality of opportunity; non-discrimination; social participation and inclusion; accessibility; equality between men and women; and respect for the evolving capacities of children with disabilities and respect to preserve their identities. With regards to health, Article 25 specifies that people with disabilities have the right to enjoy the highest attainable

standard of physical and mental health without discrimination because of their disability [15].

Australia was one of the first countries to ratify the CRPD in 2008, giving further legitimacy to the *Disability Discrimination Act*. The CRPD is a legally binding international document. Countries that are party to the treaty are legally and ethically obligated to protect the rights, dignity and equality of people with disabilities, as well as raising awareness of their human rights. Governments are required to implement legislation to ensure the recognition of these rights, in all laws, regulations, policies and programmes, and across different sectors and institutions. Furthermore, Australia ratified the Optional Protocol to the CRPD in 2009, making the government accountable to the Committee on the Rights of Persons with Disabilities and committing to periodic reporting and monitoring of the implementation of the CRPD.

As a result of the recognition by the Australian Government of the considerable disadvantage experienced by Australians with disabilities, the National Disability Strategy 2010-2020 was developed to provide a 10-year policy framework to ensure the principles of the CRPD were implemented in policies to improve the lives of Australians with disabilities, their families and their carers. The aim of the National Disability Strategy is to provide an integrated national approach to remove the barriers faced by people with disabilities, and ensure they have the opportunity to fully participate in all spheres of society [5]. The National Disability Strategy is structured around six priority areas of policy action including inclusive and accessible communities; rights protection, justice and legislation; economic security; personal and community support; learning and skills; and health and well-being [5].

As part of the strategy, the Productivity Commission led a public inquiry in 2010 into long-term care and support schemes for people with disabilities and received more than 1000 public submissions from people with disabilities, disability advocates and the disability sector [16]. The inquiry highlighted problems with the existing system and resulted in the development of the NDIS. The NDIS was launched in July 2013 in several regions in Australia. The full scheme has been implemented progressively since July 2016 and is intended to be fully operational by 2020. The NDIS has been recognised as the most significant economic and social reform since the introduction of Medicare in the 1980s [17]. It is an insurance-based scheme providing a person-centred approach to disability support services, with individualised packages of funding based on assessments of people's support needs [18]. The NDIS is designed to provide people with choice and control about how to use their funding packages to access the supports and services to meet their needs and maximise their independence and social participation. While the

NDIS represents a significant improvement in disability support services for Australians with disabilities, only those aged younger than 65 years with permanent and severe disabilities (approximately 10% of those with disabilities) will be eligible to receive the NDIS. Therefore, the NDIS needs to be complemented by other strategies to address the disadvantage experienced by Australians with disabilities and remove the barriers that prevent them from meaningfully participating in society. This implies that all government sectors need to continue to progress the rights of people with disabilities, both those who are eligible for the NDIS and those who are not.

2.1.3 Monitoring progress in relation to the CRPD

Despite these extensive policy reforms, people with disabilities in Australia still experience considerable levels of disadvantage. There is even evidence that some of the inequalities experienced by people with disabilities, such as inequalities in education, have increased in recent years [19]. Furthermore, people with disabilities in Australia experience poorer outcomes (and greater relative inequalities) than people with disabilities in other similar countries. For example, Australians with disabilities have the highest relative risk of living in poverty (defined as households with less than 60% of the median disposable income) and the lowest relative income of all the countries in the Organisation for Economic Co-operation and Development, and one of the lowest relative employment rates [8]. This is in comparison to other countries, such as Sweden, Norway and Slovakia, where there are very small or no inequalities between people with and without disabilities.

By ratifying the CRPD and the Optional Protocol, the Australian Government committed to take measures to promote the rights and freedoms of people with disabilities and, as such, is reviewed every four years on its progress. The latest review took place in September 2019, conducted by a committee of experts from the United Nations who summarised and documented the current state of disability rights in Australia, including both positive and negative aspects, in the Civil Society CRPD Shadow Report. Positive developments included the adoption of legislative, policy and administrative measures such as the *National Disability Insurance Scheme Act 2013*, but there were also areas for concern such as the ongoing poverty, disadvantage and human rights violations experienced by people with disabilities as well as their poor health, and barriers to accessing adequate, affordable and accessible health services [20, 21].

The Department of Social Services commissioned a review of the National Disability Strategy in 2018 to inform the development of the national disability framework beyond 2020. The review, published in March 2019, found that though the goals of the Strategy

were valuable and well aligned with the CRPD, there were areas for concern relating to the ongoing disadvantage experienced by people with disabilities including participation in employment and education, availability of affordable and accessible housing, and the prevalence of violence and abuse, [22]. For example, the review highlighted that the economic security of people with disabilities had deteriorated in recent years despite the introduction of new employment strategies. The review also reported concerns about the interface between the National Disability Strategy and the NDIS.

2.1.4 Need for data and research

As well as highlighting the ongoing disadvantage experienced by people with disabilities, these reviews and reports also underscored the urgent need for better disability data and research evidence on the impacts of policies to improve the social and economic circumstances of people with disabilities. The Productivity Commission inquiry report identified “poor data” on disability and a “poor evidence base” as problems for monitoring outcomes for people with disabilities and the effect of interventions [16]; the Civil Society CRPD Shadow Report described concerns about the lack of nationally consistent measures of disability [20]; and the Department of Social Services review of the National Disability Strategy recommended better data collection, more creative use of existing data, and further research to monitor outcomes and identify priority areas for policy [22].

This is not a newly identified issue. The need for better disability data has been widely acknowledged in many reports internationally since the 1980s to facilitate better understanding of the experiences of people with disabilities as a population group. The World Programme of Action Concerning Disabled Persons (1982) [23], the Standard Rules on the Equalisation of Opportunities for Persons with Disabilities (1993) [24], the United Nations International Seminar on the Measurement of Disability (2001), the United Nations CRPD (2007) [15], and the World Report on Disability (2011) [25] all explicitly state the need for collection of better disability data in order to improve the comparability of statistics, inform policy change, monitor the impact of policies and programmes, and ultimately, protect the rights of people with disabilities [26]. The lack of good-quality consistent data on disability has been a major obstacle to developing evidence-based policies and interventions to address the systematic and systemic disadvantage experienced by people with disabilities in Australia and internationally, and protect their rights [27, 28].

At a time of significant policy reform in Australia, the data suggesting that the disadvantage experienced by people with disabilities has not ameliorated over the last decades underscores the need for research about how to address the disadvantage to

improve the lives of people with disabilities and meet Australia's obligations under the CRPD. The lack of progress highlights the importance of this thesis, emphasising the need for better evidence to understand *how* to address the disadvantage experienced by people with disabilities to reduce the health inequalities. This research in this thesis, conducted within the Australian context, is urgently needed to bridge the evidence gap and facilitate the development and implementation of evidence-based policies that will most effectively address the disadvantage faced by Australians with disabilities and improve their health and well-being.

2.2 Conceptualising and measuring disability

2.2.1 Overview

Disability is a complex, multidimensional and dynamic construct [25]. It results from the interaction between a person's health conditions or impairment and the environment in which they live. The concept of disability has evolved greatly over time, has distinct meanings in different contexts and settings, and has been heavily contested. Historically, in many contexts, disability was seen to be caused by divine punishment, karma or moral failing [29]. By the end of the nineteenth century, particularly in high income countries such as Australia, disability was seen as a purely medical condition, a negative health outcome to be prevented and a failure of public health prevention [30]. The concept of disability evolved greatly in the latter half of the twentieth century, assuming a completely different position in which disability was conceived as a purely social phenomenon, resulting from social oppression [31], environmental barriers and discrimination [32]. This change represents a paradigm shift from a view that disability is defined by people's incapacity, entirely determined by bodily impairments, to one in which disability is determined entirely by social oppression, social relations and social barriers [33].

Disability research has been subject to definitional problems due to the complexity, diversity, and continuous evolution of the concept of disability. There have been large inconsistencies between the changing theoretical conceptualisation of disability and its operationalisation in practice leading to substantial variation in how disability has been measured in national surveys, research and health care services. It is important to understand the different conceptualisations of disability because how disability is conceptualised directly impacts on how it is considered (and measured) within public health research and guides formulation and implementation of disability policies.

2.2.2 Conceptualising disability

This section describes, compares and reviews the most common conceptual models of disability including the medical, social and biopsychosocial models and distinguishes the characteristics central to the biopsychosocial model of disability which is applied throughout this thesis.

The medical model

The classical approach of conceptualising disability according to people's impairments is known as the medical model of disability. Under the medical model, disability is a feature of the individual, a biological deficit directly caused by a disease, trauma or health condition, which impairs physiological or cognitive functioning [34]. Impairments represent a deviation from population standards of 'normality' in the biomedical status of the body and its functions [35]. In this conceptualisation of disability, there is a strong emphasis on categorising people with disabilities in terms of their impairment or health condition rather than focusing on people's level of functioning. The medical model implies that public health responses to disability should focus on preventing, curing or treating conditions associated with disability through medical care at the person level, such as through pharmaceutical or surgical treatment, rehabilitation and institutional care [36-38].

The medical model was heavily criticised in the 1970s and 1980s by disability activists and Disabled People's Organisations (advocacy groups run by people with disabilities for people with disabilities) because of its reductionist approach which did not recognise the physical, social, cultural and economic causes of disability; these were assumed to be inevitable consequences of living with an impairment [39, 40]. Additional criticisms of a purely medical view of disability relate to the fact that some types of impairment are not curable by medical intervention. Furthermore, the restrictions and barriers that people with disabilities experience do not result only from their impairments, therefore it was argued that disability cannot be seen purely as a medical issue [36, 41, 42]. These critiques, led by disability theorists, changed the way many people thought about disability and promoted a paradigm shift to the social model of disability. However, the medical model continues to be influential, particularly in clinical circles [43].

The social model

The social model of disability was developed by members of the disability rights movement in the United Kingdom in the 1970s [44]. In contrast to the medical model, it theorises that disability is caused by the barriers that people face interacting with the physical and social environment in which they live [34, 45, 46]. It shifts the focus away

from features of individuals, repositioning disability as a socially created problem caused by attitudes and other features of the social environment which disadvantages people with disabilities and excludes them from fully participating in society [36]. The social model makes a clear distinction between impairments and disability. Impairments consist of the health conditions experienced by individuals, whereas disability represents the social situation of people with impairments, defined as "something imposed on top of […] impairments" [47] and "the disadvantage or restriction of activity caused by a contemporary social organisation which […] excludes them from participation in the mainstream of social activities" [44].

Under the social model of disability, the role of public health shifts to one in which improving population health involves taking action to remove the social barriers that are causing the disability rather than addressing disability by providing medical care to individuals [48]. This implies that public health responses to disability should focus on modifications to the social, physical, economic and political environment, for example by ensuring accessible communities and environments for people with mobility impairments.

However, the social model of disability has been criticised for its narrow focus, completely disregarding the role of people's impairments. While it led to great successes to unite the disability rights movement by strengthening disability advocacy, raising awareness and promoting disability equality, the use of this model alone to conceptualise disability does not represent the full lived experiences of people with disabilities. The social model aims to break the link between impairment and disability [49]. However, impairment and disability are not distinct concepts, there is a definitive causal link between them. The presence of an impairment is not sufficient to cause disability, but it is a necessary component [33]. Without acknowledging this causal relationship, there is no distinction between disability and any other form of social oppression [33]. Considered alone, the social model overstates the social causes of disability, which has the consequence of failing to accurately describe the complexity of the interaction between people's health conditions and the environment in which they live, and can lead to inadequate recognition of people's health care needs [33, 50].

Closely linked to the social model of disability, and for some indistinguishable, is the human rights model of disability, which positions (and values) disability as a natural part of human diversity [51]. It recognises that people with disability have the same rights as everyone else in society, as embedded in the CRPD, and argues for the removal of barriers to participation that people face in society.

The biopsychosocial model

Both the medical model and the social model of disability have been criticised for failing to acknowledge the complexity of the experience of disability, the importance of both individual and social components. Several disability theorists have highlighted the need to acknowledge both individual restrictions (intrinsic limitations) and social barriers (extrinsic limitations) in conceptualising disability [33, 52-55]. In public health, impairments cannot be completely ignored because the characteristics of people's impairments, such as type, severity, visibility, permanence, age and mode of acquisition, have different implications for people's health, functioning and health care needs [56]. Therefore, in the context of public health, neither the medical nor the social model is sufficient alone to understand disability.

The biopsychosocial model, also known as the interactional view of disability, incorporates both medical and social models of disability into a single framework. It conceptualises disability as a complex phenomenon which results from the dynamic interaction between health conditions, individual factors and the environment [25, 41]. The biopsychosocial model considers the individual's experience of a health condition in the context of a wide range of individual, physical and social factors - such as age, gender, family circumstances, socio-economic characteristics, the built environment, and the intersection between each of these - but also in the wider social, economic and political context - such as people's experience of discrimination, negative attitudes and the impact of social policies [57]. Therefore disability is the product of multiple individual and social forces [50]. This conceptualisation entails that everyone in the population experiences disability to some extent, therefore dismantling the dichotomous division between people with and without disabilities [56].

The biopsychosocial model of disability has implications for public health. It infers that both medical approaches and modifications to people's social, physical, economic and political environments are required to increase the inclusion and participation of people with disabilities and improve their health [36]. Furthermore, the acknowledgement of the diversity of people's experiences and needs infers that public health actions such as screening and health promotion campaigns need to actively include and target people with disabilities, recognising their diverse health care needs and ensuring equal access to health services. This also requires public health research and data to better understand public health issues as they relate to people with disability.

Throughout this thesis, I adopt the biopsychosocial approach, conceptualising people with disabilities as a diverse group of people with a shared experience of limitations to

functioning as a result of the interaction between their impairments and the environment in which they live.

2.2.3 Measuring disability

The way in which disability is conceptualised has important implications for how disability is defined and measured in research and practice, and for formulation and implementation of policies. As a result of the changing conceptualisation of disability and the increasing recognition that disability arises because of both a person's impairment and the environment in which they live (in line with the biopsychosocial model), the definition of disability has evolved from a more medicalised definition - with disability defined in terms of presence of health conditions - to one focusing on people's level of functioning within the environment in which they live, determined by individual, social and environmental characteristics. Defining disability in terms of people's functioning with the context of their environment aligns well with a measurement of disability on a continuum representing a range of experiences of functioning, rather than a measure which creates a dichotomy between people with and without disabilities. The strength of defining disability in terms of functioning is that it can better represent the complexity of the biopsychosocial conceptualisation of disability. However, this is also its principal limitation as it is difficult to operationalise and consistently define a complex multidimensional concept.

There is a need to ensure that disability is measured in consistent ways, for example for estimating changes in the prevalence of disability, monitoring how the circumstances of people with disabilities have changed over time, or making cross-national comparisons. Despite the need for consistency in measurement of disability, the use of multiple definitions of disability may be necessary in practice because different definitions may be required for distinct purposes [36]. For example, defining disability to determine eligibility for accessing welfare services will be very different to the definition used to estimate prevalence of disability in the population.

The recognition of the need for better disability data and comparable disability statistics (described in section 2.1.4) led to the development of several measurement and classification instruments. Though we are moving towards a common conceptualisation of disability this has not translated into a consistent measurement of disability at a global or even a national level. The biopsychosocial model of disability has been operationalised in diverse ways in instruments to measure disability [58, 59]. The next sections describe and critique some of the commonly used measurement tools including the International Classification of Functioning, Disability and Health (ICF), which is

internationally recognised as the gold standard tool for classifying disability but is not easily operationalised in population censuses and surveys; several Australian disability measurement tools based on the ICF, including the disability measure used throughout this thesis; the World Health Organization (WHO) Disability Assessment Schedule 2.0; and, the Washington Group Questions.

The International Classification of Functioning, Disability and Health

The International Classification of Impairments, Disabilities and Handicaps (ICIDH), the precursor to the ICF, was developed by the WHO in 1980 to provide a coherent unifying framework for classifying disability [60]. It was designed to complement the International Classification of Diseases, the standard diagnostic tool to classify mortality and causes of death. The ICIDH focuses on classifying disability as consequences of disease or injury and their implications for the individual, with a strong emphasis on the medical aspects of disability rather than the barriers to functioning experienced by the individuals. In parallel with the move away from the medical model of disability, the ICIDH became strongly criticised for a number of reasons. Firstly, it was criticised for the inadequate recognition of the role of the environment in how it classified disability. Secondly, because of the strong assumptions about dichotomous states of normality and abnormality, argued to be culturally, temporally and situationally dependent [49], it was thought that it was unlikely to produce measures of disability that were consistent or comparable across cultures, contexts and time points. Thirdly, the dichotomous classification was seen as an oversimplification of the concept of disability [61].

In response to these criticisms and the move away from the medical model of disability, the ICIDH classification system was revised by the WHO and reissued as the ICF which was endorsed by all WHO member states and approved by the World Health Assembly in 2001 [62]. The ICF represents a shift from a medicalised framework of disability to a more dynamic interactive framework describing functioning and disability, which incorporates social and environmental influences as contextual factors, thus aligning with the biopsychosocial model of disability. The ICF serves as a scientific tool to study disability, putting the emphasis on classifying people's level of health and functioning associated with health conditions (rather than the health conditions *per se*) [62]. It aims to provide a globally accepted language and framework to structure disability data and statistics and a universal standard for the collection of data on disability that are consistent and comparable across time and between different contexts, sectors and disciplines.

The major difference between the ICIDH and the ICF is the conceptualisation of the causal nature of the disability. In the ICIDH, as in the medical model of disability, the cause of the disability is the psychological, physiological or anatomical characteristics of the individual. In contrast, the ICF makes no distinction between different types of impairment. It attributes disability to the interaction between health conditions or impairments, personal characteristics (such as age, sex, education, socio-economic status, resilience and personality) and environmental factors (physical, social and attitudinal environment including the socio-political environment, discrimination, characteristics of the built environment, and climate) and aims to capture these components to give a comprehensive view of the biological, individual and social perspectives of health and functioning.

The structure of the ICF is based on parallel concepts of *functioning* and *disability,* which represent outcomes of the interaction between the individual and contextual factors to describe health within the context of society. As illustrated in Figure 2.1, functioning and disability can be experienced as effects or restrictions at one or more of three levels: at a bodily level (physiological function or body structures), at a person level (activity limitations) or at a societal level (participation restrictions). Functioning and disability are also influenced by contextual factors, including environmental factors (external influences), coded as facilitators and barriers to activities at the physical, social and attitudinal levels, and personal factors (internal influences), which although are recognised as important, are not coded in the ICF because of the large societal and cultural differences. Each component of the ICF consists of multiple domains with multiple units of measurement, in such a way that the ICF classification produces thousands of codes describing health and functioning on a continuum rather than a dichotomous classification of disability. As such, different thresholds can be created for different purposes, providing greater flexibility [62].

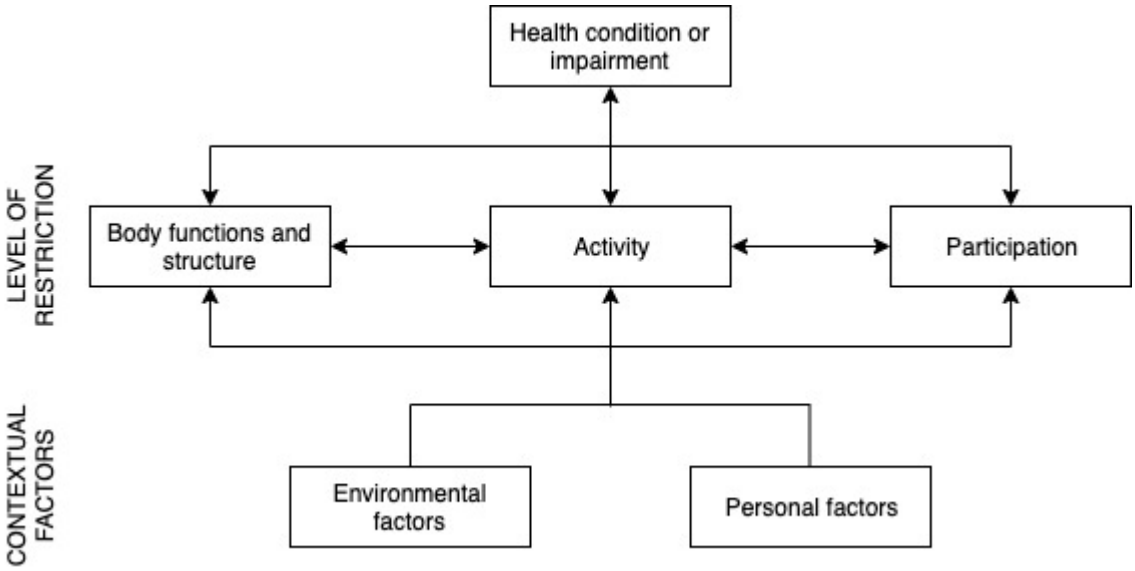

Figure 2.1 Structure of the ICF framework, adapted from [62]

The ICF has been argued to be a universal multipurpose tool that provides a broad description of health and functioning within the context of society. It was designed to improve the consistency of disability measurement with an ultimate aim to achieve better population health, increase social inclusion, improve health policies and systems, and promote equity between people with and without disabilities. Furthermore, by recognising that everyone has a range of functions and activities that they cannot do (thereby experiencing some level of disability), it has the potential to change the way public health views disability [63].

The ICF definition has been widely acknowledged as the best classification system for disability, and therefore its use has been recommended across all sectors in public health – in research, population censuses and surveys, and for allocation of services. However, though the ICF provides a useful framework to define disability, it is such a complex tool that it does not readily provide an operational set of questions that can be incorporated into surveys [64, 65]. A simple way of measuring functioning in a consistent way based on the ICF framework is required. Several questionnaires have been designed based on the ICF framework. Table 2.1 describes the measurement tools which have been used in the Australian context, including the disability question included in the Household, Income and Labour Dynamics in Australia (HILDA) Survey used in the analyses contributing to this thesis.

Table 2.1 Disability measurement tools used in Australia

Name and uses	Definition of disability	Brief summary	Strengths and limitations
Household, Income and Labour Dynamics in Australia Survey disability question	A single question to identify disability. Further information on broad impairment types and severity is collected in some but not all waves of the survey [66].	Disability is identified as a long-term health condition, impairment or disability that restricts everyday activities, has lasted for six months or more, and cannot be corrected by aids or medication. Examples are displayed on a show card by the interviewer (e.g., hearing problems, limited use of legs, mental illness which requires help or supervision).	**Strengths**: The question requires respondents to identify health conditions that restrict everyday activities, reflecting the barriers to participation outlined in the ICF. **Limitations**: Data are not collected on disability severity or broad impairment type at every wave of data collection. The use of a single question to identify disability overestimates the prevalence of disability.
Survey of Disability, Ageing and Carers disability module	166-question disability module to measure disability, including severity, broad impairment types and the underlying conditions causing the disability.	Disability is identified through limitations to functioning, generating a severity scale representing the extent to which a limitation impacts on core activities (mobility; self-care; communication) [67, 68]. Detailed information is also gathered on impairment types and diagnosis and a large number of questions relate to barriers to participation and people's need for assistance.	**Strengths**: It is the most comprehensive measure of disability and recommended as the gold standard including for estimates of disability prevalence. The questions are based on the ICF, bringing together questions relating to health conditions and how they relate to restrictions at a person or societal level. The survey differentiates between people who have long-term health conditions that limit their ability to carry out core activities and those who have long-term conditions but are not restricted in their ability to perform core activities.
Australian Bureau of Statistics short disability module – used	A set of 16 questions used to measure disability, including	Disability is identified through limitations to functioning, combining responses from	**Strengths**: The questions are compatible with the activities and participation components of the ICF and are based directly on the questions

in several surveys including the General Social Survey; the Personal Safety Survey; the National Health Survey; and the Survey of Income and Housing	severity and broad impairment types.	different questions to generate a severity scale representing the extent to which a limitation impacts on core activities. Information is also gathered on broad impairment types.	used in the Survey of Disability, Ageing and Carers. **Limitations**: The use of this shorter set of questions has been criticised as it overestimates the number of people with less severe forms of disability [67].
Australian Institute of Health and Welfare standardised disability flag – used in the National Prisoner Health Data Collection and the Specialist Homelessness Services Collection	A module comprising 10 questions used to measure disability, including severity.	Disability is identified through limitations to everyday activities in eight domains of functioning (self-care; mobility; communication; learning; home management; managing tasks/situations; personal relationships; and community life) with information recorded on severity of those limitations.	**Strengths**: It is based on the ICF and designed to be broadly consistent with the Australian Bureau of Statistics short disability module [69]. **Limitations**: The disability flag does not collect data on impairment type. The use of a shorter set of questions is also likely to overestimate the number of people with disabilities.
WHO Disability Assessment Schedule 2.0 (WHODAS) – used in the 2007 Australian National Survey of Mental Health and Well-being and currently used to determine support needs for some NDIS participants	A 36-item module to measure disability, producing an overall continuous severity score ranging from 0 (no disability) to 100 (full disability) and a severity score for each domain of functioning.	It identifies disability as limitations to functioning in six domains including cognition; mobility; self-care; interacting with others; life activities (domestic, leisure, work and school); and participation in community activities.	**Strengths**: It was developed to produce a consistent measure across different settings, independent of impairment type, grounded in the conceptual framework of the ICF, applicable both in general population surveys and clinical settings [70]. The use of a continuous measure of disability aligns well with the conceptualisation of the ICF. **Limitations**: It has not been widely adopted in population surveys in Australia.

Washington Group questions – included in Ten to Men [71] and in the supplementary disability survey conducted in 2016 on a subsample of the respondents from the 2015 Survey of Disability, Ageing and Carers	A short set of six questions used to measure disability including severity of restrictions.	Disability is identified as limitations to functioning in six domains (seeing; hearing; walking; cognition; self-care; and communication) with scoring to record the severity of those limitations (no difficulty; some difficulty; a lot of difficulty; cannot do at all).	**Strengths**: Developed to support collection of internationally comparable data, these questions generate consistent measures of functioning regardless of nationality/culture [26, 72]. The questions are based on the ICF, are easy to implement, flexible in their scoring (use of different thresholds), and they have been extensively tested [73]. **Limitations**: The questions do not adequately capture mental health conditions [73], underestimate the prevalence of disability, and have low concordance with the Survey of Disability, Ageing and Carers disability measure, particularly for people with less severe disability [74].

2.2.4 Summary

There has been a multitude of theoretical studies debating how disability should be conceptualised and defined, however there is a gap between these theoretical conceptualisations of disability and how they are operationalised in practice which has led to a lack of consistency in how disability is measured at an empirical level. While it seems that globally we are moving towards a common conceptualisation of disability through the widespread recognition of the ICF as the best classification system for disability, this has not translated into consistent measurement of disability at a global or national level. The disability measurement tools described in Table 2.1 are all based on the ICF framework; however, they produce different estimates of the prevalence of disability in Australia because of the difficulties associated with reducing such a complex framework into a short set of questions. This has implications for the comparability of estimates between populations groups and over time, highlighting the need for consistency in the measures of disability used in practice. But until we have better quality data, we need to use the best data available to understand and monitor outcomes for people with disability, being explicit about how disability is defined in the data and highlighting any limitations pertaining to those data.

2.3 Disability and health inequalities

This section focuses on disability-related health inequalities. It starts with a historical and current account of how disability is viewed and addressed in public health, arguing that disability should be viewed as an exposure (rather than an outcome). Then it goes on to describe differences in health outcomes between people with and without disabilities, making the distinction between health inequalities and health inequities, and discusses how the inequalities may arise and how to address them.

2.3.1 Disability as a public health issue

Despite people with disabilities being a substantial population group – with almost one in five Australians reporting a disability – disability has largely been excluded from the public health agenda, including in policy, practice and research [30, 75]. There has been substantial debate about whether disability is a public health issue. Historically, in line with the medical model, disability was seen as a failure of public health, in which disability and ill-health were seen as counterparts of a single concept. Public health was concerned with the prevention of disability, rather than addressing the poor health outcomes and needs of people with disabilities to reduce their disease burden [76].

Currently, despite the widespread acknowledgement of a biopsychosocial approach to disability, disability is still commonly thought of as a negative health outcome to be prevented in public health. For example, in the Global Burden of Disease studies, disability is conceived in terms of losses to health associated with health outcomes [77]. Disability weights are assigned to specific health outcomes by a group of health professionals to represent the severity of the health outcome on a scale ranging from 0 (representing perfect health) to 1 (representing death). The weights are then used to calculated disability adjusted life years, which represent total disease burden in the population of interest by combining years lost due to death and years lost due to disability for people living with each specific health condition and are used to prioritise health resources [78]. This approach to quantifying disability does not align with the biopsychosocial conceptualisation of disability, which would not equate years of life living with disability as years of life *lost* [79]. Furthermore, the disability weights are assigned by health professionals, rather than by people with lived experience of disability, according to perceived desirability of health conditions rather than measuring their impact on health and functional limitations [80].

In this thesis, I argue that, in line with the biopsychosocial model of disability in which people experience disability (and health inequalities) as a result of the interaction between their health conditions and the environment in which they live, disability should be conceived as an exposure rather than an outcome in public health. People with disabilities should be viewed as a subgroup of the population that experiences different health outcomes and has diverse health care needs, for whom concerted and targeted efforts are needed to reduce current health inequalities and target the causes of poor health.

2.3.2 Health outcomes of people with and without disabilities

Australians with disabilities experience poorer health outcomes compared to those without disabilities across a range of indicators including self-reported health [81, 82], mental health [82-84], and chronic conditions such as diabetes [85], hypertension [81, 82] and heart disease [85]. They also have a higher prevalence of risk factors for disease including high rates of smoking and obesity [81, 82], poor diet [82] low rates of physical activity [85], high measured blood pressure [82] and poor access to health services [25, 82] including breast and cervical cancer screening [85].

Some of these health inequalities are unavoidable, directly resulting from the underlying health condition causing the disability [28], such as the increased risk of congenital heart disease for people with Down syndrome [86, 87]. However, there is increasing evidence

that people with disabilities also experience substantial differences in health outcomes which are unrelated to their disability [28, 34, 88]. These differences in health are instead determined by social forces including social structures, culture, norms, political and economic environment and institutional policies which influence the social determinants of health [89]. For example, people with intellectual disabilities experience poor oral health outcomes [90], but this is not related to the intellectual disability in and of itself. Rather, it is a consequence of the social determinants of health – the "conditions in which people are born, grow, live, work, and age" [91], which incorporate people's social experiences, environments and behaviours but also social processes and structures, and the pathways by which they affect health [92].

These avoidable differences in health have been argued to represent health inequities – unjust inequalities in health that arise because of the unfair distribution of the underlying social determinants of health [28, 34, 93]. In this thesis, I refer to the health differences between people with and without disabilities as *disability-related health inequalities*, acknowledging that this incorporates both avoidable health inequities that may be amenable to policy intervention and unavoidable differences relating to people's disabilities.

The increasing evidence of the importance of the social determinants of health in contributing to the health inequalities experienced by people with disabilities underscores the need for research in this field to be embedded within a social epidemiology framework. Social epidemiology explicitly investigates how social circumstances, processes and structures (and their interactions) affect the health of individuals and populations [94], with an aim to improving population health and reducing health inequalities within (and between) populations.

The biopsychosocial model of disability inherently aligns with a social epidemiological framework for investigating health inequalities because it acknowledges the impact of people's personal circumstances and the environment in which they live on their health and well-being. In light of this conceptualisation of disability in which people are disabled by their environments as well as their impairments, disability does not necessarily equate to ill-health, therefore it is possible for people with disabilities to live healthy and fulfilled lives [95]. This underscores the need for public health to view disability and health as interrelated but distinct concepts [34], viewing disability as an exposure with an aim to maximise the health potential of people with disabilities.

2.3.3 Understanding the social determinants of disability-related health inequalities

In order to be able to reduce disability-related health inequalities, it is important to understand the causes of the inequitable distribution of health. People with disabilities experience considerable disadvantage in terms of a range of structural characteristics and conditions which constitute the social determinants of health, for example poverty, education and employment. These different dimensions of social and economic characteristics relate, intersect and mutually reinforce one another in complex ways to create patterns of health inequalities experienced by people with disabilities [96]. As a result of these multiple, intersecting relationships, the social pathways that produce disability-related health inequalities are likely to be complex. A better understanding of the possible pathways is vital to generating policy-relevant evidence about interventions that may be effective in tackling these inequalities.

Firstly, there are several mechanisms by which people with disabilities may experience socio-economic disadvantage. Socio-economic disadvantage is likely to increase the risk of people developing disability. Strong national and international research supports this *social causation* hypothesis, by which exposure to socio-economic disadvantage causes onset of disability [97-99]. However, disability may also lead to changes to people's socio-economic circumstances. There is also evidence supporting this alternative hypothesis, whereby disability results in downward social mobility in relation to a range of socio-economic circumstances such as education, employment, income and occupation [100-102]. Most likely, the existence of disability-related health inequalities represents an interplay between these different mechanisms [103].

Secondly, it is important to understand how socio-economic disadvantage contributes to health inequalities for people with disabilities. The poor health of people with disabilities may result from changes to people's socio-economic circumstances as a result of disability or may result from more distal social forces including social structures, culture, norms, the political and economic environment and institutional policies which shape the socio-economic circumstances of people with disabilities. For example, the experience of discrimination may impact on health directly as a result of the experience of disablism itself, or indirectly, operating through socio-economic factors such as employment leading to social exclusion. As such, it is important to strive to understand both the contribution of more proximal causes and the more distal *causes of the causes* [104].

2.3.4 Addressing the social determinants of disability-related health inequalities

Because disability-related health inequalities exist, in part, as a result of the social inequalities faced by people with disabilities, they are in principle avoidable and

amenable to policy intervention [105]. To reduce current health inequalities, action needs to be taken on the social determinants of these health inequalities [106]. The Commission on Social Determinants of Health was created in 2005 to develop a global agenda for health equity grounded in evidence-based research [91]. The Rio Political Declaration on Social Determinants of Health, to which Australia is a signatory, was adopted in 2011 [107]. It expresses a global commitment to implementing a social determinants of health approach to reduce health inequalities, by taking informed action on these determinants in order to "close the gap", with specific attention to the needs of vulnerable groups. However, there was no mention of people with disabilities as a vulnerable group. This underscores the invisibility of disability in the various discourses on the social determinants of health despite the increasing evidence on the large health inequalities [88, 108].

Unlike other marginalised groups, such as racial or ethnic minority groups, there is sparse evidence about the socio-economic determinants of the health inequalities faced by people with disabilities. This gap in knowledge is a significant impediment to the development of effective public health and social policies, and it is ethically imperative for it to be integrated into the public health agenda. Without research specifically into the experiences of people with disabilities, policies which do not take into account the specific contexts faced by people with disabilities are likely to fail to improve health outcomes. Evidence-based research about the socio-economic drivers of the health inequalities, as well as the impacts of social, economic and political context, is necessary to understand the complex relationships between disability, social disadvantage and health. It is important to understand the mechanisms by which disability-related health inequalities arise, which may identify modifiable factors that could be targeted in policies and interventions. In summary, there is a need for evidence that can be translated into policy and practice to develop effective strategies and interventions to improve health, reduce inequalities and operationalise people with disabilities' right to the highest attainable level of health.

2.3.5 Summary

The goal of public health is to improve population health, through prevention and health promotion, addressing the social determinants of health, and by adhering to principles of health equity [109]. As such, improving the health of Australians with disabilities should be a priority area for public health, both with an aim to increase the overall health of the population and to promote health equity.

However, Australia currently lacks knowledge on the socio-economic determinants of disability-related health inequalities, and therefore there is a lack of evidence on how to target health and social policies to improve the health of Australians with disabilities and reduce the health inequalities. There is a clear need to strengthen disability research to better understand the causes of the health inequalities and the mechanisms by which they operate. In this thesis, I directly address this research need using mental health as an example. The research in this thesis aims to bridge the evidence gap, improving understanding of the causes of the disability-related mental health inequalities and producing policy-relevant findings about how to address the poor mental health of people with disability and advance health equity.

2.4 Disability and mental health

This section focuses on disability-related mental health inequalities, synthesizing and reviewing the literature. It starts by presenting statistics describing the mental health and mental ill-health of people with disabilities in Australia to justify the urgent need for research in this area. It then describes the evidence relating to the presence of a causal relationship between disability and mental health, focusing on longitudinal studies which have examined the mental health effects resulting from disability acquisition. Finally, this section synthesizes and reviews the literature examining socio-economic characteristics as effect modifiers and mediators of the relationship between disability acquisition and mental health.

2.4.1 Prevalence of mental health and mental illness for people with disabilities

There is extensive evidence that people with disabilities in Australia have higher rates of mental ill-health and mental disorders compared to people without disability. For example, a recent report by the Australian Institute for Health and Welfare drawing on data from the National Health Survey, a large representative national survey conducted in 2017-2018, estimated that adults with disabilities were four times more likely to experience high or very high levels of psychological distress, with 32% of people with disabilities reporting psychological distress compared to 8% of people without disability [110]. The most comprehensive data source for evidence on mental ill-health and mental disorders in Australia is the Survey of Mental Health and Well-being. However, this is not a regularly conducted survey, the most recent one being conducted in 2007. This survey estimated that 37.1% of people with severe or profound disabilities had experienced a mental disorder in the previous year compared to 27.5% of people with mild or moderate disabilities and 14.8% of people with no disability [111].

There is also evidence that people with disabilities experience poorer mental health and well-being. Data on measures of mental well-being can be obtained in other surveys such as the HILDA Survey, the data source used in this thesis, which collects data on mental health and well-being using a continuous mental health score. The most recent data available in HILDA was from 2017 and estimated that people with disabilities had a mental health score that was eight-points lower than people without disability (a higher score represents better mental health), representing a large clinically meaningful difference in mental health [112, 113].

2.4.2 Disability acquisition as a determinant of mental health

Research from longitudinal studies has provided strong evidence that disability acquisition is associated with substantial mental health deterioration, using various measures of mental health and mental illness. Examining incident disability – using longitudinal data to identify a group of people followed up over time who acquire a disability during their participation in a study – rather than examining the mental health of people with prevalent (existing) disability provides a stronger analytic design for causal inference. It allows better identification of the causal effect of disability on mental health because it makes it possible to compare people's mental health before and after the disability to understand how disability itself affects people's mental health (further discussed in the Methods, sections 3.1.2 and 3.3.1).

A study in the United States explicitly examined the direction of the association between disability and mental health using structural equation models and found evidence that disability led to changes in depressive symptoms, but that depressive symptoms did not lead to changes in disability, suggesting that disability was a determinant of poor mental health but not vice versa [114]. These results were consistent with the evidence from longitudinal studies suggesting that disability acquisition was associated with an increase in depressive symptoms [115-118], increased odds of psychological distress [119], higher risk of psychological problems [100], lower life satisfaction [120-122], and poorer psychological well-being [120, 123]. Two Australian studies of young adults and one Korean study of older adults used longitudinal growth curve models to examine trajectories of mental health after disability acquisition and found some evidence of an association with a decline in mental health but only for a small proportion of the sample (approximately 20% in each of the studies), suggesting that there may be substantial heterogeneity in the effect [101, 124, 125].

These studies used a variety of different statistical approaches to quantify the effect of disability acquisition on mental health including path analysis, structural equation

modelling, stepwise regression models, multilevel models, fixed and random effects models, zero-inflated negative binomial models, semi-first difference panel models, logistic regression models, within-person analysis of variance, and longitudinal growth curve models. Despite the diverse statistical methods used to approach the research question, the range of settings and study populations in which the studies were conducted, the different measures of disability and mental health or mental illness used, the findings of the studies provide convincing evidence that disability acquisition is causally associated with a decline in people's mental health.

2.4.3 The influence of socio-economic characteristics on the relationship between disability and mental health

Studies examining the mechanisms by which disability leads to a deterioration in mental health are scarce. With regards to the role of socio-economic factors in influencing the association between disability acquisition and mental health, there is limited longitudinal research examining effect modification or mediation by socio-economic characteristics, the results of which are inconsistent.

Effect modification by socio-economic characteristics

Three longitudinal studies in the United Kingdom, the United States and Germany examined whether there were differences in the association between disability acquisition and mental health according to people's education using longitudinal data from young adults who acquired a disability [126] and older adults who acquired a physical disability [127, 128]. All three studies found evidence that the association differed by education - that the magnitude of the effect on mental health was greater for those with low compared to high education [126-128].

Four studies examined financial resources as an effect modifier of the association. A study of working age Australians found evidence that the association varied according to people's wealth prior to disability, with the largest effects for people in the lowest and middle tertiles of wealth [84]. A similar analysis of older adults in the United States found that the negative effect of disability on subjective well-being was greater in individuals with below average wealth [129]. Financial assets were found to predict trajectories of depressive symptoms for older adults who acquired a physical disability in the United States [127]. In contrast, there was no evidence of effect modification of the association between disability acquisition and life satisfaction by wealth or income in a study of Swiss adults [130].

There was evidence of effect modification by unemployment from a single study of working age people in the Netherlands [131]. The negative mental health effect

associated with the experience of both disability acquisition and a transition into unemployment was greater than the sum of each individually.

Only one study examined housing characteristics as effect modifiers – a study of working age Australians which found evidence that the association varied according to housing characteristics prior to disability; the largest declines were seen for people in unaffordable housing and insecure housing tenures [83].

Two studies of adults in the United States investigated whether the effect of disability on depressive symptoms varied according to people's marital status and neither found any evidence of a difference in the size of the effect between people who were or were not married [132, 133].

Social support was found to be an effect modifier of the association between disability and poor mental health in studies of older adults in Sweden and the United States, with smaller negative effects for people with greater social support [134, 135]. A study of adults in Switzerland found evidence that religiousness and number of friends, but not emotional support, modified the effect of disability acquisition on life satisfaction [130]. Furthermore, four cross-sectional studies found evidence that having greater social support was associated with smaller magnitudes of the association between disability and poor mental health [102, 136-138].

The review of the literature indicated that this is an understudied area, but the few studies that were identified generally supported the hypothesis that socio-economic characteristics modified the effect of disability on mental health. The majority of studies examining material socio-economic characteristics such as education, financial resources, employment and housing, found that socio-economic factors influenced the magnitude of the effect of disability on mental health. The evidence about the effect of social support as a modifier of the association between disability and mental health was also convincing, however there was no evidence of effect modification by marital status.

Despite most these studies being longitudinal in their design, there were methodological limitations in the statistical approaches applied. Quantifying the effect of disability on mental health and the extent to which it is modified by socio-economic characteristics is challenging because of the complexity of the relationship between disability and mental health and the risk of bias from confounding, selection bias and information bias that arises when using observational data (further discussed in the Methods chapter, section 3.1). Four of the studies were cross-sectional in design, and six examined prevalent (rather than incident) disability. Nine of the studies only looked at older or younger samples, limiting the representativeness of the findings. Of the five studies which used

a longitudinal design to quantify the effect of disability acquisition on mental health for a sample with a broad age range, only four used statistical approaches to attempt to better control for confounding by modelling within-person effects [83, 84, 130, 131]. Furthermore, no single study examined a comprehensive range of socio-economic characteristics as effect modifiers to understand their relative importance in modifying the effect of disability on mental health. Because of these limitations, it is difficult to draw conclusions about the influence of socio-economic characteristics in modifying the effect of disability on mental health.

The existing literature underscores the need for better evidence about the extent to which socio-economic characteristics modify the effect of disability on mental health in the Australian context. Furthermore, it draws attention to the need to address the methodological limitations of previous studies.

Mediation by socio-economic characteristics

Only four studies have examined mediation of the effect of disability on mental health using longitudinal data, with all studies quantifying the effect operating through psychosocial socio-economic factors.

A Canadian study of couples aged 65 years and older found no evidence of mediation of the effect of disability on psychological distress through marital support [139] and similarly a study of older adults in China found no evidence of a mediating effect of social interaction on life satisfaction and happiness [140]. A study of people aged 65 years and older in the United States examined whether the association was mediated by changes to social support and psychological resources. It found that social support accounted for only 6% of the total effect of disability acquisition on depressive symptoms whilst psychosocial resources (self-esteem and sense of control) accounted for 43% [141]. The final longitudinal study consisted of older adults aged 74 years and older in Taiwan and examined the effect of disability on depressive symptoms, quantifying the mediated effect operating jointly through activity restrictions, perceived stress and social support [142]. The study examined depressive symptoms at two separate time points, and results suggested that 87% of the association was explained by these mediators after four years of follow up, increasing to 99% after eight years.

All of these studies examined mediation of the effect by psychosocial factors. The estimates of proportion of the effect that was mediated by these psychosocial characteristics varied immensely, which is unsurprising because the studies were conducted in different settings, used a variety of definitions of disability and disability acquisition, examined diverse outcomes including depressive symptoms, psychological

distress, life satisfaction and happiness, and examined mediation through various psychosocial factors including marital support, social interaction, social support, psychological resources, and through activity restrictions, perceived stress and social support examined jointly. There is some suggestion from these studies that very little of the effect of disability on mental health was explained by psychosocial factors such as marital support, social support and social interaction but a greater proportion was explained through psychological resources such as self-esteem, sense of control and stress.

However, there were substantial limitations in the methodological approach of all four of these studies, which need to be considered when interpreting the findings. Though all of the studies described were longitudinal in design, only one study examined people who acquired a disability, allowing for better control for confounding [141]. Most importantly, none of the studies applied a causal mediation analysis approach to address the limitations of traditional approaches to mediation which are prone to bias (discussed in detail in the Methods chapter, section 3.5.5). Therefore, these estimates of mediated effects are likely to be biased and should be interpreted in light of these substantial limitations.

The review of the existing literature demonstrates the scarcity of the evidence relating to mediation of the effect of disability acquisition on mental health by socio-economic characteristics. It highlights the need for research using longitudinal data, applying causal mediation methods to minimise bias, and conducted within the Australian context to better understand the socio-economic mechanisms driving the disability-related mental health inequalities.

2.5 Conclusion

Since the introduction of the NDIS, disability is high on the policy agenda. While the NDIS is an important step to improve the lives of people with disabilities, key social determinants of health such as education, employment and provision of affordable housing will not be addressed by the NDIS. Furthermore, only a small proportion of Australians with disabilities will be eligible for the NDIS, therefore it is unlikely to eliminate the socio-economic disadvantage faced by Australians with disabilities and improve their poor mental health. Additional policy strategies are needed to address the current mental health inequalities.

This chapter has outlined an important evidence gap. There is a lack of knowledge about how to target health and social policies to improve the mental health of Australians with

disability. It has also highlighted the need for more robust evidence on the causal mechanisms by which socio-economic factors lead to disability-related mental health inequalities to address the limitations of existing research.

The research contributing to this thesis has been conceived to bridge this evidence gap and address this important and under researched public health issue at a time that is particularly opportune given the current major reforms to the disability policy landscape. The analyses in the thesis investigate effect modification and mediation by socio-economic characteristics to shed light on the mechanistic pathways leading from disability acquisition to poor mental health. In order to address the limitations of previous research, I apply innovative epidemiologic and statistical methods including causal mediation analysis, propensity score methods using inverse probability weighting, fixed effects models, quantitative bias analysis and multiple imputation. In such a way, the thesis aims to generate high-quality, methodologically robust, policy-relevant research about how to take evidence-based action on the social determinants of health to improve the mental health of people with disabilities in Australia.

Chapter 3 Methods

This chapter starts by summarising the methodological challenges encountered in this thesis and then describes the approaches used to address them. It details the causal inference framework that informed the choice of the epidemiological and statistical methods including the use of longitudinal data with repeated measures over time, and the use of methods including analyses of effect modification, causal mediation methods, propensity scores using inverse probability weighting, fixed effects models, quantitative bias analysis and multiple imputation.

3.1 Methodological challenges

This thesis uses observational data to estimate the causal effect of disability on mental health. In this section, I discuss methodological issues common to all analyses contributing to this thesis, which have motivated the epidemiological and statistical approaches that I have used to analyse the data. These include the complexities of the pathways between disability and mental health; confounding; selection bias; and information bias.

3.1.1 Complexity of pathways between disability and mental health

Understanding the mechanisms that underlie health inequalities is crucial in informing interventions to reduce them. In situations where it is not possible to intervene on the exposure of interest, such as disability or disability acquisition, understanding the extent to which an exposure-outcome relationship (disability-related mental health inequalities) is attributable to a third variable can provide insight into how the effects of *upstream* determinants of health (such as disability) are explained by more *downstream* risk factors (such as employment), identifying potential modifiable targets for policy interventions.

The relationship between disability and mental health is complex because disability may be both a cause and a consequence of poor mental health. There are three plausible pathways that may explain the mental health disparities that exist between people with and without disabilities (Figure 3.1). Firstly, and the focus of this thesis, is the causal effect of disability acquisition on mental health, depicted by the solid box in Figure 3.1, whereby disability causes a deterioration in mental health either through direct or indirect pathways. Secondly, the mental health disparities experienced by people with disabilities could be due to reverse causation, by which people with poor

mental health are at higher risk of acquiring a disability [97], illustrated by the dotted box in Figure 3.1. Finally, the association may be explained by confounding by variables which are common causes of both disability acquisition and poor mental health, shown by the dashed arrows in Figure 3.1, for example socio-economic disadvantage which has been shown to be a determinant of both disability [98, 99] and poor mental health [143, 144]. Most likely, the association between disability and mental health represents an interplay between these different causal processes, which work in concert to create an accumulation of advantage or disadvantage over the life course [103].

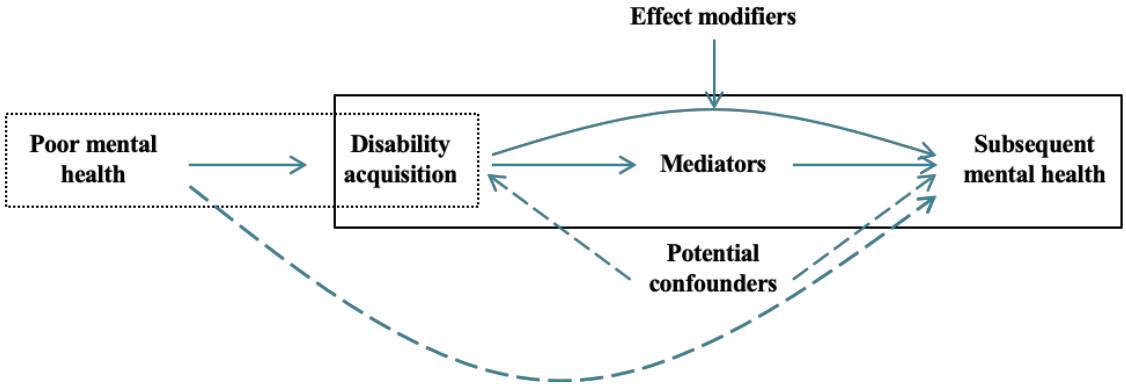

Figure 3.1 Simplified causal diagram depicting potential causal mechanisms, including causal effects (direct and indirect through mediators), reverse causation and confounding

The causal pathway of interest, from disability acquisition to poor mental health, is itself likely to encompass complex causal mechanisms. It is likely to result from a complex intersection of multiple component causes – the effect may be mediated by downstream risk factors and it may be modified by other exposures leading to heterogeneity in the effect. In addition, it might not be only separate pathways that are important in creating inequalities but 'synergistic effects of multiple exposures. The complexity of the pathways linking disability acquisition and mental health makes it difficult to disentangle and isolate the individual causal processes at play, which in turn makes it hard to design interventions to reduce existing mental health inequalities.

3.1.2 Confounding

In epidemiology, randomised controlled trials provide the highest level of evidence for establishing valid causal relationships between exposures and outcomes [145]. By randomising people to either receive the intervention (exposure) or not, with a large enough sample size, the exposure groups become *exchangeable* with respect to the outcome measure. Exchangeability with respect to an outcome occurs when the covariates that influence the outcome are balanced between the exposure groups, such

that if the exposures had been exchanged between the comparison groups, we would expect to see the same outcome distribution associated with each outcome.

However, studies examining disability as an exposure have to rely entirely on observational data as disability cannot be randomly assigned to study participants. In observational studies, participants are not randomly assigned to an exposure. Therefore, the comparison groups of people who are exposed to a risk factor and those who are not exposed to the risk factor are likely to be systematically different to each other. These differences may introduce bias due to confounding. Confounding of the effect of an exposure on an outcome occurs as a result of a variable that is a shared common cause of both exposure and outcome (Figure 3.2). It induces a spurious association between the exposure and the outcome, making it difficult to identify causal effects from observed associations.

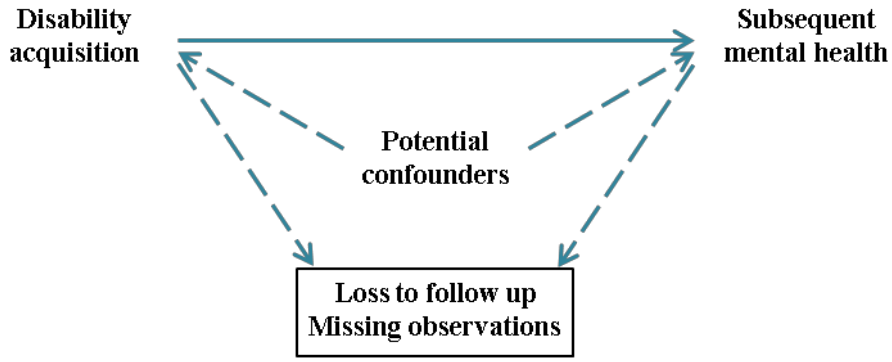

Figure 3.2 Simplified causal diagram depicting bias due to confounding and selection bias

Some of these differences will be measurable and therefore the common cause (or causes) can be adjusted, stratified or conditioned on in the analyses, eliminating (or diminishing) the spurious component of the association [146]. However, some of the spurious association may persist, as a result of residual confounding due to unmeasured or mismeasured confounders.

Generally in the population people with disabilities differ from those without disability in many ways including their age and socio-economic characteristics. When we compare people with and without disability in order to draw conclusions about differences in their mental health arising as a result of their disability, we need to account for the differences in mental health between the groups that exist because of confounding by other covariates (such as imbalances between the groups in the distribution of age and socio-economic characteristics). If all confounders are measured (without error) in the observational dataset and can therefore be adjusted for, then the exposure groups would

be said to be exchangeable conditional on a set of covariates. If confounders are not recorded in the dataset, or are poorly measured, the groups would not be exchangeable, leading to bias in the estimated effects from residual confounding.

With an exposure that changes over time, longitudinal observational studies provide one way of isolating the downstream causal effects of the exposure on the outcome, separately from the effects of reverse causation and confounding. Alternatively, quasi-experimental studies can also provide estimates of the causal effect of disability, for example where disability is the consequence of a chance occurrence such as a bus collision [147].

3.1.3 Selection bias

Selection bias produces biased estimates of effect resulting from the selection of participants in the study. It occurs if the relationship between the exposure and outcomes is different in the participants compared to non-participants [148]. Missing data are unavoidable in longitudinal studies that follow the same individuals over time, arising from non-response to the survey, loss to follow up (drop out) from the survey, and missing observations for variables recorded in the dataset. If missing data are influenced by both the exposure and the outcome (or their causes), the results of an analysis including only those participants with complete data will be biased. Conditioning on the missing data (by restricting the sample to those with complete data) induces a (non-causal) statistical association between the exposure and the outcome due to collider-stratification bias, a type of selection bias. The missing data act as a collider of the association, a common effect of the exposure and the outcome, leading to selection bias resulting from conditioning on a collider (Figure 3.2) [149, 150].

This thesis uses longitudinal observational data, therefore there will inevitably be problems of missing data. Because we are defining our exposed group as people who acquire a disability during their participation in the study, they are already participants of the survey when they acquire the exposure, therefore missing data due to non-response to the survey will not be affected by the exposure or the outcome. However missing data due to loss to follow up and missing observations are likely to be affected by both disability acquisition and mental health which may lead to selection bias.

3.1.4 Information bias

Another type of bias in estimating effects, information bias, arises from mismeasurement of the variables of interest to the analysis [148]. Information bias can occur from measurement error of the outcome (mental health), misclassification of the exposure

(disability), or mismeasurement of the confounders and mediators in the analysis because all variables are self-reported.

Measurement error can be independent or dependent, and non-differential or differential [151, 152]. Measurement error is said to be *independent* if the measured values of the exposure and outcome are statistically independent conditional on the true values of the exposure and outcome, or *dependent* if the measured values are statistically dependent conditional on the true values [151]. Measurement error (of the outcome) is said to be *non-differential* if the measurement error of the outcome does not depend on the true value of the exposure (and vice versa for measurement error of the exposure), or *differential* if the measurement error of the outcome depends on the true value of the exposure (and vice versa). Therefore, measurement error can be independent non-differential, dependent non-differential, independent differential or dependent differential. All types of measurement error can produce bias in the results, but the biases arising from dependent differential measurement error are most problematic as the magnitude and direction of the effect of bias on the results cannot be predicted [151].

Relating to the research questions in this thesis, where both exposure and outcome are self-reported, any measurement error in the reporting of the exposure is also likely to occur in the outcome, leading to bias due to dependent measurement error. For example, if an individual systematically over-reports both the extent of their disability and mental ill health, they may appear to be more highly correlated than they truly are, causing bias away from the null. It is also possible that the measurement error is differential. For example, people with disabilities may systematically report their mental health differently compared to people without disability. Therefore, the measurement error affecting the analyses in this thesis is likely to be dependent differential.

3.2 A causal inference framework

The methodological challenges described in the section above highlight the need for caution in the interpretation of estimated statistical associations between disability and mental health as causal effects. To address these challenges throughout this thesis, innovative methods grounded in the causal inference framework have been used to generate robust estimates of effect, which can be interpreted as causal effects. The purpose of this section is to introduce the causal inference framework that has been applied throughout this thesis and its relevance to the study of disability and mental health.

3.2.1 Causal inference in epidemiology

Epidemiology aims to establish associations between exposures and outcomes in order to develop interventions to reduce or eliminate exposure to the risk factor to improve health outcomes. To infer that changing an exposure will change an outcome, there needs to be an understanding about cause and effect - whether exposure to a risk factor *causes* an outcome. Therefore, at its core, epidemiology is interested in estimating causal effects rather than statistical associations [153].

Traditional approaches in epidemiology have been reluctant to infer causal relationships from observational studies because they are prone to confounding, reverse causation, selection bias and information bias. However, with many research questions in the field of social epidemiology only answerable using observational data, it is important to establish a best practice approach to infer causation from observational studies, to guide researchers as to how to use the best possible evidence to answer important epidemiological questions.

A causal inference framework for analyses of observational data is necessary to formulate causal assumptions underpinning the analyses, link statistical models to the data, and draw conclusions from the results, given the causal assumptions being made [154]. The recognition for the need to implement observational analyses within a causal inference framework has led to the development and application of a wide range of analytic methods to estimate causal effects, to enable inference of causation from estimated statistical associations.

3.2.2 The counterfactual approach

The counterfactual approach is one approach to causal inference in epidemiology. It defines causal effects as comparisons of potential outcomes under different interventions (or exposures) [155]. Therefore, a causal effect is estimated by comparing a particular health outcome of interest when an individual is exposed with the health outcome when the same individual is unexposed. However, the fundamental problem of causal inference is that we can never simultaneously observe counterfactual exposure states within the same individual – we only ever observe one *factual* exposure state, but not the *counterfactual* state. Therefore it is impossible to observe individual-level causal effects of an exposure on an outcome [156].

However, with appropriate data and a set of clearly stated assumptions, the average causal effect in the population can be estimated by comparing average outcomes between exposed and unexposed individuals. The average causal effect estimated in

the sample can approximate the true causal effect if the exposure groups are exchangeable.

In well-conducted randomised controlled trials, exchangeability of comparison groups is achieved by randomly assigning some people to receive the intervention and others not, making the groups comparable. Therefore, comparing the observed outcomes between the exposure groups provides an estimate of the average causal effect in the population.

In observational studies however, finding a surrogate for the theoretical counterfactual individual is more challenging. The counterfactual framework proposes that analyses of observational studies should mimic key aspects of randomised controlled trials, with individual-level exposures viewed as interventions, allowing the findings to be interpreted using counterfactual reasoning [157]. Many of the features of randomised experiments that make them so appealing for causal inference can be replicated in the design (and analysis) of observational studies [155]. An in-depth understanding of the "intervention" assignment – i.e., how the exposure of interest is distributed in the population – can be used to reconstruct a hypothetical underlying randomised controlled trial that would have led to the observed data [155]. Causal inference from observational studies relies on the assumption of conditional exchangeability (that the exposure groups are exchangeable conditional on a set of covariates), meaning that within subsets of the population the exposure is effectively randomised [158]. Therefore, a key focus of analyses of observational data becomes adequate adjustment for these covariates [159].

The counterfactual framework formally articulates the set of assumptions and conditions under which causal effects can be estimated [160] and provides a set of methodological tools to investigate cause-effect relationships.

3.2.3 Causal diagrams

Counterfactual reasoning is often supported by causal diagrams [161]. Causal diagrams, also known as Directed Acyclic Graphs, are visual representations of the causal relationships believed to exist between different variables in an analysis, therefore illustrating the hypothesised causal story linking an exposure and an outcome [154]. Causal diagrams depict causal effects using arrows between variables.

They are useful tools for guiding epidemiological analyses within a causal inference framework, their principal use being for clarifying assumptions about confounding and guiding covariate selection [162]. Their mathematical properties can be used to determine whether conditional exchangeability can be achieved (based on the hypothesised causal structure between the variables), using an algorithm known as d-separation [163], and therefore whether causal effects can be inferred from the data.

Aside from providing guidance as to which variables should be adjusted for in an analysis to control for confounding, causal diagrams can also provide insight into residual confounding from unmeasured confounders and selection bias [162].

As such, causal diagrams explicitly illustrate the causal assumptions being made in an analysis, the accuracy of which can then be openly debated and challenged [164]. While it is clear that causal diagrams provide a powerful tool to improve epidemiological analyses, the validity of results informed by causal diagrams depends on the accuracy of the hypothesised causal relationships. Detailed information on study design, temporal ordering, biological mechanisms and epidemiological principles are necessary to construct valid causal diagrams [165]. Throughout this thesis, causal diagrams were constructed using the most recent evidence from the literature to ensure they were as accurate as possible.

3.2.4 Summary

In this thesis, all analyses have been grounded in counterfactual reasoning and guided by causal diagrams to maximise causal inference. Though the many methodological challenges mean that strong assumptions are necessary to infer causation from estimated associations, the causal inference approach strengthens the research by making the assumptions underpinning the analyses explicit and interpreting the results of the analyses in light of the causal assumptions being made and the limitations of the data.

3.3 The Household, Income and Labour Dynamics in Australia Survey

In order to address the methodological challenges posed by the complex nature of the relationship between disability and poor mental health, and the risk of bias from confounding, selection bias and information bias, I examined the research questions of interest using a large nationally representative longitudinal dataset. Throughout this thesis, data from the HILDA Survey were used to estimate the effect of disability acquisition on mental health and illustrate how socio-economic characteristics influence this relationship.

HILDA is a household-based longitudinal study of Australian individuals and households that has been conducted annually since 2001 [166]. The HILDA Survey is a rich data source – it has detailed information on the demographic, social, economic, and health characteristics of individuals and their families collected using a combination of interviews with trained personnel and self-completed questionnaires.

Designed as a panel study, the HILDA Survey follows the same group of individuals over time, recording changes to their circumstances. The original sample recruited in 2001 consisted of 13,969 participants from 7682 households, randomly sampled using a national probability sample of private dwellings to be representative of the Australian population. The sampling strategy consisted of a three-stage area-based design, in which areas were first selected with probability proportional to size calculated using the number of dwellings reported in the 1996 Census, then dwellings were selected with equal probability of selection within each area, and finally households were selected within each dwelling [166]. Data were collected on each household member, and face-to-face interviews were sought from all household members aged 15 years and above. The household response rate in the first wave of the survey was 66%. Of the 15,127 eligible adults in these households, 13,969 completed the survey (92%) [66].

In subsequent waves, all existing survey members were invited to take part in the survey as well as household members attaining the age of 15 years and new household members if new households were formed. Inclusion of these new households into the survey maintains sample representativeness. A top up sample of 2000 households was added in 2011 to allow better representativeness of the Australian population and increase the sample size of the survey [167]. The top up sample was selected using the same multistage sampling methods as the original sample. Household response rates in subsequent waves ranged between 87% in wave 2 and 67% in wave 17 [66].

The analyses in this thesis use data from the first 17 waves of HILDA, including 253,182 observations from 31,206 participants.

The choice of this dataset was based on the following features:

i) HILDA is the only nationally representative longitudinal survey in Australia;

ii) It has a large sample size and survey participants have been followed up for 17 years;

iii) The data are collected annually, with data on disability, mental health and socio-economic characteristics measured at every wave;

iv) The disability question is based on the ICF definition of disability;

v) It collects data on a commonly used mental health score which has been validated for use in the Australian population;

vi) Data are collected on a comprehensive range of socio-economic characteristics;

vii) The longitudinal nature of the data enables characterisation of changes to people's circumstances over time (annually).

The study protocol was approved by the Melbourne School of Population and Global Health Human Ethics Advisory Group (ID 1545138.1).

3.4 Variables

3.4.1 Exposure: disability acquisition

In all analyses in this thesis, I used the longitudinal nature of the data to model incident disability to understand the effect of acquiring a disability on mental health, rather than examining people with prevalent (existing) disability. Identifying a group of people who changed disability status enabled comparisons of people's circumstances prior and subsequent to disability to isolate the effect of disability on people's mental health.

Information on disability was collected from participants in every wave of the HILDA Survey in the face-to-face questionnaire using a single question asking participants if they had an "impairment, disability or long term health condition, which restricts their everyday activities that had lasted, or was likely to last, for a period of six months or more". From 2003 onwards, respondents who reported a disability were then asked to identify the type of impairments that they experienced from a list provided by the interviewer (e.g., hearing problems, limited use of legs, mental illness which requires help or supervision). Across all 17 waves of data, the average prevalence of disability in the sample was 27%.

Disability acquisition was coded from the single question about the presence of disability, defined as two consecutive waves reporting no disability immediately followed by two consecutive waves reporting a disability. Two consecutive waves of disability were used to identify people with longer-term disabilities. Furthermore, using two consecutive waves in which individuals reported a disability excludes people with transient disability and may also reduce potential information bias from mismeasurement of disability, which is more likely to occur using a single report of disability.

3.4.2 Outcome: mental health

The Mental Health Inventory (MHI), one of the eight subscales of the Short Form 36 (SF-36) health questionnaire, was used in all analyses in this thesis to measure people's mental health. This variable was recorded in every wave of the HILDA Survey, therefore it can be used to explore changes in people's mental health over time, and how those changes relate to changes in disability.

The SF-36 is a commonly-used general health questionnaire which has been validated for use in the Australian population using the HILDA Survey [168]. The SF-36 comprises eight subscales measuring multiple health dimensions, including physical functioning, vitality, bodily pain, general health perception, physical role functioning, emotional role functioning, social role functioning and mental health, which are constructed using weighted averages of the questions relating to each subscale and transformed into scales ranging from 0 to 100. Two overall summary measures of health and well-being representing physical health and mental health can also be constructed by aggregating the eight individual subscales using principal components analysis [169, 170].

The MHI is the mental health subscale of the SF-36. It assesses symptoms of depression, anxiety and psychological well-being over the previous four weeks. It is created using five questions, each scored using five response categories, and the total scores are transformed into a continuous variable ranging from 0 to 100, with higher scores representing better mental health. The MHI is a well validated measure of mental health status, and has been shown to be psychometrically sound [171], with high reliability and acceptable internal consistency and discriminant validity using the HILDA Survey [168, 169]. It is an effective screening tool for mood and anxiety disorders and severe depressive symptomatology [172-175]. Previous research has suggested that a difference of four to five-points on the MHI scale is likely to reflect a minimally important clinical difference in mental health [112, 113].

3.4.3 Socio-economic characteristics

HILDA collects data on a wide range of social and economic variables. I applied a broad conceptualisation of socio-economic characteristics to represent the social and economic determinants of health and health inequalities. These were then used in analyses to understand their influence on the relationship between disability acquisition and mental health. They included education, employment, occupation, income, wealth, financial hardship, financial satisfaction, housing tenure, housing affordability, relationship status, children, social support, frequency of socialising, relationship with parents, smoking, alcohol consumption, physical activity, body mass index, diet and sleep. Most of these variables were recorded at every wave of data collection, therefore it is possible to examine changes in socio-economic characteristics that occur as a result of disability acquisition, and how such changes influence the relationship between disability and mental health.

3.5 Effect modification analysis

3.5.1 What is effect modification?

As described in Section 3.1, there are complex mechanisms leading from disability to poor mental health. Effect modification of the relationship by another exposure is one manifestation of the complexity of this relationship [176].

There are many examples, particularly in social epidemiology, where the effect of one exposure depends on the presence or absence of another exposure, or where the magnitude of the association between an exposure and outcome differs across levels of a third variable [177]. This phenomenon is known as effect modification. It leads to heterogeneity of effects across subgroups of people with a shared characteristic in the population. This is particularly common when there are complex mechanisms by which an exposure leads to an outcome, where multiple component causes may be required to cause an outcome.

The terminology effect modification and interaction are often used interchangeably, however they are conceptually different. Effect modification relates to whether the effect of an exposure on an outcome differs across levels of a third variable, whereas interaction relates to the effect of two exposures together on an outcome [178, 179]. The aim of this thesis is to understand how the effect of disability acquisition on mental health is influenced by socio-economic characteristics so relates to effect modification rather than interaction.

3.5.2 Why study effect modification?

Effect modification tells us for whom an effect occurs, or for whom the effect is of smaller or greater magnitude. Such analyses can be used to identify subgroups who are particularly vulnerable to poor outcomes, for whom interventions may be more beneficial. But it can also shed light on mechanism, providing insight as to why it is of a particular magnitude. Furthermore, in situations where we cannot intervene on the exposure, such as with disability, understanding determinants of the magnitude of the association may help identify targets for interventions that could eliminate some of the effect of the exposure on the outcome.

3.5.3 Measures and scale of effect modification

Effect modification can be quantified by estimating the difference between the joint effect of the exposure and the effect modifier on the outcome (Y_{11}-Y_{00}, i.e. the difference between the outcome when an individual is exposed to both the exposure and the effect modifier Y_{11} and the outcome when an individual is exposed to neither Y_{00}) and the effect

of each of the exposure and the effect modifier on the outcome individually ([Y_{10}-Y_{00}]+[Y_{01}-Y_{00}]), thereby quantifying the excess effect attributable to exposure to both risk factors simultaneously [180].

Known as the relative excess risk due to interaction (RERI), it can be calculated by:

$$Effect\ modification\ on\ the\ additive\ scale = E[Y_{11}] - E[Y_{10}] - E[Y_{01}] + E[Y_{00}]$$

This difference is a quantification of the excess effect of the additive scale.

It is also possible to quantify the excess effect on the multiplicative scale using a log-linear regression model. It can be calculated by:

$$Effect\ modification\ on\ the\ multiplicative\ scale = \frac{E[Y_{11}]/E[Y_{10}]}{E[Y_{01}]/E[Y_{00}]}$$

It is possible to have effect modification on the additive scale but not the multiplicative scale, and vice versa. It has been argued that the additive scale is of greater public health importance because it provides insight as to whether the effect of the exposure on the outcome differs between subgroups of the population [181, 182]. Therefore, effect modification on the additive scale has been used throughout this thesis.

3.6 Causal mediation analysis

3.6.1 What is mediation?

Mediation is another manifestation of the complexity of the relationship leading from disability to poor mental health. Mediation occurs when an exposure affects an outcome through an intermediary variable (or variables) – the exposure influences this intermediary variable, which in turn influences the outcome. Therefore mediation relates to the processes that explain observed relationships between exposures and outcomes [183]. Understanding and quantifying the phenomenon of mediation allows for a better understanding of the mechanistic relations between variables, and therefore a better understanding of how exposures lead to health outcomes, and the relative strength of different pathways [176].

Mediation analysis quantifies the proportion of a total effect of an exposure on an outcome that is occurring through a particular intermediate variable, the mediator, and what portion is occurring through other pathways. It aims to decompose the total effect of an exposure on an outcome into the proportion of the effect that occurs through a mediator, the *indirect effect*, and the effect not through the mediator, the *direct effect*.

3.6.2 Why study mediation?

Understanding the mechanisms by which exposures affect outcomes can be of vital importance to public health. Analysis of mediation allows epidemiologists to go beyond describing risk factors for diseases to developing explanatory theories of causation for how diseases arise. Therefore, it can provide aetiologically important information which can strengthen understanding of causal effects as well as clarifying specific mechanistic pathways [183].

Mediation concerns causal explanation for relationships between exposures and outcomes. Analyses of mediation therefore can produce useful, policy-relevant evidence on the proportion of an exposure's effect on an outcome that operates through an intermediary factor. Importantly, it can be used to estimate the impact of public health interventions, or refine existing interventions, by targeting the most important components of the pathways from exposures to diseases, particularly in settings where it is not possible or effective to intervene directly on the exposure. For example, understanding the pathways leading from disability acquisition to poor mental health would enable quantification of how much mental health inequalities could be reduced over time by intervening on important intermediary variables such as employment or social support.

3.6.3 Traditional approaches to mediation analysis

The concept of mediation analysis is not new. It emerged from methods of genetic path modelling, a special case of structural equation modelling describing directed dependencies between variables by modelling covariance and correlation matrices, developed by Wright (1921) in the 1920s [184]. Mediation analysis became popularised in the 1970s and 1980s. Mediation analysis was first introduced to epidemiology in 1973 using an approach commonly known as the *difference method* [185], and in the psychology literature in the mid-1980s using an approach known as the *product method* [186]. The difference method and the product method have been widely used in the health and social sciences and will be hereafter referred to as the traditional methods for mediation analysis. Both of these approaches use parametric statistical methods that rely on combinations of regression coefficients to estimate direct effects and indirect effects that occur through the mediator of interest [187]. Using both of these approaches, the interpretation of the total effect is the overall effect of the exposure on the outcome; the direct effect is the effect of the exposure on the outcome at a fixed level of the mediator; and the indirect effect is the effect of the exposure on the outcome that operates through the mediator.

The difference method is the most common approach used in epidemiology, first described by Susser (1973) [185]. This method fits two regression models for the outcome conditional on the exposure and any potential confounders, one with adjustment for the mediator and one without, and compares the coefficient for the exposure in models with and without the mediator. The model with the mediator provides an estimate of the direct effect of the exposure on the outcome and the model without the mediator estimates the total effect of the exposure on the outcome. Any attenuation of the effect on adjustment for the mediator is interpreted as indication of mediation, with the indirect effect calculated as the difference between the unadjusted and adjusted estimated exposure effects.

The product method was popularised by Baron and Kenny (1986) in a highly influential paper which discussed the distinction between mediation and moderation in the context of linear structural equation models [186]. In this approach, two regression models are fitted. In the same way as in the difference method, the direct effect is estimated as the coefficient for the exposure from a regression model for the outcome conditional on the exposure, mediator and any potential confounders. An additional model is fitted, regressing the mediator on the exposure and potential confounders. The indirect effect is estimated from the product of the exposure coefficient in the mediator model and the mediator coefficient in the outcome model. The rationale for this approach is that mediation depends on the extent to which the exposure changes the mediator, and the extent to which the mediator changes the outcome.

These methods are algebraically equivalent if both the outcome and mediator are continuous and modelled using correctly specified linear regression models, however the results will differ in scenarios with binary mediator or outcome variables in which logistic regression models are used [188], suggesting that one or both of these approaches must produce biased estimates [189, 190].

3.6.4 Limitations with traditional approaches to mediation

The traditional approaches to mediation analysis are prone to bias. Firstly, the traditional approaches presuppose no interactions between the effect of the exposure and the mediator on the outcome, which can lead to biased results and invalid conclusions. It is possible for heterogeneity to exist in the effect of the exposure on the outcome across different levels of the mediator, even in such a way that the effects operate in different directions. By assuming no interaction, the effect would be averaged over the different levels of the mediator, which could substantially bias the estimate of the direct effect. Secondly, there are strong assumptions about no unmeasured confounding which can

introduce bias. In addition to the assumption that is made in any epidemiologic analysis of the relationship between an exposure and an outcome about no unmeasured confounding of the exposure-outcome relationship, in mediation analyses it is also important to account for confounding of the exposure-mediator relationship, and particularly problematic is the assumption about no confounding of the mediator-outcome relationship. Even if the exposure is randomised, as in a randomised controlled trial, a mediation analysis will not necessarily be free of confounding because the mediator is unlikely to also have been randomly allocated and therefore there may be confounding of the mediator-outcome relationship [191], which can substantially bias the estimates of direct and indirect effects. Thirdly, traditional methods cannot model non-linear associations between the exposure, mediator and outcome [192, 193].

The traditional approaches will give a valid estimate of direct and indirect effects (with a causal interpretation) for a continuous outcome and a continuous mediator if (1) the outcome model without an interaction between exposure-mediator is correctly specified and (2) the no unmeasured confounding assumptions hold, i.e. that the covariates included in the regression models include all sets of potential confounders. However, there are many situations in which mediation analyses using traditional approaches will produce biased estimates.

3.6.5 Counterfactual approach to mediation analysis

With the increasing interest in causal inference in statistics, coupled with the exponential increase in computing power and the availability of ‘big data’, there has recently been a renewed flurry of interest in methods of mediation analysis. Novel approaches based on a causal inference framework have been developed which seek to address the limitations of traditional approaches, supplying definitions of direct and indirect effects that allow for decomposition of the total effect in the presence of exposure-mediator interactions and non-linear associations between the exposure, mediator and outcome.

Causal mediation analysis provides a general approach to decomposing total effects into direct and indirect effects irrespective of the statistical models (e.g. logistic regression for binary outcomes) and of interactions between the exposure and mediator. In this framework, differences in outcomes are estimated between counterfactual scenarios with defined values of the exposure and mediator. The advantage of this counterfactual framework is that it clarifies which effects are being estimated and the assumptions necessary to identify direct and indirect effects from data, which were often not brought to focus in traditional methods. The methods still include the same confounding assumptions as the traditional approaches, but there is an important emphasis on

clarifying the confounding assumptions required for the total, direct and indirect effect estimates to have a causal interpretation. This guides researchers to draw causal diagrams to identify measured and unmeasured confounders and to inform sensitivity analyses.

Definitions

Robins and Greenland (1992) and Pearl (2001) formulated counterfactual-based definitions of total, direct and indirect effects that can be estimated from regression models [191, 192].

The controlled direct effect (CDE) estimates the effect of an exposure on an outcome while fixing the mediator at a particular value *m*. It is calculated as the difference between two counterfactuals Y_{1m} and Y_{0m}, which represents the difference between the outcome Y when the individual is exposed (a=1) and the mediator set to a value *m* and the outcome when the individual is unexposed (a=0) and the mediator is set to *m*.

$$CDE(m) = E[Y_{1m} - Y_{0m}]$$

If the estimated CDE is non-zero, then it can be concluded that there is a direct effect of the exposure on the outcome which is not occurring through the mediator. The CDE is an interventionist approach which can be interpreted as the effect of an intervention that sets the mediator to a particular value for the whole population (e.g. an income value), with direct relevance to questions about policy. Because the mediator is set at a particular value, it is possible to estimate a series of CDEs, fixing the mediator at various different values (e.g. low, middle, and high values of income). In the presence of an interaction between the exposure and the mediator, we would expect there to be differences in the CDE of the exposure on the outcome at different levels of *m*.

The natural direct effect (NDE) estimates the effect of an exposure on the outcome while fixing the mediator to the value it would naturally take in the absence of the exposure. It is calculated as the difference between two counterfactual outcomes, the potential outcome when the individual is *exposed* and the mediator is set to the level it would naturally take in the absence of the exposure M_0 and the potential outcome when the individual is *unexposed* and the mediator is set to M_0.

$$NDE = E[Y_{1M_0} - Y_{0M_0}]$$

In contrast to the CDE, the estimation of the NDE allows the total effect of an exposure on an outcome to be decomposed into a direct and an indirect component and to calculate the proportion of the total effect which is due to mediation. Therefore this is the most appropriate measure of interest for understanding the mechanistic pathways that

are operating from the exposure to the outcome because it provides an estimate of what would have happened to the outcome for an exposed individual if the exposure had not caused the mediator but everything else had remained constant [194]. Another motivation for choosing the NDE is that the interpretation of the NDE may correspond to a more realistic substantive research question than the CDE which fixes the value of the mediator for the whole population at a single fixed value. The NDE addresses this problem because it fixes the mediator to the level that would naturally arise in the absence of the exposure for each individual in the population. For the purpose of this thesis, the NDE is likely to represent a more meaningful construct, providing an estimate of the effect of disability on mental health if the socio-economic characteristics of people with disabilities were no different to those of people without disability, rather than fixing socio-economic characteristics at a single value for the entire population.

The natural indirect effect (NIE) estimates the effect of the mediator on the outcome while fixing the exposure to a particular value. It is calculated as the difference between two counterfactual outcomes, when the individual is exposed and the mediator is set to the value it would naturally take in the *presence* of the exposure and the outcome when the individual is exposed and the mediator is set to the value it would naturally take in the *absence* of the exposure. Therefore, it represents the change we would expect to observe in a given outcome if we were to change the mediator to the value it would be expected to take under the counterfactual value of the exposure but without actually changing the exposure. For the NIE to be non-zero, the mediator would have to be different at different levels of the exposure ($M_1 \neq M_0$), therefore the exposure has to have an effect on the mediator.

$$NIE = E[Y_{1M_1} - Y_{1M_0}]$$

The total causal effect (TCE) estimates the total effect of the exposure on the outcome, calculated as the difference between the outcome when the individual is exposed and the outcome when the individual is unexposed. It can be decomposed into the sum of the NDE and the NIE.

$$TCE = E[Y_{1M_1} - Y_{0M_0}] = E[(Y_{1M_1} - Y_{1M_0}) + (Y_{1M_0} - Y_{0M_0})]$$

The proportion mediated (PM) measures the proportion of the effect of the exposure on the outcome that is mediated by the intermediary variable, calculated as the NIE over the TCE. This can also be interpreted as the proportion of the effect that would be removed if the pathway through the mediator was removed, therefore it captures the relative importance of the pathway in explaining the effect of the exposure on the outcome.

$$PM = \frac{NIE}{TCE}$$

It is important to note that this measure can be problematic, with poor precision and large confidence intervals, because it combines the variance of the NIE and the TCE. Therefore, it is not a good measure for testing for mediation but is useful as an intuitive measure of the extent of mediation.

Alternative decompositions

One of the key advantages of the counterfactual approach to mediation is that it can decompose the total effect into direct and indirect components in the presence of exposure-mediator interactions. Accounting for such interactions is necessary for estimating unbiased direct and indirect effects but understanding the contribution of exposure-mediator interaction is also important for fully accounting for the mediated effect, capturing the complexity of the dynamics of mediation, and increasing power to detect mediation [176].

The exposure-mediator interaction can either be included in the estimate of the NDE or the NIE. Therefore, there are two possible decompositions of the total effect into direct and indirect components that can be carried out, which differ in how they account for the exposure-mediator interaction. Natural effects can be further divided into pure and total effects, distinguished by differential inclusion of the exposure-mediator interaction - the interaction component being included in the total effect, not in the pure effect [195, 196]. Therefore, if there is no exposure-mediator interaction, the pure and total effects will be the same, but if exposure-mediation interaction is present, then the pure and total effects will be different.

The most common decomposition is the one described above, which decomposes the total effect into the pure NDE and the total NIE [191]. As defined above, the pure NDE represents the effect of changing the exposure on the outcome while fixing the mediator to the value it would naturally take in the *absence* of the exposure and the total NIE represents the effect of changing the mediator to the value it would take under counterfactual values of the exposure while fixing the exposure to be *present*.

An alternative is to decompose the total effect into the total NDE and the pure NIE. The total NDE represents the effect of changing the exposure on the outcome while fixing the mediator to the value it would naturally take in the *presence* of the exposure.

$$NDE_{total} = E[Y_{1M_1} - Y_{0M_1}]$$

The pure NIE represents the effect of changing the mediator to the value it would take under counterfactual values of the exposure while fixing the exposure to be *absent*.

$$NIE_{pure} = E[Y_{0M_1} - Y_{0M_0}]$$

In the more commonly used decomposition, the interaction is included in the indirect effect. This has been argued to be preferable because it captures the entirety of the effect that is mediated therefore provides evidence as to the operation (rather than just the presence) of mediation [197, 198]. Therefore, this is the decomposition used in the causal mediation analysis in this thesis.

If the interaction mechanism itself is of substantive interest, the TCE can be further decomposed to isolate the interaction component. VanderWeele (2014) developed an approach to decompose the TCE into four separate components: the CDE, the reference interaction, the mediated interaction and the pure NIE [199].

The CDE, the component due to neither interaction nor mediation, as defined above, is the direct effect of the exposure A on the outcome Y when the mediator is fixed at a particular value *m* for the whole population.

The reference interaction, the component due to interaction but not mediation, is the excess effect of the exposure on the outcome which exceeds the sum of the effect of the exposure and mediator considered separately that operates only if the mediator is present in the absence of exposure (i.e. M_0=1).

$$reference\ interaction = E[(Y_{1M_1} - Y_{1M_0} - Y_{0M_1} + Y_{0M_0})(M_0)]$$

The mediated interaction, the component due to both mediation and interaction, is the excess effect of the exposure on the outcome which exceeds the sum of the effect of the exposure and mediator considered separately that operates only if the exposure has an effect on the mediator (i.e. M_1-M_0≠0).

$$mediated\ interaction = E[(Y_{1M_1} - Y_{1M_0} - Y_{0M_1} + Y_{0M_0})(M_1 - M_0)]$$

The pure NIE, the component due to mediation but not interaction, as defined above, is the effect of the exposure on the outcome mediated by differential exposure to the mediator. It is only non-zero if the exposure has an effect on the mediator and the mediator affects the outcome when the population is not exposed.

The four-way decomposition approach quantifies the effect of the exposure-mediator interaction and unifies analytically the concepts of mediation and interaction (or effect modification), providing insight into the distinct roles of mediation and interaction in explaining the relationship between an exposure and an outcome [199].

Assumptions

Causal mediation methods still rely on the same three assumptions that are required for causal inference generally [200]. These are: positivity (there must be both exposed and unexposed participants at every combination of values of the observed confounders); consistency (that an individual's potential outcome under the observed exposure will be precisely equal to the observed outcome); and exchangeability (that individuals are comparable so that counterfactuals can be estimated by comparing individuals in the sample) [200]. There are also four additional assumptions about confounding that are required for estimates to have a causal interpretation.

i) No unmeasured confounding of the exposure-outcome relationship;

ii) No unmeasured confounding of the mediator-outcome relationship;

iii) No unmeasured confounding of the exposure-mediator relationship;

iv) No exposure-induced mediator-outcome confounding [176].

Under these assumptions, we can relate our counterfactual definitions of the CDE, NDE and NIE to empirical expressions that we can estimate from data.

Limitations of natural direct and indirect effects

- Cross-world counterfactuals

Counterfactuals are used to define natural direct and indirect effects at the individual level, which allow us to identify average effects for the population under a number of relatively strong assumptions (described above). However, in practice, for each individual, we only observe one of these scenarios (either exposed or unexposed). But hypothetically we can imagine (and estimate) two separate potential outcomes, one for each exposure level. For the estimation of the NDE and the NIE, additional potential outcomes are estimated, for different values of the mediator in addition to the potential outcomes for different values of the exposure (also known as "nested counterfactuals"). As such, the inferences that are being made are more complicated and more reliant on assumptions as we are observing only one of the counterfactuals that we are estimating for each individual. The estimation of natural direct and indirect effects relies on so-called *cross-world* counterfactuals, which represent for example, the potential outcome that would have been observed had the individual had the exposure X=1 and the mediator that would have been observed under X=0. Therefore, this approach estimates potential outcomes which are unobservable even with experimental data, as it requires the combination of logically incompatible exposure states [201].

- Many unmeasured mediators

Any direct path from exposure to outcome includes a number of infinitesimally small steps operating through intermediate variables (measured or unmeasured) [195, 202]. For estimating the total effect, these are irrelevant as long as their error terms are not correlated with the outcome. However, for the estimation of the NDE and NIE, this can be problematic. If the mediator of interest is not the very first of these steps, any preceding intermediate steps on the pathway from exposure to outcome would be potential confounders of the mediator-outcome relationship which could bias the estimation of the NDE and NIE [195, 202]. These preceding mediators would simultaneously be mediators of the exposure-outcome relationship and confounders for the effect of the mediator of interest on the outcome. Conditioning on such variables will remove bias from confounding but this will block part of the direct effect from exposure to outcome, leading to a bias in the estimate of the NDE. This is likely to be particularly problematic in situations where there is a large time lag between the exposure and the mediator of interest.

- Policy significance

There has been ongoing debate about whether the interpretation of direct and indirect effects is of policy significance because they cannot be interpreted as the effects of real-life interventions [191, 192, 203, 204]. However, when mediators represent socio-economic characteristics, as in this thesis, gaining an understanding of potential outcomes that would have been observed if there were no socio-economic inequalities between people with and without disabilities, may give insight into the proportion of the disability-related mental health inequalities that is avoidable and could be eliminated through policy intervention.

3.6.6 Multiple mediators

When multiple mediators are of interest natural direct and indirect effects can be estimated in different ways depending on the causal structure of the mediators. The simplest approach would be to estimate NIEs through each mediator considered separately, but this will only produce unbiased estimates if the mediators are causally unrelated. If the mediators are causally related, it is possible to decompose the TCE into the NDE and NIE operating through multiple mediators jointly, and if the causal ordering between each of the mediators is known, then the mediators can be examined sequentially to estimate path-specific NIEs [205]. For example, in a situation with two causally ordered mediators M1 and M2, with M1 causally preceding M2, sequential causal mediation analysis enables estimation, in model 1, of the NIE through M1

including paths that act through causal descendants of M1 but excluding the path that only acts through M2, and, in model 2, the NIE through both M1 and M2, consisting of all paths except for the direct path from the exposure A to the outcome Y (Figure 3.3). Therefore, the effect operating through M2 can only be inferred from the effect operating through M1 and the joint effect through M1 and M2. Further limitations include the additional assumptions required about the causal structure of the mediators to estimate NIEs through distinct pathways as well as the potential for bias if the mediators share common causes.

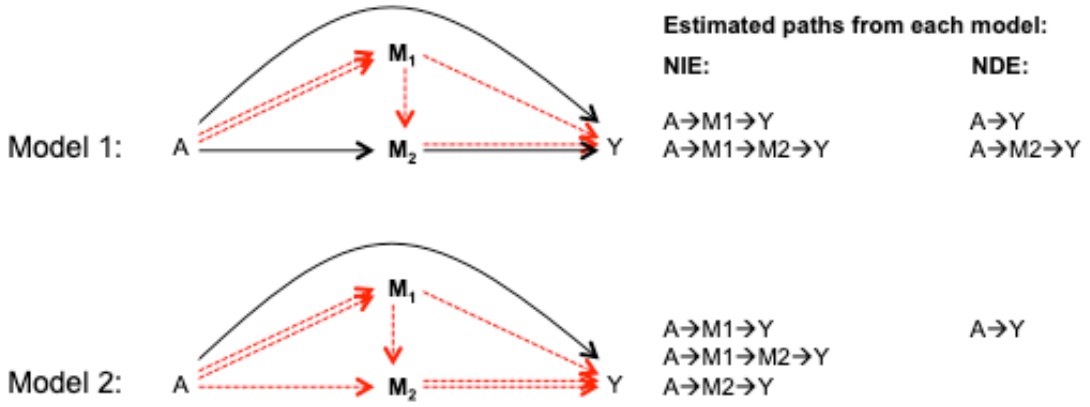

Figure 3.3 Simplified causal diagrams illustrating estimated paths from the exposure A to the outcome Y in a sequential mediation analysis with two causally-related mediators M1 and M2 – the NDE is represented by solid black lines and the NIE by dashed red lines

3.6.7 Interventional effects

An alternative approach, estimating stochastic interventional effects, was developed to address some of the limitations of natural effects identified above [206, 207]. In contrast to natural effects, which estimate individual-level effects of shifting the value of the mediator under different exposure scenarios, interventional effects quantify population-average effects representing the effect of shifting the distribution of the mediator in the population to what would be expected if everyone in the population was exposed or unexposed [208].

The interventional direct effect (IDE) estimates the population-average effect on the outcome of changing the exposure under a stochastic intervention G_0. G_0 sets the value of the mediator for each person in the population at a value randomly drawn from the conditional distribution of the mediator among the unexposed, given each individual's set of observed covariates.

$$IDE = E[Y_{1G_0} - Y_{0G_0}]$$

The interventional indirect effect (IIE) estimates the population-average effect on the outcome of shifting the mediator from the distribution that would be expected if everyone were exposed to the distribution that would be expected if everyone were unexposed given each individual's set of observed covariates. Therefore, the IIE estimates the effect of equalising the distribution of the mediator in one exposure group to be the same as that of another exposure group.

$$IIE = E[Y_{1G_1} - Y_{1G_0}]$$

Several reasons have been proposed for choosing to quantify interventional effects instead of natural effects. Interventional effects are particularly attractive for the analysis of multiple mediators because they allow quantification of effects via distinct pathways even if the structural dependence between mediators is unknown [206]. They also require one fewer assumption than the estimation of natural effects - they can be estimated in the presence of exposure-induced mediator-outcome confounding, thus avoiding the cross-world assumption. In the absence of such confounding, interventional effects for a single mediator are analogous to population-average estimates of natural direct and indirect effects [209]. Furthermore, interventional effects estimate the impact of population-level interventions on the mediator, which may have greater policy relevance than natural effects which estimate individual causal effects [208]. Particularly for analyses of health inequalities in which we are interested in understanding the effect of potential interventions on the mediator rather than the exposure, contrasting population distributions of the mediator rather than predicted values of the mediator for each individual under counterfactual exposure states, may be of greater policy significance [209, 210].

3.7 Propensity score methods

3.7.1 Why use propensity score methods?

As described in section 3.1, observational data for comparisons of health outcomes between groups of individuals are prone to problems of confounding if covariates differ substantially between the exposure groups. Propensity score methods provide an alternative method to traditional regression models for confounder adjustment and can provide an unbiased estimate of the causal effect of an exposure on an outcome in the presence of confounding [211-213]. It has been suggested that propensity score methods can better adjust for confounding compared to traditional multivariable linear regression models because they mimic an experimental approach [211]. Furthermore, the assumptions of exchangeability (whether the covariates that influence the outcome

are balanced between the exposure groups) and positivity (whether there is overlap in the distribution of covariates between the exposure groups) can be easily verified. Propensity scores reduce the characteristics of each individual in the sample into a single composite score [214], so that the analysis model estimates the effect of the exposure on the outcome adjusted for differences in all observed characteristics using a single covariate. Therefore, propensity score methods are particularly beneficial in analyses with many potential confounders. Additional advantages are that propensity score approaches are more robust to model misspecification and that the propensity score model, and therefore the choice of confounders in the main analysis, is conducted without considering the outcome variable, which avoids the possibility of the analysis strategy being driven by prior beliefs about expected association or results of the analysis.

In propensity score approaches, confounder adjustment is a two-step process. First, a regression model is fitted for the exposure with potential confounders as explanatory variables to derive a propensity score for each individual. The propensity score is the probability that an individual is exposed (acquires a disability) conditional on a group of chosen (observed) covariates [213]. Each person's propensity score is unknown but can be estimated from the data based on their observed covariates, typically using logistic regression. Second, these propensity scores are then used to balance covariates between the exposure groups and estimate unbiased exposure-outcome effects [213]. At every value of the propensity score, the observed covariates (i.e., the baseline characteristics of exposed and unexposed individuals) will be balanced [211]. As such, the exposure groups become exchangeable conditional on the propensity score, as they are comparable with regards to all measured potential confounders [213]. However, it is important to note that, whether using the propensity score approach or adjusting for all measured confounders in a regression model, neither will adjust for potential confounding by unmeasured variables.

3.7.2 Propensity score using inverse probability weighting

Propensity scores can be used in several ways to adjust for potential confounding, including methods based on stratification, matching, weighting, and inclusion of the propensity score as a covariate in the exposure-outcome regression model [215]. I chose to use inverse probability of treatment weighting because:

i) it does not discard any data (unlike matching exposed and non-exposed using propensity scores);

ii) simulation studies have suggested it produces the most valid estimates (compared to stratification or including the propensity score as a covariate in the exposure-outcome regression model) [216-218];

iii) the method is easily adapted to incorporate multiple imputation of missing data.

Unlike the other methods, inverse probability weighting does not attempt to create balanced exposure groups with the same value of the propensity score. Instead, it uses propensity scores to create an inverse probability weight (IPW) for each individual in the sample. The weights are used to simulate a *pseudo-population* that represents the samples that would have been observed if everyone in the sample had experienced both disability and no disability, to estimate a counterfactual outcome for each individual in the sample. As such, the exposure groups become exchangeable conditional on all measured potential confounders [213].

3.7.3 Estimation of the propensity score

As defined above, the propensity score represents the probability that an individual is exposed conditional on their baseline characteristic. Propensity scores can be estimated in different ways. In the analysis which used a propensity score approach in this thesis, I chose to estimate propensity scores by fitting a logistic regression model for disability acquisition including baseline covariates as explanatory variables to derive the predicted probability of disability acquisition (i.e. estimated propensity score) for each individual. The propensity score was generated using variables measured prior to each individual's first report of disability, to ensure that the groups were exchangeable prior to reporting a disability, so that any differences in mental health between the groups could be attributable to the effect of disability acquisition. There has been substantial debate in the literature about which variables to include to best estimate the propensity score. It has been argued that the propensity score should include all potential confounders as well as variables that are predictors of the outcome, but not the exposure, which increases precision without introducing bias [219]. In my analysis, I included variables that were considered to be potential confounders as well as variables that were predictors of the outcome (Figure 3.4).

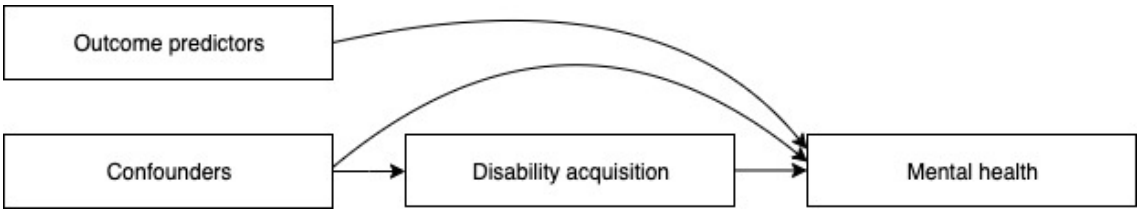

Figure 3.4 Simplified causal diagram demonstrating variables contributing to the propensity score

Propensity scores can also be estimated using machine learning methods, addressing limitations of logistic regression models including potential for model misspecification and restrictions to statistical modelling assumptions [220]. Machine learning approaches have been suggested as alternatives to overcome some of these issues, especially when there are a large number of covariates [221], because they are model-free and non-parametric [220]. Machine learning methods include a range of different statistical approaches that use classification and prediction algorithms to predict outcomes based on a set of covariates. Whilst logistic regression techniques estimate parameters based on an a priori specified data model, machine learning techniques do not make any assumptions about the data mechanism, they use trends in the data to predict outcomes using a learning algorithm, rather than estimating parameters [221]. Boosted regression trees, which partition the data into a number of different groups, have been proposed as an alternative to logistic regression to estimate propensity scores, particularly in models with a large number of covariates, because they may be less sensitive to model misspecification and problems of collinearity [222, 223]. I also estimated the propensity score using boosted regression trees, however this did not materially affect results of the analysis, therefore logistic regression was used to generate propensity scores in the analysis.

3.7.4 Estimation of inverse probability weights

Inverse probability weights were estimated for each individual in the sample from their propensity scores. Exposed individuals were allocated a weight of $\frac{1}{propensity\ score}$ and the unexposed a weight of $\frac{1}{1-propensity\ score}$. Very large weights and extreme outliers can arise which disproportionately influence results, therefore weights were trimmed at the 99th percentile [224].

3.7.5 Diagnostics

Diagnostics can be performed to assess balance between the groups. The distribution of the estimated propensity scores can be compared between the exposed and unexposed groups in the unweighted and weighted samples to ensure overlap between

the distributions, testing the assumption of positivity has been achieved by the inverse probability weights.

To verify whether the assumption of exchangeability is met, that confounding variables are balanced between the exposure groups, standardised differences between exposed and unexposed groups for each covariate can be calculated in the weighted sample. Standardised differences represent the difference in means between the comparison groups in units of the pooled standard deviation, therefore they are not influenced by sample size and can be used to compare the relative balance of covariates measured using different units [225]. Standardised differences of less than 10% are considered to reflect good balance of confounders [225, 226].

3.7.6 Estimation of the average causal effect

Propensity score methods estimate marginal effects, or average causal effects, which represent the population-average causal effect, i.e. the difference between two outcomes: the mean outcome if everyone in the population was exposed and the mean outcome if everyone in the population was unexposed [215]. The analysis of the main effect was estimated by fitting a linear regression model for the outcome (mental health) with the exposure (disability acquisition) as the only independent variable, including the inverse probability weights as probability weights. The average causal effect is estimated by fitting a regression model for the outcome with the exposure as the only independent variable and the weights included as probability weights.

3.8 Fixed effects analysis

3.8.1 Why use fixed effects models?

As described previously, observational data are prone to bias from confounding. This is particularly problematic in studies which aim to estimate causal effects of disability because people with and without disabilities have very different characteristics, and these differences may lead to bias in the effect estimates.

Longitudinal data with repeated measures on the same individuals over time can be used to examine how changes in exposures relate to changes in outcomes. One analytic method to analyse longitudinal data which is used extensively in econometrics, known as fixed effects analysis, can reduce the impact of some types of confounding and improve causal estimation [227, 228].

Fixed effects models are regression models which aim to estimate the causal effect of an exposure on an outcome by modelling the within-person variation in exposures and

outcomes. In its simplest form, fixed effects regression estimates the difference in a person's outcome between two time points, one in which they were exposed and one in which they were unexposed. As such the effect of the exposure on the outcome can be estimated for each person in the sample, which can be averaged across the whole sample to get an estimate of the population-average causal effect. This approach can be extended to multiple waves of data, comparing mean outcome measurements in waves in which a person was exposed and waves in which they were unexposed.

Each individual acts as their own control, thereby controlling for characteristics of the individual which do not vary with time [229]. Because they estimate within-person effects, fixed effects models implicitly eliminate confounding by both observed and unobserved time-invariant variables, by removing the time invariant variation between individuals [230]. However, fixed effects models do not control for confounding by time-varying variables, which need to be included as covariates in the regression model.

This is particularly attractive in the field of social epidemiology (and in studies estimating causal effects of disability) where exposure-outcome relationships are likely to be confounded by a large number of different individual factors, such as demographic, socio-economic, and behavioural characteristics, and contextual factors, such as socio-economic, cultural and environmental conditions [231], many of which will be stable over time.

3.8.2 Estimating fixed effects regression models

In order to estimate within-person effects, variation between individuals is removed by mean centring all time-varying variables so that they are expressed as deviations from the individual-specific means. Therefore, all individuals in the analysis have the same mean values for each time-varying variable contributing to the analysis (exposure, outcome and covariates), which eliminates between-person variance and time-invariant confounding. As such, effect estimates represent within-person effects.

Fixed effects models can be estimated in Stata using the *xtreg* command with the *fe* option, without the need to mean centre each variable in the analysis because Stata does this by default.

3.8.3 Limitations of fixed effects models

The main advantage of fixed effects models is that only within-individual variation is modelled, thereby removing confounding from time-invariant characteristics. However, this also poses a limitation and can lead to imprecise estimates. As the regression parameters are generated from information on changes within individuals, it is necessary

to check that there is enough variation within-person in the exposure and outcome variables over time in the sample, or the parameter estimates will be generated from a very small sample size. This is of less concern for a continuous variable such as mental health (as opposed to a binary variable), which is likely to display greater variation within-individuals over time.

Furthermore, because regression coefficients in fixed effects models are generated from changes in exposures and outcomes within-individuals, as well as impacting on the precision of estimates, there are also consequences to the generalisability of the estimates. The effect estimates from fixed effects models can only be generalised to those people in the population who experience a change in the exposure. The models do not tell us anything about the effect of the exposure on the outcome for people who do not change exposure status, such as people with permanent disability.

Another limitation is that the effect of time-invariant characteristics cannot be estimated [230]. For example, when interested in the effect of changing disability status on mental health, no regression coefficient will be estimated for the effect of ethnicity. It is possible however, to examine the interaction between disability and ethnicity on the outcome, to understand how the effect of disability on mental health differs for people of different ethnicities.

Finally, the models assume that the effect of the exposure on the outcome is contemporaneous as they are measured in the same wave. However, it may be that the effects of disability on mental health persist or change over time, which cannot be captured in fixed effects models. Because the exposure and outcome are measured in the same wave, there is also the risk of reverse causation, whereby the outcome may be causing the exposure, which cannot be disentangled in fixed-effects models. More complex dynamic fixed-effects models and cross-lagged fixed-effects models which can incorporate time-lagged variables may be necessary to capture these effects [230, 232].

3.9 Quantitative bias analysis

3.9.1 Why use quantitative bias analysis?

Though analytic approaches aiming to minimise bias from confounding from measured variables are commonly used, little attention is paid to bias from unmeasured confounding. One approach that can be used is quantitative bias analysis. Quantitative bias analysis aims to quantify the error in the estimation of the effect of an exposure on

an outcome to understand how much the estimated effect may differ from the true effect [233].

In the analyses in this thesis, the problem of unmeasured confounding became particularly apparent in the sequential causal mediation analysis (described in section 5.1). Causal mediation analysis methods estimating natural direct and indirect effects rely on four strong assumptions about no unmeasured confounding to have a causal interpretation. Though measures were taken to adjust for exposure-outcome and exposure-mediator confounding, there was potential for residual confounding of the mediator-outcome relationship (see Figure 3.5).

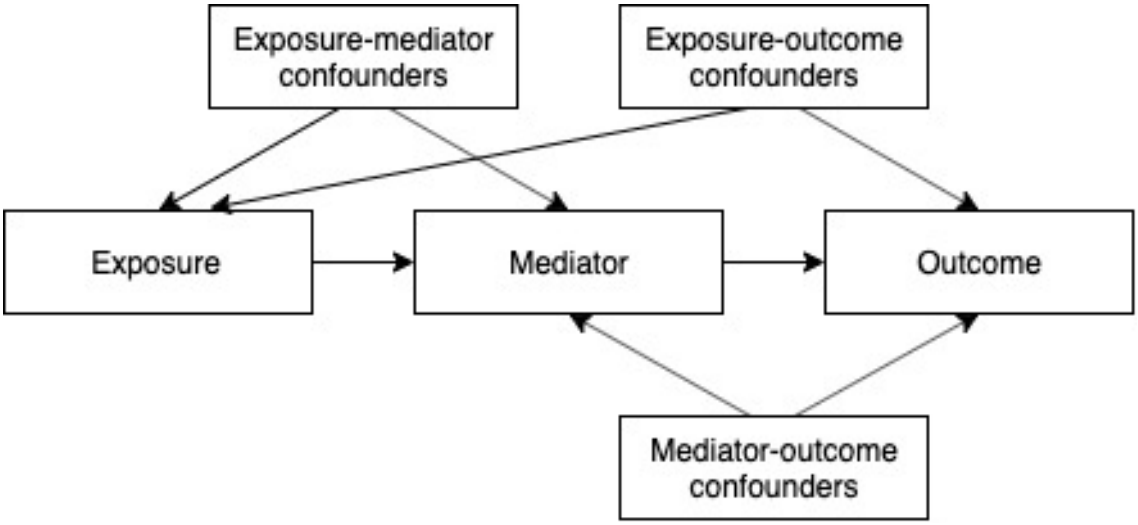

Figure 3.5 Simplified causal diagram illustrating exposure-outcome, exposure-mediator and mediator-outcome confounding

3.9.2 Quantitative bias for estimation of natural direct and indirect effects

The problem of unmeasured mediator-outcome confounding in the estimation of natural direct and indirect effects can be addressed by performing a quantitative bias analysis which assesses the sensitivity of the results to the assumption of no unmeasured confounding of the mediator-outcome association as proposed by VanderWeele (2010) [234]. The quantitative bias analysis involves estimating the effect of a potential unmeasured binary confounder of the association between the mediator and the outcome on the natural direct and indirect effects.

This is achieved by estimating a bias parameter for the NDE and the NIE. The bias parameter was estimated by positing a range of plausible values for two of the parameters, *gamma* and *delta*. Gamma (γ) measures the magnitude of the combined effect of the unmeasured confounder U on both the mediator and the outcome, and delta

(δ) measures the difference in the prevalence of U between people who were exposed and unexposed.

$$Bias\ (NDE) = \delta\gamma$$

$$Bias\ (NIE) = -\delta\gamma$$

The estimates of the natural direct and indirect effects can then be recalculated according to different scenarios (different magnitudes of the bias parameter) to assess how strong the potential confounding would have to be to eliminate the natural direct and indirect effect, and whether this is likely to happen within the range of plausible values.

3.10 Multiple imputation

3.10.1 Why use multiple imputation?

Missing data are common and often inadequately handled, leading to potential selection bias in the estimated effects. Multiple imputation is a statistical approach used to minimise selection bias from missing data and may increase precision of estimates. It creates a number of imputed datasets in which missing observations are replaced with plausible values, then estimates the effect of interest in each imputed dataset, followed by calculation of an average estimate across the imputed datasets and a corresponding standard error which accommodates the within- and between-imputation variance using Rubin's Rules [235, 236].

3.10.2 Missing data mechanisms

Missing data can arise for different reasons. The missingness mechanism determines the analytic strategy that should be used to deal with the missing data [237, 238], however it is not always possible to know the mechanism explaining the missing data.

Data are missing completely at random (MCAR) if the probability of a variable being missing does not depend on the values of observed or missing data. If data are missing completely at random, then complete case analyses will produce unbiased estimates of effect (though less precise).

Data are missing at random (MAR) if the probability of a variable being missing does not depend on the values of missing data, conditional on the observed data. Complete case analyses are likely to produce biased estimates of effect, therefore other analytic strategies are needed to account for the missing data, such as multiple imputation and inverse probability weighting.

Data are missing not at random (MNAR) if the probability of a variable being missing depends on the values of missing data, even after conditioning on the observed data [237, 238]. If the missing data are MNAR, multiple imputation is likely to produce biased estimates and other methods are required such as sensitivity analyses to assess the impact of departures from the MNAR assumption on the results. Of note, it is not possible to assess from the data, whether the missing data are MAR or MNAR.

3.10.3 Multiple imputation analysis

Because of the large amount of missing data in the HILDA Survey, multiple imputation was performed to deal with the missing data in all analyses. A comparison of measured characteristics between participants with and without missing data in the sample suggested that the data were not MCAR but it was plausible that the data were MAR. Therefore, multiple imputation was considered to be an appropriate analytic method to maximise the validity of the findings.

Multiple imputation imputes missing observations by drawing from the posterior predictive distribution of the unobserved data given the observed data. The imputed datasets are created using chained equations, in which univariate imputation models are specified for each variable with missing data, and the model cycles through each variable with missing data in turn (*mi impute chained* command in Stata). All variables included in the epidemiological analysis should be included in the imputation model, as well as auxiliary variables - additional variables which are associated with the variable with missing data or are predictive of the missingness of that variable.

The target analysis can then be conducted on each of the imputed datasets, and the estimates are combined using Rubin's Rules, which additionally takes into account the variability between the estimates from the imputed datasets, to provide an overall estimate and standard error [236].

3.10.4 Combining multiple imputation and inverse probability weighting

There has been considerable debate in the literature about how to combine inverse probability weighting with multiple imputation, primarily concerning whether the propensity score analysis should be performed *within* each imputed dataset and then combined, or whether the propensity scores should be combined *across* the imputed datasets. Simulation studies have provided evidence that the *within* approach minimises bias [239, 240]. Therefore in the analysis using inverse probability weighting, propensity scores and inverse probability weights were generated within each imputed dataset and estimates for the effect of interest were then combined using Rubin's Rules.

3.10.5 Combining multiple imputation and causal mediation analysis

To conduct the causal mediation analyses using multiple imputation, the imputed datasets were created and the causal mediation analysis was performed on each imputed dataset. Standard errors for the estimates in each imputed dataset were generated using bootstrapping with 200 replications. The mean of the estimates from each imputed dataset was calculated to give an overall estimate of the NDE, NIE and TCE. Standard errors were derived using Rubin's Rules for combining the between-imputation and within-imputation variance (using the bootstrap standard error for each imputed dataset).

3.11 Conclusion

The counterfactual approach to causal inference has been used throughout this thesis to provide a theoretical framework and a set of analytic tools to best answer the aims and objectives of the thesis. In order to generate the most causally-robust evidence and address the methodological challenges described above, I use longitudinal data to characterise changes in people's circumstances over time and apply innovative statistical methods to conduct analyses informed by causal diagrams to minimise the effect of potential biases from confounding, information bias and selection bias.

Furthermore, because of the complexity of the causal relationships linking disability and poor mental health, I have chosen to use epidemiological methods that seek to explain the mechanisms by which disability leads to poor mental health to add to the evidence as to *how* and *why* people with disability experience large mental health inequalities. A better understanding of the mechanistic pathways improves causal inference but also generates policy-relevant evidence, providing insight into the effect of potential interventions on mental health inequalities and the circumstances under which they will work [183]. The strong focus on causal inference will ensure that the findings of the research can be translated into effective policy interventions that will improve mental health and reduce inequalities.

Chapter 4 Effect modification

4.1 Disability acquisition and mental health: effect modification by a broad range of demographic and socio-economic characteristics

4.1.1 Introduction

In this first chapter of results, I present the findings of an analysis which examines a wide range of different demographic and socio-economic characteristics to determine whether they influence the magnitude of the effect of disability acquisition on mental health. Understanding how the effect of disability acquisition is differential across population subgroups is important in identifying vulnerable subgroups of people with disabilities who are likely to experience greater mental health declines, and therefore may benefit most from targeted social and health policies interventions. Differential effects also provide insight into the mechanism by which disability acquisition leads to a decline in mental health, identifying contributing factors that influence the magnitude of the effect, which can shed light on the complex ways in which disability and contributing factors interact to affect poor mental health.

The analysis uses a propensity score approach with inverse probability weighting. As well as providing an alternative to multivariable regression for controlling for confounding that has been argued to better achieve exchangeability of comparison groups, propensity score methods can explicitly assess whether the assumptions of exchangeability and positivity– two of the key assumptions for causal inference – have been met.

4.1.2 Disability acquisition and mental health: an analysis of excess mental health inequalities according to demographic and socio-economic characteristics using propensity score models with inverse probability of treatment weighting

This section consists of the following article [1]:

Aitken Z, Simpson JA, Bentley R, Kavanagh AM. *Disability acquisition and mental health: effect modification by demographic and socioeconomic characteristics using data from an Australian longitudinal study.* BMJ Open, 2017. 7(9): e016953.

Introduction

Currently, one in five Australians has a disability [241]. People with disabilities experience substantial health inequalities compared to those without disabilities, reporting poorer health across a wide range of indicators including mental health [83, 84]. For example, in Australia, a large survey found that 48% of people reporting severe

disabilities experienced mental health problems compared to 6% of those without disabilities [81].

Evidence from longitudinal studies has demonstrated that disability acquisition is associated with a deterioration in mental health [83, 84, 101, 116, 124, 126, 129, 141], suggesting a causal relationship between disability and poor mental health. However, not all people who acquire a disability experience a decline in their mental health [102], there is substantial heterogeneity in the magnitude of the association. Understanding this heterogeneity may be aetiologically informative and may shed light on the structural drivers of the inequalities [242, 243]. Intersectionality theory has been posited as a way to understand how social identities (e.g. gender) and positions (e.g. socio-economic disadvantage) interact to shape people's experiences [244]. This approach suggests that social determinants of health cannot be understood independently; rather the focus should be on understanding how factors intersect and mutually reinforce each other in their health impacts [245]. Bauer argues that intersectionality theory can be operationalised in epidemiological studies by fitting interactions between different exposure variables [244]. In this study, we explore how demographic, social and economic factors interact with disability to modify its effect on mental health.

There is limited evidence about how the association between disability and mental health varies according to demographic and socioeconomic characteristics. Four longitudinal studies have examined whether the association between disability acquisition and mental health differs according to socioeconomic characteristics. Two studies of working aged Australians found evidence that the association varied according to housing characteristics and wealth; the largest mental health declines were seen for people in unaffordable and insecure housing [83] and with low wealth [84]. Similarly, a study of people approaching retirement age in the United States found that the negative effect of disability on subjective wellbeing was greater in individuals with low wealth prior to disability acquisition [129]. Finally, a study of individuals aged 33 years from the United Kingdom found larger effects of disability acquisition on psychological distress for people with low education [126]. None of the studies comprehensively examined a range of factors that influence the effect of disability acquisition on mental health to fully understand important demographic and socioeconomic determinants, and their relative importance.

A better understanding of the characteristics that determine the magnitude of the mental health effects for people who acquire a disability will provide information that can be used to inform the development of targeted social and health policies for people with disabilities most likely to experience poorer mental health. Such interventions are likely

to improve the mental health of people with disabilities, but may also have implications for long term health and welfare costs associated with disability. This analysis uses data from a longitudinal study to model relationships between disability acquisition and mental health, testing for effect modification by demographic and socioeconomic characteristics prior to disability and quantifying excess mental health effects associated with these characteristics.

Methods

Data source

The Household, Income and Labour Dynamics in Australia (HILDA) Survey is a longitudinal study of Australian households, conducted annually since 2001. The survey collects information about the demographic, social, economic, and health characteristics of individuals using a combination of interviews and self-completion questionnaires. Data are collected on all household members, with interviews conducted with those aged 15 years and older. The original sample included 13 969 participants from 7682 households, randomly sampled using a national probability sample of private dwellings. In later waves, continuing survey members included all participants from the original sample, any children born or adopted in the household and new partners. Additional households were added in later waves to maintain representativeness, with a sample size after 14 waves of 28 794 people. Response rates were above 70% for new participants and above 90% for continuing respondents. Full details of HILDA are available elsewhere [246].

Disability acquisition

Information on disability was collected from participants in every wave, defined in HILDA as “a long-term health condition, impairment or disability that restricts you in your everyday activities, and has lasted or is likely to last for six months or more”. If participants reported a disability, they were then asked to describe the type of impairment, such as limited use of fingers or arms, or problems with eyesight that could not be corrected with glasses or contact lenses. Disability was not disaggregated into different impairment types because we lacked power to examine differences by disability characteristics but also to make the results relevant to disability policies which generally do not address different types of impairments specifically. The analysis was restricted to the most recent four waves of the survey (2011 to 2014) to enable selection of comparable groups to maximise exchangeability. Participants were included in the analysis if they were disability-free for the first two consecutive waves of the analysis followed by either two consecutive waves of disability (disability group) or two

consecutive waves of no disability (control group). We used two consecutive waves of disability so as to exclude people with transient disability and to reduce the potential for measurement error. Eligibility for inclusion required participation in all four waves and response to the disability question at every wave.

Mental health

The Short Form 36 is a widely-used general health questionnaire that has been validated for use in the Australian population [168]. It comprises eight subscales measuring various components of health and two overall summary measures. The Mental Health Inventory (MHI), one of the subscales, is comprised of five items relating to mental health over the previous four weeks, specifically measuring symptoms of depression, anxiety and psychological wellbeing. Each item is scored using five response categories, and the total scores are transformed into a scale ranging from 0 to 100, with higher scores reflecting better mental health.

Covariates

Covariates were measured in the first wave contributing to the analysis, as a measure of circumstances prior to disability acquisition. Demographic characteristics consisted of age, sex and country of birth. Socioeconomic characteristics included education, employment, income (population quintiles of equivalised household disposable income), financial hardship, housing tenure, housing affordability (unaffordable defined as households in the lowest 40% of the income distribution with housing costs exceeding 30% of their gross income), relationship status, and children. Social support was constructed using the average of 10 items addressing aspects of emotional support (rated on a 7-point Likert scale) [247], categorised into tertiles. Wealth was defined as household assets minus debt and categorised into tertiles, recorded in 2014 as wealth questions were not asked in 2011. Lifestyle factors included alcohol consumption, smoking, physical activity and self-reported Body Mass Index (BMI). Baseline mental health was included as a covariate. Specific categories of each variable are described in Table 1.

Propensity score approach

Propensity score methods provide an alternative method to adjust for confounding compared to traditional regression models and are particularly useful in analyses with many potential confounders [212, 213]. We estimated a propensity score for each individual by fitting a logistic regression model for disability acquisition including all covariates (potential confounders and predictors of mental health)(see Figure 4.1) [219]. We employed Inverse Probability Weighting (IPW), which uses propensity scores to

create a weight for each individual [158]. The distribution of the weights was examined using boxplots (Figure 4.4 in supplementary file 1). Some very large weights resulted from people with disabilities with very low propensity scores, therefore we trimmed weights at the 99th percentile [224].

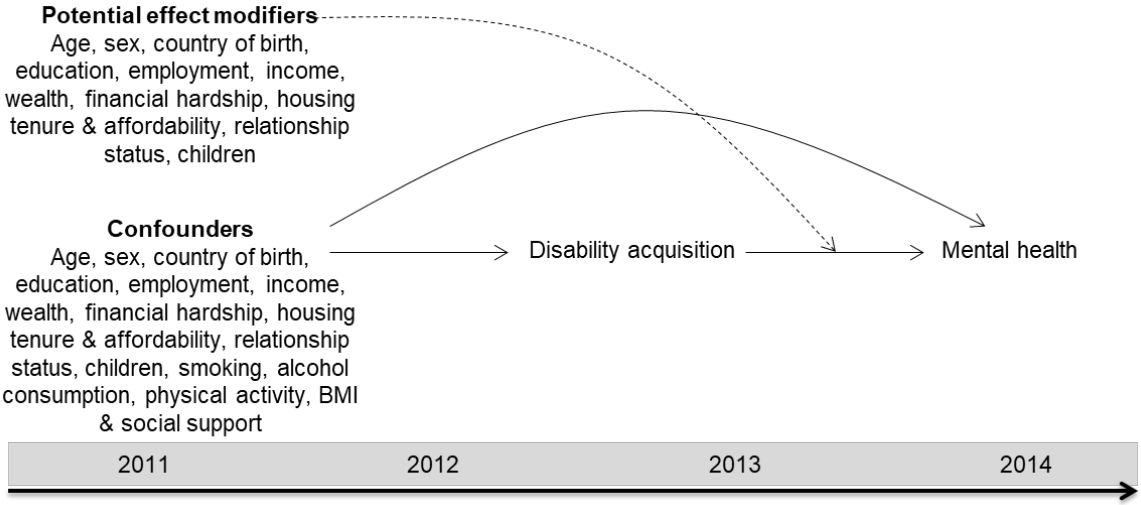

Figure 4.1 Causal diagram depicting potential confounders and effect modifiers of the association between disability acquisition and mental health

The average causal effect (ACE) which represents the mean causal effect for all individuals in the population was estimated from a linear regression model with IPW as probability weights, using robust estimators given that the weights are estimated [248]. To verify whether balance of the confounding variables was achieved, percentage standardised differences between the groups were calculated for each covariate, with differences less than 10% considered to reflect good balance [226].

Statistical analysis

Descriptive analyses were conducted, summarising mental health for people who acquired a disability and those who did not, as well as demographic, socioeconomic and mental health characteristics prior to disability acquisition.

The ACE of disability acquisition on mental health at the final wave was estimated using linear regression models for mental health with disability acquisition as the sole independent variable with IPW weights. We tested for additive effect modification by demographic and socioeconomic factors identified a priori that were thought to show evidence for effect heterogeneity based on substantive knowledge, including age, sex, country of birth, education, employment, income, wealth, financial hardship, housing tenure, housing affordability, relationship status and children. We quantified excess effects associated with each characteristic, which is the additional difference in mental

health which exceeds the sum of each factor considered separately, and the effect of disability acquisition within each stratum of the effect modifier. We included an interaction term between disability acquisition and each effect modifier in turn, and tested for effect modification using likelihood ratio tests and Wald tests. Analyses were conducted using Stata/SE 12.1 [249].

Missing data

Investigation and handling of missing data was conducted for people who were eligible for inclusion in the sample, therefore people who were lost to follow up were excluded from the analyses. The distribution of baseline covariates was compared between those with and without missing observations. Missingness was associated with the values of measured variables, suggesting that the data were not missing completely at random. Therefore, multiple imputation using chained equations with 50 imputations was employed to maximise the validity of the findings as this approach assumes the data are missing at random, that is, systematic differences between missing and observed values are explained by differences in observed data [235]. The multiple imputation model included all variables in the target analysis as well as additional auxiliary variables (further details in supplementary file 2).

Sensitivity analyses

A series of sensitivity analyses were conducted to test the robustness of findings. Firstly, we repeated analyses using alternative propensity score approaches (stratification and covariate adjustment). Secondly, we conducted a complete case analysis. Thirdly, we excluded people who acquired psychological impairments, as we would expect them to have poorer mental health associated with disability.

Results

A total of 8323 individuals were eligible for inclusion in the analysis, 387 with disability and 7936 controls. Complete data were available for 6461 participants (78%); Figure 4.2 describes eligibility criteria and missing data. Data were missing for mental health, financial hardship, housing tenure and affordability, relationship status, social support, alcohol consumption, smoking, physical activity and BMI. People with missing data had poorer mental health and were more likely to experience socioeconomic disadvantage across all indicators (Table 4.4 in supplementary file 2).

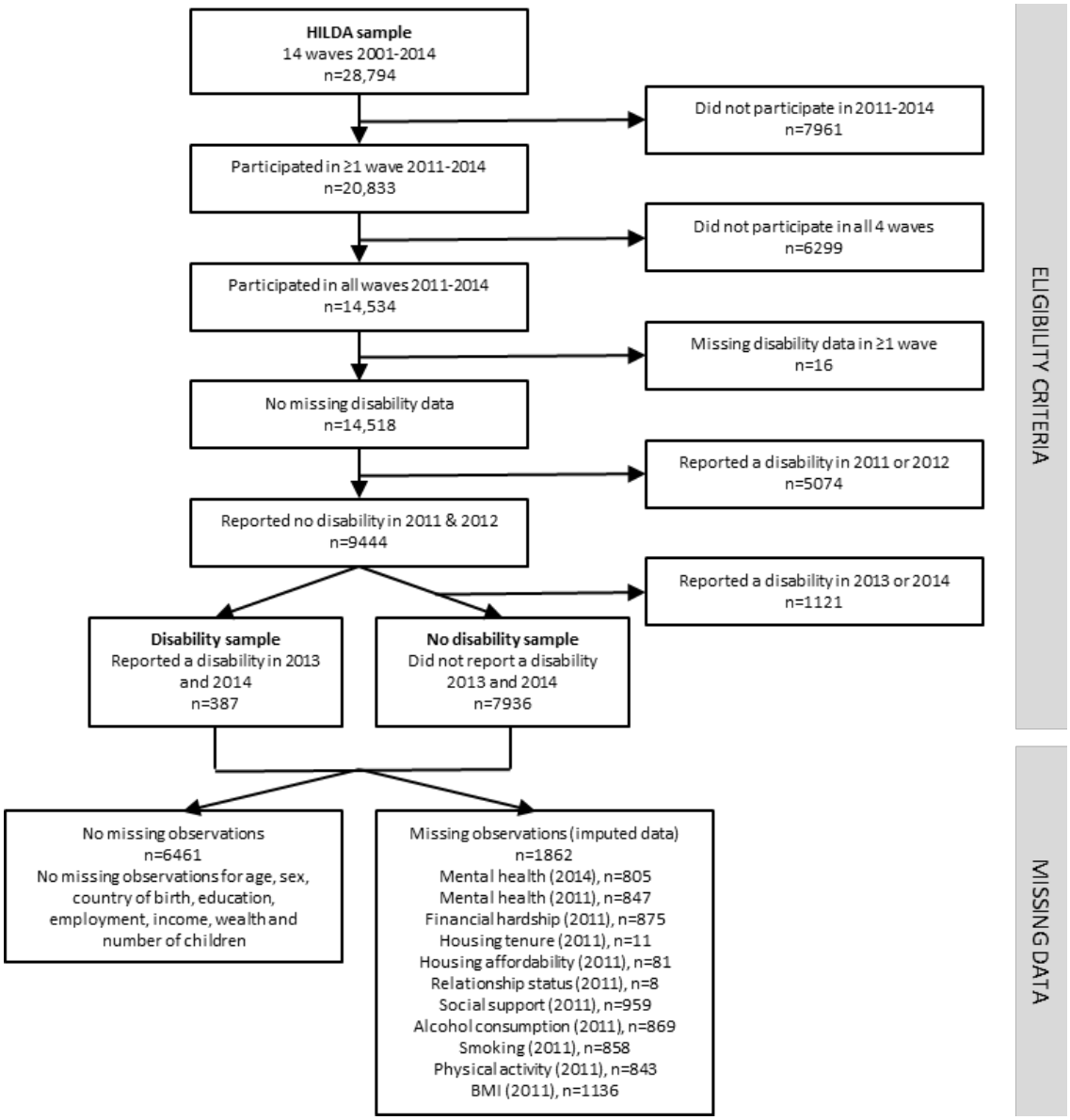

Figure 4.2 Flow diagram showing sample selection and missing data

There were differences in the distribution of covariates at baseline between people who acquired a disability and those who did not (Table 4.1). People who acquired a disability were older and more likely to be male and born outside of Australia. They experienced greater socioeconomic disadvantage including low education, employment rates, income, wealth and social support, greater financial hardship, higher rates of smoking, less physical activity and higher BMI, though were similar with regards to housing tenure, affordability and alcohol consumption. Those with disabilities were more likely to be in a relationship and have children. The groups differed in terms of mental health, both before and after disability. At the final wave, those with disabilities had poorer mental health compared to those without (70.9 compared to 77.6). But even at baseline, those in the

disability sample had poorer mental health compared to people who did not acquire a disability, with mean MHI scores of 73.8 compared to 77.9 (Table 4.1).

Table 4.1 Demographic, socioeconomic and mental health characteristics of people with and without disabilities, Australia, 2011-2014

	Disability n=311		No disability n=6150	
	n	%	n	%
Age				
<30	43	13.8	1901	30.9
30-44	66	21.2	1957	31.8
45-69	101	32.5	1568	25.5
60+	101	32.5	724	11.8
Sex				
Men	160	51.4	2928	47.6
Women	151	48.6	3222	52.4
Country of birth				
Australia	241	77.5	4862	79.1
Other	70	22.5	1288	20.9
Education				
Bachelor or higher	56	18	1820	29.6
Secondary, certificate, diploma	149	47.9	2902	47.2
Did not complete secondary	106	34.1	1428	23.2
Employment				
Employed	193	62.1	4810	78.2
Unemployed	9	2.9	176	2.9
Not in the labour force	109	35	1164	18.9
Income				
Q5 (highest)	56	18	1662	27
Q4	62	19.9	1495	24.3
Q3	68	21.9	1252	20.4
Q2	62	19.9	1170	19
Q1 (lowest)	63	20.3	571	9.3
Wealth				
High	114	36.7	2497	40.6
Medium	106	34.1	2052	33.4
Low	91	29.3	1601	26
Financial hardship				
Prosperous/very comfortable	39	12.5	1278	20.8
Reasonably comfortable	170	54.7	3386	55.1
Just getting by/very poor	102	32.8	1486	24.2
Housing tenure				
Outright owner	120	38.6	1773	28.8
Mortgager	107	34.4	2683	43.6
Private renter	67	21.5	1483	24.1
Public renter	7	2.3	80	1.3
Other	10	3.2	131	2.1
Housing affordability				
Affordable	289	92.9	5731	93.2
Unaffordable	22	7.1	419	6.8
Relationship				
Yes	229	73.6	4204	68.4
No	82	26.4	1946	31.6

Children				
No	82	26.4	2396	39
Yes	229	73.6	3754	61
Alcohol consumption				
Never	53	17	920	15
Rarely	115	37	2171	35.3
1-2 times/week	52	16.7	1340	21.8
≥3 times/week	91	29.3	1719	28
Smoking				
Never smoked	146	46.9	3676	59.8
Ex-smoker	102	32.8	1483	24.1
Current	63	20.3	991	16.1
Physical activity				
≥4 times/week	110	35.4	2294	37.3
1-3 times/week	122	39.2	2612	42.5
<1 time/week	79	25.4	1244	20.2
Mental health (mean (SD))	311	70.9 (19.2)	6150	77.6 (14.9)
Mental health at baseline (mean (SD))	311	73.8 (18.4)	6150	77.9 (14.3)
BMI (mean (SD))	311	27.4 (5.3)	6150	25.8 (5.0)
Social support (mean (SD))	311	5.3 (1.1)	6150	5.6 (1.0)

These differences highlighted the need for a propensity score approach to ensure adequate control for confounding. The estimated propensity score model indicated that the strongest predictors of disability acquisition were age, education, unemployment, wealth, relationship status and mental health at baseline. The distribution of propensity scores differed between people with and without disabilities, but the IPW balanced the distribution between the groups (Figure 4.5 in supplementary file 3).

The IPW performed well at balancing the baseline covariates, with standardised differences between the groups much lower in the weighted sample. After IPW, the groups were comparable in terms of mental health at baseline, with MHI scores of 77.0 for people with disability and 77.7 for those without disability, a standardised difference of 4% compared to 25% in the unweighted sample. The groups also became more comparable in terms of all other baseline covariates, with standardised differences less than 10% for most variables, except age, children, smoking status and BMI, which were all under 17% (Figure 4.3 and Table 4.6 in supplementary file 4).

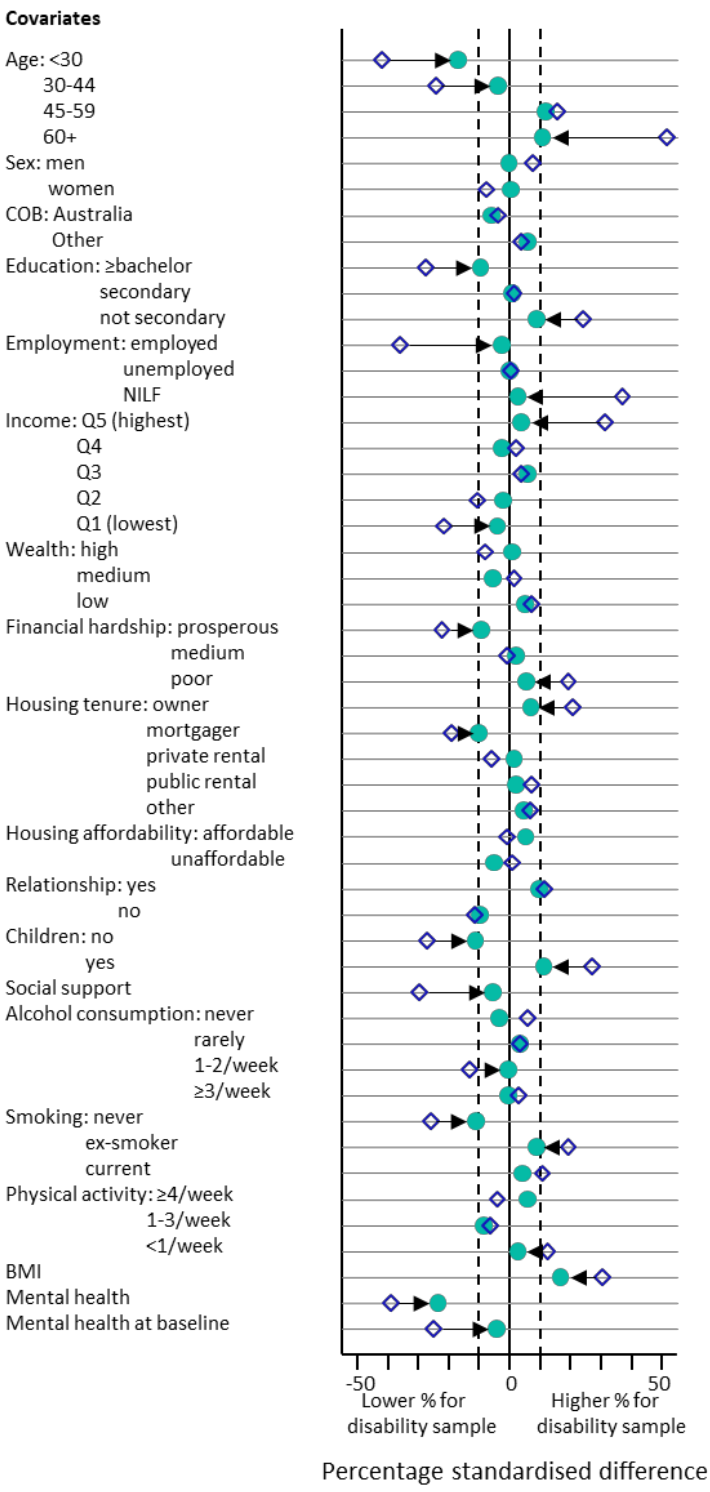

Figure 4.3 Percentage standardised differences between people with and without disabilities for each covariate before and after IPW on the propensity score (unweighted sample ◇ ; IPW weighted sample: ●) ; the dashed lines indicate the 10% differences which reflect good balance of confounders.

The ACE of disability acquisition on mental health was estimated to be a five-point decline in MHI score (estimated mean difference: -5.1, 95% CI -7.2, -3.0). The joint

effects of disability and socioeconomic characteristics were largest for people who acquired a disability who were public renters (-23.2, 95% CI -33.8, -12.6), unemployed (-17.2, 95% CI -30.7, -3.7), had low income (-15.0, 95% CI -20.9, -9.1), poor wealth (-13.2, 95% CI -17.3, -9.1), experienced financial headship (-12.5, 85% CI -16.1, -8.8), were in unaffordable housing (-13.1, 95% CI -22.0, -4.3), and those in the younger age groups (<30: -15.5, 95% CI -20.0, -10.9; 30-44: -14.3, 95% CI -18.9, -9.8) (Table 4.2, column 1).

Table 4.2 Regression coefficients representing joint and components effects of disability and each socioeconomic factor on the mental health score (n=8323)

	Joint effect [a]		Effect of disability[b]		Effect of socioeconomic factors[c]		Excess effect[d]		Test for interaction
Socioeconomic characteristics	Coeff	95% CI	Coeff	95% CI	Coeff	95% CI	Coeff	95% CI	P value
Age (years)									
<30	-15.5	-20.0, -10.9	-6.6	-11.1, -2.1	-8.9	-10.0, -7.8	-2.1	-7.5, 3.4	
30-44	-14.3	-18.9, -9.8	-7.0	-11.5, -2.5	-7.3	-8.4, -6.2	-2.5	-7.9, 2.9	
45-69	-9.2	-12.6, -5.9	-4.1	-7.4, -0.8	-5.1	-6.3, -4.0	0.4	-4.0, 4.9	
60+	-4.5	-7.5, -1.6	-4.5	-7.5, -1.6	0		0		p=0.663
Sex									
Men	-5.4	-8.5, -2.3	-5.4	-8.5, -2.3	0		0		
Women	-6.1	-8.9, -3.2	-4.9	-7.8, -2.1	-1.2	-1.9, -2.3	0.4	-3.8, 4.7	p=0.836
Country of birth									
Australia	-5.8	-8.2, -3.4	-5.8	-8.2, -3.4	0		0		
Other	-3.5	-7.6, 0.7	-2.9	-7.1, 1.3	-0.5	-1.4, 0.3	2.9	-1.9, 7.7	p=0.240
Education									
Bachelor or higher	-3.6	-7.9, -0.8	-3.6	-7.9, -0.8	0		0		
Secondary, certificate, diploma	-5.6	-8.6, -2.6	-4.9	-7.9, -1.9	-0.7	-1.5, 0.1	-1.3	-6.6, 4.0	
Did not complete secondary	-8.3	-12.1, -4.5	-6.6	-10.5, -2.8	-1.7	-2.6, -0.7	-3.1	-8.9, 2.8	p=0.581
Employment									
Employed	-4.7	-7.2, -2.3	-4.7	-7.2, -2.3	0		0		
Unemployed	-17.2	-30.7, -3.7	-10.9	-24.5, 2.8	-6.3	-8.6, -4.1	-6.1	-20.0, 7.7	
Not in the labour force	-5.4	-9.1, -1.7	-5.9	-9.6, -2.1	0.4	-0.5, 1.3	-1.1	-5.6, 3.4	p=0.629
Income									
Q5 (highest)	-1.1	-4.9, 2.7	-1.1	-4.9, 2.7	0		0		
Q4	-4.6	-9.0, -0.3	-4.1	-8.5, 0.2	-0.5	-1.5, 0.4	-3.1	-8.9, 2.7	
Q3	-6.1	-10.1, -2.2	-4.5	-8.6, -0.5	-1.6	-2.6, -0.6	-3.5	-9.0, 2.1	
Q2	-8.5	-13.6, -3.3	-6.3	-11.4, -1.1	-2.2	-3.2, -1.2	-5.2	-11.6, 1.2	
Q1 (lowest)	-15.0	-20.9, -9.1	-12.5	-18.5, -6.5	-2.5	-3.9, -1.2	-11.4	-18.5, -4.3	p=0.034
Wealth									

High	-4.3	-7.5, -1.0	-4.3	-7.5, -1.0	0		0		
Medium	-6.3	-9.5, -3.0	-4.0	-7.3, -0.7	-2.3	-3.0, -1.5	0.2	-4.4, 4.9	
Low	-13.2	-17.3, -9.1	-7.4	-11.6, -3.3	-5.8	-6.7, -4.9	-3.2	-8.4, 2.1	p=0.388
Financial hardship									
Prosperous/very comfortable	-3.4	-9.4, 2.6	-3.4	-9.4, 2.6	0		0		
Reasonably comfortable	-6.5	-9.5, -3.6	-4.8	-7.6, -1.9	-1.8	-2.7, -0.9	-1.4	-8.2, 5.3	
Just getting by/very poor	-12.5	-16.1, -8.8	-6.3	-10.0, -2.7	-6.1	-7.2, -5.1	-3.0	-10.0, 4.1	p=0.675
Housing tenure									
Outright owner	-5.2	-8.8, -1.5	-5.2	-8.8, -1.5	0		0		
Mortgager	-7.6	-11.0, -4.2	-5.2	-8.6, -1.8	-2.5	-3.3, -1.6	0.0	-5.0, 5.0	
Private renter	-9.9	-14.2, -5.6	-4.6	-8.9, -0.3	-5.3	-6.3, -4.3	-0.6	-5.0, 6.2	
Public renter	-23.2	-33.8, -12.6	-14.8	-25.8, -3.7	-8.4	-11.6, 5.2	-9.6	-21.3, 2.1	
Other	-10.7	-21.5, 0.1	-6.9	-18.0, 4.1	-3.8	-6.1, -1.4	-1.8	-13.4, 9.9	p=0.500
Housing affordability									
Affordable	-4.7	-6.9, -2.6	-4.7	-6.9, -2.6	0		0		
Unaffordable	-13.1	-22.0, -4.3	-10.0	-18.9, -1.0	-3.2	-4.6, -1.7	-5.2	-14.4, 4.0	p=0.265
Relationship									
Yes	-3.7	-6.1, -1.4	-3.7	-6.1, -1.4	0		0		
No	-11.5	-15.5, -7.4	-8.8	-12.9, -4.8	-2.6	-3.4, -1.9	-5.1	-9.8, -0.4	p=0.033
Children									
Yes	-5.4	-8.0, -2.9	-5.4	-8.0, -2.9	0		0		
No	-7.7	-11.3, -4.1	-5.0	-8.6, -1.4	-2.7	-3.4, -2.0	0.4	-4.0, 4.9	p=0.851

[a]The coefficient represents the joint effect of disability and each category of the covariate on mean mental health score, compared to those with no disability in the baseline socioeconomic category

[b]The coefficient represents the effect of disability on mean mental health score, compared to those with no disability, within each category of the covariate

[c]The coefficient represents the effect of each socioeconomic factor on mean mental health score, compared to baseline socioeconomic category, for people with no disability

[d]The excess effect is the value of the interaction term on an additive scale, which represents the additional effect of disability and each socioeconomic factor on mean mental health score

There was strong evidence from the tests for interaction of an excess effect between disability and income and disability and relationship status on mental health (Table 4.2, column 5), with excess effects of more than five points for people who were not in a relationship (-5.1, 95% CI -9.8, -0.4) and more than 11 points for those in the lowest income category (-11.4, 95% CI -18.5, -4.3) (Table 4.2, column 4). As such, the effects of disability on mental health were more detrimental among people with lower income compared to higher income (Q1: -12.5, 95% CI -18.5, -6.5; Q5: -1.1, 95% CI –4.9, 2.7) and people who were not in a relationship (-8.8, 95% CI -12.9, -4.8) compared to those who were (-3.7, 95% CI -6.1, -1.4) (table 4.2, column 2). There were also large excess effects for other socioeconomic characteristics, with particularly large effects for those unemployed, with low wealth, experiencing financial hardship, unaffordable housing and for public renters, suggesting that these characteristics also modified the effect of disability acquisition on mental health, though the interaction terms were not statistically significant.

The first sensitivity analysis using alternative propensity score approaches did not materially change the results, suggesting that models with IPW were robust. In the second sensitivity analysis, the complete case analysis, despite only small changes in the magnitude of individual coefficients, the gradients in the excess effects across categories of socioeconomic characteristics were more pronounced, and as a result there was additional evidence for effect modification for education, wealth, housing tenure and housing affordability. The third sensitivity analysis excluding people with psychological impairments reduced the number of people acquiring a disability from 387 to 316. For most covariates, the effect estimates between disability acquisition and mental health were slightly attenuated and the confidence intervals were wider (and no longer statistically significant), however the patterns of association were similar. A notable exception was the difference in the magnitude of effect according to relationship status, for which an excess effect was no longer apparent.

Discussion

This is the first study to examine a broad range of socioeconomic and demographic characteristics to understand the relative importance of socioeconomic influences on the effect of disability acquisition in adulthood on mental health. Our finding that disability interacts with socioeconomic characteristics to shape mental health underlines the importance of considering intersectionality in studies of disability and health inequalities [244]. There was strong evidence that the effect was greater for people on low income and those not in a relationship, and there were also large differences in the magnitude of the effect according to employment status, wealth, financial hardship, housing tenure

and housing affordability, though the interaction terms were not statistically significant. The results were similar to other studies, finding some evidence of differences in the effect by wealth [84, 129], housing tenure and affordability [83], though effect modification by education was less pronounced than in other studies [126].

In this study, people who acquired a disability experienced on average a 5-point decline in mental health, substantially exceeding a three-point difference considered to represent a clinical meaningful change [169]. Importantly, the results suggest that the mental health effects are heterogeneous, with some people experiencing much larger mental health declines, for example a 12.5 point decline for those in the lowest income quintile.

This study benefited from a number of strengths. The longitudinal nature of the data enabled identification of incident cases of disability and characteristics prior to disability. We used IPW weighting to control for confounding, to ensure that the groups were comparable prior to disability acquisition, strengthening our ability to interpret estimates as causal effects of disability on mental health. We trimmed weights at the 99th percentile to avoid large weights leading to inflated standard errors, resulting in poorer balance between the groups, with standardised differences of four covariates ranging from 10% to 17%. While this is a limitation, we believed this was preferable to extreme weights. Finally, results from other propensity score approaches including covariate adjustment and stratification were similar, suggesting that the IPW models were robust.

Our analysis has a number of limitations. We tested for effect modification across many variables, which increased the risk of type I error. However the variables were selected a priori and all associations were in the expected direction, therefore unlikely to have arisen by chance. Additionally, only two of the variables showed statistical evidence of effect modification, despite large differences in the magnitude of the effect estimates. Though we had a large sample and a continuous outcome, the power to detect interactions was low because only 5% of study participants acquired a disability. Missing data may have introduced selection bias. The complete case analysis suggested effect modification by a greater number of socioeconomic characteristics compared to results using multiple imputation. This was explained by the relatively poorer mental health of disabled individuals with high socioeconomic status who had missing data compared to those with complete data, which led to an overestimation of gradients in the complete case analysis. However multiple imputation is likely to have corrected for this selection bias since the method requires the less stringent assumption of missing at random (i.e. missingness depends on measured covariates as observed in our study), whereas the complete case analysis assumes the participants with missing data represent a random sample of those that were intended to be observed (missing completely at random). We

did not account for survey weights, which may affect estimated standard errors, however adjustment for the survey weights in conventional linear regression models did not substantially change the results. Finally, there was potential for dependent misclassification bias, where misclassification of the outcome depends on misclassification of the exposure, because both disability and mental health were self-reported.

Though the patterns of association were generally similar, there were some differences in the results of the sensitivity analysis excluding people with psychological impairments, particularly the attenuation of effect modification by relationship status. One assumption underpinning the propensity score approach is the no-multiple-versions-of-treatment assumption, which stipulates that the potential outcomes under each level of the exposure are well defined and take on a single value [250]. It is possible that the effect of disability on mental health may vary according to the "version" of the exposure, such as different types or severity of disability, however we lacked power to examine differences by disability characteristics. Therefore estimated effects should be interpreted as average causal effects of disability on mental health, which may affect the transportability of results as the estimated effect depends on the distribution of disability characteristics in the sample [251]. People with severe disabilities are less likely to participate in HILDA, therefore our results are likely to underestimate the population effect of disability acquisition on mental health.

The finding that the effect of disability acquisition on mental health depends on people's socioeconomic characteristics has important implications for disability, social and public health policies. People who are socioeconomically disadvantaged (particularly those with low income and not in a relationship) are a vulnerable subgroup of people with disabilities who are likely to experience greater mental health effects, and therefore may benefit most from targeted social and health policies interventions. Therefore, addressing the social determinants of health of people with disabilities, such as interventions that improve people's financial situation, for example investing in employment and education services for people with disabilities, may have substantial beneficial impacts on mental health. Such interventions have implications for the mental health and wellbeing of people with disabilities and their families, as well as for long term health and welfare costs. It is also important that high quality mental health services are accessible and affordable for people who acquire a disability particularly if they are socioeconomic disadvantaged or not in a relationship.

4.1.3 Supplementary material for section 4.1.2

Supplementary file 1. Distribution of inverse probability weights

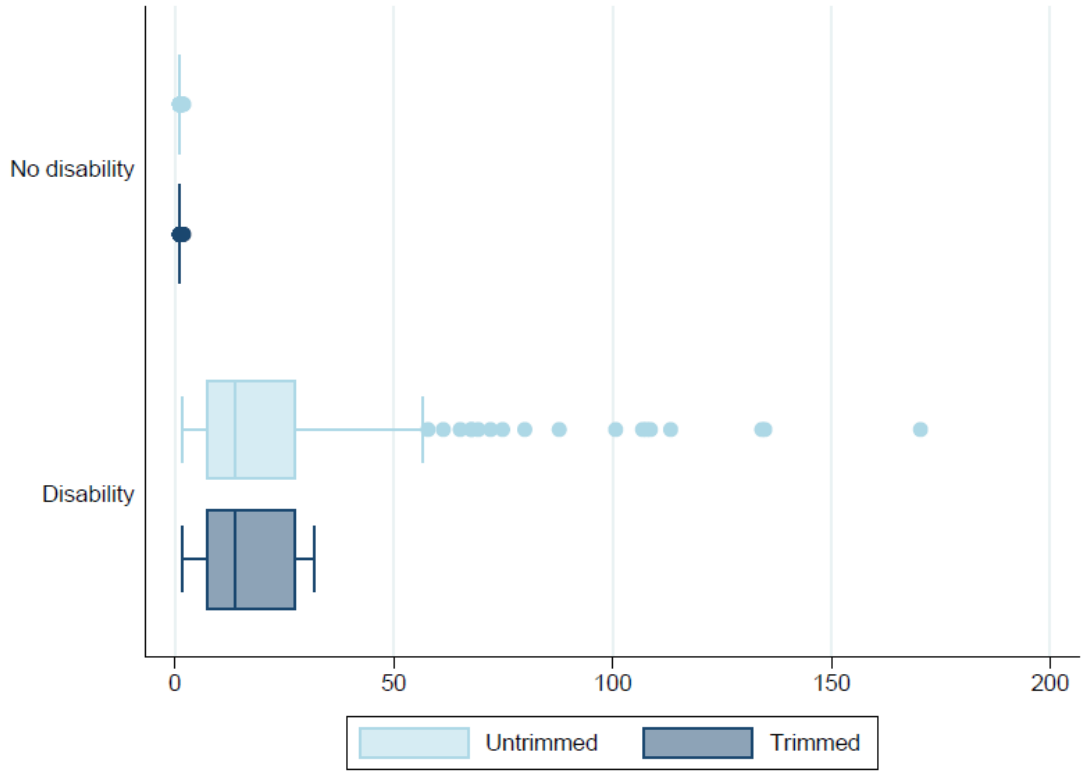

Figure 4.4 Box plot displaying the distribution of non-trimmed and trimmed inverse probability of treatment weights, for people with and without disabilities, displaying the median (vertical line), the 25th and 75th percentiles (box), and outliers (dots)

Supplementary file 2. Description of missing data and multiple imputation

Missing data

There were missing observations for mental health in 2014 and at baseline, financial hardship, housing tenure, housing affordability, relationship status, social support, alcohol consumption, smoking, physical activity and BMI (see Table 4.3).

Table 4.3 Missing observations for each variable in the analysis

		Missing	
	Wave	n	%
Mental health	2014	805	8.5%
Mental health	2011	847	9.0%
Disability acquisition	2011-2014	0	0.0%
Age	2011	0	0.0%
Sex	2011	0	0.0%
Country of birth	2011	0	0.0%
Education	2011	0	0.0%
Employment	2011	0	0.0%
Income	2011	0	0.0%
Wealth	2014	0	0.0%
Financial hardship	2011	875	9.3%
Housing tenure	2011	11	0.1%
Housing affordability	2011	81	0.9%
Relationship status	2011	8	0.1%
Children	2011	0	0.0%
Social support	2011	959	10.2%
Alcohol consumption	2011	869	9.2%
Smoking	2011	858	9.1%
Physical activity	2011	843	8.9%
BMI	2011	1,136	12.0%

There was evidence that people with missing observations were systematically different from those without disabilities (see Table 4.4). They had poorer mental health and experienced greater socioeconomic disadvantage across every covariate examined but had similar demographic characteristics. Therefore, we concluded that the data were not missing completely at random, but that it was plausible that the data may be missing at random (that the probability of missingness was conditional on the observed data but not the missing data).

Table 4.4 Comparison of fully observed variables between people with complete (n=6461) and incomplete data (n=1862), Australia, 2011-2014

Covariate	Complete data		Incomplete data		
	n	mean (SD)	n[a]	mean (SD)	p value
Mental health (2014)	6461	77.3 (15.2)	1057	74.9 (16.4)	p<0.001
Mental health (2011)	6461	77.7 (14.5)	1015	75.2 (15.5)	p<0.001
Social support	6461	5.6 (1.0)	903	5.5 (1.0)	p=0.004
BMI	6461	25.9 (5.0)	726	25.6 (5.2)	p=0.078
	n	%	n	%	p value
Disability acquisition					
Disability	311	4.8	76	4.1	
No disability	6150	95.2	1786	95.9	p=0.186
Age					
<30	1944	30.1	885	47.5	
30-44	2023	31.3	522	28.0	
45-69	1669	25.8	332	17.8	
60+	825	12.8	123	6.6	p=0.327
Sex					
Men	3088	47.8	922	49.5	
Women	3373	52.2	940	50.5	p=0.190
Country of birth					
Australia	5103	79.0	1451	77.9	
Other	1358	21.0	411	22.1	p=0.327
Education					
Bachelor or higher	1876	29.0	374	20.1	
Secondary, certificate, diploma	3051	47.2	917	49.3	
Did not complete secondary	1534	23.7	571	30.7	p<0.001
Employment					
Employed	5003	77.4	1371	73.6	
Unemployed	185	2.9	97	5.2	
Not in the labour force	1273	19.7	394	21.2	p<0.001
Income					
Q5 (highest)	634	26.6	339	18.6	
Q4	1232	24.1	405	21.6	
Q3	1320	20.4	369	19.8	
Q2	1557	19.1	402	21.8	
Q1 (lowest)	1718	9.8	347	18.2	p<0.001
Wealth					
High	2611	40.4	479	25.7	
Medium	2158	33.4	588	31.6	
Low	1692	26.2	795	42.7	p<0.001
Financial hardship					
Prosperous/very comfortable	1317	20.4	169	17.1	
Reasonably comfortable	3556	55.0	506	51.3	
Just getting by/very poor	1588	24.6	312	31.6	p<0.001
Housing tenure					
Outright owner	1893	29.3	362	19.6	
Mortgager	2790	43.2	729	39.4	
Private renter	1550	24.0	654	35.3	
Public renter	87	1.4	61	3.3	
Other	141	2.2	45	2.4	p<0.001
Housing affordability					
Affordable	6020	93.2	1591	89.3	

Unaffordable	441	6.8	190	10.7	p<0.001
Relationship					
Yes	4433	68.6	1016	54.8	
No	2028	31.4	838	45.2	p<0.001
Children					
Yes	3983	61.7	1011	54.3	
No	2478	38.4	851	45.7	p<0.001
Alcohol consumption					
Never	973	15.1	194	19.5	
Rarely	2286	35.4	383	38.6	
1-2 times/week	1392	21.5	217	21.9	
≥3 times/week	1810	28.0	199	20.0	p<0.001
Smoking					
Never smoked	3822	59.2	552	55.0	
Ex-smoker	1585	24.5	210	20.9	
Current	1054	16.3	242	24.1	p<0.001
Physical activity					
≥4 times/week	2404	37.2	354	34.7	
1-3 times/week	2734	42.3	401	39.4	
<1 time/week	1323	20.5	264	25.9	p<0.001

[a]the number of observations for each covariate for people with incomplete data (i.e. participants with missing observations for at least one variable in the analysis for whom data were available for each specific covariate)

Multiple imputation

Missing data may lead to biased estimates of effect if participants with complete data differ from those with missing data and excluding participants with any missing data from the analysis reduces the statistical power [235]. Therefore, we conducted multiple imputation to reduce bias and potentially increase the power and precision of the analysis.

We created 50 imputed datasets using chained equations, in which univariate imputation models were specified for each variable with missing data, and the model cycled through each variable with missing data in turn (*mi impute chained* command in Stata). All variables included in the epidemiological analysis were included in the imputation model, as well as auxiliary variables, additional variables which were associated with the variable with missing data or predicted missingness of that variable. For each variable with missing data, the auxiliary variables included in the imputation model are listed in Table S2.3. The target analysis was conducted on each of the 50 imputed datasets, and the estimates were combined using Rubin's rules to provide an overall estimate with a standard error, which takes into account the variability between the estimates from the imputed datasets [236].

Table 4.5 Variables included in the imputation model for each covariate with missing data

Covariate	Wave	Variables in the imputation model
Mental health Alcohol consumption Smoking Physical activity BMI	2014 2011 2011 2011 2011	All variables in the epidemiological analysis; mental health (2010, 2012, 2013), self-rated health (2011, 2014); prior socio-economic characteristics (2010): education, employment, financial hardship, housing tenure, housing affordability, relationship status; parental occupation (2011); lifestyle factors (2014): alcohol consumption, smoking, physical activity, BMI; Socio-Economic Indexes For Areas (SEIFA) index of relative socio-economic disadvantage score (2011)
Financial hardship Housing tenure Housing affordability Relationship status Social support Mental health	2011 2011 2011 2011 2011 2011	All variables in the epidemiological analysis; mental health (2010, 2012, 2013), self-rated health (2011, 2014); prior socio-economic characteristics (2010): education, employment, financial hardship, housing tenure, housing affordability, relationship status; parental occupation (2011); SEIFA index of relative socio-economic disadvantage score (2011)

Supplementary file 3. Propensity score distribution

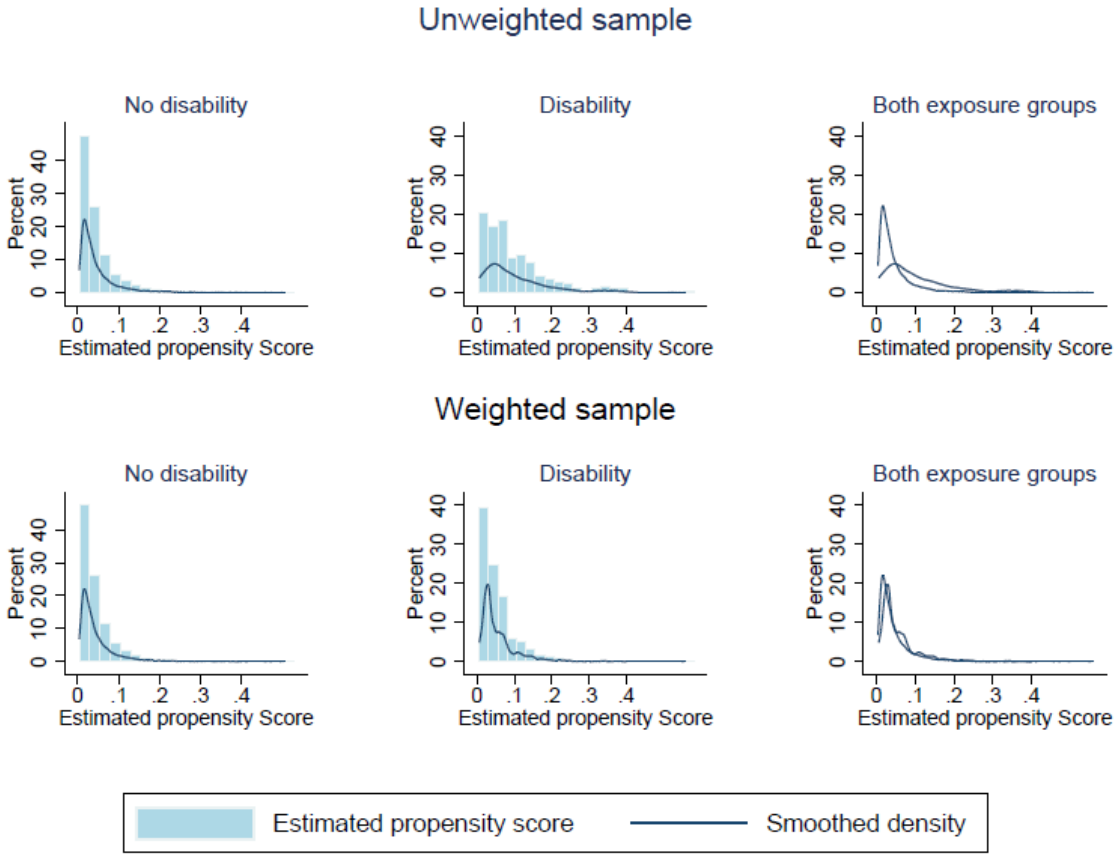

Figure 4.5 Propensity score distribution in the whole sample and the weighted sample

Supplementary file 4. Standardised differences in baseline covariates between people with and without disabilities

Table 4.6 Standardised differences between people with and without disabilities in the unweighted and weighted samples, for each covariate

	Unweighted sample			Weighted sample[a]		
	Disability n=311 %	No disability n=6150 %	Standardised difference %	Disability n=311 %	No disability n=6150 %	Standardised difference %
Age						
<30	13.8	30.9	-41.9	22.7	30.1	-16.9
30-44	21.2	31.8	-24.2	29.6	31.3	-3.8
45-69	32.5	25.5	15.4	31.2	25.8	11.9
60+	32.5	11.8	51.5	16.6	12.8	10.8
Sex						
Men	51.4	47.6	7.7	47.7	47.8	-0.2
Women	48.6	52.4	-7.7	52.3	52.2	0.2
Country of birth						
Australia	77.5	79.1	-3.8	76.5	79.0	-5.9
Other	22.5	20.9	3.8	23.5	21.0	5.9
Education						
Bachelor or higher	18.0	29.6	-27.5	24.8	29.0	-9.6
Secondary, certificate, diploma	47.9	47.2	1.4	47.6	47.2	0.8
Did not complete secondary	34.1	23.2	24.2	27.6	23.8	8.8
Employment						
Employed	62.1	78.2	-35.9	76.4	77.4	-2.6
Unemployed	2.9	2.9	0.2	2.9	2.9	-0.1
Not in the labour force	35.0	18.9	36.9	20.8	19.7	2.7
Income						
Q5 (highest)	20.3	9.3	31.3	10.9	9.8	3.7
Q4	19.9	19.0	2.3	18.1	19.1	-2.6
Q3	21.9	20.4	3.7	22.9	20.4	5.9
Q2	19.9	24.3	-10.6	23.3	24.1	-1.9
Q1 (lowest)	18.0	270.	-21.7	24.9	26.6	-3.9

Wealth						
High	36.7	40.6	-8.1	40.9	40.4	0.9
Medium	34.1	33.4	1.5	30.8	33.4	-5.6
Low	29.3	26.0	7.2	28.3	26.2	4.9
Financial hardship						
Prosperous/very comfortable	12.5	20.8	-22.3	16.8	20.4	-9.3
Reasonably comfortable	54.7	55.1	-0.8	56.2	55.1	2.3
Just getting by/very poor	32.8	24.2	19.2	27.0	24.6	5.6
Housing tenure						
Outright owner	38.6	28.8	20.7	32.6	29.3	7.1
Mortgager	34.4	43.6	-19.0	38.3	43.2	-9.9
Private renter	21.5	24.1	-6.1	24.6	24.0	1.5
Public renter	2.3	1.3	7.2	1.6	1.4	1.9
Other	3.2	2.1	6.7	2.9	2.2	4.4
Housing affordability						
Affordable	92.9	93.2	-1.0	94.5	93.2	5.3
Unaffordable	7.1	6.8	1.0	5.5	6.8	-5.3
Relationship						
Yes	73.6	68.4	11.6	73.0	68.6	9.7
No	26.4	31.6	-11.6	27.0	31.4	-9.7
Children						
No	26.4	39.0	-27.1	33.0	38.4	-11.2
Yes	73.6	61.0	27.1	67.0	61.6	11.2
Alcohol consumption						
Never	17.0	15.0	5.7	13.8	15.0	-3.6
Rarely	37.0	35.3	3.5	37.1	35.4	3.4
1-2 times/week	16.7	21.8	-12.9	21.4	21.6	-0.4
≥3 times/week	29.3	28.0	2.9	27.8	28.0	-0.5
Smoking						
Never smoked	46.9	59.8	-25.9	53.7	59.1	-11.0
Ex-smoker	32.8	24.1	19.3	28.4	24.5	8.8
Current	20.3	16.1	10.8	17.9	16.3	4.2
Physical activity						

≥4 times/week	35.4	37.3	-4.0	40.2	37.2	6.1
1-3 times/week	39.2	42.5	-6.6	38.2	42.3	-8.4
<1 time/week	25.4	20.2	12.4	21.6	20.5	2.8
	Mean (SD)	Mean (SD)	%	Mean (SD)	Mean (SD)	%
Mental health	70.9 (19.2)	77.6 (14.9)	-39.0	73.5 (5.9)	77.5 (19.5)	-23.4
Mental health at baseline	73.8 (18.4)	77.9 (14.3)	-25.0	77.0 (5.3)	77.7 (18.8)	-4.3
BMI	27.4 (5.3)	25.8 (5.0)	30.6	26.8 (1.7)	25.9 (6.6)	16.8
Social support	5.3 (1.1)	5.6 (1.0)	-29.7	5.6 (1.2)	5.6 (0.3)	-5.5

[a]The estimated propensity scores were used to create inverse probability of treatment weights for each individual in the sample. Some very large weights resulted from people with disabilities with very low propensity scores, therefore weights were trimmed at the 99th percentile of the distribution.

4.2 Disability acquisition and mental health: effect modification by employment characteristics

4.2.1 Introduction

In my first analysis of effect modification, I examined a wide range of socio-economic characteristics to examine whether these influenced the magnitude of the effect of disability acquisition on mental health. The results of this study suggested that, despite only income and relationship status demonstrating statistically significant differences in the size of the estimated effects, socio-economic variables generally influenced the magnitude of the mental health decline associated with disability acquisition, such that people who experienced socio-economic disadvantage experienced larger deteriorations in mental health after acquiring a disability. Therefore, the findings suggest that socio-economic characteristics are important contributing factors that interact with disability to influence effect on mental health.

With regards to employment, these results suggested that employment status modified the magnitude of the effect of disability acquisition on mental health, with an average mental health decline of 4.7-points for people who were in employment compared to 10.9-points for those who were unemployed and 5.9-points for those who were not in the labour force. The mental health decline was more than double the magnitude for those who were unemployed compared to the employed. However, surprisingly the test for interaction did not imply that there was a difference in the magnitude of the effect estimates. This is likely due to low power of interaction tests to detect effects but could also be due to bias in the estimates due to confounding.

In this section, I present the results of a further analysis in which I investigate effect modification by employment in more detail, further disaggregating the employment variable to better understand how characteristics of people's employment may influence the effect of disability on mental health. Using fixed-effects models to estimate the within-person effects of disability on mental health, thus better controlling from confounding by time-invariant characteristics, the analysis compares the magnitude of the effect of disability acquisition on mental health for people employed in different occupation types (low, medium and high skilled occupations) and different contract types (permanent, fixed-term, casual and self-employed) and well as those who were unemployed and those not in the labour force.

4.2.2 Does the effect of disability acquisition on mental health differ by employment characteristics? A longitudinal fixed-effects analysis

This section consists of the following article [2]:

Aitken Z, Simpson JA, Bentley R, Milner A, LaMontagne AD, Kavanagh AM. *Does the effect of disability acquisition on mental health differ by employment characteristics? A longitudinal fixed-effects analysis.* Social Psychiatry and Psychiatric Epidemiology, published online 24 October 2019.

Introduction

Globally, it is estimated that 15% of the population currently live with a disability [25]. People with disabilities experience large mental health inequalities compared to those without disability [81, 101, 252]. There is evidence from longitudinal studies that disability acquisition leads to deterioration in people's mental health, suggesting the existence of a causal relationship between disability and poor mental health [3, 83, 84, 117, 121, 126, 129]. Notably, there is a great deal of heterogeneity in the magnitude of this effect; some people experience substantial mental health declines whereas others experience little or no decline [102].

According to the International Classification of Functioning, Disability and Health (ICF) framework, disability results from the interaction between people's health conditions, individual characteristics and social factors [62]. Conceived in this way, social factors and socio-economic inequalities are likely to affect how a health condition or impairment impacts on people's functioning and restrictions to participation in society. This highlights the importance of examining the interaction between disability and social factors when examining mental health effects associated with disability acquisition.

Previous research has suggested that socioeconomic disadvantage exacerbates the negative effect of disability acquisition on mental health. Six longitudinal studies have examined whether the association between disability acquisition and mental health differs according to people's socioeconomic characteristics prior to disability, and found evidence that the association varied by income [1], housing characteristics [83], social support [3], relationship status [1], education [126], and wealth [84, 129]. Importantly, these results suggest that favourable socioeconomic circumstances may provide a buffer against the detrimental effect of disability on mental health. The evidence about how employment characteristics affect the relationship between disability and mental health is sparse. An Australian study found larger negative effects for people who were unemployed prior to disability, though the study did not have the statistical power to detect an interaction [1].

This is an important gap in the literature. Evidence about the psychosocial benefits of employment on mental health in the general population, such as the positive impacts of high psychosocial job quality on mental health [253, 254] and the negative effects

associated with transitions from employment to unemployment [255], have led us to hypothesize that characteristics of people's employment may attenuate the effect of disability on people's mental health. Casual or temporary employment has been hypothesised to adversely affect mental health [256], but previous studies in the Australian general working population have been null [257]. Nevertheless, casual employment—due to its lack of security, paid sick leave, and other adverse working conditions—could be more important for people with disability and could modify the disability acquisition-mental health relationship. A better understanding of the effects of employment characteristics may identify subgroups of people who are particularly vulnerable to large mental health declines associated with disability acquisition and may give insight into the mechanisms linking disability and mental health [176], which could inform the development of targeted social and health policies.

This analysis uses data from a large Australian longitudinal study to model relationships between disability acquisition and mental health in working age individuals, testing how employment characteristics and disability acquisition combine to influence mental health. We test for effect modification by employment characteristics prior to disability acquisition using two characteristics of people's employment, occupational skill level and contract type, to quantify excess mental health effects of disability acquisition associated with these characteristics.

Methods

Data source

We used data from 17 waves of the Household, Income and Labour Dynamics in Australia (HILDA) Survey, a nationally representative longitudinal study of Australian households and individuals which collects information annually on a wide range of social, demographic, health and economic characteristics [258]. The original panel was selected in 2001 and included 13,969 individuals from 7682 households, sampled using a national probability sample of private dwellings. In subsequent waves, survey members included all original participants, household members attaining the age of 15 years, and new participants when existing participants formed new households. A top up sample was added in 2011 to maintain representativeness. At each wave, data were collected on each household member, and face-to-face interviews were sought from all household members aged 15 years or above. The response rate for participation in the survey was 80% and attrition between waves was about 6%. We used data from individuals aged 25 to 64 years to represent the working age population.

Mental health

Mental health was measured in every wave using the Mental Health Inventory (MHI), one of the eight subscales of the Short Form 36 (SF-36) health questionnaire. The SF-36 is a widely used self-completion measure of health status that has been validated for use in the Australian population and to detect within-person changes in health over time [169]. The mental health subscale assesses symptoms of anxiety, depression and positive aspects of mental health. It has been shown to be psychometrically sound [171], and an effective screening tool for mood and anxiety disorders and severe depressive symptomatology [172-175, 259-262]. It includes five questions relating to mental health over the previous four weeks, each scored using five response categories, which are summed and rescaled into a continuous measure ranging from 0 to 100. Higher scores represent better mental health.

Disability acquisition

Information on disabilities was collected in every wave. Participants were asked if they had an "impairment, long-term health condition or disability which restricts their everyday activities that had lasted, or was likely to last, for a period of six months or more". Participants were defined as having acquired a disability if they reported, in consecutive years, two waves with no disability followed immediately by two waves with a disability. A minimum of two consecutive waves was used so as to identify people with relatively longer-term disability [120, 263, 264]. All consecutive waves in which individuals reported a disability subsequent to disability acquisition were also included in analyses as well as all consecutive waves reporting no disability prior to disability acquisition (minimum of four, maximum of 17 consecutive waves contributed for each person). If participants reported more than one episode of disability acquisition, only the first episode was included.

Employment characteristics

Occupational skill level was defined using the Australian and New Zealand Standard Classification of Occupations, a skill-based classification of occupations. Information on occupation was combined into a measure with three mutually exclusive categories: high skill jobs (managers; professionals); medium skill jobs (technicians and trades workers; community and personal service workers; clerical and administrative workers); and low skill jobs (sales workers, machinery operators and drivers, labourers). Contract typewas categorised as permanent (ongoing) employment; fixed term contract; self-employed (employee of one's own business or self-employed); and casual (in a casual or temporary contract or employed through a labour-hire firm or temporary employment agency). For

each variable, we generated response categories to additionally specify people who were not in the labour force (not actively seeking employment, for various reasons including education, retirement, infirmity/disability, or household duties) and unemployed (actively seeking employment or unable to find work in the last four weeks). To represent employment characteristics prior to disability acquisition, variables were recorded as time-invariant, measured two waves prior to the first year of reported disability.

Missing data

We used multiple imputation (MI) using chained equations with 50 imputations to maximise the validity of findings (note, <0.1% missing data for disability so these individuals were excluded). This approach assumes that the missing data were missing at random, that the systematic differences in the distribution of missing and observed variables are explained by differences in observed data [235]. The imputation model included all variables in the fixed-effects analysis as well as additional auxiliary variables including self-rated health, education, household income, relationship status and children. To account for potentially important interactions in the analysis model (i.e. ensure congeniality between the analysis and imputation model), we imputed missing values separately for waves with and without disability to account for interactions [265].

Statistical analysis

The characteristics of the sample with complete data were described at baseline (participants' first wave contributing to the sample) and employment characteristics were summarised two waves prior to the first year of reported disability. Mean mental health scores for waves with and without disability were compared across categories of employment characteristics. For each individual, mean mental health was calculated for waves in which they reported a disability and waves in which they reported no disability (within-person mean mental health), and these were then pooled to summarise the mean mental health scores for the sample (between-persons).

We used fixed-effects longitudinal linear regression models to estimate the association between disability acquisition and mental health score, using the imputed data. Regression coefficients from the models describe within-person estimated mean differences in mental health scores between waves in which they reported a disability and waves in which they reported no disability. Fixed-effects models estimate changes in outcomes associated with changes in exposure status within individuals, rather than between individuals, therefore controlling for individual-level factors that do vary over time. In this way, the models remove bias from time-invariant confounding from both measured and unobserved variables [230]. Covariates were included in regression

models if they were deemed to be potential time-varying confounders, i.e., common causes of both disability acquisition and mental health. We considered a single covariate, age measured as a continuous variable (see Figure 4.6). Though income, household structure and relationship status could be conceived as potential confounders, they were not included as covariates in models as they were likely to be affected by disability acquisition and therefore be potential mediators of the association. Including mediators as covariates in the models would bias the effect estimates by adjusting for variables on the causal pathway between the exposure and outcome.

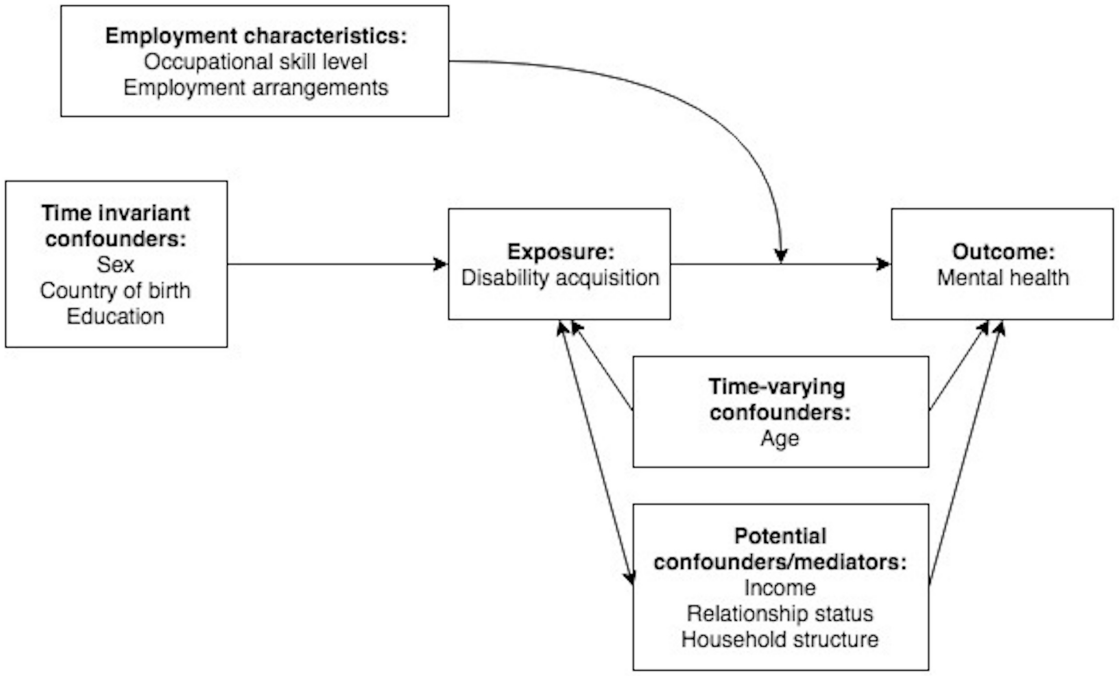

Figure 4.6. Causal diagram illustrating postulated causal relationships between disability acquisition and mental health

To assess whether the association between disability acquisition and mental health varied by prior employment characteristics, we included an interaction term between disability acquisition and employment characteristics, with separate models for each employment characteristic. We assessed whether there was statistical evidence of effect modification on the additive scale using Wald tests assessing whether the interaction term coefficients were different to zero. Analyses were conducted in Stata/SE 15 [249], using the *mi estimate* and *xtreg* commands with fixed-effects estimators and robust standard errors to fit regression models. A complete case analysis on the sample of people with complete data on all variables was carried out as a comparison to the primary analysis using MI.

Sensitivity analysis

We conducted two sensitivity analyses to test the robustness of our findings. Firstly, we excluded people acquiring disability relating to a psychological impairment as the mental health effect and the influence of employment characteristics may differ for this subgroup. Secondly, we restricted the waves of data contributing to the sample to the two waves following disability acquisition, because some people may have only two waves of data prior or post disability, whereas other may have up to 15 and the fixed effects models assume that a contemporaneous effect that does not change over time.

Results

Across the 17 waves of data, there were 2096 individuals of working age who met our definition of disability acquisition contributing a total of 16,949 observations, with a mean number of eight observations per person (four preceding disability and four subsequent to disability on average). Complete data were available for 2072 people and 15,586 observations, with missing data for 8% of observations (Figure 4.7). Of these, 1998 people had at least one wave of data in which they reported no disability and one wave of data in which they did report a disability (15,410 observations), and therefore contributed to the sample for the complete case analysis.

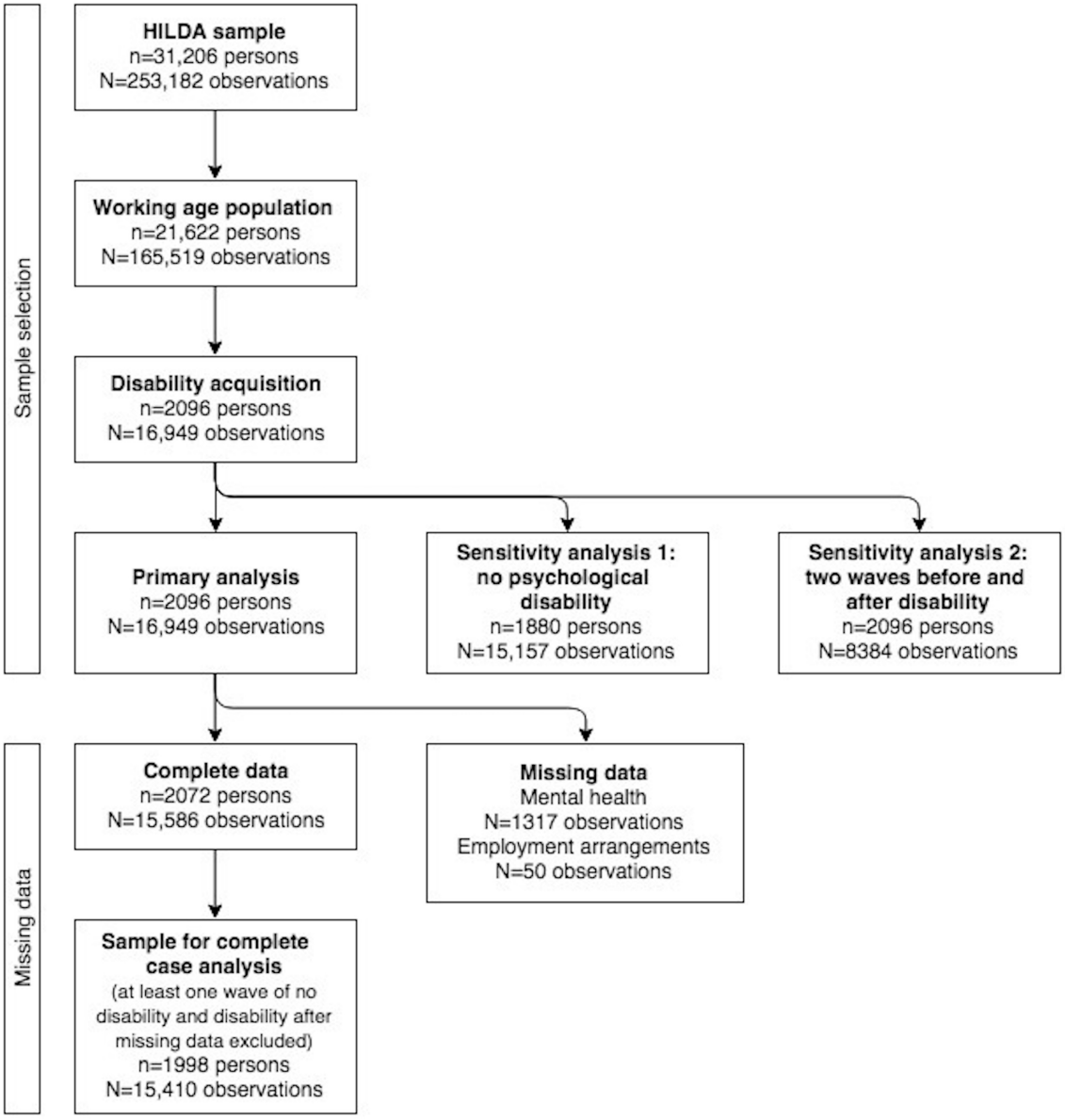

Figure 4.7 Flow diagram illustration selection of eligible sample and details of missing data

Table 4.10 in the supplementary material compares the characteristics of observations with complete data versus missing data (see supplementary material). Missing observations were more common for people who were younger, male, born outside of Australia, not in a relationship, with poorer mental health, and those who experienced socioeconomic disadvantage, including low education, low income and low skilled jobs.

Descriptive analyses

At baseline, the mean age of participants in the sample was 42 years, 54.5% were women, 78.9% were born in Australia and 31.9% had not completed secondary school education (Table 4.7). Prior to disability, 37.2% were in high skilled jobs, 39% were in

medium skilled jobs, and 23.8% in low skilled jobs; 60% were in permanent employment, 7% on fixed-term contracts, 18.1% self-employed, and 15% in casual employment; 3.7% were unemployed and 18.1% were not in the labour force (Table 4.8). MHI scores were on average four points lower in waves with disability compared to waves with no disability (68.5 versus 72.1, Table 4.8). Both in waves with and without disability, people who were employed had the highest MHI scores and those who were unemployed had the lowest scores. Prior to disability, MHI scores were similar across categories of occupational skill level, but subsequent to disability acquisition, there was a gradient of decreasing mean MHI scores with declining occupational skill level. MHI scores were higher for people who were self-employed and in permanent employment, and lower for those in fixed-term and casual employment, both before and after disability acquisition.

Table 4.7 Sample characteristics at baseline (n=1998)

	n	%
Age (years), mean (SD)	42.3	10.2
Sex		
Men	910	45.6
Women	1088	54.5
Country of birth (n=1996)		
Australia	1574	78.9
Other	422	21.1
Education (n=1997)		
Less than secondary	637	31.9
Secondary/certificate/diploma	898	45.0
University education	462	23.1

Table 4.8 Distribution of employment characteristics two waves prior to disability acquisition for those who were employed and mean within-person mental health score in waves reporting no disability and waves reporting disability (n=1998)

	Baseline		Mean MH No disability	Mean MH Disability
	n	%	Mean (SD)	Mean (SD)
Whole sample	1998	100.0	72.1 (14.8)	68.5 (17.1)
Occupational skill level				
High skill	582	29.1	72.9 (13.8)	70.6 (16.5)
Medium skill	609	30.5	73.3 (14.0)	69.9 (16.8)
Low skill	372	18.6	72.3 (15.5)	67.1 (17.5)
Unemployed	73	3.7	67.1 (15.7)	63.5 (17.1)
Not in the labour force	362	18.1	69.5 (16.4)	65.1 (17.3)
Contract type				
Permanent	937	46.9	73.0 (14.3)	69.6 (17.1)
Fixed term	109	5.5	70.1 (14.6)	66.6 (17.8)
Self-employed	283	14.2	74.6 (13.5)	71.6 (16.1)
Casual	234	11.7	71.9 (14.9)	67.9 (16.4)
Unemployed	73	3.7	67.1 (15.7)	63.5 (17.1)
Not in the labour force	362	18.1	69.5 (16.4)	65.1 (17.3)

Regression analyses

The overall effect of disability acquisition on mental health was estimated to be more than a four-point decline on the MHI scale (estimated mean difference in MHI between waves with and without disability: -4.3, 95% CI -5.0, -3.5, Table 4.9). There was strong evidence of effect modification of the relationship between disability acquisition and mental health by occupational skill level (p=0.026) but not contract type (p=0.800).

Table 4.9 Results of the primary analysis: linear fixed-effects regression coefficients for the estimated within-person mean difference in mental health score between waves reporting disability and no disability and interaction terms representing the additional within-person effect of disability on mental health for categories of employment characteristics separately (n=2096, N=16,949)

	Effect of disability		Interaction term		P value
	Coeff[a,b]	95% CI	Coeff	95% CI	
Overall effect	-4.3	-5.0, -3.5			
Occupational skill level					
High skill	-3.0	-4.2, -1.7	0		
Medium skill	-4.0	-5.2, -2.8	-1.0	-2.7, 0.7	
Low skill	-6.1	-7.6, -4.5	-3.1	-5.0, -1.2	
Not in the labour force	-4.8	-6.2, -3.3	-1.8	-3.6, 0.1	
Unemployed	-4.6	-8.0, -1.2	-1.6	-5.2, 2.0	p=0.026
Contract type					
Permanent	-4.0	-5.0, -3.0	0		
Fixed term	-4.2	-7.3, -1.1	-0.2	-3.4, 2.9	
Self-employed	-3.6	-5.4, -1.8	0.4	-1.7, 2.4	
Casual	-5.3	-7.3, -3.3	-1.3	-3.5, 0.8	
Not in the labour force	-4.7	-6.2, -3.3	-0.8	-2.5, 1.0	
Unemployed	-4.6	-6.1, -3.1	-0.6	-4.1, 2.9	p=0.800

[a]results were obtained using multiple imputation using chained equations with 50 imputed datasets
[b]models were adjusted for age

People in low skilled jobs experienced on average a six-point decline in MHI when they acquired a disability (-6.1, 95% CI -7.6, -4.5) compared to a four-point decline for people in medium skilled jobs and a three-point decline for those in high skilled jobs. People who were unemployed or not in the labour force also experienced large mental health declines of almost five-points on the MHI scale. For contract type, the effects were similar across the categories, with the largest effect for people in casual employment who experienced on average a 5.3-point decline in MHI. The results of the complete case analysis (Table 4.11 in the supplementary material) and the sensitivity analyses (Table 4.12 in the supplementary material) were similar to the primary analysis.

Discussion

The findings of this study suggest that type of occupation prior to disability acquisition influences the magnitude of the effect of disability on mental health, but not contract type. People in low skilled jobs had more than twice the mental health decline associated with disability acquisition compared to those in high skilled jobs. People in low skilled jobs experienced a six-point decline in mental health score, which is considerably larger than the four to five-point difference considered to represent a clinically meaningful difference in mental health [112, 113]. People who were unemployed or not in the labour force prior to disability acquisition also experienced large mental health declines.

This study had a number of strengths. It used longitudinal data from a large nationally representative longitudinal study of Australian households. We used fixed-effects regression, using a within-person analytic design to control for time-invariant individual characteristics. Fixed-effects regression can thus minimise the risk of bias from confounding by unmeasured (or poorly measured) variables that are stable over time.

There were also a number of limitations. There were missing data for 9% of observations in the sample. Examination of the patterns of missingness suggested that there were differences in the observed characteristics of people with missing observations compared to those with complete data, which could have led to selection bias. We used multiple imputation to assess the impact of selection bias from missing data on the results and found very similar results to the complete case analysis, suggesting that missing data is unlikely to have substantially biased the results. There is also the possibility of attrition bias, as we did not impute data for people who were lost to follow up, however attrition was low, on average 6% between waves. Another limitation is dependent misclassification bias, which results from the dependency between misclassification of the exposure and outcome. As both disability and mental health were self-reported, it is likely that there was measurement error in the reporting of both measures, and a possibility that the measurement errors were correlated. However, the fixed-effects approach can address this problem to some extent, accounting for any measurement error for an individual that is stable over time, such as negative affectivity. There may have been residual confounding by time-varying confounders. Income, household structure and relationship status are likely to be confounders but also mediators of the association and were therefore not included in the models. However, their inclusion did not affect the effect estimates, therefore residual confounding due to these variables is unlikely. The fixed effects models assume a contemporaneous effect of disability on mental health that does not change over time. While this is a strong assumption, as for some individuals, the effect may decrease over time, restricting the

analysis to two waves before and after disability acquisition did not materially change the findings. Disability acquisition was defined as two consecutive waves of no disability followed immediately by two consecutive waves of disability. This narrow definition may not capture all forms of disability, however it identifies impairments that are less likely to be transient in presentation. Furthermore, people with severe disabilities are likely to be underrepresented within the sample, particularly those with intellectual or psychological impairments, because they may be less likely to be respond to the survey and because the survey samples households from private dwellings only, thereby excluding people with more severe disability who may be living in care facilities. This may lead to an underestimation of the true effect of disability on mental health.

The findings are consistent with previous research which found that the effect of disability on mental health was greater for people who were unemployed [1] and those who experience socioeconomic disadvantage across a number of indicators [1, 3, 83, 84, 126, 129]. Our study identified people in low skilled occupation as a subgroup of people with disabilities who are particularly vulnerable to poor mental health, with some evidence of larger effects than for those who were unemployed or not in the labour force prior to disability.

There are a number of explanations for the gradient in the magnitude of mental health decline according to people's occupational skill level including economic, psychosocial and other pathways. It may be that people in low skilled occupations are more likely to lose their job when they acquire a disability, with impacts to income and financial security; identity, status and self-esteem; social contact and social support; and health-related behaviours [266]. Alternatively, people in low skilled jobs may experience poorer working conditions such as high job demands and low flexibility, which may further contribute to the negative psychosocial impact of disability [267]. It was not possible to investigate the mechanisms by which this effect was operating in this study because the effect modifiers were measured prior to disability acquisition. Future research should aim to disentangle the pathways explaining the effect modification.

Despite the limitations of the study, the robust statistical methods, the consistency of the results with previous research and the magnitude of the effects estimates highlight the importance of people with disabilities' occupation for their mental health. The findings suggest that interventions on employment characteristics could mitigate the effect of disability on mental health and reduce the impact of structural inequalities on mental health [268]. In addition to provision of high-quality accessible and affordable mental health services for people with disabilities, social interventions that focus on increasing employment rates, improving the sustainability of employment, and providing

employment services and education and training opportunities for people who acquire a disability, particularly for people in low skilled occupations, may improve mental health outcomes.

4.2.3 Supplementary material for section 4.2.2

Table 4.10 Distribution of demographic, socio-economic and employment characteristics and mental health for those with complete data and those with missing data

	Complete data n=15,410		Missing data n=1,539		P value
	n	%	n	%	
Age (mean, SD)	46.4	10.1	43.3	10.2	<0.001
Sex					
Men	6888	44.7	783	50.9	
Women	8522	55.3	756	49.1	<0.001
Country of birth					
Australia	12,257	79.5	1115	72.5	
Other	3144	20.4	422	27.4	
Missing	9	0.1	2	0.1	<0.001
Education					
Less than secondary	4731	30.7	563	36.6	
Secondary/certificate/diploma	7144	46.4	728	47.3	
University education	3530	22.9	247	16.1	
Missing	5	0.1	1	0.1	<0.001
Household income quintiles					
Q1 (lowest)	2175	14.1	311	20.2	
Q2	2611	16.9	293	19.0	
Q3	3149	20.4	312	20.3	
Q4	3476	22.6	297	19.3	
Q5 (highest)	3999	26.0	326	21.2	<0.001
Relationship status					
In a relationship	11262	73.1	1027	66.7	
Not in a relationship	4148	26.9	512	33.3	<0.001
Occupational skill level					
High skill	4561	29.6	367	23.9	
Medium skill	4678	30.4	411	26.7	
Low skill	2935	19.1	398	25.9	
Unemployed	2706	17.6	277	18.0	
Not in the labour force	530	3.4	86	5.6	<0.001
Contract type					
Permanent	7273	47.2	613	39.8	
Fixed term	796	5.2	81	5.3	
Self-employed	2154	14.0	227	14.8	
Casual	1951	12.7	205	13.3	
Unemployed	2706	17.6	277	18.0	
Not in the labour force	530	3.4	86	5.6	
Missing	0	0	50	3.3	<0.001
Mental health (mean, SD)	70.5	18.7	64.6	20.4	<0.001

Table 4.11 Results of the complete case analysis: linear fixed-effects regression coefficients and interaction terms for categories of employment characteristics (n=1998)

	Effect of disability		Interaction term		P value
	Coeff[a]	95% CI	Coeff	95% CI	
Overall effect	-3.7	-4.4, -3.0			
Occupational skill level					
High skill	-2.4	-3.6, -1.2	0		
Medium skill	-3.4	-4.6, -2.2	-1.0	-2.6, 0.7	
Low skill	-5.6	-7.1, -4.0	-3.2	-5.0, -1.3	
Not in the labour force	-4.5	-6.0, -3.0	-2.1	-3.9, -0.2	
Unemployed	-3.9	-7.1, -0.7	-1.5	-4.8, 1.9	p<0.001
Contract type					
Permanent	-3.4	-4.4, -2.5	0		
Fixed term	-3.1	-6.0, -0.2	0.3	-2.6, 3.3	
Self-employed	-3.2	-4.9, -1.4	0.3	-1.7, 2.3	
Casual	-4.7	-6.8, -2.6	-1.3	-3.5, 0.9	
Not in the labour force	-4.5	-6.0, -3.0	-1.1	-2.8, 0.6	
Unemployed	-3.9	-7.1, -0.7	-0.4	-3.7, 2.8	p=0.250

[a]models were adjusted for age

Table 4.12 Results of the sensitivity analyses: linear fixed-effects regression coefficients and interaction terms for categories of employment variables separately for (1) the analysis removing people with psychological impairments (n=1880, N=15157) and (2) the analysis using the sample restricted to two waves preceding and two waves following disability acquisition (n=2096, N=8384)

	Effect of disability		Interaction term		P value
	Coeff[a,b]	95% CI	Coeff	95% CI	
(1) Psychological impairments removed					
Overall effect	-3.7	(-4.4, -3.0)			
Occupational skill level					
High skill	-2.3	(-3.5, -1.1)			
Medium skill	-3.6	(-4.9, -2.4)	-1.4	(-3.0, 0.3)	
Low skill	-5.5	(-7.0, -4.0)	-3.2	(-5.0, -1.4)	
Not in the labour force	-3.9	(-5.5, -2.3)	-1.6	(-3.5, 0.3)	
Unemployed	-4.9	(-8.4, -1.3)	-2.6	(-6.2, 1.1)	p=0.010
Contract type					
Permanent	-3.4	(-4.4, -2.5)			
Fixed term	-3.2	(-6.1, -0.3)	0.2	(-2.7, 3.2)	
Self-employed	-3.6	(-5.4, -1.7)	-0.1	(-2.2, 2.0)	
Casual	-4.4	(-6.3, -2.5)	-0.9	(-3.0, 1.1)	
Not in the labour force	-3.9	(-5.5, -2.3)	-0.4	(-2.2, 1.4)	
Unemployed	-4.9	(-6.4, -3.3)	-1.4	(-5.0, 2.2)	p=0.919
(2) Sample restricted to two waves preceding and two waves following disability acquisition					
Overall effect	-2.8	(-4.1, -1.5)			
Occupational skill level					
High skill	-1.5	(-3.2, 0.2)			
Medium skill	-2.9	(-4.5, -1.3)	-1.4	(-3.1, 0.3)	
Low skill	-4.1	(-6.0, -2.2)	-2.6	(-4.6, -0.6)	
Not in the labour force	-3.7	(-5.7, -1.8)	-2.2	(-4.2, -0.2)	
Unemployed	-2.0	(-6.0, 2.0)	-0.5	(-4.4, 3.5)	p=0.080
Contract type					
Permanent	-2.6	(-4.1, -1.2)			
Fixed term	-3.0	(-6.4, 0.3)	-0.4	(-3.8, 3.0)	
Self-employed	-2.0	(-4.1, 0)	0.6	(-1.3, 2.5)	
Casual	-3.4	(-5.7, -1.2)	-0.8	(-3.0, 1.4)	
Not in the labour force	-3.7	(-5.7, -1.8)	-1.1	(-2.9, 0.7)	
Unemployed	-2.0	(-3.9, 0)	0.7	(-3.2, 4.6)	p=0.732

[a]results were obtained using multiple imputation using chained equations with 50 imputed datasets

[b]models were adjusted for age

4.3 Disability acquisition and mental health: effect modification by social support

4.3.1 Introduction

The two previous chapters examined the effect of socio-economic characteristics prior to disability on the association between disability and mental health to try to better understand how these characteristics influence the effect of acquiring a disability on mental health. The purpose of these analyses was to identify vulnerable subgroups of the population of people with disabilities and to shed light on the mechanisms by which disability leads to a decline in mental health. However, with these potential effect modifiers measured prior to disability, the results only provide insight into how prior characteristics influence the effect, which is only part of the causal story. The effect by which disability affects people's mental health is also likely to be modified by changes to people's socio-economic characteristics subsequent to disability acquisition.

To further disentangle the mechanistic pathways leading from disability acquisition to poor mental health, and how much of the effect operates through socio-economic characteristics, it is important to also understand how disability acquisition affects these characteristics, and how in turn this leads to changes to people's mental health. This can be achieved by measuring socio-economic characteristics subsequent to disability acquisition as well as prior.

Therefore in this chapter, I present the findings of a study in which I examine the effect of a single socio-economic characteristic – social support – measured both prior and subsequent to disability. The analysis quantifies effect modification by social support, comparing the magnitude of the effect of disability acquisition on mental health for people who experienced high or low social support before acquiring a disability and high or low social support and after disability acquisition. This enables a better understanding of how prior characteristics affect the mental health effect but also how changes to social support subsequent to disability affect the association between disability and mental health.

4.3.2 Does social support modify the effect of disability acquisition on mental health? A longitudinal study of Australian adults

This section consists of the following article [3]:

Aitken Z, Krnjacki L, Kavanagh AM, LaMontagne AD, Milner A. *Does social support modify the effect of disability acquisition on mental health? A longitudinal study of Australian adults.* Social Psychiatry and Psychiatric Epidemiology, 2017. 52(10): 1247-1255.

Introduction

Nearly twenty percent of Australians report a disability, a prevalence similar to other developed countries [25, 269]. People with disabilities are one of the most socioeconomically disadvantaged groups in society [269]. In Australia, as in most other developed countries, people with disabilities are less likely to be employed and complete post-secondary education, have lower income [19], wealth [270] and social support [11] compared to those without disability. Disabled people have poorer physical and mental health than those without disability, and many of the conditions they experience are not directly related to their impairment (e.g. high rates of diabetes and depression for people with intellectual impairments) [25]. There is growing evidence to suggest that the poorer health of people with disabilities is at least partly explained by their disadvantaged living circumstances [93, 102].

According to the International Classification of Functioning, Disability and Health (ICF) framework, disability results from the interaction between health conditions, personal attributes and environmental factors [62]. Environmental factors are included as key determinants of disability in the ICF, therefore recognising the influence of the physical, social and attitudinal environment in facilitating or restricting people's functioning, activities and participation in society [25, 271]. Conceived in this way, characteristics of people's social environment, such as their ability to access social support, may influence the impact of impairments on activity limitations and participation restriction.

Social support refers to people's perceptions of available social resources that are provided to them by other people in either a primary group (small, enduring, intimate, informal, commonly family/close friends) or secondary group (larger, interactions guided by rules, regulations, and hierarchical positions, including work, voluntary, and religious organisations) [272]. There is strong evidence that the receipt of social support is associated with improvements in health [273-275]. There is also some evidence that social support can act as a "buffer" that protects individuals from experiencing poor mental health during times of life stress [247, 274, 276]. Therefore it is plausible that social support may protect people with disabilities against poor mental health. It is also important to note that social support differs between men and women [277]}, and there is evidence of gender differences in the protective effect of social support [273] and its relationship with health outcomes [278] at a general population level. Therefore the way in which social support buffers the effect of disability acquisition on mental health may also differ by gender.

A number of studies have examined the effect of acquiring a disability on mental health, which consistently reported an increase in psychological distress, depressive symptoms, and a decline in mental health and wellbeing following disability acquisition [83, 84, 121, 126, 129]. However, not all people who acquire a disability experience deterioration in their mental health [102]. The impacts of significant life events, such as the acquisition of a disability, on mental health may depend on individual resources such as personal resilience [279], experience of economic hardship [99], education [126], and social support [280]. The effect of disability acquisition on mental health has been shown to be modified by socioeconomic disadvantage prior to disability acquisition, including low levels of education [126], low wealth [84, 129], and insecure and unaffordable housing [83]. However, few studies have examined whether social support influences the mental health of people who acquire a disability. A study of Australian adolescents examined whether mental health inequalities between people with and without disabilities were modified by social support and found larger mental health inequalities for those with low compared to higher social support [102]. A Finnish study found that increased social support (having good neighbours) and access to instrumental support buffered the effect of receiving a disability pension on mental distress [276]. However, these studies examined existing disability rather than acquisition of disability.

In this study, we examined whether social support protects people who acquire a disability from deterioration in mental health. We hypothesised that people with lower levels of social support would experience larger mental health declines than those with higher levels of social support. We examined differences in the magnitude of mental health effects according to categories of social support, measured both prior and subsequent to disability acquisition, and the interaction between them. We also examined whether the associations were different between men and women.

Methods

Data source

The Household, Income and Labour Dynamics in Australia (HILDA) Survey is a longitudinal nationally representative study of Australian households and individuals, which has been conducted in annual waves since 2001. The original panel included 13,969 individuals from 7682 households, sampled using a national probability sample of private dwellings [246]. Data were collected on a range of life domains from household members aged 15 years and above. In later waves, survey members included all participants from the original panel and household members attaining the age of 15 years, as well as new participants added as a result of changes in household composition

if new households were formed by existing survey participants. While original sample members were sample from private dwellings, existing survey members remained part of the sample and were followed up even if they moved into non-private dwellings such as a residential care home [281]. Response rates were above 75% for initial wave respondents and were above 94% for continuing participants between waves (attrition rate of less than 6% on average). Fourteen waves of HILDA data were included in the analysis (2001-2014).

Outcome variable

Mental health was assessed using the five item Mental Health Inventory (MHI), a subscale of the SF-36 general health survey. The MHI is a measure of general mental health; it assesses symptoms of depression and anxiety and positive aspects of mental health in the previous four weeks. This scale was constructed from the five items that best predicted the summary score of the 38-item Mental Health Inventory used in the Medical Outcomes Study questionnaire and has been shown to have reasonable validity and be an effective screening instrument for mood disorders or severe depressive symptomatology [175, 262]. The MHI is a continuous measure ranging from 0 to 100, with higher scores representing better mental health.

Disability measures

Information on long-term health conditions and disabilities was collected in all waves using a definition derived from the ICF framework [282]. Participants were asked if they had an ‘impairment, long-term health condition or disability which restricts their everyday activities that had lasted, or was likely to last, for a period of six months or more’. Participants who reported a disability were then asked a subsequent question specifying the type of disability, such as of sight, hearing and speech problems; limited use of arms or fingers and feet or legs; or nervous or emotional condition which requires treatment, which allowed us to classify types of impairment into broad categories including sensory and speech, physical, intellectual and psychological impairments.

Participants were defined as having acquired a disability if they did not report a disability for two consecutive waves followed by two consecutive waves with a reported disability, as used in previous studies of disability acquisition [83, 84]. All consecutive waves in which individuals did not report a disability prior to disability acquisition and all consecutive waves reporting a disability subsequent to disability acquisition were included, therefore two or more waves before disability and two or more waves after disability. We used a minimum of two waves with disability so as to exclude people with

transient disabilities. If participants reported more than one episode of disability acquisition (4% of the sample), only the first episode was included.

Social support

The HILDA Survey includes questions about perceived social support. These items have been used to create a social support scale in previous studies [247, 283-285]. We constructed a social support scale derived from 10 items which address aspects of emotional support (e.g., *When I need someone to help me out, I can usually find someone*; *People don't come and visit as much as I would like*). Items were rated on a 7-point Likert scale, and the Cronbach's alpha was 0.84 across all waves. The social support scale was created by reverse coding the items relating to negative social support and averaging the ten items in the scale, to create a measure with higher scores representing greater social support. The continuous scale was strongly positively skewed; therefore we dichotomised it into a binary variable based on a median split (low (1-5.59) and high (5.6-7)). Social support was measured before and after disability acquisition, using data reported one year prior to disability acquisition and one year after.

Other variables

Other variables were considered as potential confounders of the association between disability acquisition and mental health. Age was collapsed into four categories: 15-29, 30-44, 45-59 and 60 years and older. Employment status was measured as employed (working for pay at least one hour per week, unemployed (those who are actively seeking employment or currently unable to find work), not in the labour force (not actively seeking employment). Household disposable income was calculated by summing the income components for all adults in the household, with imputed values computed for missing variables using the methods described elsewhere (20% imputed values for observations in the sample) [286]. Household disposable income was equivalised using the modified Organisation for Economic Co-operation and Development (OECD) scale [287] and converted to quintiles of the Australian population distribution using statistics published by the Australian Bureau of Statistics [288]. We also included education (postgraduate or bachelor degree; diploma or certificate; completion of secondary education; non-completion of secondary education), household structure (couple with no children; couple with children; lone parent with children; lone adult; other) and relationship status (married or de facto; separated, divorced, widowed; single).

Statistical analysis

Mean MHI scores were presented for age, relationship status, household structure, education, employment and income by disability status. Mean MHI scores were also

summarised by categories of social support prior and post disability, by disability status and gender. For each individual, a mean MHI score was computed for waves in which they reported a disability and waves in which they did not report a disability (within-person means, pooled across time). These means were then averaged to represent the overall mean MHI score (between-persons) by disability status.

We used longitudinal linear regression models with fixed-effects estimators to estimate the association between disability acquisition and mental health. All analyses were stratified by gender, with results presented separately for men and women. Coefficients from the models describe average within-person differences in MHI scores between waves in which individuals reported no disability and waves in which they reported disability. Because they estimate within-person effects, fixed-effects models remove bias from measured and unmeasured time-invariant confounding [230]. We controlled for age, employment status, household income, education, household structure and relationship status as potential time-varying confounders.

To assess whether the association between disability acquisition and mental health varied by social support, we included a product term between disability acquisition, prior social support, and social support subsequent to disability, and assessed whether there was statistical evidence of effect measure modification with either social support variable (or both simultaneously) using the P values of the product terms and likelihood ratio tests for interaction. We also included interaction terms between disability and age based on a priori hypotheses that the effect of disability acquisition on mental health is likely to differ by age, which was confirmed by likelihood ratio tests from comparisons of models with and without interaction terms.

All analyses were conducted in Stata/SE 12 [289], using the *xtreg* command with fixed-effects estimators and robust standard errors, and the *lincom* command for linear combination of coefficients to compute effect estimates and 95% confidence intervals for categories of social support. The data were extracted from HILDA using the Add-On package PanelWhiz for Stata [290].

Sensitivity analyses

We conducted a sensitivity analysis to test the robustness of our findings. We excluded people in the sample who reported psychological impairments (defined as nervous or emotional condition which requires treatment or any mental illness which requires help or supervision). People with psychological impairments are likely to report poorest mental health and there is evidence that they have low levels of social support [11], therefore it is possible that having a psychological impairment may modify the effect, in

that the relationships between disability, social support and mental health may differ for this subgroup of individuals compared to other types of impairment.

Results

There were 2679 persons (20,798 observations) who met our criteria for disability acquisition. Complete data were available for 82% of individuals, resulting in a final analytic sample of 2200 persons (15,724 observations), including 1233 women and 967 men. The mean number of observations (contributed annual waves of data) per person was 7. Further details of sample selection and missing data are in Figure 4.8.

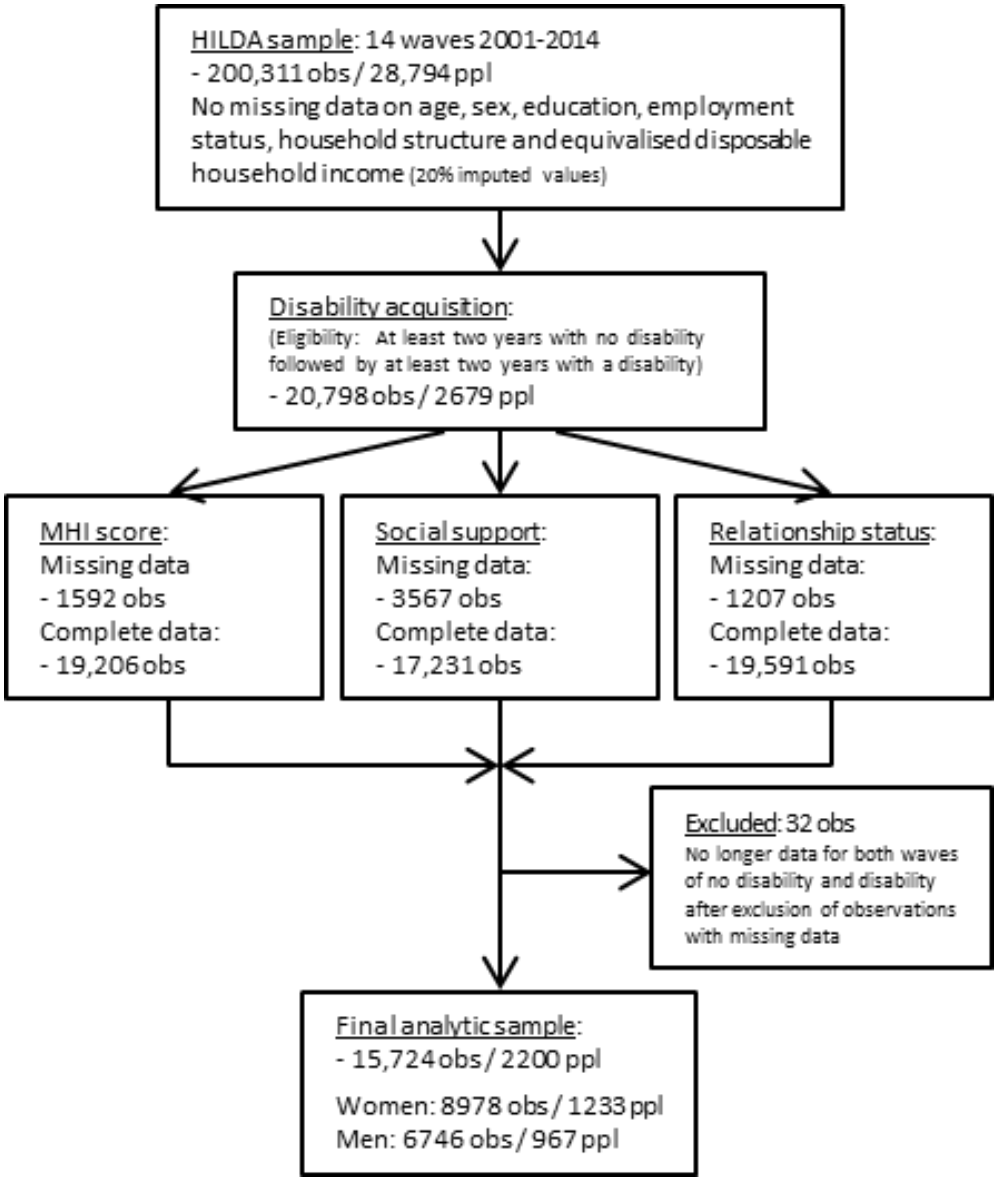

Figure 4.8 Flow diagram of sample selection and missing data

At baseline entry into the analytic sample (first wave reporting no disability), over a third of the sample were younger than 45 years, and nearly a third were aged 45 to 59 years

and another third were 60 years or older (Table 4.13). More than half of the sample were women (56%), 65% were in a relationship, 42% had not completed secondary education and 58% were employed. There were 22% in the highest income quintile while 19% were in the lowest.

Table 4.13 Characteristics of sample (at baseline entry into the analytic sample, n=2200 persons) and pooled MHI scores in all waves preceding disability and all waves following disability acquisition (observations=15,724)

Characteristics	Distribution		Mean pooled MHI score	
	n	%	Measured in waves preceding disability	Measured in waves following disability
Whole Sample	2200	100	74.5 (15.1)	71.2 (16.5)
Age group (years)				
<30	347	15.8	69.5 (16.7)	63.6 (18.9)
30-44	533	24.2	71.7 (15.5)	66.3 (17.8)
45-59	658	29.9	73.8 (14.7)	69.7 (16.4)
60+	662	30.1	79.7 (13.6)	76.2 (14.7)
Sex				
Women	1233	56.1	72.9 (15.6)	69.6 (16.6)
Men	967	44.0	76.9 (14.1)	73.2 (16.2)
Relationship status				
Married/de facto	1426	64.8	75.5 (14.7)	72.7 (15.9)
Separated/divorced/widowed	430	20.0	73.6 (16.3)	69.7 (17.6)
Single	344	15.6	69.6 (16.2)	63.3 (18.9)
Household structure				
Couple no children	808	36.7	77.5 (14.4)	74.5 (16.0)
Couple with children	807	36.7	72.7 (15.4)	68.4 (16.7)
Lone parent with children	183	8.3	68.6 (17.6)	63.7 (19.1)
Lone person	326	14.8	72.7 (16.7)	69.1 (18.6)
Other	76	3.5	71.3 (16.7)	65.8 (18.7)
Education				
Bachelor/postgraduate	409	18.6	75.4 (13.7)	73.3 (15.0)
Diploma/certificate	613	27.9	75.5 (14.6)	71.8 (16.7)
Completed secondary	259	11.8	71.5 (15.8)	68.1 (17.6)
Not completed secondary	919	41.8	73.9 (15.9)	70.1 (17.2)
Employment				
Employed	1278	58.1	74.3 (14.5)	70.9 (16.0)
Unemployed	80	3.6	65.2 (19.5)	59.3 (21.7)
Not in the labour force	842	38.3	75.2 (16.4)	71.0 (18.3)
Income				
Q5 - High	489	22.2	76.5 (14.7)	73.8 (15.8)
Q4	440	20.0	74.9 (15.4)	72.1 (16.8)
Q3	443	20.1	74.2 (16.1)	70.3 (18.0)
Q2	409	18.6	74.1 (16.1)	69.7 (18.7)
Q1 - Low	419	19.1	73.9 (17.2)	69.5 (18.6)

Table 4.13 also shows the average MHI scores for waves before and after disability acquisition for each covariate. The MHI score was lower in waves when disability was

reported than before disability acquisition (71.2 versus 74.5). Both before and after disability acquisition, MHI scores were positively associated with age and higher for men than women. There were distinct socioeconomic gradients in the MHI scores, with lower mental health in more disadvantaged groups (people who were single, lone parent families, low education, unemployed, and low income) compared to higher socioeconomic groups.

In terms of social support, the proportion people reporting low social support was greater for men, with 44.4% of men reporting low social support both before and after disability acquisition compared to 36.5% of women (Table 4.14). For both women and men, social support declined after disability acquisition. For women, 13.9% experienced a decline in social support between waves before and after disability acquisition, compared to 16.9% of men. MHI scores were lower following disability acquisition than preceding disability acquisition, lower for women compared to men, and lower for those with low social support. For example, for women with high social support before and low social support after disability acquisition, their pooled MHI score in all waves preceding disability was 74.0 compared to 65.5 in waves following disability acquisition.

Table 4.14 Pooled MHI scores for all waves preceding disability and all waves following disability acquisition, by category of social support before and after disability acquisition

Social support one year before disability acquisition	Social support one year after disability acquisition: High				Social support one year after disability acquisition: Low			
	n	%	Pooled MHI score Mean (SD)		n	%	Pooled MHI score Mean (SD)	
			Preceding disability	Following disability			Preceding disability	Following disability
Women (n=8978 observations)								
High	490	39.7	79.9 (11.9) [a]	77.5 (13.1)	171	13.9	74.0 (14.1)	65.5 (16.8)
Low	122	9.9	72.8 (14.1)	73.4 (14.1)	450	36.5	64.8 (16.2)	61.5 (16.3)
Men (n=6746 observations)								
High	270	27.9	84.2 (9.9)	82.6 (10.4)	163	16.9	80.7 (10.1)	73.8 (15.2)
Low	105	10.9	78.6 (13.7)	77.1 (15.0) [b]	429	44.4	69.7 (14.6)	66.2 (16.5)

[a] This corresponds to the mean of women's pooled MHI scores in all waves preceding disability for those who reported high social support one year before disability and high social support one year after disability acquisition

[b] This corresponds to the mean of men's pooled MHI scores in all waves following disability for those who reported low social support one year before disability and high social support one year after disability acquisition

Regression analyses

There was statistical evidence of an additive three-way interaction between disability and social support prior and social support subsequent to disability acquisition (test for interaction: $p<0.001$), therefore interaction terms were included between disability acquisition and social support in the regression models.

We quantified the deterioration in mental health between waves in which women and men did not report a disability and the waves in which they did, according to their reported level of social support before and after disability. Though the effects of disability acquisition on mental health were much larger for women, for both women and men there was a consistent pattern of association with social support. The effect of disability acquisition on mental health was greatest for women and men who experienced a change from high to low social support. For women, those who experienced a decline in social support had a 12.5 point difference in MHI score between waves preceding and following disability acquisition (-12.5, 95% CI -14.7, -10.2) compared to a six point decline for those with high social support both before and after disability (-6.4, 95% CI -8.3, -4.4) (Table 4.15). The excess effect on mental health of declining social support was greater than six points (interaction term: -6.1, 95% CI -7.9, -4.4). There was also a very large mental health deterioration for women who had low social support both before and after disability acquisition (-7.8, 95% CI -9.7, -5.9). For men, the largest effect was for those who experienced a decline in social support (-6.8, 95% CI -9.2, -4.4), followed by those with consistently low social support (-4.8, 95% CI -7.0, -2.6), and the smallest effect for those with consistently high social support (-2.5, 95% CI -4.7, -0.3).

Table 4.15 Linear fixed-effects regression coefficients for the within-person difference in MHI score between waves preceding and following disability acquisition, presented by social support categories before and after disability acquisition with coefficients for the interaction terms

	Social support one year after disability acquisition					
	High			Low		
Social support one year before disability acquisition	coeff	95% CI	P value	coeff	95% CI	P value
Women (n=8978 observations)						
Linear regression coefficient[a]						
High	-6.4	-8.3, -4.4	<0.001	-12.5	-14.7, -10.2	<0.001
Low	-4.4	-6.9, -1.9	0.001	-7.8	-9.7, -5.9	<0.001
Interaction term[b]						
High	0			-6.1	-7.9, -4.4	<0.001
Low	2.0	0.0, 4.0	0.055	-1.4	-2.7, -0.1	0.030
Men (n=6746 observations)						
Linear regression coefficient[a]						
High	-2.5	-4.7, -0.3	0.027	-6.8	-9.2, -4.4	<0.001
Low	-2.7	-5.3, -0.1	0.043	-4.8	-7.0, -2.6	<0.001
Interaction term[b]						
High	0			-4.3	-6.1, -2.5	0.001
Low	-0.2	-2.2, 1.8	0.858	-2.3	-3.7, -0.9	<0.001

[a] Adjusted for age, household structure, marital status, education, employment, quintiles of equivalised household disposable income and age*disability product term

[b] The coefficient represents the additional effect of disability acquisition on mean MHI score within-persons for each category of the change in social support variable compared to the reference category (high social support prior and post disability)

Sensitivity analysis

Results were robust to the sensitivity analysis. Exclusion of people with psychological impairments attenuated the results slightly although the gradient was still evident; the magnitude of the reduction in MHI in the high to low social support group was 11 points (-11.1, 95% CI -13.6, -8.7) compared to six points among people in the high to high social support group (-6.2, 95% CI -8.3, -4.1) for women and there was a six point reduction for men in the high to low group (-5.9, 95% CI -8.4, -3.5) compared to a two point reduction in the consistently high social support group (-1.9, 95% CI -4.2, 0.3) (Table 4.16).

Discussion

The magnitude of the effect of disability acquisition on mental health was much larger for women than men, but there was a consistent pattern of association with social support for both women and men. There was evidence of effect measure modification by social support: the magnitude of change in mental health following disability acquisition varied greatly according to social support before and after disability acquisition. People whose social support changed from high to low experienced the greatest negative effects of disability, followed by people who had consistently low social support and those who remained with high social support following disability acquisition. The smallest negative effect was seen for people who experienced improvement in social support. This could suggest that social support acted as a protective factor for mental health. However, it is important to interpret these effects in conjunction with the mean MHI score within each subgroup. Though those experiencing consistently low social support did not experience the largest mental health declines, their mental health scores were substantially lower than the other groups, both before and after disability acquisition.

Consistent with our hypothesis, people with low social support *subsequent* to disability acquisition experienced the poorest mental health. People who changed from high to low social support experienced the largest declines, and people with consistently low social support, despite having smaller magnitude of decline, experienced the poorest mental health. Contrary to our hypothesis, high levels of social support *prior* to disability acquisition did not protect against mental health deterioration.

Our findings are consistent with studies in Finland and Australia, which found that social support buffered the effect of having a disability on poor mental health [102, 276]. This study builds on existing research by examining disability acquisition – incident rather than prevalent disability – and by investigating the effect of social support before and after disability acquisition, to better understand the causal relationships between disability, social support and mental health. These results suggest social support

subsequent to disability acquisition buffers the adverse impacts of disability acquisition on mental health, which may be explained by provision of emotional support to prevent negative emotional and behavioural responses [273], or through informational and instrumental support, for example access to information related to health or access to health care [273].

There were substantial differences in the magnitude of the effect for women and men. The effect of disability on mental health was about two-fold higher in women than men, across all categories of social support. The observed gender difference may relate to psychological attributes related to vulnerability to life events [291] and differences in stress responses which may make women more vulnerable to poor mental health [292]. Interestingly, the difference in the relative magnitude of the effect according to social support categories was similar between women and men.

In this paper, we examined within-person change in mental health and observed substantial and clinically significant levels of change. People whose social support changed from high to low following disability acquisition had a seven (men) to 13 point (women) (~50 to 80% of one standard deviation) reduction in their mental health, substantially exceeding a three-point difference on the scale considered clinically meaningful [169]. By comparison, within-person change in mental health associated with the onset of unemployment in the general population is in the range of 2-4 points on the MHI scale [247].

This study has a number of strengths. First, it is based on a large population-based longitudinal survey in which 2200 people acquired a disability over 14 years. Second, causally-robust fixed-effects regression was used to control for time-invariant confounding and important time-varying confounders were adjusted for. Third, we used a comprehensive measure of social support measured before and after disability acquisition.

There are also potential limitations, including unmeasured confounding, dependent misclassification bias and selection bias. The fixed-effects analysis mitigates against time-invariant confounding including unmeasured variables (e.g., negative affectivity) and dependent misclassification bias, whereby misclassification of the exposure and the outcome are correlated, because it estimates within-person effects. There is potential for unmeasured time-varying confounding, however we controlled for key socioeconomic characteristics that were thought to be the most important potential confounders. Selection bias arising from missing data is another potential limitation, but loss to follow-up in HILDA was low (<10% for most waves). The use of a short five-item questionnaire

to measure mental health, in contrast to a more comprehensive questionnaire, means that it does not serve as a diagnostic tool for mental health disorders. Rather, the MHI scale of the SF-36 has proved to be a successful screening tool for mood disorders and severe depressive symptomatology in the general population [262], making it a good instrument to measure general mental health and wellbeing for use in population surveys. We could not examine differences in the association by impairment type or severity of disability. Information on type of impairment was asked of participants who reported a long-term health condition; however we lacked power to examine associations for different types of impairments, and questions relating to severity of disability were included in only three waves of the survey (2004, 2009 and 2013). Finally, HILDA is likely to under-represent people with severe disabilities and communication impairments for whom social support may be even more important. We used a single broad question asked in HILDA to define disability, and as a result, the prevalence of disability is much higher than the prevalence estimated in other surveys such as the Survey of Disability Ageing and Carers (SDAC), an Australian survey which uses a much more lengthy and comprehensive tool to define disability (27.2% in HILDA 2012 versus 21.2% for the population aged 15 years and above in SDAC 2012) [241]. The use of a more inclusive and broader question in our sample is likely to pick up relatively milder cases of disability, which may underestimate the effect of disability acquisition on mental health as well as the influence of social support.

Conclusions

This analysis found that social support is an important resource that protects individuals against the negative mental health effects of disability acquisition. People who experienced a decline in social support and those with consistently low social support were particularly vulnerable to poor mental health subsequent to disability acquisition. Furthermore, we found that the effects of disability acquisition on mental health were of much greater magnitude for women than for men. The magnitude of the mental health effects associated with low social support, combined with the modifiability of social support, highlights the importance of these findings for developing new policy and practice strategies to support people at the time of disability acquisition, and to promote social support for people with disabilities.

4.3.3 Supplementary material for section 4.3.2

Table 4.16 Linear fixed-effects regression coefficients for the within-person difference in MHI score between waves preceding and following disability acquisition excluding those acquiring a psychological impairment, presented by social support categories before and after disability acquisition with coefficients for the interaction terms

	Social support one year after disability acquisition					
		High			Low	
Social support one year before disability acquisition	coeff	95% CI	P value	coeff	95% CI	P value
Women (n=8226 observations)						
Linear regression coefficient[a]						
High	-6.2	-8.3, -4.1	<0.001	-11.1	-13.6, -8.7	<0.001
Low	-2.8	-5.5, -0.2	0.035	-7.0	-9.2, -4.9	<0.001
Interaction term[b]						
High	0			-5.0	-6.8, -3.2	<0.001
Low	3.3	1.3, 5.3	0.001	-0.9	-2.2, 0.4	0.180
Men (n=6442 observations)						
Linear regression coefficient[a]						
High	-1.9	-4.2, 0.3	0.097	-5.9	-8.4, -3.5	<0.001
Low	-2.3	-5.0, 0.4	0.095	-4.0	-6.3, -1.8	<0.001
Interaction term[b]						
High	0			-4.0	-5.8, -2.2	<0.001
Low	-0.4	-2.4, 1.6	0.720	-2.1	-3.5, -0.7	0.003

[a] Adjusted for age, household structure, marital status, education, employment and quintiles of equivalised household disposable income
[b] The coefficient represents the additional effect of disability on mean MHI score within-persons for each category of the change in social support variable compared to the reference category (high social support prior and post disability)

Chapter 5 Mediation

5.1 Sequential causal mediation analysis to estimate natural direct and indirect effects through multiple mediators

5.1.1 Introduction

The three analyses in the previous chapter provided evidence that the effect of disability acquisition on mental health differs according to people's socio-economic characteristics prior to disability, suggesting that socio-economic factors play a significant role in determining the magnitude of the effect of disability acquisition on mental health. The third analysis also examined effect modification by social support *subsequent* to disability acquisition, to determine whether changes to social support subsequent to disability acquisition influenced the magnitude of the association. This analysis found evidence that changes to people's social support subsequent disability (as well as social support prior to disability) influenced the magnitude of the effect of disability acquisition on mental health, suggesting that changes to socio-economic characteristics as a result of disability may be implicated in explaining the mental health decline associated with disability.

Chapter 6 further examines the mechanism leading from disability acquisition to poor mental health, using a causal mediation approach to understand how changes to people's socio-economic characteristics as a result of disability acquisition lead to a deterioration in people's mental health.

The first causal mediation analysis examines a wide range of socio-economic characteristics measured subsequent to disability acquisition to quantify how much of the effect of disability acquisition on mental health operates through these factors. The causal mediation analysis decomposes the total effect of disability acquisition on mental health into the effect operating through these socio-economic factors, the NIE, and the effect operating through other pathways, the NDE. The socio-economic characteristics are categorised into three broad groups: material, psychosocial and behavioural factors, an approach previously used to explain income-related health inequalities. The indirect effects are estimated sequentially, firstly quantifying the effect operating through material socio-economic factors, then the joint indirect effect through material and psychosocial factors, and finally the joint indirect effect through material, psychosocial and behavioural factors. This analytic approach provides an understanding of the relative contribution of these three groups of socio-economic characteristics to the effect of disability acquisition on mental health.

5.1.2 Do material, psychosocial and behavioural factors mediate the relationship between disability acquisition and mental health? A sequential causal mediation analysis

This section consists of the following article [4]:

Aitken Z, Simpson JA, Gurrin L, Bentley R, Kavanagh AM. *Do material, psychosocial and behavioural factors mediate the relationship between disability acquisition and mental health? A sequential causal mediation analysis.* International Journal of Epidemiology, 2018. 47(3): 829-840.

Introduction

Currently more than a billion people, approximately 15% of the world's population, live with a disability [25]. People with disabilities experience substantial health inequalities and are at high risk of poor mental health [83, 84]. A causal relationship between disability and poor mental health has been suggested from analyses of prospective cohort studies [83, 84, 101, 116, 124, 126, 129, 141]. However, the mechanism by which disability leads to deterioration in mental health is poorly understood.

There are a number of different potential explanations for a causal link between disability and poor mental health. Supported by theoretical and empirical studies of the mechanisms underlying income-related health inequalities, three frameworks have become well established in explaining how social determinants influence health: material, psychosocial and behavioural pathways [293-296]. Firstly, the material pathway, by which differential exposure to structural and material living conditions leads to health inequalities, which posits that material conditions such as poverty and economic deprivation affect health directly but also have indirect effects by enabling access to better living circumstances such as access to health care [297]. Secondly, the psychosocial perspective emphasises the importance of psychosocial and stress-related risk factors on health, with inequalities arising from the unequal distribution of psychosocial factors such as social support, home-work balance and personal control [298]. Thirdly, differences in health-related behaviour are thought to contribute to health inequalities, for example, smoking, physical activity, and diet [299]. There has been considerable debate regarding the relative importance of these factors in explaining social inequalities in health [294]. Most empirical studies have argued for the significance of material pathways [294, 300], postulated to have a greater relative contribution because they exert both a direct effect on health as well as an indirect effect through psychosocial and behavioural pathways [295, 301].

With regards to the mechanisms driving disability-related mental health inequalities, disability acquisition may lead to changes in material, psychosocial and behavioural

factors, which could explain, or mediate, the observed mental health deterioration. At present, it is not clear to what extent the effect of disability on mental health operates through these proposed pathways or through other mechanisms. Evidence regarding the importance of different pathways between disability and mental health is sparse; the research has mainly been conducted in cross-sectional studies of people with chronic illness, has only examined psychosocial pathways, and no study has examined multiple pathways simultaneously. Three studies examined mediation of the effect through psychosocial resources, and found evidence that some of the effect of disability acquisition on depressive symptoms [141] and depression was operating through this pathway [141, 302, 303]. Understanding the mechanisms underpinning these mental health inequalities is an important public health question because socioeconomic intermediary variables are potential modifiable targets for interventions to mitigate the adverse effects of disability on people's mental health [183].

In this study, we use data from four waves of a longitudinal study of Australian adults and apply recently developed methods - sequential causal mediation analysis – to estimate the relative importance of three distinct mechanistic pathways leading from disability acquisition to poor mental health, quantifying the indirect effects through material, psychosocial and behavioural factors (Figure 5.1). Material factors are likely to affect mental health directly as well as indirectly through psychosocial factors such as latent consequences of employment (e.g., purposeful time use, self-esteem) [304], and behavioural factors. Similarly, psychosocial factors are thought to exert a direct effect on mental health, and an indirect effect through behavioural factors.

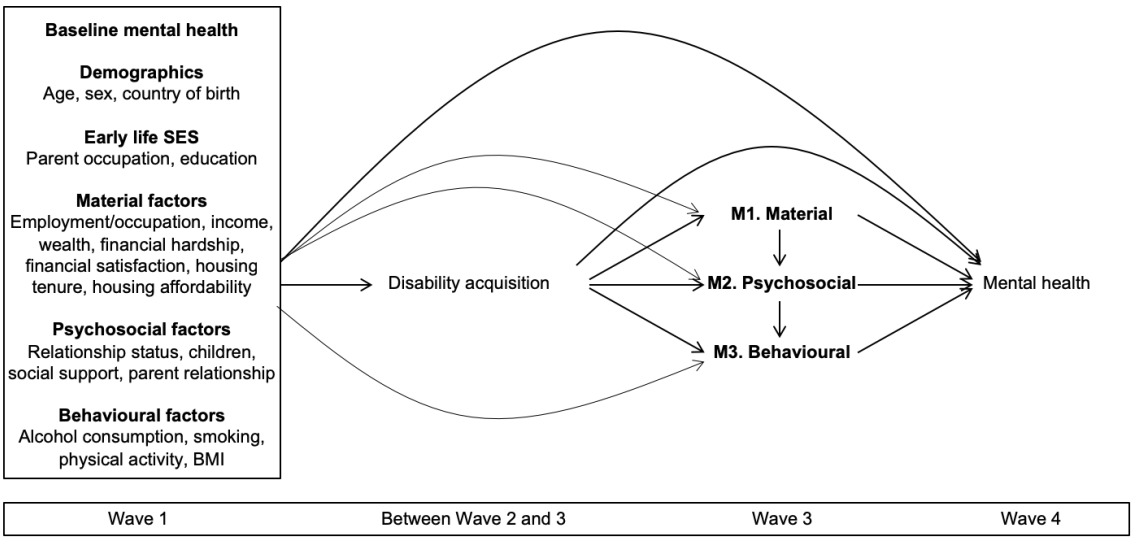

Figure 5.1 Casual diagram illustrating postulated causal relationships between disability acquisition and mental health

Methods

Data source

The Household, Income and Labour Dynamics in Australia (HILDA) Survey is a longitudinal study of Australian households, conducted annually since 2001 [246]. The survey collects information on demographic, social, economic, and health characteristics of individuals using a combination of interviews and self-completed questionnaires. The original sample included 13 969 participants from 7682 households, randomly sampled using a national probability sample of private dwellings. A top-up sample was added in 2011 to maintain representativeness, leading to a sample size after 14 waves of 28 794 people. On average for all waves of the survey, response proportions were 80% (ranging from 70% to 92%) and attrition was 5.7% between waves, ranging from 3.5% in 2014 to 13.2% in 2002. The analysis used four waves of the survey (2011 to 2014) to establish a temporal sequence between disability acquisition, the mediators, and mental health.

Disability acquisition

Information on disability was collected in every wave, using a single question defining disability as “an impairment, disability or long-term health condition, which restricts everyday activities that had lasted for six months or more”. Disability acquisition was defined as two waves reporting no disability, followed immediately by two consecutive waves reporting a disability. We used two consecutive waves of disability so as to exclude people with transient disability and to reduce the potential for measurement error, a definition used in previous studies examining disability acquisition [120, 263, 264, 305]. Participants who acquired a disability were compared to those who reported no disability in any of the four waves. People who reported other patterns of exposure, such as a single wave of disability, were excluded. Eligibility for inclusion required participation and response to the disability question at all four waves.

Mental health

Mental health was assessed in the final wave (2014) using the Mental Health Inventory (MHI), a subscale of the Short Form 36 (SF-36; a widely-used general health questionnaire that has been validated in the Australian population using data from the HILDA Survey) [168]. The MHI is a well validated and reliable measure of mental health status [169]. It measures symptoms of depression, anxiety and psychological wellbeing and has been shown to be an effective screening tool for mood and anxiety disorders and severe depressive symptomatology in comparisons with established mental health, wellbeing, and depression scales [172-175] as well as studies comparing against clinical diagnoses [259-262]. The MHI has been shown to be psychometrically sound, with high

internal consistency, discriminant validity, and high test-retest reliability [171]. It includes five items relating to mental health over the previous four weeks, each scored using five response categories. Total scores were transformed into a scale with a mean score of 74 (range: 0-100), as per standard practice, with higher scores reflecting better mental health. Previous research has suggested that a difference of four to five-points on the MHI scale is likely to reflect a minimally important clinical difference in mental health [112, 113].

Mediators

Mediator variables, described in Table 5.1, were measured in the third wave (2013). The choice of variables and their classification into three broad categories was motivated by reviewing empirical studies examining different explanations for socioeconomic inequalities in health [294, 306-308] and selecting similar variables available in the HILDA Survey where possible.

Table 5.1 Description of mediator variables

Variables	Type	Definition/categorisation
Material factors:		
Occupation	Categorical	High skill; medium skill; low skill job; unemployed/not in labour force
Weekly income	Continuous	Equivalised household disposable income, $AUD
Financial hardship	Categorical	Prosperous/very comfortable; reasonably comfortable; just getting along/poor/very poor
Financial satisfaction	Continuous	Satisfaction with financial circumstances, ranked using an 11-point Likert scale ranging from “totally dissatisfied” to “totally satisfied”
Housing tenure	Categorical	Outright owner; mortgager; renter
Housing affordability	Binary	Unaffordable defined as households in the lowest 40% of the income distribution with housing costs exceeding 30% of their gross income
Psychosocial factors:		
Relationship status	Binary	Yes; no
Children	Binary	Yes; no
Social support	Continuous	Constructed using the average of 10 questions addressing aspects of emotional support, each rated on a 7-point Likert scale[247]
Socialising	Continuous	Frequency of socialising with friends or relatives, rated on a 7-point Likert scale ranging from daily to less than once every three months
Parent relationship	Continuous	Satisfaction with relationship with parents, rated on an 11-point Likert scale ranging from “completely dissatisfied” to “completely satisfied”
Behavioural factors:		
Smoking	Categorical	Never; ex-smoker; current
Alcohol consumption	Categorical	Never; rarely; 1-2 days per week; >2 days per week
Physical activity	Categorical	>3 times per week; 1-3 times per week; less than once a week
Body Mass Index	Continuous	Self-reported, kg/m^2
Healthy diet index	Continuous	Ranging from 0 “unhealthiest” to 4 “healthiest”, derived from four binary questions: eating fruit every day; eating vegetables every day; eating fatty foods less than once a month; drinking low fat milk[309]
Quality of sleep	Continuous	Rated on a 4-point Likert scale ranging from 1 “very good” to 4 “very bad”

Baseline covariates

Baseline covariates were measured in the first wave (2011), as a measure of people’s circumstances prior to disability acquisition. It is well documented in the literature that

the incidence of disability is socially patterned, with people who experience socioeconomic disadvantage being more likely to acquire a disability [263, 310, 311]. Furthermore, according to the International Classification of Functioning, Disability and Health (ICF) framework, disability results from the interaction between health conditions, personal attributes and environmental factors [62, 282]. Conceived in this way, personal attributes such as the experience of financial strain, or characteristics of people's social environment, such as their ability to access social support, are key determinants of disability as they influence the impact of people's impairments on activity limitations and restriction to participation.

Demographic characteristics included age, sex and country of birth (Australia; other) and socioeconomic characteristics included education (bachelor degree and above; completion of secondary education; did not complete secondary) and parental occupation (high skill; medium skill; low skill or not in the labour force). Baseline levels of material, psychosocial and behavioural variables were recorded, categorised as described above, except for diet index and sleep quality, which were not measured in 2011. Mental health at baseline was measured using the MHI.

Sequential causal mediation approach

Mediation analysis aims to determine the extent to which an association between an exposure (here, incident disability) and an outcome (mental health) is due to the effect of the exposure on an intermediate variable (the mediator) which then influences the outcome. It aims to partition the total (causal) effect (TCE) of the exposure on the outcome into the effect that acts through the mediator, the indirect effect, and the effect of exposure on outcome through mechanisms other than those that involve the mediator, the direct effect ("direct" in the sense that it by-passes the putative mediator). We sought to decompose the effect of disability acquisition on mental health into natural direct effects (NDE) and natural indirect effects (NIE) through material, psychosocial and behavioural factors using a sequential approach to causal mediation analysis (further details in Supplementary File 1) [205]. This approach allows for mediation analysis through multiple causally-related mediators and accommodates exposure-mediator interactions, one of the main sources of potential bias of the traditional approach to mediation. Based on our assumptions about the causal ordering of the mediators, this approach enabled us to estimate, in model 1, the NIE through material factors (including paths that act through causal descendants of material factors but excluding paths that act only through psychosocial and/or behavioural factors), in model 2, the NIE through both material and psychosocial factors (and through their causal descendants but excluding the path that acts only through behavioural factors), and in model 3, the NIE

through material, psychosocial and behavioural factors, consisting of all possible paths except for the “direct” path from exposure to outcome (Figure 5.2).

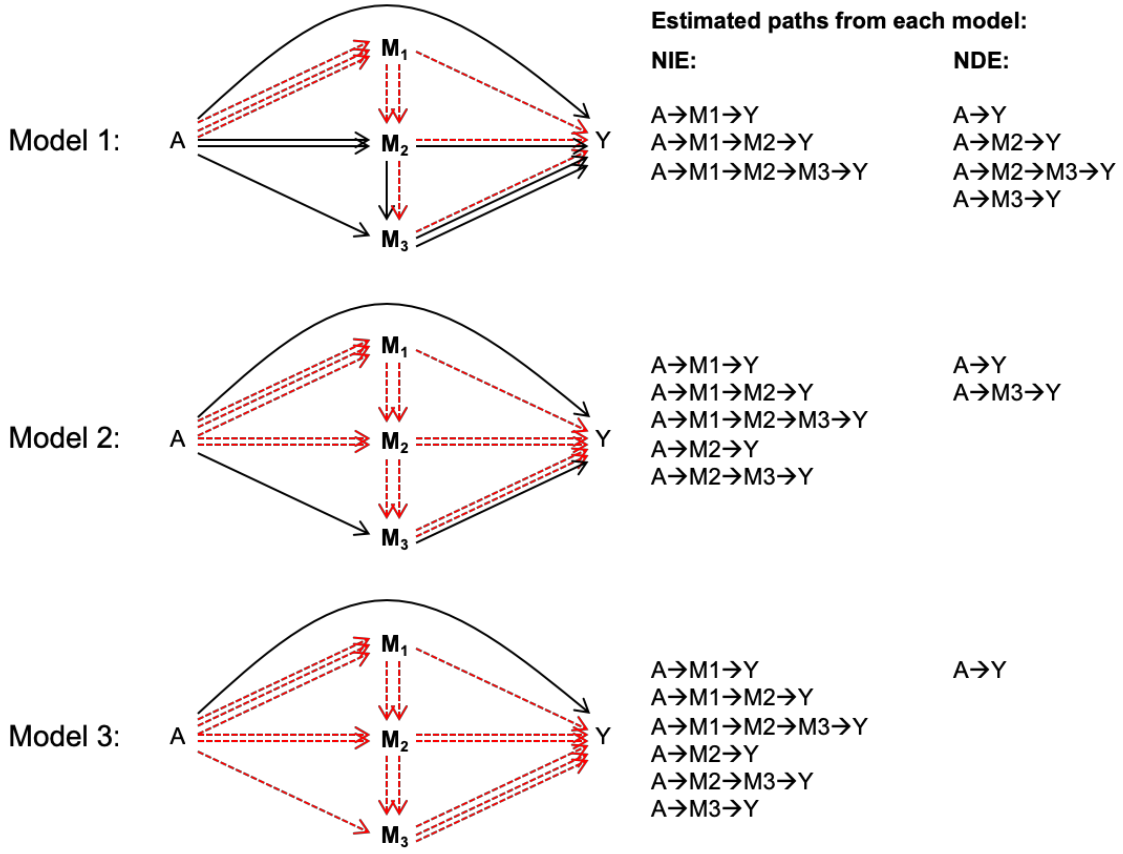

Figure 5.2 Simplified causal diagrams illustrating estimated paths in models 1-3, the NDE illustrated by the black lines (-) and the NIE by the red dashed lines (- -) (A, disability acquisition (exposure of interest); Y, mental health (outcome); Mediators - M1, material factors; M2, psychosocial factors; M3, behavioural factors)

Statistical analysis

We used a weighting approach to estimate the marginal TCE, NDE and NIE for each set of mediators (further details in Supplementary file 1). Inverse probability weighting was used to achieve exchangeability between the comparison groups and thus to account for possible confounding of the exposure-mediator and exposure-outcome associations by measured covariates [158, 205, 312]. The MHI was modelled as a continuously-valued outcome using linear regression models with and without the mediators, including all baseline variables as covariates. Interactions were included between the exposure and mediator variable if removal of an interaction term substantially changed the estimates of the NDE and NIE [176], measured as a change in the estimate of greater than half a standard error. Bootstrapping with 200 replications was used to calculate 95% confidence intervals.

Missing data

There were missing observations for the outcome, as well as several baseline covariates and mediators (Table 5.5, Supplementary file 2). The distribution of baseline covariates was compared between participants with and without missing observations to determine whether missingness was associated with the values of measured variables. Participants with missing data had poorer mental health and greater socioeconomic disadvantage across all measures compared to those with complete data (Table 5.6, Supplementary file 2), suggesting that the data were not missing completely at random. Multiple imputation (MI) using chained equations with 50 imputations was performed to optimise the validity of the findings. The imputation models included all variables in the target analysis as well as additional auxiliary variables (Table 5.7, Supplementary file 2).

The sequential mediation analysis was conducted on each of the 50 imputed datasets and the mean of the estimates from each imputed dataset was calculated to give an overall MI estimate of the NDE and NIE. Standard errors were derived using Rubin rules for combining the between-imputation and within-imputation variance (obtained by bootstrapping the NDE and NIE estimates) [236].

Sensitivity analyses

Three sensitivity analyses were conducted to test the robustness of findings. First, we performed a bias analysis for unmeasured confounding, which assessed the sensitivity of the results to unmeasured confounding of the mediator-outcome association, positing a range of plausible values for the strength of association of the potential confounder with mental health and the difference in prevalence of this confounder between those with and without disability (further details in Supplementary file 3) [234]. Second, we removed participants with psychological impairments, defined as nervous or emotional conditions which require treatment, or any mental illness which requires help or supervision, as the effect of acquiring a psychological impairment on a general mental health score is likely to be different to other types of impairments. Third, we conducted a complete case analysis.

Results

Of the 28 794 people who participated in at least one wave of HILDA between 2001 and 2014, 14 534 participated in all four waves 2011 to 2014, and 14 518 of these (99.9%) responded to the disability question in all four waves. A total of 8323 individuals satisfied the definition of disability acquisition or reported no disability in any of the four waves, making them eligible for inclusion in the analysis (Figure 5.3, Supplementary file 2).

Complete data for all baseline covariates, mediators and mental health score were available for 4305 individuals (52% of the eligible sample).

Baseline characteristics

At baseline, people who went on to acquire a disability were older than those without disability (mean age of 53 versus 41 years, Table 5.2). They had poorer education, with 33.6% not completing secondary education compared to 24.9% of those without disability, were more likely to be unemployed or not in the labour force (38.8% versus 22.7%), had a lower mean weekly income (AU$834 versus AU$987), and experienced greater financial hardship (34.1% versus 25.1% reported being very poor or just getting by). People with disabilities were more likely to be in a relationship (71.3% versus 65.3%) and have children (72.9% versus 59.4%), more likely to be current (21.2% versus 17.2%) or ex-smokers (31.8% versus 23.7%), less likely to exercise regularly (34.2% versus 37.0%), and had higher mean BMI (27.4 versus 25.8kg/m^2). At baseline, they also reported poorer mental health than those without disability (mean MHI score of 73.3 versus 77.6).

Table 5.2 Distribution of baseline characteristics for people who acquired a disability and the control sample (n=8323)

	Disability n=387		No disability n=7936	
	n	%	n	%
Age, years (mean (SD))	387	52.5 (18.1)	7936	41.2 (15.4)
Sex				
Men	193	49.9	3817	48.1
Women	194	50.1	4119	51.9
Country of birth				
Australia	297	76.7	6257	78.8
Other	90	23.3	1679	21.2
Parent occupation				
High skill	181	47.4	3988	51.1
Medium skill	129	33.8	2645	33.9
Low skill/never worked	72	18.9	1168	15.0
Missing	n=5		n=135	
Education				
Bachelor or higher	66	17.1	2184	27.5
Secondary, certificate, diploma	191	49.4	3777	47.6
Did not complete secondary	130	33.6	1975	24.9
Occupation				
High skill	75	19.4	2373	29.9
Medium skill	97	25.1	2395	30.2
Low skill	65	16.8	1364	17.2
Unemployed/not in the labour force	150	38.8	1799	22.7
Missing	n=0		n=5	
Income, weekly $AUD (mean (SD))	387	833.9 (476.8)	7936	986.9 (496.5)
Wealth				
High	138	35.7	2952	37.2
Medium	130	33.6	2616	33.0
Low	119	30.8	2368	29.8
Financial hardship				
Prosperous/very comfortable	42	12.0	1444	20.3
Reasonably comfortable	188	53.9	3874	54.6
Just getting by/very poor	119	34.1	1781	25.1
Missing	n=38		n=837	
Financial satisfaction (mean (SD))[a]	387	6.4 (2.3)	7931	6.7 (2.0)
Missing	n=0		n=5	
Housing tenure				
Outright owner	148	38.2	2107	26.6
Mortgager	126	32.6	3393	42.8
Other	113	29.2	2425	30.6
Missing	n=0		n=11	
Housing affordability				
Affordable	348	91.1	7263	92.4
Unaffordable	34	8.9	597	7.6
Missing	n=5		n=76	
Relationship				
Yes	276	71.3	5173	65.3
No	111	28.7	2755	34.8
Missing	n=0		n=8	
Children				

No	105	27.1	3224	40.6
Yes	282	72.9	4712	59.4
Social support (mean (SD))[b]	347	5.3 (1.1)	7017	5.6 (1.0)
Missing	n=40		n=919	
Frequency of socialising (mean (SD))[c]	347	3.8 (1.6)	7068	3.4 (1.4)
Missing	n=40		n=868	
Relationship with parents (mean (SD))[d]	197	7.9 (2.2)	5833	8.1 (2.0)
Missing	n=190		n=2103	
Alcohol consumption				
Never	59	16.9	1108	15.6
Rarely	129	36.9	2540	35.8
1-2 times/week	62	17.7	1547	21.8
≥3 times/week	100	28.6	1909	26.9
Missing	n=37		n=832	
Smoking				
Never smoked	164	47.0	4210	59.2
Ex-smoker	111	31.8	1684	23.7
Current	74	21.2	1222	17.2
Missing	n=38		n=820	
Physical activity				
≥4 times/week	120	34.2	2638	37.0
1-3 times/week	137	39.0	2998	42.1
<1 time/week	94	26.8	1493	20.9
Missing	n=36		n=807	
BMI, kg/m^2 (mean (SD))	332	27.4 (5.3)	6855	25.8 (5.0)
Missing	n=55		n=1081	
Mental health inventory (mean (SD))[e]	351	73.3 (18.9)	7125	77.6 (14.4)
Missing	n=36		n=811	

[a] Satisfaction with financial circumstances, ranked using an 11-point Likert scale ranging from “totally dissatisfied” to “totally satisfied”
[b] Constructed using the average of 10 questions addressing aspects of emotional support, each rated on a 7-point Likert scale
[c] Frequency of socialising with friends or relatives, rated on a 7-point Likert scale ranging from daily to less than once every three months
[d] Satisfaction with relationship with parents, rated on an 11-point Likert scale ranging from “completely dissatisfied” to “completely satisfied”
[e] Measured using five questions from the SF-36, each of which is scored using five response categories, and the total scores are transformed into a scale ranging from 0 to 100, with higher scores reflecting better mental health

Sequential causal mediation analysis

Interactions between the exposure and the following mediator variables were included in the regression models: material factors including occupation, housing affordability, housing tenure and satisfaction with financial circumstances, psychosocial factors including social support, frequency of socialising and relationship status, and behavioural factors including smoking, alcohol consumption, physical activity, BMI and diet.

The TCE of disability acquisition was estimated to be a 5.3-point reduction in MHI score (95% CI -6.8, -3.7, Table 5.3). In the sequential approach we first considered the mediated effect through material factors and estimated a mean 1.7 points decline (95%

CI -2.8, -0.6) in MHI was occurring through material factors, which corresponds to 32.1% of the total effect. We then considered the additional effect of psychosocial factors and found that 33.2% was explained by both material and psychosocial factors (NIE: -1.7, 95% CI -3.0, -0.5), and the additional effect of behavioural factors explained 38.6% of the decline (NIE: -2.0, 95% CI -3.4, -0.6).

Table 5.3 Total causal effect (TCE), natural direct effect (NDE) and natural indirect effect (NIE) of disability acquisition on mental health, with mediation through material factors, psychosocial and behavioural factors

	Material factors	+ psychosocial factors	+ behavioural factors
	Coef.* (95% CI)	Coef.* (95% CI)	Coef.* (95% CI)
TCE	-5.3 (-6.8, -3.7)	-5.3 (-6.8, -3.7)	-5.3 (-6.8, -3.7)
NDE	-3.6 (-5.4, -1.8)	-3.5 (-5.3, -1.7)	-3.2 (-5.1, -1.4)
NIE	-1.7 (-2.8, -0.6)	-1.7 (-3.0, -0.5)	-2.0 (-3.4, -0.6)
Proportion of effect explained (%)	32.1 (10.1, 54.1)	33.2 (8.5, 58.0)	38.6 (11.4, 65.9)

*These primary analysis results were obtained using multiple imputation using chained equations with 50 imputed datasets

Sensitivity analysis

The results were robust to the changes implied by the scenarios in the sensitivity analyses. The bias analysis demonstrated that the estimated indirect effects were unlikely to be explained by unmeasured confounding (Supplementary file 3). Removing disabled people with psychological impairments (41 of 387) attenuated the effect estimates, however, the proportion of the effect mediated increased slightly. For the complete case analysis, only small changes in the magnitude of individual coefficients were observed (Table 5.4).

Table 5.4 Results of the sensitivity analyses showing total causal effect (TCE), natural direct effect (NDE) and natural indirect effect (NIE) of disability acquisition on mental health, with mediation through material, psychosocial and behavioural factors

	Material factors	+ psychosocial factors	+ behavioural factors
	Coef. (95% CI)	Coef. (95% CI)	Coef. (95% CI)
Psychological impairments removed*			
TCE	-4.3 (-5.9, -2.7)	-4.3 (-5.9, -2.7)	-4.3 (-5.9, -2.7)
NDE	-2.7 (-4.6, -0.8)	-2.6 (-4.4, -0.8)	-2.2 (-4.0, -0.3)
NIE	-1.6 (-2.7, -0.5)	-1.7 (-3.0, -0.5)	-2.1 (-3.5, -0.8)
Proportion of effect explained (%)	37.7 (7.0, 68.4)	40.2 (8.8, 71.6)	49.8 (14.6, 84.9)
Complete case analysis			
TCE	-5.1 (-7.7, -2.5)	-5.1 (-7.7, -2.5)	-5.1 (-7.7, -2.5)
NDE	-3.3 (-5.8, -0.8)	-3.2 (-5.7, -0.7)	-3.1 (-5.7, -0.5)
NIE	-1.7 (-3.6, 0.1)	-1.9 (-4.1, 0.3)	-2.0 (-4.5, 0.6)
Proportion of effect explained (%)	34.4 (0.2, 68.7)	36.9 (-4.4, 78.2)	38.9 (-10.0, 87.8)

*These sensitivity analysis results were obtained using multiple imputation using chained equations with 50 imputed datasets

Discussion

Interpretation of findings

In this analysis, we found that 32% of the effect of disability acquisition on mental health was mediated by material factors, with only a negligible proportion explained by the addition of psychosocial factors and 5% by behavioural factors. This is consistent with the majority of the literature explaining health inequalities, which found that health differences are predominantly attributable to material factors [294, 300]. The results were not consistent with studies which had shown that psychosocial resources accounted for some of the effect of disability on depression [141, 302, 303], however these pathways are not mutually exclusive and it is possible that a large proportion of the effect through material factors is also operating through psychosocial pathways. Previous studies did not use a sequential causal mediation approach, which allows estimation of the additional contribution of psychosocial factors beyond the effect that is operating through material factors [205].

The effect sizes estimated in this study were of clinical significance. Study participants who acquired a disability experienced on average a five-point decline in mental health, exceeding the four to five-point difference considered to represent a clinically meaningful change [112, 113, 169]. The effect mediated through material factors was estimated to be 32.1%, which can be interpreted as the proportion of the mental health decline that could be avoided if people with disabilities experienced the same material socioeconomic circumstances as those without disabilities. About two fifths of the effect (38.6%) was explained by all three sets of mediators, leaving a large proportion of the effect unexplained – it seems unlikely that the remaining 61.4% of the total effect is not mediated by any other factors and is therefore a true “direct” effect. This is perhaps not surprising, as despite measuring a broad range of socioeconomic characteristics, these measures capture only a snapshot of people’s socioeconomic experiences at one point in time [313] and do not capture the broader structural, political and economic processes they experience [294]. Additionally, there were some factors that were not recorded in HILDA which could be important mediators, such as experience of discrimination, sense of personal control (asked only in 2011 and 2015), psychosocial working conditions and personal-work balance (asked only for those people who were employed). Therefore the effect operating through psychosocial pathways may be underestimated.

Strengths and limitations

This study used data from a large longitudinal survey in Australia. The longitudinal nature of the data meant that we could characterise disability acquisition, based on a sample of

people who reported no disability for two waves followed immediately by two waves of disability. Furthermore, we could measure disability acquisition, mediators and mental health at different time points, to establish a temporal sequence between them, and control for prior values of the mediator and mental health score so that the results can be interpreted as effects of changes in the mediators on the outcome. We used causal sequential mediation methods, which can address the limitations of traditional mediation methods, generating unbiased estimates of mediation through multiple causally-ordered mediators, given a set of clearly specified assumptions of no confounding.

There were also limitations with this study. The analysis rests on several strong assumptions about no confounding between disability acquisition, mediators and mental health. We used inverse probability weighting to account for (measured) confounding of the disability-mental health and the disability-mediator relationships. For the assumption relating to no uncontrolled confounding of the mediator-outcome relationship, we conducted a bias analysis which suggested that the NDE and NIE were unlikely to be explained by confounding by measured or unmeasured variables. The weighting approach is sensitive to outcome model misspecification, which can lead to biased estimates of natural direct and indirect effects, however this approach was deemed most appropriate because of the large number of mediators [205] Furthermore, to ensure best specification of the outcome model, interactions between the exposure and each mediator were considered and tested. There were strong assumptions about the causal ordering of the mediators. The direction of causality between these contributory factors is likely to be bi-directional, for example, the relationship between employment and social support. This may have led to overestimation of the proportion of the effect operating through material factors if these are consequences of psychosocial and behavioural factors, rather than a cause of them. However, for most of the variables considered, the effect is likely to be causally ordered from material to psychosocial to behavioural factors. There was a large proportion of missing data and this was higher in participants with poorer mental health and greater socioeconomic disadvantage, however, the use of multiple imputation as the primary analysis should have reduced this selection bias.

The concepts of disability and mental health are related, which makes it difficult to isolate the causal effect of one on the other. To address this limitation, firstly we chose to use the mental health subscale of the SF-36 health questionnaire (MHI-5), rather than the summary mental health score (MCS), therefore selecting parts of the SF-36 questionnaire that were less likely to overlap with the definition of disability. Furthermore, we conducted a sensitivity analysis in which we excluded people with psychological impairments to further minimise overlap between the concepts, which did not change the

interpretation of the results though the magnitude of effect estimates was slightly attenuated. When we excluded people with psychological impairments, the proportion of the effect mediated was slightly larger. It is plausible that the mechanisms are different for people with psychological impairments compared to other types of disability, though the relative proportion through each of the three pathways was similar. It would be interesting to look at differences in these effects according to types of impairments, however we lacked power to examine differences by disability characteristics. Finally, people with severe disabilities are less likely to participate in HILDA, therefore our results are likely to underestimate the population effect of disability acquisition on mental health.

Conclusions

The finding that the effect of disability acquisition on mental health operates predominantly through material factors has important policy implications. These results highlight that social policy reforms that reduce socioeconomic disadvantage among people who acquire a disability will improve mental health. This could be achieved through better social protection, including income support, but also through improved educational and employment opportunities for people with disabilities and access to affordable housing. It is important to further disentangle the mechanisms involved in the material pathway, to better understand the relative importance of specific factors and which social determinants are driving the mental health inequalities. This will help to better target policy interventions to improved the mental health of people with disabilities.

5.1.3 Supplementary material for section 5.1.2

Supplementary file 1. Causal mediation analysis

Traditional regression-based approaches to mediation analysis are prone to biased estimates of direct and indirect effects because (1) they presuppose no interactions between the exposure and mediator, (2) cannot deal with non-linearities, and (3) make strong assumptions about the absence of confounding [176]. The causal mediation analysis approach that we have used seeks to address the limitations of the traditional approach by supplying definitions of direct and indirect effects based on the potential outcomes framework that allows for decomposition of the total effect in the presence of non-linearities and interactions [191, 192]. The potential outcomes approach has been used to clarify what effects are being estimated and under what assumptions, which were often not brought to focus in traditional methods. In this framework, the estimation of effects is achieved by comparing potential outcomes under different hypothetical values of the exposure and the mediator.

Definitions

For a single mediator, Robins and Greenland (1992) and Pearl (2001) specified definitions of these effects based on differences between possibly counterfactual ("counter to fact") scenarios with defined values of the exposure and mediator [191, 192]. Consider an exposure A, a mediator M and an outcome Y. If the binary exposure has as its possible values a (exposed) and a^* (unexposed), let M_a denote the potential value of the mediator if the exposure was set to a, and Y_{am} the potential value of the outcome if the exposure were set to a and the mediator set to m. We use the *composition assumption* to define the single-subscripted potential value Y_a as $Y_a = Y_{aMa}$. The Total Causal Effect (TCE) is the difference between the outcome when the individual is exposed and the outcome when the individual is unexposed, and can be written as $E[Y_a] - E[Y_{a^*}] = E\left[Y_{aM_a}\right] - E\left[Y_{a^*M_{a^*}}\right]$. The Natural Direct Effect (NDE) is the difference between the outcome when the individual is exposed and the mediator set to the value it would have taken had the individual been unexposed and the outcome when the individual is unexposed and the mediator is set to its natural unexposed value, defined as $E\left[Y_{aM_{a^*}}\right] - E\left[Y_{a^*M_{a^*}}\right]$. The Natural Indirect Effect (NIE) is the difference between the outcome when the individual is exposed and the mediator is set to its natural exposed value and the outcome when the individual is exposed and the mediator set to the value it would have taken had the individual been unexposed, defined as $E\left[Y_{aM_a}\right] - E\left[Y_{aM_{a^*}}\right]$. From these definitions, we see that TCE = NDE + NIE so that the total causal effect decomposes into a direct effect through the exposure (where the mediator takes the value it would have taken if the individual was unexposed) and an indirect through the mediator (where all individuals are assumed to be exposed).

Assumptions

Under several no confounding assumptions, the counterfactual definitions of the direct and indirect effects can be related to empirical expressions that can be estimated from data.

i) No unmeasured confounding of the exposure-outcome relationship (conditional on covariates C);

ii) No unmeasured confounding of the mediator-outcome relationship (conditional on covariates C and exposure A);

iii) No unmeasured confounding of the exposure-mediator relationship (conditional on covariates C);

iv) No exposure-induced mediator-outcomes confounding (conditional on covariates C).

Multiple mediators

This framework has recently been extended to allow for the examination of exposure-outcome associations where there are potentially multiple mediators even if the mediators influence each other in a series of distinct causal paths from exposure to outcome, as they do in our example. Examining the natural indirect effect of each mediator separately is not an appropriate strategy if the goal is to partition the total causal effect because it will not be equal to the sum of the individual mediated effects unless the mediators are independent conditional on the exposure. VanderWeele and Vansteelandt (2014) proposed estimating the joint indirect effect through all mediators, thus quantifying all indirect pathways *en bloc* [205]. Furthermore, if causal ordering between the mediators is known, then further decomposition can be made by sequentially adding each mediator at a time, however it may not be possible to identify – and thus estimate –indirect pathways through all distinct sub-paths in the causal diagram even if all variables are measured without error and we assume that relationships between potential outcomes can be captured by a nonparametric structural equation model [205].

In our analysis, we have three causally-ordered mediators: material factors, which affect psychosocial factors, which in turn affect behavioural factors. Given the assumptions about their causal ordering, the indirect effects of disability acquisition on mental health through these mediators can be estimated sequentially. Firstly, the NIE through material factors can be estimated, which includes paths occurring through its causal descendants (psychosocial and behavioural factors). Conversely the NDE through all other pathways can be estimated, which includes the effect occurring through none of the mediators as well as the effects through psychosocial and behavioural factors that do not operate through material factors (see Figure 2). Secondly, the NIE through both material and psychosocial factors can be estimated (including paths that subsequently include behavioural factors), which estimates the contribution of psychosocial factors in addition to material factors. Here, the NDE estimates the effect through paths other than material and psychosocial factors. Thirdly, the NIE through material, psychosocial and behavioural factors can be estimated which represents the joint effect of all three mediators, and the NDE represents the direct effect of disability on mental health not occurring through any of these pathways, that is, through any material, psychological or behavioural factors. Taking the difference between the second (material + psychological) and third (material + psychological + behavioural) NIE's produces an NIE that can be

interpreted as the indirect effect of disability acquisition on mental health that passes only through behavioural factors. This is the only one of the three factors for which such a calculation is possible since both material and psychological factors have multiple paths to the outcome that Avin et al (2005) showed using the theory of graphical models cannot be distinguished [314].

Estimation

We used a weighting approach that required fitting models for both the outcome and the exposure. A logistic regression model for disability acquisition including all baseline covariates was fitted, and predicted values were used to estimate a propensity score for each individual in the sample and generate Inverse Probability Weights (IPW) [158]. Quantifying the marginal NDE and NIE required the estimation of the population averages of three potential outcomes: $E\left[Y_{aM_a}\right]$, $E\left[Y_{a^*M_{a^*}}\right]$, and $E\left[Y_{aM_{a^*}}\right]$ [205]. All predicted values were generated for the sample of people without disability, generating predicted values based on alternate hypothetical scenarios about disability acquisition and values of material, psychosocial and behavioural factors. The IPW were used to shift the distribution of confounders from the mean nested counterfactual for people without disability, the unexposed, to the whole population. $E\left[Y_{a^*M_{a^*}}\right]$ was estimated by taking the weighted mean mental health score among people without a disability. $E\left[Y_{aM_a}\right]$ was estimated as a weighted average of the predicted mental health scores that people without disability would have experienced had they acquired a disability. This was estimated by fitting a linear regression model for mental health including disability acquisition and all baseline covariates, but not the mediator. Coefficients from the model were used to predict each person's mental health score, fixing the covariate value of the disability acquisition variable to estimate their potential outcome had they acquired a disability. $E\left[Y_{aM_{a^*}}\right]$ was estimated as a weighted average of predicted mental health scores that people without a disability would have experienced had they acquired a disability but with the mediator value that would have naturally occurred if they had remained disability-free. This was estimated by fitting a linear regression for mental health, including disability acquisition, all baseline covariates and the mediator, and fixing the disability coefficient to estimate the potential outcome had they acquired a disability but using the individual's own values (that it would naturally take with no disability) of the mediator. This approach was extended to account for each set of mediators in turn.

Supplementary file 2. Missing data

Eligibility criteria

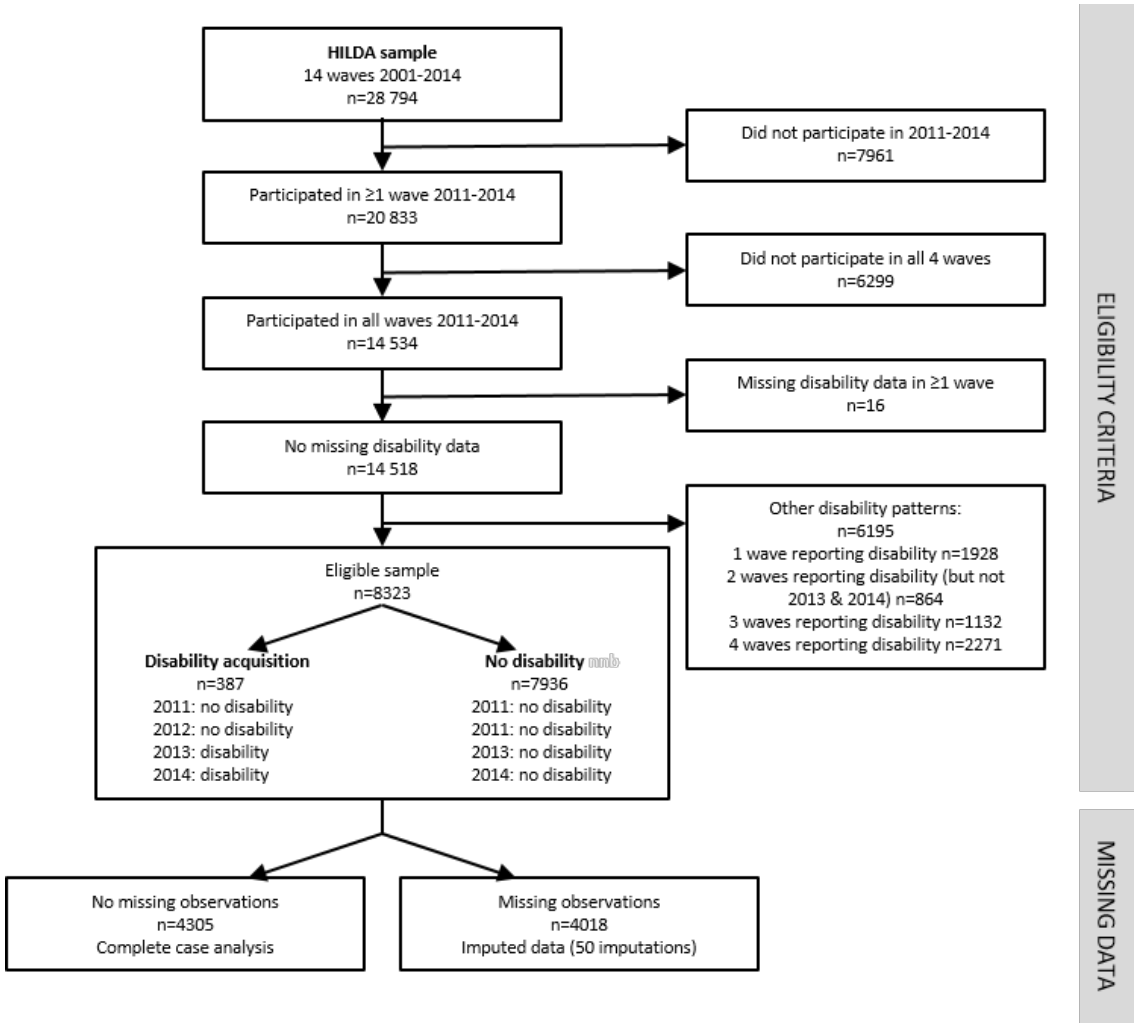

Figure 5.3 Flow diagram showing sample selection and missing data

Missing data

There were missing observations for the outcome, as well as for some of the baseline covariates and mediators (Table 5.5). Of the 8323 individuals eligible for inclusion, there were 4305 with complete data (52%) and 4018 with at least one missing observation (48%).

Table 5.5 Missing observations for each variable in the analysis

		Missing	
	Wave	n	%
Outcome			
Mental health	2014	805	9.7
Baseline covariates			
Mental health	2011	847	10.2
Parental occupation	2011	140	1.7
Occupation	2011	5	0.1
Financial hardship	2011	875	10.5
Satisfaction with finances	2011	5	0.1
Housing tenure	2011	11	0.1
Housing affordability	2011	81	1.0
Relationship status	2011	8	0.1
Social support	2011	959	11.5
Socialising	2011	908	10.9
Relationship with parents	2011	2,293	27.6
Alcohol consumption	2011	869	10.4
Smoking	2011	858	10.3
Physical activity	2011	843	10.1
Body Mass Index (BMI)	2011	1,136	13.6
Mediators			
Occupational skill level	2013	4	0.0
Financial hardship	2013	898	10.8
Satisfaction with finances	2013	2	0.0
Housing tenure	2013	8	0.1
Housing affordability	2013	74	0.9
Relationship status	2013	5	0.1
Social support	2013	974	11.7
Frequency of socialising	2013	941	11.3
Relationship with parents	2013	2,488	29.9
Alcohol consumption	2013	915	11.0
Smoking	2013	896	10.8
Physical activity	2013	875	10.5
BMI	2013	1,087	13.1
Sleep quality	2013	931	11.2
Healthy diet index	2013	894	10.7

Distribution of baseline covariates between people with and without missing data

There was evidence that people with missing observations were systematically different from those without missing observations at baseline (Table 5.6). People with incomplete data were older, more likely to be born outside of Australia, and experienced greater socioeconomic disadvantage across almost every covariate examined. Therefore, missingness was associated with the values of measured variables, suggesting that the data were not missing completely at random (MCAR). We deemed that it was plausible that the data may be missing at random (MAR - that the probability of missingness was conditional on the observed data but not the missing data) [235].

Table 5.6 Comparison of fully observed baseline variables between people with complete (n=4305) and incomplete data (n=4018)

	Complete data n=4305		Incomplete data n=4018		P value
	n	%	n	%	
Age, years (mean (SD))	4305	38.3 (12.3)	4018	45.5 (18.0)	<0.001
Sex					
Men	2049	47.6	1961	48.8	
Women	2256	52.4	2057	51.2	0.270
Country of birth					
Australia	3503	81.4	3051	75.9	
Other	802	18.6	967	24.1	<0.001
Parent occupation					
High skill	2306	53.6	1863	48.0	
Medium skill	1462	34.0	1312	33.8	
Low skill/never worked	537	12.5	703	18.1	<0.001
Education					
Bachelor or higher	1317	30.6	933	23.2	
Secondary, certificate, diploma	2114	49.1	1854	46.1	
Did not complete secondary	874	20.3	1231	30.6	<0.001
Occupation					
High skill	1461	34.0	987	24.6	
Medium skill	1354	31.5	1138	28.3	
Low skill	751	17.5	678	16.9	
Unemployed/not in the labour force	736	17.1	1213	30.2	<0.001
Income, weekly $AUD (mean (SD))	4305	3.5 (1.3)	4018	3.1 (1.4)	<0.001
Wealth					
High	1575	36.6	1515	37.7	
Medium	1490	34.6	1256	31.3	
Low	1240	28.8	1247	31.0	0.004
Financial hardship					
Prosperous/very comfortable	898	20.9	588	18.7	
Reasonably comfortable	2341	54.4	1721	54.8	
Just getting by/very poor	1066	24.8	834	26.5	0.039
Financial satisfaction (mean (SD))[a]	4305	6.7 (2.0)	4013	6.6 (2.1)	0.040
Housing tenure					
Outright owner	933	21.7	1322	33.0	
Mortgager	2075	48.2	1444	36.0	
Other	1297	30.1	1241	31.0	<0.001
Housing affordability					
Affordable	3984	92.5	3627	92.1	
Unaffordable	321	7.5	310	7.9	0.476
Relationship					
Yes	2910	67.6	2539	63.3	
No	1395	32.4	1471	36.7	<0.001
Children					
No	1901	44.2	1428	35.5	
Yes	2404	55.8	2590	64.5	<0.001
Social support (mean (SD))[b]	4305	5.7 (0.9)	3059	5.6 (1.0)	<0.001
Frequency of socialising (mean (SD))[c]	4305	3.4 (1.4)	3110	3.5 (1.5)	0.002
Relationship with parents (mean (SD))[d]	4305	8.2 (1.9)	1725	8.0 (2.1)	0.012
Alcohol consumption					
Never	632	14.7	535	17.0	

Rarely	1565	36.4	1104	35.1	
1-2 times/week	988	23.0	621	19.7	
≥3 times/week	1120	26.0	889	28.2	<0.001
Smoking					
Never smoked	2650	61.6	1724	54.6	
Ex-smoker	944	21.9	851	26.9	
Current	711	16.5	585	18.5	<0.001
Physical activity					
≥4 times/week	1560	36.2	1198	37.7	
1-3 times/week	1868	43.4	1267	39.9	
<1 time/week	877	20.4	710	22.4	0.007
BMI, kg/m^2 (mean (SD))	4305	25.7 (5.0)	2882	26.1 (5.2)	<0.001
Mental health inventory (mean (SD))[e]	4305	77.2 (14.3)	3171	77.6 (15.1)	0.227

[a] Satisfaction with financial circumstances, ranked using an 11-point Likert scale ranging from "totally dissatisfied" to "totally satisfied"
[b] Constructed using the average of 10 questions addressing aspects of emotional support, each rated on a 7-point Likert scale
[c] Frequency of socialising with friends or relatives, rated on a 7-point Likert scale ranging from daily to less than once every three months
[d] Satisfaction with relationship with parents, rated on an 11-point Likert scale ranging from "completely dissatisfied" to "completely satisfied"
[e] Measured using five questions from the SF-36, each of which is scored using five response categories, and the total scores are transformed into a scale ranging from 0 to 100, with higher scores reflecting better mental health

Multiple imputation

Missing values were imputed by drawing from the posterior predictive distribution of the unobserved data given the observed data. We created 50 imputed datasets using chained equations, in which univariate imputation models were specified for each variable with missing data, and the model cycled through each variable with missing data in turn (*mi impute chained* command in Stata). All variables included in the epidemiological analysis were included in the imputation model, as well as auxiliary variables, additional variables which were associated with the variable with missing data or predicted missingness of that variable. For each variable with missing data, the auxiliary variables included in the imputation model are listed in Table 5.7. The analysis was conducted on each of the 50 imputed datasets, and the estimates were combined using Rubin's Rules to provide an overall estimate with a standard error, which takes into account the variability between the estimates from the imputed datasets [236].

Table 5.7 Variables included in the imputation model for each covariate with missing data

Covariate	Variables in the imputation model
Mental health (2011, 2014)	
Mental health	All variables in the epidemiological analysis, mental health (2010, 2012, 2013), self-rated health (2010-2014), material factors (2010, 2012, 2014), psychological factors (2010, 2012, 2014), behavioural factors (2010, 2012, 2014), SEIFA score (2011)
Material factors (2011, 2014)	
Parental occupation (2011 only) Occupation Financial hardship Satisfaction with finances Housing tenure Housing affordability	All variables in the epidemiological analysis, mental health (2010, 2012, 2013), self-rated health (2010-2014), material factors (2010, 2012, 2014), SEIFA score (2011)
Psychosocial factors (2011, 2014)	
Relationship status Social support Socialising Relationship with parents	All variables in the epidemiological analysis, mental health (2010, 2012, 2013), self-rated health (2010-2014), psychological factors (2010, 2012, 2014), SEIFA score (2011)
Behavioural factors (2011, 2014)	
Alcohol consumption Smoking Physical activity BMI Sleep Diet	All variables in the epidemiological analysis, mental health (2010, 2012, 2013), self-rated health (2010-2014), behavioural factors (2010, 2012, 2014), SEIFA score (2011)

Supplementary file 3. Quantitative bias analysis

Bias analysis for unmeasured mediator-outcome confounding

We performed a bias analysis that assessed the sensitivity of the results to the assumption of no unmeasured confounding of the mediator-outcome association, according to the method described by VanderWeele (2010) [234]. The bias analysis involved estimating the effect of a potential unmeasured binary confounder (denoted U) of the association between one of the mediators and mental health on the NDE and NIE. This was repeated for a range of plausible values of gamma, a parameter measuring the magnitude of the combined effect of U on both the mediators and mental health, and delta, a parameter measuring the difference in the prevalence of U between those with and without disability. Plausible values of gamma were selected, ranging from -1.8 to 1.8, based on the magnitude of the largest coefficient of the measured confounders in the multivariable linear regression model for mental health (those in public rental compared to home owners: estimated mean difference=1.8). Plausible values of delta

were selected to range between 0 and 15%, based on the largest difference in prevalence of observed baseline covariates between people who acquired a disability and those who did not (difference in prevalence of never smoking was 15%).

Results of bias analysis

Figures 5.4 and 5.5 give the values of γ and δ that would eliminate the effects. The solid line curve represents the values of γ and δ that would reverse the sign of the point estimate for the NDE and NIE, and thus eliminate the effect. In Figure 5.4, values of δ and γ that lie below the solid line would reverse the sign of the NDE point estimate, and those below the dashed line would make the NDE no longer statistically significant. IN Figure 5.5, values of δ and γ that lie above the solid line would reverse the sign of the NIE point estimate, and those above the dashed line would make the NIE no longer statistically significant. The dashed lines represent the 95% confidence intervals around the point estimate, therefore the values of γ and δ that would make the estimates of the NDE and NIE no longer statistically significant.

For both the NDE (Figure 5.4) and the NIE (Figure 5.5), much larger values of γ and δ than those considered plausible would be necessary to eliminate the effect or make the effects no longer statistically significant. Therefore, the estimated direct and indirect effects seem to be unlikely due to the presence of mediator-outcome confounding.

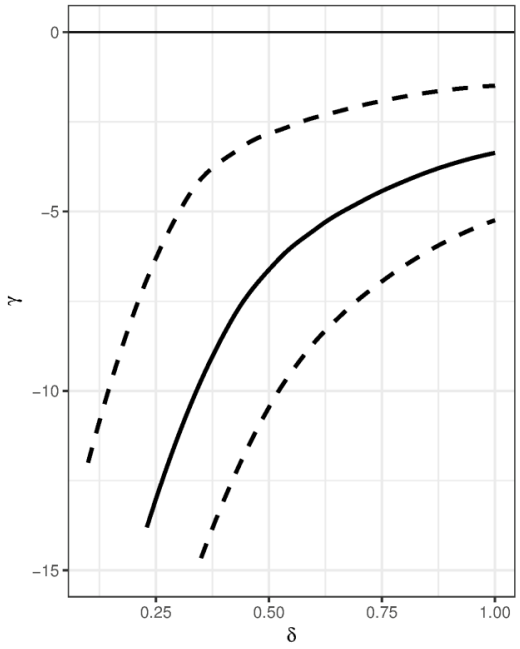

Figure 5.4 Values of δ and γ that would reverse the sign of the NDE point estimate

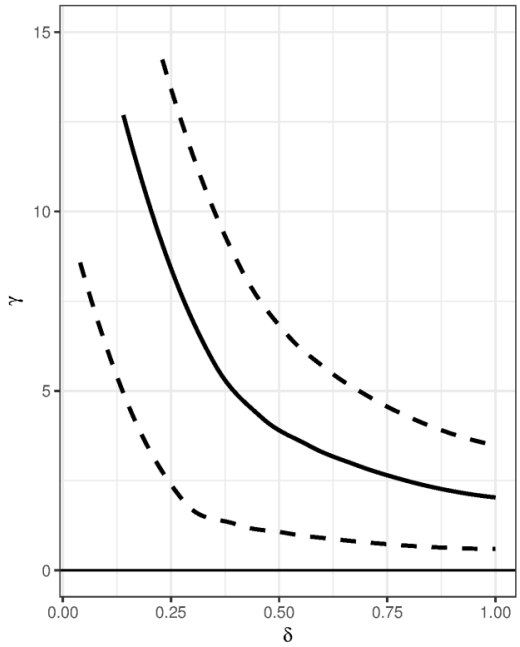

Figure 5.5 Values of δ and γ would reverse the sign of the NIE point estimate

5.2 Interventional effects to estimate direct and indirect effects through multiple mediators

5.2.1 Introduction

After establishing that a third of the effect of disability acquisition on mental health was explained by material socio-economic factors in the previous causal mediation analysis, this analysis aims to further disentangle which specific factors are driving the effect. A limitation of the causal mediation approach estimating natural direct and indirect effects, as detailed in section 3.6.6, is that with multiple causally ordered mediators, it is not possible to estimate the indirect effects operating through distinct pathways. In Section 5.2.2 of this chapter, I apply a novel approach, estimating interventional direct and indirect effects to quantify how much of the effect of disability acquisition on mental health operates through two distinct (but causally-related) material socio-economic characteristics: employment and income. In contrast to natural effects, interventional effects enable estimation of indirect effects through distinct pathways even if the mediators are causally related.

5.2.2 How much of the effect of disability acquisition on mental health is mediated through employment and income? A causal mediation analysis quantifying interventional indirect effects.

Background

In Australia, 18% of the population live with a disability [6]. People with disabilities experience large health inequalities compared to those without disability, a large proportion of which are unrelated to the impairment causing the disability [28, 81, 252, 315]. They also report poorer mental health, including high rates of depression and anxiety, psychological distress and lower mental well-being scores [1, 3, 83, 84, 118, 121, 126, 141]. The relationship between disability and poor mental health is likely to be bidirectional, with disability causing a deterioration in people's mental health and poor mental health increasing risk of disability [316]. Several longitudinal studies have quantified the causal effect of disability on mental health, using repeated measures data and examining incident disability to isolate the direction of effect [83, 84, 101, 116, 124, 126, 129, 141]. These studies have provided a body of evidence suggesting that disability leads to a deterioration in people's mental health, but the underlying mechanism is poorly understood [4, 317]. This is an important omission as understanding the pathways by which the effect operates may highlight targets for interventions to mitigate the adverse effects of disability on people's mental health [183].

The social model of disability theorises that disability is caused by the physical and social environment in which people live [29]. In contrast to the medical model of disability in which disability is determined by people's health conditions or impairments, it shifts the focus away from features of individuals, making a clear distinction between impairments and disability. The social model conceptualises disability as a socially-created problem caused by attitudes and other features of the social environment which disadvantages people with disabilities and excludes them from fully participating in society [49]. In this conceptualisation, mental health inequalities exist as a result of the circumstances in which people with disabilities live; therefore disability-related mental health inequalities are likely to operate through socio-economic pathways.

There are a number of different mechanisms by which socio-economic characteristics could explain the mental health inequalities experienced by people with disabilities. Theoretical and empirical studies examining the causes of income-related health inequalities have suggested three main mechanistic pathways: material, psychosocial, and behavioural frameworks [293-296]. The material framework posits that material conditions such as poverty and economic deprivation have direct effects on health outcomes but also indirect effects through access to better living conditions such as health care [297]. The psychosocial mechanism operates through differential exposure to psychosocial resources and stress-related risk factors, which causes differences in health [298]. Finally, differences in health-related behaviours are likely to affect health, such as risk taking behaviour, alcohol consumption, smoking, diet and physical activity [299]. Most empirical studies examining these mechanisms have found evidence of a greater relative importance of material factors in explaining income-related health inequalities [294, 300].

In a previous analysis (section 5.1.2), I quantified how much of the effect of disability acquisition on mental health operated through each of these three mechanistic pathways, finding that the effect operated predominantly through material factors rather than psychosocial or behavioural factors [4]. The results suggested that 39% of the effect of disability on mental health could be explained by socio-economic circumstances, with the largest proportion of the effect (32%) mediated by material factors including employment, occupation, income, financial hardship, financial satisfaction, and housing tenure and affordability. The analysis used a sequential approach which, in the presence of multiple causally-ordered mediators, can estimate the indirect effect through the first mediator and the indirect effect operating through both the first and subsequent mediators jointly. This method cannot estimate the effect through each of the multiple mediators separately without additional strong assumptions. Furthermore, in this

analysis seven different material socio-economic characteristics were examined jointly, therefore there remains a lack of understanding about which specific material factors drive the effect of disability acquisition on mental health, limiting the development of effective interventions to improve the mental health of people with disabilities.

Novel causal mediation methods, known as interventional effects, allow quantification of indirect effects through distinct pathways in the presence of multiple mediators even if the structural dependence between the mediators is unknown [206] and can model the effects of population-level interventions, which may be more relevant for policy [208]. In this analysis, using data from four waves of the HILDA Survey (2011-2014) and restricting the sample to the working age population (25 to 64 years), I aim to further disentangle the mechanism linking disability and poor mental health, quantifying interventional effects for mediation analysis to examine path-specific effects occurring through two material factors: employment and income.

Methods

Data source

I used data from the HILDA Survey, a large longitudinal study of Australian households, which collects information on a wide range of demographic, social, economic, and health characteristics. The initial survey was conducted in 2001, with annual ongoing follow-up surveys of the original participants. The original sample included 13,969 participants from 7682 households, randomly sampled using a national probability sample of private dwellings. A top-up sample was added in 2011 to maintain representativeness, leading to a sample size after 14 waves of 28,794 people. On average across all waves of the survey, response rates were 80% and attrition was 6% between waves. This analysis was restricted to working age participants (25 to 64 years) because it examined the effect operating through employment. I chose to use data from the same four waves of the survey as were used in the previous analysis described in section 5.1.2 (2011 to 2014) to establish a temporal sequence between baseline characteristics (measured in 2011), disability acquisition (occurring between 2012 and 2013), the mediators (measured in 2013), and mental health (measured in 2014, Figure 5.6).

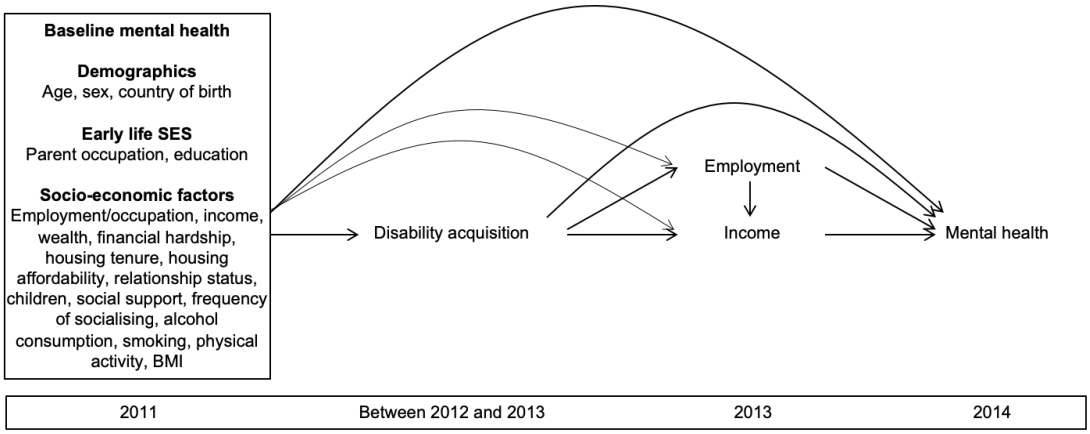

Figure 5.6 Causal diagram illustrating postulated causal relationships between disability acquisition and mental health

Mental health

Mental health was assessed in the final wave (2014) using the MHI, a subscale of the SF-36 - a widely used general health questionnaire that has been validated in the Australian population using data from the HILDA Survey [168]. The MHI is a well validated and reliable measure of mental health status [169]. It measures symptoms of depression, anxiety and psychological well-being and has been shown to be psychometrically sound [171] and an effective screening tool for mood and anxiety disorders and severe depressive symptomatology [172-175]. The MHI includes five items relating to mental health over the previous four weeks, each scored using five response categories. Total scores were transformed into a scale with a mean score of 74 (range: 0-100), with higher scores reflecting better mental health. Previous research has suggested that a difference of four to five-points on the MHI scale is likely to reflect a minimally important clinical difference in mental health [112, 113].

Disability acquisition

Information on disability was collected in every wave, using a single question defining disability as “an impairment, disability or long-term health condition, which restricts everyday activities that had lasted for six months or more”. Disability acquisition was defined as two waves reporting no disability (2011 and 2012), followed immediately by two consecutive waves reporting a disability (2013 and 2014). Two consecutive waves of disability were used in order to exclude people with transient disability, a definition used in previous studies examining disability acquisition [120, 263, 264] and all analyses in this thesis. Participants who acquired a disability were compared to those who reported no disability in any of the four waves. Any other patterns of exposure, such as

a single wave reporting a disability, were excluded. Eligibility for inclusion required participation and response to the disability question at all four waves.

Mediators

Two mediator variables were examined, measured in 2013, employment status and income. Employment was categorised as a binary variable, defined as people who were currently employed versus those who were either unemployed (actively seeking employment in the previous four weeks) or not in the labour force (not actively seeking employment). Income was measured as a continuous variable describing people's weekly disposable income (in $AUD) truncated at the 1st and 99th percentiles.

Baseline covariates

Baseline variables that were potential confounders of the association between disability and mental health were included as covariates in regression models. This included all variables that were common causes of both the exposure and outcome, assessed using a causal diagram (Figure 5.6). Baseline covariates were measured 2011, as a measure of people's circumstances prior to disability acquisition. Demographic characteristics included age, sex and country of birth (Australia; other) and socio-economic characteristics included parental occupation (high skill; medium skill; low skill or not in the labour force), education (bachelor degree and above; completion of secondary education; did not complete secondary), occupation (high skill, medium skill, low skill, unemployed/not in the labour force), income (weekly personal disposable income), wealth (tertiles of net wealth), financial hardship (prosperous/very comfortable; reasonably comfortable; just getting along/poor/very poor), housing tenure (outright owner; mortgager; renter), housing unaffordability (defined as households in the lowest 40% of the income distribution with housing costs exceeding 30% of their gross income), relationship status (yes/no), children (yes/no), social support (constructed using the average of 10 questions addressing aspects of emotional support, each rated on a 7-point Likert scale) [247], frequency of socialising with friends or relatives (rated on a 7-point Likert scale ranging from less than once every three months to daily), smoking status (never; ex-smoker; current), alcohol consumption (never; rarely; 1-2 days per week; >2 days per week), physical activity (>3 times per week; 1-3 times per week; less than once a week) and BMI (kg/m^2).

Mediation analysis

Mediation analysis aims to determine the extent to which the effect of an exposure on an outcome is due to the effect of the exposure on an intermediate variable (the mediator), which in turn influences the outcome. It aims to decompose the total effect of

the exposure on the outcome into the portion of the effect that acts through the mediator, the *indirect effect*, and the portion of the effect of exposure on outcome through other mechanisms, the *direct effect* ("direct" in the sense that it by-passes the putative mediator) [176].

There has recently been a major revival of mediation analysis with the development of counterfactual-based definitions of direct and indirect effects, generating new causal inference approaches for mediation analysis which address the problems of bias encountered by traditional approaches to mediation. The counterfactual framework has been used to clarify what effects are being estimated and the assumptions necessary to identify direct and indirect effects from data, which were often not brought to focus in traditional methods [191, 192]. This causal inference framework estimates the TCE and decomposes it into the NDE and the NIE under a set of identification assumptions, which include four assumptions about no unmeasured confounding: of (1) the exposure-outcome relationship, (2) the mediator-outcome relationship, (3) the exposure-mediator relationship, and (4) no exposure-induced mediator-outcome confounding [203].

As described in section 3.6.5, the NDE estimates the effect of an exposure on the outcome while fixing the mediator to the value it would naturally take in the absence of the exposure, thereby representing the effect of the exposure on the outcome through pathways other than the mediator of interest. It is defined as:

$$NDE = E[Y_{1M_0} - Y_{0M_0}],$$

where Y denotes the outcome of interest, A the binary exposure with two values A=1 (exposed) and A=0 (unexposed), and M the mediator. Counterfactuals are denoted by Y_{aM_a}, for example Y_{1M_1}, representing the value of the outcome that would have been observed had the individual been exposed and the mediator was set to the value it would have naturally taken if the individual were exposed ($M_{a=1}$).

The NIE estimates the effect of the mediator on the outcome while fixing the exposure to a particular value for the whole population; it represents the change we would expect to observe in a given outcome if we would change the mediator to the value it would take under the counterfactual value of the exposure but without actually changing the exposure. It is defined as:

$$NIE = E[Y_{1M_1} - Y_{1M_0}].$$

In situations where multiple mediators are of interest, a sequential approach to mediation analysis can be taken, as in the analysis presented in section 5.1.2, which allows decomposition of the TCE into a NDE and NIE through multiple mediators and estimation

of path-specific effects, but requires assumptions to be made about the causal ordering between the mediators [205].

Recently there has been substantial debate about the usefulness of estimating the NDE and NIE because these *natural* effect estimates cannot be interpreted as the effects of real life interventions [202] and the approach relies on very strong identification assumptions, particularly the use of cross-world counterfactuals [201]. The estimation of natural direct and indirect effects relies on cross-world counterfactuals such as Y_{10} which represents the expected value of the outcome had the person been exposed but the mediator set to the value it would have taken if the individual were not exposed. This counterfactual represents the predicted value of the outcome under conflicting interventions on the exposure, logically incompatible exposure states which can therefore neither be enforced experimentally nor tested empirically [192, 318]. Furthermore, in situations with multiple mediators, there is no insight into indirect effects through distinct pathways without additional strong assumptions. Vansteelandt and Daniel (2017) present an alternative approach to mediation analysis, which estimates IDEs and IIEs [206]. These are not defined in terms of cross-world counterfactuals and can therefore be identified under weaker assumptions (they do not require assumption (4)). As described in section 3.6.7, the IDE estimates the effect on the outcome of changing the exposure under a stochastic intervention G_0 which sets the value of the mediator for each person at a value randomly drawn from the conditional distribution of values of the mediator among the unexposed, given the observed covariates. It is defined as:

$$IDE = Y_{1G_0} - Y_{0G_0}.$$

The IIE estimates the effect on the outcome of shifting the mediator from the distribution that would be expected if everyone were unexposed to that if everyone were exposed, i.e. the effect of equalising the distribution of the mediator in one exposure group to be the same as that of another exposure group, defined as:

$$IIE = Y_{1G_1} - Y_{1G_0}.$$

Interventional effects are particularly attractive for the analysis of multiple mediators because they allow quantification of effects via distinct pathways even if the structural dependence between mediators is unknown [206]. Furthermore, interventional effects quantify average population-level effects representing the effect of shifting the distribution of the mediator in the population to what would be expected if everyone in the population was exposed. This population average effect is a more realistic estimand than natural effects which estimate individual causal effects [208].

Statistical analysis

The TCE was estimated by fitting a linear regression model for mental health conditional on disability acquisition and baseline covariates, generating a predicted mean mental health score for each individual had they acquired a disability and had they remained disability-free.

The interventional effects approach estimates the IDE and IIE. Assuming that employment (M1) causally precedes income (M2, Figure 1), the interventional effects approach enabled estimation of the IIE through employment, the IIE through income and the IIE resulting from the dependence between employment and income.

Four models were fitted to the data to estimate the IDE and IIEs:

Model 1: a linear regression model for mental health conditional on disability acquisition, employment, income, and all baseline covariates, including an interaction between disability acquisition and employment.

Model 2: a logistic regression model for employment conditional on disability acquisition and baseline covariates.

Model 3: a linear regression model for income conditional on disability acquisition, employment, and all baseline covariates, including an interaction between disability acquisition and employment (model for M2 conditioning on M1).

Model 4: a linear regression model for income conditional on disability acquisition and all baseline covariates (model for M2 marginalising over M1).

Models 3 and 4, although likely to be incompatible, allow estimation of indirect effects through different pathways (M1, M2 and through their dependence) because conditioning on M1 allows estimation of the joint distribution of M1 and M2, whereas marginalising over M1 allows estimation of the independent distributions of M1 and M2.

The IDE and IIE were estimated using the Monte-Carlo approach described in Vansteelandt and Daniel (2017) [206]. This involved sampling values of the mediators for each individual by taking one million random draws from the fitted distributions of the mediators for different counterfactual exposure scenarios. These sampled values were then used to predict expected values of the outcome under different counterfactual scenarios of the exposure and mediator. Bootstrapping with 200 replications was used to calculate 95% confidence intervals.

Missing data

There were missing observations for the outcome, mediators and some baseline covariates (Table 5.8). The variables with the greatest amount of missing data were mental health (9-10%), health behaviours (smoking, alcohol consumption, physical activity: 10%), BMI (13%), social support and frequency of socialising (11%) and financial hardship (10%).

Table 5.8 Number (%) of missing observations for mental health, mediators and baseline covariates

		Missing	
	Wave	n	%
Mental health	2014	506	9%
Mediators			
Employment status	2013	0	0%
Weekly income (AUD)	2013	0	0%
Baseline covariates			
Age	2011	0	0%
Sex	2011	0	0%
Country of birth	2011	0	0%
Parent occupation	2011	107	2%
Education	2011	0	0%
Occupation	2011	2	<1%
Weekly income (AUD)	2011	0	0%
Wealth	2011	0	0%
Financial hardship	2011	564	10%
Housing tenure	2011	4	<1%
Housing affordability	2011	151	3%
Relationship status	2011	1	<1%
Children	2011	0	0%
Social support	2011	609	11%
Frequency of socialising	2011	603	11%
Smoking status	2011	561	10%
Alcohol consumption	2011	567	10%
Physical activity	2011	550	10%
BMI (kg/m^2)	2011	721	13%
Mental health at baseline	2011	555	10%

The distribution of baseline covariates (measured in 2011) was compared between participants with and without missing observations to determine whether missingness was associated with the values of measured variables. Participants with missing data had poorer mental health, were younger, were more likely to be born outside of Australia and experienced greater socio-economic compared to those with complete data (Table 5.9), suggesting that the data were not MCAR.

Table 5.9 Distribution of disability, mental health, mediators and baseline covariates between participants with complete data on all variables required (i.e. complete cases) and those with missing observations

	Complete cases (n=4311)		Missing observations (n=1341)	
	n	%	n	%
Exposure: Disability acquisition				
Yes	182	4.2	51	3.8
No	4129	95.8	1290	96.2
Outcome: Mental health (mean, SD)	77.3	14.8	74.5	16.5
M1: Employment				
Employed	3679	85.3	1117	83.3
Unemployed/not in the labour force	632	14.7	224	16.7
M2: Income (AUD, mean, SD)	1065.4	653.0	1003.2	649.1
Baseline covariates:				
Age (mean, SD)	44.7	10.1	42.0	10.0
Sex				
Men	2071	48.0	653	48.7
Women	2240	52.0	688	51.3
Country of birth				
Australia	3328	77.2	969	72.3
Other	983	22.8	372	27.7
Parent occupation				
High skilled	624	14.5	213	17.3
Medium skilled	1458	33.8	404	32.7
Low skilled/not in the labour force	2229	51.7	617	50.0
Education				
Higher education	1563	36.3	389	29.0
Secondary education	2050	47.6	681	50.8
Less than secondary	698	16.2	271	20.2
Occupation				
High skilled	1719	39.9	464	34.7
Medium skilled	1427	33.1	429	32.0
Low skilled	620	14.4	223	16.7
Unemployed/not in the labour force	545	12.6	223	16.7
Weekly income (AUD, mean, SD)	1002.2	613.0	901.7	5923.0
Wealth				
Highest tertile	1770	41.1	402	30.0
Middle tertile	1593	37.0	489	36.5
Lowest tertile	948	22.0	450	33.6
Financial hardship				
Prosperous/very comfortable	802	18.6	116	14.9
Reasonably comfortable	2383	55.3	418	53.8
Just getting along/poor/very poor	1126	26.1	243	31.3
Housing tenure				
Outright owner	1022	23.7	212	15.9
Mortgage	2112	49.0	626	46.8
Renter	1177	27.3	499	37.3
Housing affordability				
Affordable	4029	93.5	1053	88.5
Not affordable	282	6.5	137	11.5
Relationship status				
In a relationship	3479	80.7	986	73.6

Not in a relationship	832	19.3	354	26.4
Children				
Yes	3142	72.9	978	72.9
No	1169	27.1	363	27.1
Social support (mean, SD)	5.6	1.0	5.5	1.0
Frequency of socialising (mean, SD)	3.6	1.4	3.6	1.5
Smoking status				
Never	2349	54.5	393	50.4
Ex	1171	27.2	198	25.4
Current	791	18.4	189	24.2
Alcohol consumption				
Never	482	11.2	119	15.4
Rarely	1451	33.7	292	37.7
1-2 days per week	980	22.7	174	22.5
>2 days per week	1398	32.4	189	24.4
Physical activity				
>3 times per week	1470	34.1	264	33.4
1-3 times per week	1859	43.1	308	38.9
Less than once per week	982	22.8	219	27.7
BMI (kg/m^2, mean, SD)	26.5	5.0	26.4	5.2
Mental health at baseline (mean, SD)	77.4	14.4	75.8	14.8

Multiple imputation using chained equations with 50 imputations was performed to maximise the information available and reduce bias due to the missing data. The imputation models included all variables in the target analysis as well as additional auxiliary variables including mental health (2010, 2012, 2013), self-rated health (2010-2014), socio-economic characteristics (2010, 2012) except education, parent occupation and wealth which changed minimally over time, and SEIFA score (2011). The mediation analysis was conducted on each of the 50 imputed datasets and the mean of the estimates from each imputed dataset was calculated to give an overall estimate of the total, direct and indirect effects. Standard errors were derived using Rubin's Rules for combining the between-imputation and within-imputation variance, with the latter obtained by bootstrapping of each imputed dataset [236]. Results from the multiple imputation (primary analysis) were compared to those of the complete case analysis (secondary analysis restricted to 4311 participants).

Sensitivity analysis

The effect on mental health of acquiring a psychosocial impairment is likely to be different to the mental health effect associated with acquiring other types of impairments. Therefore as a sensitivity analysis, I removed participants who acquired psychosocial impairments, defined as nervous or emotional conditions which require treatment, or any mental illness which requires help or supervision, and assessed whether the magnitude of the effect differed for people who acquired non-psychosocial impairments.

Results

Sample selection

Of the 28,794 people who participated in the first 14 waves of the HILDA Survey, 14,534 participated in all four waves between 2011 and 2014, of which 10,450 were of working age and had complete data on disability (Figure 5.7). 5652 people were eligible for inclusion in the sample based on their reported disability status in the four waves: 233 (4.1% acquired a disability) and 5419 did not report a disability in any of the waves of analysis. Complete data for the exposure, mediators, confounders and outcome were available for 4311 participants, 76% of the sample.

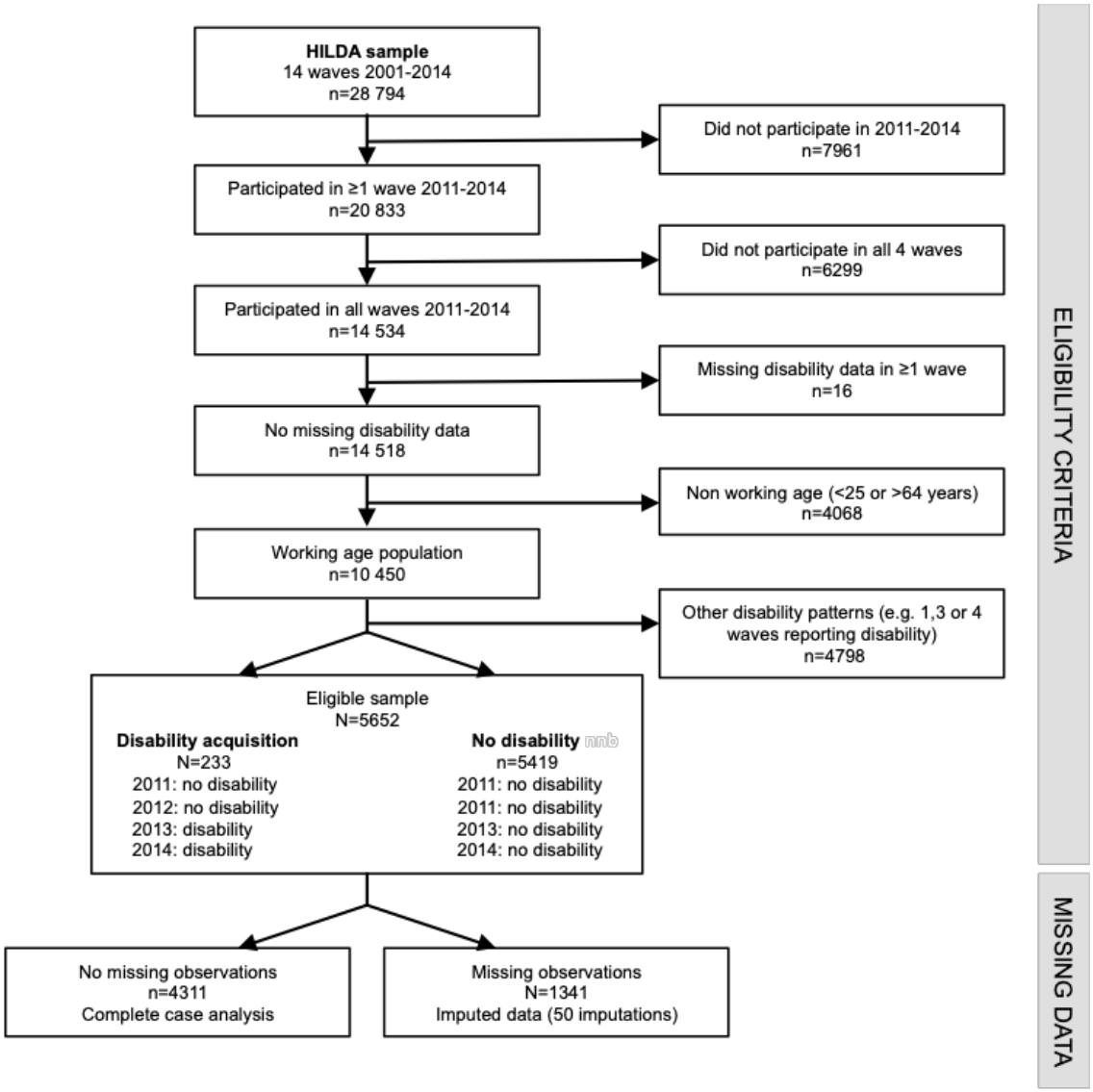

Figure 5.7 Flow chart illustrating sample selection and missing data

Descriptive analysis

Baseline demographic and socio-economic characteristics of the sample were described for a randomly selected imputed dataset (Table 5.10). At baseline, prior to reporting a disability, people in the disability sample were older, had lower mental health, and were more likely to experience socio-economic disadvantage including: low education; low skilled occupation, unemployment or not in the labour force; low income; financial hardship; unaffordable housing; lower social support and frequency of socialising; more likely to be current smokers; less likely to exercise; and higher BMI. They were similar to those without disability in terms of sex, country of birth, parents' occupation, wealth, housing tenure, relationship status, number of children, and alcohol consumption.

Table 5.10 Distribution of baseline demographic, socio-economic and mental health characteristics for people who acquired a disability and those with no disability for a randomly selected imputed dataset

	No disability (n=5429)		Disability (n=233)	
	n	%	n	%
Age (mean, SD)	43.9	10.1	48.3	9.6
Sex				
Men	2607	48.1	117	50.2
Women	2812	51.9	116	49.8
Country of birth				
Australia	4116	76.0	181	77.7
Other	1303	24.1	52	22.3
Parent occupation				
High skilled	812	15.0	43	18.5
Medium skilled	1817	33.5	80	34.3
Low skilled/not in the labour force	2790	51.5	110	47.2
Education				
Higher education	1906	35.2	46	19.7
Secondary education	2611	48.2	120	51.5
Less than secondary	902	16.7	67	28.8
Occupation				
High skilled	2119	39.1	65	27.9
Medium skilled	1780	32.9	77	33.1
Low skilled	799	14.7	44	18.9
Unemployed/not in the labour force	721	13.3	47	20.2
Weekly income (AUD, mean, SD)	984.9	612.6	827.0	519.5
Wealth				
Highest tertile	2088	38.5	84	36.1
Middle tertile	1998	36.9	84	36.1
Lowest tertile	1333	24.6	65	27.9
Financial hardship				
Prosperous/very comfortable	978	18.1	26	11.2
Reasonably comfortable	2959	54.6	122	52.4
Just getting along/poor/very poor	1482	27.4	85	36.5
Housing tenure				
Outright owner	1173	21.7	61	26.2

Mortgage	2638	48.7	102	43.8
Renter	1608	29.7	70	30.0
Housing affordability				
Affordable	4983	92.0	206	88.4
Not affordable	436	8.1	27	11.6
Relationship status				
In a relationship	4280	79.0	185	79.4
Not in a relationship	1139	21.0	48	20.6
Children				
Yes	1479	27.3	53	22.8
No	3940	72.7	180	77.3
Social support (mean, SD)	5.6	1.0	5.2	1.1
Frequency of socialising (mean, SD)	4.4	1.4	3.9	1.6
Smoking status				
Never	2929	54.1	99	42.5
Ex	1414	26.1	73	31.3
Current	1076	19.9	61	26.2
Alcohol consumption				
Never	643	11.9	31	13.3
Rarely	1824	33.7	96	41.2
1-2 days per week	1262	23.3	44	18.9
>2 days per week	1691	31.2	62	26.6
Physical activity				
>3 times per week	1828	33.7	70	30.0
1-3 times per week	2316	42.7	90	38.6
Less than once per week	1275	23.5	73	31.3
BMI (kg/m^2, mean, SD)	26.5	5.0	28.2	5.5
Mental health at baseline (mean, SD)	77.3	14.2	70.2	18.7

Mediation analysis

The TCE of acquiring a disability was estimated to be a five-point decline in mental health (estimated mean difference: -4.8, 95% CI -7.0, -2.7, Table 5.11). The IIE through employment was estimated to be a 0.5-point difference (-0.5, 95% CI -1.0, 0.0) in mental health score, accounting for 10.6% of the total effect (95% CI -1.0, 22.1), and there was no evidence that income explained any of the effect (0.0 95% CI 0.0, 0.1). There was also no evidence for an IIE resulting from the dependence between employment and income (0.0, 95% CI 0.0, 0.0). The results of the complete case analysis supported the findings of the multiple imputation analysis, with a slight attenuation of the magnitude of the TCE and IDE and wider confidence intervals (Table 5.11). The complete case estimate of the IIE through employment was estimated to explain 16% of the total effect, and there was no evidence of mediation through income.

Table 5.11 Results of the interventional mediation analyses estimating total, direct and indirect effects of disability acquisition on mental health, with mediation through employment (M1) and income (M2)

	Coeff	95% CI	% mediated
Multiple imputation:			
TCE	-4.8	-7.0, -2.7	
IDE	-4.0	-6.1, -1.9	
IIE through employment (M1)	-0.5	-1.0, 0.0	10.6 (-1.0, 22.1)
IIE through income (M2)	0.0	0.0, 0.1	-0.5 (-1.6, 0.6)
IIE from dependence of M1 & M2	0.0	0.0, 0.0	0.0 (-0.1, 0.1)
Complete case analysis:			
TCE	-4.1	-6.4, -1.8	
IDE	-2.9	-5.0, -0.8	
IIE through employment (M1)	-0.7	-1.4, 0.1	16.0 (-3.6, 35.6)
IIE through income (M2)	0.0	-0.1, 0.1	0.0 (-3.1, 3.2)
IIE from dependence of M1 & M2	0.0	-0.1, 0.1	-0.1 (-3.2, 3.0)

Sensitivity analysis

The sensitivity analysis in which participants with psychosocial impairments were excluded from the sample produced a TCE that was slightly smaller in magnitude (estimated mean difference: -3.8, 95% CI -6.0, -1.6, Table 5.12) but a similar proportion of the effect mediated by employment (11.6%) and income (0%). The complete case analysis produced similar results, though with a smaller estimated total effect (estimated mean difference: -2.9, 95% CI -5.2, -0.5) and a slightly higher estimated proportion of the effect mediated by employment of 20% (Table 5.12).

Table 5.12 Results of the sensitivity analysis excluding people with psychosocial impairments

	Coeff	95% CI	% mediated
Multiple imputation:			
TCE	-3.8	-6.0, -1.6	
IDE	-3.0	-5.2, -0.9	
IIE through employment (M1)	-0.4	-0.9, 0.1	11.6 (-16.2, 39.4)
IIE through income (M2)	0.0	0.0, 0.1	-0.4 (-2.9, 2.2)
IIE from dependence of M1 & M2	0.0	0.0, 0.0	0.0 (-0.3, 0.2)
Complete case analysis:			
TCE	-2.9	-5.2, -0.5	
IDE	-2.0	-4.1, 0.1	
IIE through employment (M1)	-0.6	-1.3, 0.2	20.0 (-45.2, 85.4)
IIE through income (M2)	0.0	-0.1, 0.0	0.0 (-2.8, 3.3)
IIE from dependence of M1 & M2	0.0	0.0, 0.0	0.0 (-0.3, 0.3)

Discussion

Main findings

This study estimated that 11% of the effect of disability acquisition on mental health was explained by changes to people's employment but none through income or jointly through the co-dependence of employment and income. The previous analysis which quantified the effect of disability acquisition on mental health operating through a broad range of material socio-economic factors including employment, occupation, income, financial hardship, financial satisfaction, housing tenure and housing affordability estimated that 32% of the effect operated through these mediators. In this study, I aimed to identify specific drivers of the effect by examining two of these mediators separately, choosing employment and income. Employment, categorised crudely as being in employment or not, explained a small (but potentially important) proportion of the total effect of disability acquisition on mental health.

Interpretation of results

Disability acquisition is likely to lead to a decline in people's mental health through a variety of mechanisms. There is evidence from the literature that acquiring a disability is associated with decreasing employment rates and increased risk of belonging to low income households [264], exposing people to higher levels of financial stress which may explain their poorer mental health. Changes to people's employment explained a proportion of the effect, however it was surprising that income explained none of the effect. This suggests that the benefits of employment to mental health operate through pathways other than income. It may be that the positive effects of employment on mental health result from latent benefits such as providing a time structure, social contact and social identity [304].

Strengths

This analysis applies a novel approach to mediation analysis which addresses the limitations of traditional mediation methods and sequential causal mediation methods, by estimating indirect effects through multiple mediators under weaker assumptions than NIEs. The longitudinal nature of the data, with repeated surveying of participants every year, was utilised to establish a temporal sequence between the baseline variables, exposure, mediators and outcomes to better understand the causal relationship between disability and mental health. This analysis examined incident disability, comparing people who acquired a disability to those who reported no disability, to account for differences between the groups prior to disability to maximise causal inference. It also adjusted for baseline values of the mediators and baseline mental health so that the

indirect effects can be interpreted as the effect of changes in the mediators as a result of disability acquisition.

Limitations

There were a number of limitations. Causal mediation analysis relies on strong assumptions about confounding for the estimates to have a causal interpretation, described in detail in section 3.6.5 [176]. This analysis controlled for a large number of baseline variables, measured prior to disability acquisition, including socio-economic factors and mental health, which should have accounted for any differences in measured variables between the exposure groups. However, it is possible that there may have been some mediator-outcome confounding, which may have biased the results. There were only 233 people who acquired a disability in our dataset, which resulted in low power to detect effects, particularly when partitioning the effect into direct and indirect effects through additional variables. Employment was examined as a binary variable due to limitations of the methods for estimating interventional direct and indirect effects, which have not been extended to handling mediators with more than two categories. This coarse classification of employment into two categories may have underestimated the indirect effect through employment. We examined mediators in 2013, in the same wave of data that people first reported a disability. It is possible that changes to employment and income resulting from disability acquisition may become apparent over a longer time frame than we examined in this analysis, which may have led to an underestimate of the effect operating through employment and income. All variables were self-reported and therefore prone to measurement error, which may have introduced bias if the degree of error was different between people with and without a disability. There were missing data for 24% of participants in the sample, however we used multiple imputation which is likely to have controlled for selection bias from missing data. Finally, the HILDA sampling frame included residents of private dwellings. People with severe disabilities, who may be more likely to live in non-private dwellings such as nursing homes or group homes, are likely to be under-represented in the sample.

Conclusions

The finding that changes to people's employment as a result of disability acquisition explains a proportion of the effect of disability on mental health has important policy implications. The results highlight the need to implement measures to enable people with disabilities to remain in employment and improve employment and vocational training opportunities for people who acquire a disability to improve their mental health and reduce current mental health inequalities.

Chapter 6 Unification of effect modification and mediation

6.1 A four-way decomposition of the effect of disability acquisition on mental health through employment status

6.1.1 Introduction

The three analyses in Chapter 5 demonstrated that socio-economic characteristics modify the effect of disability acquisition on mental health, suggesting that socio-economic factors are implicated in the pathway leading from disability acquisition to mental health. The two analyses in Chapter 6 further explore the mechanism leading from disability acquisition to poor mental health by decomposing the total effect into the effect operating indirectly through socio-economic characteristics and the direct effect (through other mechanisms). These analyses suggest that the effect of disability acquisition is mediated, in part, by changes to people's socio-economic characteristics resulting from disability.

However, the indirect effects estimated in Chapter 6 represent a combination of the effect due to mediation by socio-economic factors as well as the effect resulting from the interaction between the exposure and the mediator. A recent method has been proposed which can further decompose the total effect of an exposure on an outcome into the effects due to interaction only, to mediation only, to both interaction and mediation, and to neither.

In this final chapter of results, the concepts of effect modification and mediation are brought together in a single approach, quantitatively separating the components of the effect of disability acquisition on mental health which are attributable to effect modification and mediation by employment. A more nuanced understanding of the mechanism by which employment influences the effect of disability acquisition on mental health will inform the design of effective policies on employment to improve mental health and reduce disability-related mental health inequalities.

6.1.2 How much of the indirect effect of disability acquisition on mental health through employment status is operating through mediation and interaction: a four-way decomposition analysis

Background

People with disabilities have poorer mental health than those without disability. There is evidence from longitudinal studies of a causal relationship whereby disability acquisition leads to a deterioration in people's mental health [100, 101, 114-125]. However, research on the mechanistic pathways leading from disability acquisition to poor mental health is scarce. Given the increasing body of evidence suggesting that the mental health inequalities are to a large extent socially-determined, it is important to understand how socio-economic characteristics influence the relationship between disability acquisition and declining mental health to be able to inform social interventions to improve the mental health of people with disabilities. It may be that disability acquisition interacts with socio-economic characteristics leading to greater declines in mental health for subgroups of the population who experience socio-economic disadvantage. Alternatively, disability acquisition may lead to changes to people's socio-economic circumstances which in turn lead to mental health declines. Or pathways to poorer mental health may operate through a combination of these mechanisms.

In this thesis, I quantified how much of the effect of disability acquisition on mental health was due to effect modification and how much to mediation, examining several determinants of health including material, psychosocial and behavioural factors. In section 5.1.2, I quantified how much of the effect of disability acquisition on mental health was operating through three main mechanistic pathways: material, psychosocial and behavioural socio-economic characteristics. This analysis suggested that socio-economic factors explained more than a third of the causal relationship, with most of the effect (32%) explained by material characteristics including employment, occupation, income, financial hardship, financial satisfaction, and housing tenure and affordability [4]. A further analysis (section 5.2.2) investigated specific drivers of the mediated effect, including the effect of employment and income, and found that 11% of the effect was explained by employment status but none through income. These findings provide evidence that material socio-economic factors, particularly employment, mediates the relationship between disability acquisition and poor mental health. I also examined the interaction between disability acquisition and individuals' socio-economic characteristics prior to disability to understand how socio-economic disadvantage affected the magnitude of the effect of disability on mental health. The results suggested that the detrimental effect of disability acquisition on mental health was substantially greater for

socio-economic disadvantaged individuals, particularly those who were unemployed, not in the labour force, those employed in low skilled jobs (section 4.2.2) [2], people with low income (section 4.1.2) [1], and those with low social support (section 4.3.2) [3].

In this chapter, I examine the effect of employment in greater detail, with an aim to identify how much of the indirect effect of disability acquisition on mental health that operates through employment is due to mediation, interaction or both. The analytic approach, a four-way decomposition approach developed by VanderWeele (2014), enables the total effect of disability acquisition on mental health to be decomposed into the components due to mediation only, to interaction only, to both mediation and interaction, and the direct component due to neither mediation nor interaction [199]. Therefore, this approach unifies methods attributing effects to interactions and those attributing effects to mediation. The findings will provide insight into the mechanism by which employment contributes to the disability-mental health relationship and therefore may inform interventions on employment to reduce the impact of disability acquisition on mental health.

Methods

Data source

The data source used in this analysis was the HILDA Survey, a nationally representative longitudinal survey of Australian households, which collects data on a wide range of demographic, health, social and economic characteristics. The survey began in 2001 with a national probability sample of households occupying private dwellings and has been collecting data annually, following the same group of individuals over time. All household members aged 15 years and above were invited to participate in the survey, completing a face to face interview and a self-completed questionnaire. The original sample consisted of 13,969 participants from 7682 households. Original sample members were followed up in subsequent waves and all new household members were invited to participate in the survey. The sample was replenished in 2011 to maintain representativeness, adding an additional 2153 households. Across all waves, response rates were 80% and attrition was 6% between waves. This analysis used data from four waves of the survey (2011 to 2014) and was restricted to working age participants (aged 25 to 64 years).

Mental health

Mental health was assessed using the MHI, one of the eight subscales of the SF-36, a self-completed general health questionnaire which has been validated for use in research examining health characteristics and health inequalities in the Australian

population [168]. The MHI measures symptoms of depression, anxiety and psychological well-being over the previous four weeks. It is a well validated measure of mental health status [169] and an effective screening tool for severe depression and mood and anxiety disorders [172-175]. The MHI is constructed from five items in the questionnaire, each scored using five response categories, and total scores are transformed into a scale ranging from 0 to 100 with a mean of 74. Higher scores represent better mental health. Previous studies have suggested that a difference in the MHI scale of four to five points reflects a minimally important clinical change in mental health [112, 113]. Mental health was assessed in every wave of HILDA. In this analysis, the outcome mental health was measured in 2014 and baseline mental health (prior to disability) was included in the model as a potential confounder, measured in 2011.

Disability acquisition

Information on disability was asked in every wave of the HILDA Survey, using a single question asking participants whether they had an “impairment, disability or long-term health condition, which restricts everyday activities that had lasted for six months or more”. This information was used to define the exposure groups, comparing people who had acquired a disability to those who reported no disability in any of the four waves included in the analysis. Disability acquisition was defined as two consecutive waves with no disability (2011 and 2012) followed by two consecutive waves with disability (2013 and 2014). All other patterns of disability (such as a single wave of reported disability or all waves) were not eligible for inclusion in the sample as well as participants with any missing observations for the disability variable. Two consecutive waves of disability were required to identify participants with long-term disability rather than transient health conditions, a commonly used definition of disability acquisition [120, 263, 264].

Employment status

Employment status was categorised as a binary variable, defined as people who were employed versus those who were not employed (unemployed and actively seeking employment in the previous four weeks or not actively seeking employment). Employment status was measured in 2013.

Other covariates

Variables were included as covariates in regression models if they were deemed to be potential confounders of the association between disability acquisition and mental health, defined as variables that were identified as common causes of both disability acquisition and mental health using a causal diagram (Figure 6.1).

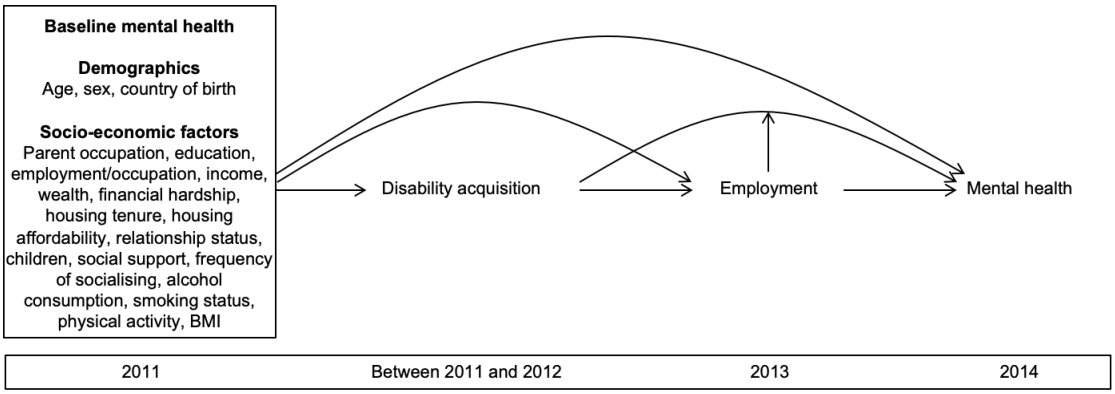

Figure 6.1 Causal diagram illustrating postulated causal relationships between exposure, mediator, outcome and covariates

All potential confounders were measured in 2011 to represent people's circumstances prior to disability acquisition. These comprised demographic characteristics including age (in years), sex (men; women), country of birth (Australia; other), socio-economic characteristics including education (bachelor degree and above; completion of secondary education; did not complete secondary), parental occupation (high skill; medium skill; low skill or not in the labour force), occupation (high skill; medium skill; low skill; unemployed/not in the labour force), income (defined as weekly personal disposable income in $AUD), financial hardship (prosperous/very comfortable; reasonably comfortable; just getting along/poor/very poor), wealth (categorised into tertiles of net wealth), tenure of housing (outright owner; mortgagee; renter), housing unaffordability (defined as households in the lowest 40% of the disposable income distribution spending more than 30% of their gross income on housing costs), relationship status (yes; no), children (yes; no), social support (a scale constructed using the average of 10 questions addressing emotional support, each rated on a 7-point Likert scale) [247], frequency of socialising with friends/relatives (rated on a 7-point Likert scale ranging from less than once every three months to daily), and health behaviours including smoking status (never; ex-smoker; current), alcohol consumption (never; rarely; 1-2 days per week; >2 days per week), physical activity (>3 times per week; 1-3 times per week; less than once a week) and BMI (in kg/m^2).

Mediation analysis

A mediation analysis aims to determine the extent to which the effect of an exposure on an outcome is occurring through an intermediate variable (the mediator). It decomposes the total effect of an exposure on an outcome into the component operating through the mediator – the indirect effect – and the component operating through other pathways – the direct effect [44].

There has recently been a major revival of mediation analysis which has led to the development of new statistical methods for mediation analysis based on the counterfactual framework to causal inference. The new methods, described in detail in section 3.6.5, have been developed to address the problems of bias encountered by traditional approaches to mediation including non-linearities and interactions between the exposure and mediator. In the counterfactual framework, the estimation of effects is achieved by comparing the outcomes of two hypothetical scenarios. These methods are grounded on counterfactual-based definitions of direct and indirect effects irrespective of statistical models and of interactions between the exposure and mediator. The counterfactual framework has been used to clarify what effects (counterfactual contrasts) are being estimated and the assumptions under which the direct and indirect effects can be identified from data, which were often not detailed in traditional approaches [45, 46].

Because mediation analysis decomposes the total effect of an exposure on an outcome into the components operating through distinct pathways, it can shed light on the mechanisms operating from exposure to outcome. For example, quantifying the magnitude of the effect of disability acquisition on mental health operating through employment (and the proportion of the total effect mediated by this characteristic) increases understanding of the relative importance of employment in explaining the mental health decline associated with disability acquisition. This evidence can then be used to inform the development of policy interventions to target modifiable characteristics on the pathway between disability acquisition and poor mental health.

Interaction analysis

The effect of an exposure on an outcome can be heterogeneous; it may vary in magnitude for different subgroups of the population or may even be beneficial to some subgroups of people and harmful to others. An interaction analysis aims to determine whether the effect of an exposure on an outcome differs for different subgroups of the population [176]. It quantifies the magnitude of the effect of an exposure on an outcome in the presence or absence of another variable, the *interacting* variable. In this case, it may be important to determine for whom the effect of an exposure on an outcome occurs, or for whom the magnitude is greatest, to identify subgroups of people who are at highest risk or may benefit the most from interventions.

Interaction analysis decomposes the effect of an exposure on an outcome in the presence of a third variable into the component attributable to the exposure only, the component attributable to the interacting variable only and the component attributable to the excess effect due to the interaction between the exposure and the interacting

variable. Quantifying the pathways operating through the joint effect of an exposure and interacting variable on an outcome can shed light on the mechanistic pathways operating between exposures and outcomes. For example, quantifying the effect of disability acquisition on mental health separately for people who are employed and not employed increases understanding of the reasons for the heterogeneity in the magnitude of the mental health decline in the population of people who acquired a disability. As well as being used to identify subgroups of people who are particularly vulnerable to mental health declines, this evidence can also draw attention to modifiable characteristics which may be targets of policy interventions to reduce the negative effect of disability on mental health.

Unification of mediation and interaction analysis using a four-way decomposition approach

Mediation and interaction can operate simultaneously. If interaction exists between the exposure and mediator, then this needs to be accounted for to fully capture the dynamics of mediation. In causal mediation analysis in which the total effect is typically decomposed into the pure NDE and the total NIE, the interaction component is captured in the total NIE. However, it is also possible to carry out a different decomposition into the total NDE and the pure NIE in which the interaction component is captured in the total direct effect.

The four-way decomposition approach developed by VanderWeele (2014) decomposes the total effect of an exposure on an outcome into four separate components: the component due to mediation only, to interaction only, to both mediation and interaction, and the direct component due to neither mediation nor interaction [199].

Specifically the TCE, defined as $Y_1 - Y_0$ is decomposed into four components [199]. In this notation, Y denotes the outcome of interest, A the binary exposure with two values A=1 (exposed) and A=0 (unexposed), and M the mediator. Counterfactuals are denoted by Y_{aM_a}, for example Y_{1M_1}, representing the value of the outcome that would have been observed had the exposure been set to 1 (exposed) and the mediator to the value it would have taken if the individual were exposed M_1.

i) The CDE, the component not due to interaction nor mediation, is the direct effect of the exposure A on the outcome Y when the mediator is fixed at a particular value M=0 for the whole population. It is defined as $Y_{1M_0} - Y_{0M_0}$.

ii) The reference interaction, the component due to interaction but not mediation, is the excess effect of the exposure on the outcome which exceeds the sum of the effect

of the exposure and mediator considered separately that operates only if the mediator is present in the absence of exposure (i.e., $M_0 = 1$). It is defined as $([Y_{1M_1} - Y_{0M_0}] - [\{Y_{1M_0} - Y_{0M_0}\} + \{Y_{0M_1} - Y_{0M_0}\}])(M_0)$, which reduces to $(Y_{1M_1} - Y_{1M_0} - Y_{0M_1} + Y_{0M_0})(M_0)$.

iii) The mediated interaction, the component due to mediation and interaction, is the excess effect of the exposure on the outcome which exceeds the sum of the effect of the exposure and mediator considered separately that operates only if the exposure has an effect on the mediator (i.e., $M_1 - M_0 \neq 0$). It is defined as $(Y_{1M_1} - Y_{1M_0} - Y_{0M_1} + Y_{0M_0})(M_1 - M_0)$.

iv) The pure mediated effect, the component due to mediation but not interaction, is the effect of the exposure on the outcome mediated by differential exposure to the mediator. It is defined as $(Y_{0M_1} - Y_{0M_0})(M_1 - M_0)$, and will only be non-zero if the exposure has an effect on the mediator and the mediator affects the outcome when the population is not exposed.

Statistical analysis

The *med4way* command in Stata was used to estimate the TCE of disability acquisition on mental health and decompose this effect into the CDE, the reference interaction, the mediated interaction and the pure mediated effect [319]. The *med4way* command computed the estimates by combining the regression parameters from two regression models: firstly, a linear regression model for the outcome (mental health) as a function of the exposure, the mediator, their interaction and the baseline covariates; and secondly, a logistic regression model for the mediator (employment status) as a function of the exposure and baseline covariates. Nonparametric bootstrapping with 500 replications was used to calculate standard errors for the estimated effects. Analyses were performed using Stata/SE 15.0 software (StataCorp, College Station, TX, USA).

The causal interpretation of the estimated effects relies on a number of assumptions about confounding necessary to identify the four components: (1) no confounding of the exposure-outcome association, (2) no confounding of the exposure-mediator association, (3) no confounding of the mediator-outcome association, (4) and no exposure-induced mediator-outcome confounding [176].

Missing data

The sample for the analysis consists of the same sample as the interventional effects analysis presented in section 5.2.2 as it uses the same waves of data, the same inclusion

criteria, and the same variables for the exposure, outcome, mediator and covariates (except for income which is not included in this analysis but had no missing data).

There were missing observations for the outcome and some of the baseline covariates for 1341 observations, 24% of the sample (see Table 5.4, section 5.2.2). The distribution of baseline characteristics and the percentage of people acquiring a disability was compared between sample participants with and without missing data to determine whether the pattern of missingness was associated with disability acquisition and other measured covariates (see Table 5.5, section 5.2.2). Individuals with missing observations had poorer mental health and experienced greater levels of socio-economic disadvantage across all socio-economic measures compared to participants with complete observations. As a result, multiple imputation using chained equations with 50 imputations was used to impute the missing data (*mi impute chained* command in Stata) assuming the data were MAR. Missing observations were imputed by drawing from the posterior predictive distribution of the missing data given the observed data. Univariate imputation models were specified for each variable with missing data. The models included the variables in the primary analysis as well as additional (auxiliary) variables which were associated with the variables with missing observations or were predictive of missingness. These auxiliary variables included mental health measured in 2010, 2012 and 2013, self-rated health from 2010 to 2014, all time-varying variables describing socio-economic characteristics measured in 2010 and 2012, and the SEIFA index of relative socio-economic disadvantage recorded from the Census in 2011.

The four-way decomposition mediation analysis was conducted on each of the 50 imputed datasets and the mean of the estimates from each imputed dataset was calculated to give an overall estimate of the estimated effects. Rubin Rules were applied to derive standard errors for the estimates, combining the between-imputation and within-imputation variance, where the latter was obtained from bootstrapping of each imputed dataset [52].

Results

Sample selection

Of the 14,534 sample members who participated in all four waves of the HILDA Survey between 2011 and 2014, 10,450 had complete data on disability and were of aged between 25 to 64 years (see Figure 5.5, section 5.2.2). Of these, 5652 were eligible for inclusion based on the definition of exposure groups: 233 acquired a disability (4.1% of the sample) and 5419 reported no disability in all four waves. Complete data on all

variables contributing to the analysis were available for 4311 participants, 76% of the sample.

Descriptive analysis

Prior to acquiring a disability, people in the disability sample were older, more socio-economically disadvantaged in terms of education, occupation, unemployment, income, financial hardship and housing affordability, had lower social support and frequency of socialising, more likely to smoke, less likely to exercise, had higher BMI and had poorer mental health compared to those with no disability in any wave (see Table 5.6, section 5.2.2). They were similar with respect to sex, country of birth, parents' occupation, wealth, housing tenure, relationship status, number of children, and alcohol consumption.

Mediation analysis

Disability acquisition was estimated to lead to a 5.28-point decline in mental health score (estimated mean difference: -5.28, 95% CI -7.32, -3.24, Table 6.1). Decomposing the TCE into direct and indirect components estimated that the CDE of disability acquisition on mental health was a decline of 3.53-points (-3.53, 95% CI -6.40, -0.67), therefore 67% of the effect of disability acquisition on mental health would remain if the mediator was uniformly fixed so that everyone in the population was employed. The remaining 33% was attributable to the indirect effect of disability on mental health operating through employment status. The reference interaction, the effect due to additive interaction between the exposure and mediator (i.e., the excess effect additional to the sum of the effect of acquiring a disability and the effect of being unemployed) for people without disability, was estimated to account for 17% of the effect (-0.92, 95% CI -1.89, 0.05). The mediated interaction accounted for 15% of the effect (-0.80, 95% CI -2.24, 0.65), which is the excess effect due to additive interaction between the exposure and mediator which operates only if disability acquisition has an effect on employment. Finally, the pure mediated effect, the effect occurring through changes to employment as a result of disability, was estimated to account for 1% of the effect (-0.04, 95% CI -0.13, 0.06).

Table 6.1 Results of the four-way decomposition analysis estimating the proportion of the effect of disability acquisition on mental health due to mediation and interaction with employment

Component	Coeff*	95% CI	% attributable
Total causal effect	-5.28	-7.32, -3.24	100%
Controlled direct effect	-3.53	-6.40, -0.67	67%
Reference interaction	-0.92	-1.89, 0.05	17%
Mediated interaction	-0.80	-2.24, 0.65	15%
Pure indirect effect	-0.04	-0.13, 0.06	1%

*estimated mean difference in MHI score, a subscale of the SF-36; CI – Confidence Interval

Discussion

Main findings

This study estimated that 33% of the total effect of disability acquisition on mental health operates through employment and 67% through other pathways. This suggests that if everyone in the population was employed, then the substantial disability-related mental health inequality could be reduced by one third.

Further decomposition of the impact of employment found that 17% of the total effect was due to the impact of disability on mental health in those who were unemployed (i.e. effect modification by employment only) and 15% due to a combination of effect modification and mediation, whereas mediation alone explained a negligible amount (1% of the total effect). These results suggest that employment is intricately involved in the relationship between disability acquisition and mental health and highlights the importance of the interaction between employment and disability acquisition in explaining the effect of disability acquisition on mental health.

Interpretation of results

The component of the effect attributable to interaction, the sum of the reference interaction and the mediated interaction, has been suggested to represent differential susceptibility, by which the susceptibility to the health consequences of an exposure depends on exposure to complementary underlying factors [320, 321]. This is a useful framework for thinking about how to take action on health inequalities. Almost all of the effect operating through employment was attributable to interaction, whereby people experienced differential susceptibility to the mental health effects associated with disability acquisition according to their employment status. The results suggest that changing the social distribution of the mediator by equalising employment outcomes

between people with and without disability, thus addressing the differential susceptibility, may reduce the mental health inequality between the groups by up to 32% [320].

Strengths

The four-way decomposition approach to examine mediation and interaction simultaneously is a novel approach developed by VanderWeele (2014) to provide greater insight into the causal processes leading from exposures to outcomes [199]. It brings together the overlapping concepts of mediation and interaction into a single analysis, quantifying the effect operating through a third variable and decomposing this indirect effect into the separate contribution of each of these mechanisms and their joint effect. Estimating the effect operating through each of the specific mechanisms can provide insight into the most effective public health policy strategies to target health inequalities [320].

The sample for analysis compared people who acquired a disability with people who reported no disability in any of the four waves. Using a measure of incident disability rather with prevalent disability enabled the analysis to be better controlled for differences in demographic, socio-economic and mental health characteristics between the exposure groups prior to disability to maximise exchangeability of the groups. Furthermore, the analysis was also adjusted for employment status prior to disability so that indirect effects operating through employment could be interpreted as the effects of changes to people's employment as a result of their disability.

Limitations

There were also limitations to the approach. The most notable limitation is the strong assumptions being made about confounding and no measurement error for the effects to have a causal interpretation. As well as the assumptions about no confounding of the exposure-outcome relationship, examining pathways through an intermediary variable includes the additional assumptions of no confounding of the exposure-mediator and mediator-outcome relationships. The analysis adjusted for differences in measured variables between the exposure groups prior to disability acquisition, which is likely to control for the majority of the confounding of the exposure-outcome and exposure-mediator relationships. However, there may still be some confounding of the mediator-outcome relationship, which is particularly problematic if the confounding variable is affected by the exposure. Exposure-induced mediator-outcome confounding may overestimate the mediated effect.

There may also have been measurement error of the exposure, mediator and outcome, as all variables were self-reported. The measurement error in these variables could be

dependent, whereby the misclassification of the exposure, mediator and outcome are not independent of one another, which has been shown to underestimate effects in interaction analyses [320, 322]. Furthermore, misclassification of the mediator has been shown to lead to substantial underestimation of the mediated effect [323], however employment was categorised as a binary variable which is likely to have been reported accurately. Therefore, though the impact of dependent measurement error on the results is hard to predict, it is most likely to lead to an underestimation of the total and indirect effects.

Employment status, the effect modifier, was categorised into a binary variable – employed versus not employed – due to restrictions of the analytic models which could only model binary or continuous mediator variables. The crude categorisation of employment may have led to an underestimation of the indirect effect because it may mask some of the nuances in the complex relationship between changes to people's employment and mental health that may be captured by other aspects of people's employment, such as occupation, and distinguishing between people who are unemployed (actively looking for employment) and those not in the labour force.

Another limitation relating to the use of a dichotomised variable as the effect modifier is that the level at which the variable is dichotomised can determine the presence and size of interaction effects [324]. However, in this context there is strong justification for comparing people who were employed to those who were not to generate policy-relevant findings.

The direct effect of disability acquisition on mental health operating through pathways other than employment was estimated as a CDE. In contrast to the NDE, which estimates the direct effect of an exposure on an outcome when the mediator is set at the value it would naturally take if the individual were unexposed, the controlled direct effect estimates the effect of an exposure on an outcome when the mediator if fixed at a particular value for the whole population. In this analysis, the CDE represented the direct effect of disability acquisition on mental health had everyone in the population been in employment, which is not a realistic public health intervention. Although it is not realistic to fix the mediator at a particular value for the entire population, the CDE can provide an estimate of the maximum impact of a potential intervention on an outcome [325].

The analysis estimated the indirect effects due to additive interaction rather than multiplicative interaction. Additive interaction was of primary interest throughout the thesis because it has been argued to be of greater public health interest [181, 182]. Furthermore, the Stata command *med4way* estimates the indirect effect due to additive

interaction only. However, testing for multiplicative interaction would be valuable to lend further support to the findings.

There were missing data for 24% of observations in the analytic sample which may have introduced selection bias, however, this should have been controlled for by the use of multiple imputation. Though the HILDA Sample is designed to be representative of the Australian population, it is unlikely to be representative of the population of people with disabilities. People with more severe impairments are less likely to participate in HILDA because the survey only samples private dwellings and because non-response to the survey is more likely for people with severe health conditions. Finally, there were only 233 people in the disability sample according to the definition of disability acquisition which meant that the analysis had low power to detect effects. This is particularly relevant in studies of mediation and interaction which rely on people being *doubly exposed* to the exposure and the mediator of interest to estimate indirect effects [320, 322].

Conclusions

Almost all of the indirect effect of disability acquisition on mental health through employment was found to be due to interaction. This has important policy implications. Policies need to target the causes of low employment rates for people with disabilities, with a focus on both individual resources and structural drivers such as investing in vocational and training opportunities, improving disability employment services and addressing discrimination in the workplace. Such policy changes would improve the mental health of people with disabilities and have the potential to reduce current mental health inequalities by a third.

However, targeting unemployment alone may not be sufficient to significantly improve mental health for the subgroup of people with disabilities, though it may reduce the proportion of people experiencing larger mental health effects associated with disability acquisition. It is important to better understand the mechanisms by which the differential susceptibility to disability acquisition is operating – how employment buffers the effect of disability on mental health – to reduce the magnitude of the effect of disability on mental health for people who are unemployed. If employment is acting through downstream risk factors, such as through social support, then it may be beneficial to people with disabilities' mental health to have social support components in employment policies or promote social support for people with disabilities via other strategies.

Chapter 7 Discussion

The research contributing to this PhD thesis investigated the socio-economic mechanisms by which disability affects people's mental health. It aimed to contribute to the development of policy interventions to improve the mental health of people with disabilities. The research responded to a recognised evidence gap, identified as a priority area for government, at a pivotal point in time given the changing disability policy landscape in Australia.

This thesis provides the first detailed and comprehensive evidence internationally demonstrating the effect of a broad range of socio-economic characteristics in shaping the mental health of people who acquired a disability. The aim of this chapter is to summarise and reconcile the results of the six distinct analyses and suggest possible explanations for the findings. The chapter will also discuss the policy implications of the results in light of the strengths and limitations of the research, and suggest avenues for future research.

7.1 Summary of key findings

The effect of disability acquisition on mental health is influenced by people's socio-economic characteristics prior and subsequent to disability.

The first three analyses (results of which are presented in Chapter 4) examined how the effect of disability acquisition on mental health varied according to socio-economic characteristics. Such analyses of effect modification provide insight into the mechanisms by which disability affects mental health by quantifying the relative effects of contributing factors on the outcome and under which circumstances the effect of disability is of a greater (or smaller) magnitude. Understanding the complex interactions between socio-economic characteristics and disability that lead to mental health deterioration may identify leverage points for policy intervention.

The first two analyses examined a wide range of socio-economic characteristics measured prior to disability acquisition to determine whether these influenced the magnitude of the effect of disability acquisition on mental health. The results demonstrated that socio-economic characteristics generally contributed to the effect of disability on mental health, with people who were socio-economically disadvantaged prior to disability experiencing larger mental health declines. The magnitude of the

mental health decline was notably large for people who were unemployed, had low income, low wealth, experienced financial hardship, lived in public housing, in unaffordable housing and those who were not in a relationship (section 4.1.2). Further disaggregation of employment characteristics demonstrated that people employed in low skilled jobs also experienced large mental health effects associated with disability acquisition (section 4.2.2).

The third analysis examined effect modification by social support, further disentangling the pathways by measuring social support both prior and subsequent to disability to better understand the temporal nature of the association. Social support both prior and subsequent to disability were found to be important determinants of the magnitude of the mental health effect, with particularly large effect sizes for people who experienced consistently low social support and those who experienced a decline in social support following disability (section 4.3.2).

The results of these three studies suggest that socio-economic characteristics are involved in the mechanistic pathways leading from disability acquisition to poor mental health. People's socio-economic circumstances affect how they respond to acquiring a disability, suggesting that socio-economic disadvantage can exacerbate the negative mental health effect and, conversely, that socio-economic advantage can provide a buffer that protects people against mental health deterioration when they acquire a disability.

The effect of disability acquisition on mental health is in part mediated by socio-economic characteristics, particularly material factors such as employment, which alone explains 11% of the effect.

The next two analyses examined whether the effect of disability acquisition on mental health was mediated by socio-economic characteristics subsequent to disability acquisition (results presented in Chapter 5). Analyses of mediation tell us about mechanism by decomposing the total effect into the portion operating through one (or several) intermediary variables and the portion of the effect operating through other pathways. Such analyses can identify targets for policy intervention that could be addressed or modified to eliminate the effect of disability acquisition on mental health operating through those indirect pathways.

The first of these analyses examined a wide range of socio-economic characteristics and quantified the indirect effect operating through these, categorised broadly into variables representing material, psychosocial and behavioural factors. Approximately a third of the effect was found to be explained by material socio-economic factors such as

employment, income, financial hardship and housing characteristics (section 5.1.2). Further investigation of the indirect effect through two distinct socio-economic characteristics in the subsequent analysis, namely employment and income, revealed that approximately 11% of the effect was explained by employment alone though none through income (section 5.2.2).

Importantly, in these analyses, by controlling for socio-economic characteristics prior to disability, the effects can be interpreted as the effect mediated by the change in people's socio-economic circumstances as a result of disability acquisition, rather than the effect of socio-economic disadvantage prior to disability.

These results highlight employment of people with disabilities as an important driver of the existing mental health inequalities and suggest that social policies addressing employment opportunities may be effective in reduce disability-related mental health inequalities.

The mechanism driving the mediated effect is through the interaction between the effects of disability and employment on mental health, rather than pure mediation of the effect of disability acquisition on mental health through employment.

Mediation can arise as a result of different mechanistic effects. It incorporates both phenomena of mediation and effect modification in its estimation. The final results chapter contributing to this thesis (Chapter 6) brings together the concepts of effect modification and mediation empirically, further decomposing the indirect effect operating through employment to separate out the components due to effect modification only, due to mediation only, due to both and due to neither. A more nuanced understanding of the mechanism by which employment mediates the effect of disability acquisition on mental health will provide evidence about how to best design policies to take action to reduce disability-related mental health inequalities.

The analysis estimated that a third of the total effect of disability acquisition on mental health was operating through employment. This is substantially greater than the 11% estimate from the previous mediation analysis estimating interventional effects. This discrepancy is likely to be related to the difference in methodological approach. IDEs can be interpreted as the average mental health difference that would remain between people with and without disabilities if the employment rate for people with disabilities was shifted to be the same as those without disability, simulating the effect of an intervention to equalise employment rates between people with and without disabilities, estimated to be 90% of the total effect. In contrast, CDEs estimate the effect of an exposure on an outcome when the mediator is fixed at a particular value for the whole population. In this

analysis, the CDE represents the average mental health difference that would remain between people with and without disabilities had everyone in the population been in employment, estimated to be 67% of the total effect. Rather than simulating the effect of a realistic policy intervention, it provides an estimate of the maximum impact of a potential intervention on mental health [325].

Further decomposition of the indirect effect through employment found that 17% of the total effect was due to effect modification by employment only, 15% due to a combination of effect modification and mediation, whereas mediation alone explained a negligible (1%) amount. These results highlight the importance of interaction between employment and disability acquisition as the mechanism driving the effect on mental health. The effect attributable to interaction has been suggested to represent differential susceptibility, by which susceptibility to the mental health consequences of disability acquisition depends on exposure to contributing risk factors, in this case, not being in employment.

To summarise, a broad range of socio-economic characteristics were found to be implicated in the mechanistic pathway leading from disability acquisition to poor mental health, with employment identified as a particularly important driver of the effect.

7.2 Strengths and limitations of the research

Before discussing the implications of the findings, it is worth reviewing the strengths and limitations of this thesis, with particular emphasis on those relating to the analyses and the data used.

7.2.1 Strengths

This research presents the first study examining the influence of a broad range of socio-economic characteristics on the effect of disability acquisition on mental health to understand their relative contribution to the effect. The strengths of the study relate to the choice of data and methods used to rigorously examine and disentangle the complex pathways operating between disability acquisition and poor mental health to generate robust estimates of the causal effect.

This study was made possible by several strengths relating to the data source. The HILDA Survey is the only nationally representative longitudinal survey of adults in Australia. It has a large sample size, collecting data annually from over 17,000 people since 2001, including data on disability, mental health and a wide range of different socio-economic characteristics. The longitudinal nature of the data enabled me to characterise changes in people's circumstances over time, relating changes to disability with changes

to people's socio-economic circumstances and their mental health. By modelling incident disability by characterising a sample of people who acquired a disability using longitudinal data, it is possible to use data collected prior and subsequent to disability to isolate the effect of disability on people's mental health.

There were several strengths of this study that relate to the analytic methods used. This is the first study to quantify direct and indirect effects of disability acquisition on mental health using state of the art causal mediation methods which address the substantial biases that traditional approaches to mediation are prone to. In addition to addressing the biases that stem from estimation of effects in the presence of exposure-mediator interactions and non-linear associations between the exposure, mediator and outcome, the counterfactual approach to mediation analysis clarifies the assumptions under which direct and indirect effects can be estimated from the data and interpreted as causal effects. The causal mediation analyses that were conducted made strong (but explicitly stated) assumptions about confounding. Exposure-mediator and exposure-outcome confounding are likely to have been mitigated by inverse probability weighting to make the exposure groups exchangeable. Though there may have been some mediator-outcome confounding, the mediation analysis presented in section 5.1.2 conducted a quantitative bias analysis to assess the potential effect of an unmeasured confounder and concluded that the indirect effect was unlikely to have been explained by confounding.

Similarly, in the analyses of effect modification there is likely to have been good control for confounding by measured variables through the propensity score approach using inverse probability weighting and through the application of fixed effects models which implicitly remove all confounding by time invariant characteristics by modelling within-person effects.

The four-way decomposition approach to examine mediation and interaction simultaneously is a novel approach to mediation analysis, and this is one of the first studies to apply this method. It provides greater insight into the mechanisms by which the mediated effect is operating, which can inform policy strategies as to how to best target and reduce health inequalities.

7.2.2 Limitations

There are also limitations to this study which need to be considered for the interpretation of the results. These relate to residual confounding; selection bias and representativeness; information bias; the definition of the exposure; the definition of the

outcome; statistical power and multiple testing; and some limitations relating to the causal mediation methods.

Though there was good control for confounding by measured variables, it is possible that there was confounding by unmeasured (or poorly measured) variables. For example, there was limited information in HILDA on childhood socio-economic circumstances and psychosocial resources and stressors, which may have led to residual confounding of the association between disability and mental health. Furthermore, even for those confounders for which there is good quality, accurately measured data, adjusting for such variables at a single point in time is unlikely to capture the complexity of the causal processes that play out over people's life course leading to differences in socio-economic characteristics between people with and without disabilities [326]. However, in this thesis I identified people who had recently acquired a disability and adjusted for socio-economic characteristics prior to disability to improve exchangeability of the exposure groups. People with incident disability are likely to be more comparable in terms of socio-economic characteristics prior to disability than people who have lived with disability for many years or those with a lifelong impairment. Therefore, this may lessen the problem of bias from residual confounding, though some will be inevitable.

Longitudinal studies are prone to selection bias arising from missing data due to non-response to the survey, loss to follow up from the survey, and missing observations for some variables among respondents. Selection bias would lead to biased estimates of effect if the missing data are influenced by both disability acquisition (the exposure) and mental health (the outcome). Because, by definition, people acquired their disability after joining the survey, the decision to participate in the survey is not affected by their exposure status, and therefore non-response to the survey will not lead to selection bias. Loss to follow up from the survey, however, is likely to be affected by both disability acquisition and people's mental health, and therefore may lead to biased estimates of effect. However, attrition from the survey was low, on average 6% between waves. Missing observations for certain questions in the survey are also likely to have been affected by both disability and mental health, and there was a large proportion of missing observations for some variables such as mental health. However, in all analyses I used multiple imputation to corrects for selection bias from missing observations under the assumption that the data were MAR, which is plausible.

The most problematic source of bias in this thesis is likely to arise from information bias because it was not addressed quantitatively in the analyses (in contrast to confounding and selection bias) and its impact on the results is difficult to assess. In the section of the thesis describing the methodological challenges in Chapter 3, the various possible

types of measurement error were described - independent non-differential, dependent non-differential, independent differential or dependent differential. Because all variables in the analyses were self-reported, there is potential for measurement error in the reporting of both the exposure and the outcome and these are likely to be statistically dependent. There was potential for misclassification of disability because of the use of a very broad definition of disability. Two consecutive waves of disability were used to characterise acquisition, which may reduce the possibility of measurement error. There was also potential for measurement error of mental health, and there is some evidence of systematic differences in reporting between people with and without disabilities [327]. As such, any measurement error is likely to be dependent differential, which is the most concerning type of information bias, and its effect on the results is difficult to assess. Using a different data source with more objective mental health outcomes such as clinical diagnoses of mental illness, use of mental health services or prescription of anti-depressants may address this problem of bias. However, examining a self-reported measure of people's mental health and well-being allows results to be interpreted in terms of determinants of good mental health for the whole population of people with disabilities rather than maintaining a narrower focus on preventing mental illness among the most severely affected.

There is also potential for measurement error in the reporting of the mediators, which has been shown to lead to an underestimation of the mediated effect [323], and measurement error in potential confounders leading to residual confounding even in the presence of independent non-differential measurement error. Whereas the between-person analyses are likely to be vulnerable to biases arising from residual confounding, the within-person analyses may be less prone to residual confounding by mismeasured covariates as the analytic design implicitly controls for all time invariant confounding.

The focus of this thesis is relatively narrow because it is restricted to those people who acquire a disability in adulthood. Consequently, the results are not representative or applicable to the wider population of people with disabilities in Australia for whom policies and strategies are needed to improve poor mental health. It also limits our understanding of the causes of disability-related mental health inequalities across the life course. However, restricting the analyses to people who acquire a disability confers substantial benefits in terms of exchangeability of the exposure groups and thereby minimises bias from confounding and reverse causation, and addresses to some extent problems of exposure-induced mediator-outcome confounding which are particularly problematic if there is a long lag time between the exposure and the outcome.

Furthermore, the HILDA sample generally is unlikely to be representative of the underlying population of people with disabilities in Australia. Though it is designed as a nationally-representative survey, the people who choose to participate are of higher socio-economic status than those people who do not participate. Additionally, it is likely to underrepresent people with severe disabilities who may be more likely to live in hospitals, nursing homes or group homes because the HILDA Survey is restricted to people living in private dwellings.

There were also limitations relating to the way in which the exposure and outcome were defined. As described in Chapter 2, the definition of disability used in the analyses contributing to this thesis was derived from the ICF and represents a broad definition of disability including many different types of impairment and on different scales of severity. The ICF emphasises that disability represents a continuum of functioning, however it is almost always dichotomised in practice which makes it more suitable for measuring and monitoring the circumstances and inequalities experienced by people with disabilities and designing services, policies and programmes. However, the dichotomous classification used in HILDA has implications for the interpretation of the findings as the broad definition leads to a diverse group of people with disabilities with different levels of functioning who are likely to experience heterogeneity in the effect of disability on mental health. There may be differences in how this heterogeneous group of people experience the effect of disability on mental health (and on their socio-economic circumstances) according to the severity of people's disabilities, impairment type, age and mode of acquisition (for example, acquiring a disability through an injury versus ageing). It would be of interest to quantify these differential effects which could be achieved through analyses of effect modification to understand the best strategies to maximise mental health for different subgroups of people with disabilities. It is an important avenue for future research, however it is beyond the scope of the thesis. Furthermore, such subgroup analyses cannot be carried out meaningfully using the HILDA data due to low power.

A limitation is also the focus on short-term mental health effects associated with disability acquisition. The effect of disability on mental health is likely to change over time, therefore examining only short-term effects limits our understanding of the effects of disability acquisition on mental health over the life course. Though we did include data for more waves following disability acquisition in the within-person analyses, 70% of the sample contributed only two or three years of mental health data following disability acquisition, therefore the results represent short-term mental health effects for most of

the sample. Future research could examine longer term mental health effects as well as heterogeneity in trajectories of mental health effects following disability acquisition.

Furthermore, the concepts of disability and mental health are related, making it difficult to isolate the causal effect of disability on mental health. Some of the people contributing to the disability sample acquired a psychosocial disability, and therefore they would be expected to experience a decline in their mental health associated with disability acquisition. To address this limitation, in all analyses except for the four-way decomposition study, I presented the results of a sensitivity analysis in which people with psychosocial impairments were excluded from the analysis. Though this tended to decrease the magnitude of the estimated effects of disability acquisition on mental health, the patterns of associations were the same and it did not change the interpretation of the findings. Though it would be interesting to specifically examine differences in the estimated effects according to impairment type, there was not sufficient statistical power to do this using the HILDA data.

The measure of mental health is also correlated with self-reported social support with a Pearson's correlation coefficient of 0.49, making it difficult to quantify the influence of social support on the relationship between disability and mental health. Using an objective mental health measure such as clinical diagnoses of mental illness, mental health services use or prescription of anti-depressants from linked administrative data could address the issue of mental health being conceptually related to disability and social support, though administrative data are prone to other biases including information bias and external validity of the sample. Using a more objective measure of social support such as a more objective quantification of people's social networks could mitigate the problem of correlated measures, however social networks represent a slightly different theoretical concept to an individual's perceived social support.

There were additional limitations relating to sample size and statistical power. Because of how I defined disability acquisition using the definition of two waves of data reporting no disability followed immediately by two waves of data with disability, the sample of people who acquired a disability was fairly small, meaning there was low power to detect effects. This is particularly relevant in studies of mediation and effect modification which rely on people being *doubly exposed* to disability and socio-economic characteristics to generate estimates of effect.

Multiple testing is also worth noting as a limitation. In the first analysis, I tested for effect modification by a large number of different socio-economic characteristics, which increases the risk of type I error, whereby results are found to be statistically significant

purely by chance. However, the variables were selected a priori and all associations were in the expected direction, therefore they were unlikely to have arisen by chance. Furthermore, the interpretation of results relied on the magnitude of the estimated effects more than the statistical tests of significance.

Finally, there were some limitations relating to the causal mediation methods. Standard causal mediation approaches only allow quantification of indirect effects through a single mediator. The first mediation analysis in this thesis used a sequential mediation analysis, which extends the causal mediation approach to examine indirect effects through multiple mediators examined jointly. However, this method makes assumptions about the direction of causal ordering between the different mediators. The second mediation analysis quantified interventional effects which can estimate effects through two distinct mediators, without the strong assumptions about causal dependencies between the mediators, but this method is limited to only two mediators.

Though the effect modification and mediation analyses examined the influence of a large number of different socio-economic characteristics, it is important to acknowledge that disability is likely to interact with multiple socio-economic factors simultaneously in complex ways. The interconnected nature of these social categorisations could not be captured quantitatively to take into account how these different axes of inequalities, and their combinations, interact to produce disability-related mental health inequalities.

While attempts to minimise bias are necessary, observational epidemiological studies will always be subject to biases and errors, particularly those addressing complex research questions such as understanding the relationship between disability acquisition and mental health. Therefore, it is important to remember that results from single studies, whether observational or experimental, need to be interpreted in light of the entire body of evidence surrounding the research question. Studies using different designs, different data sources, conducted in different contexts and populations are likely to be subjected to diverse sources of bias [328]. Therefore, integrating evidence from diverse studies – known as triangulation of evidence – will produce a stronger evidence base on which to base policy decisions.

7.3 Interpretation and implications of findings

This PhD thesis has demonstrated that socio-economic characteristics are instrumental in explaining the effect of disability acquisition on mental health. The main contribution of this thesis relates to the understanding of the mechanistic pathways leading from disability acquisition to poor mental health, which has important policy implications.

7.3.1 Comparisons with other studies

The poor mental health of people with disabilities has been widely documented in Australia and there is strong evidence from multiple studies internationally demonstrating a causal effect of disability acquisition on mental health. However, surprisingly, the causes of the mental health inequality and the mechanisms driving it have received very little attention.

This thesis comprises the first studies that have examined a broad range of different socio-economic characteristics as effect modifiers of the effect of disability acquisition on mental health. The results demonstrated that socio-economic characteristics contributed to the effect of disability on mental health, with people who were socio-economic disadvantaged prior to disability experiencing larger mental health declines. The magnitude of the mental health decline was notably large for people who were unemployed, in low skilled jobs, had low income, low wealth, experienced financial hardship, lived in public housing, in unaffordable housing, those who were not in a relationship and had poor social support. The results are consistent with previous studies of effect modification which examined individual characteristics as effect modifiers, which found evidence of effect modification by education, employment, financial resources, housing, and social support.

This is the first application of causal mediation methods to examine how much of the effect of disability acquisition on mental health was explained by socio-economic characteristics. The results suggested that a large proportion of the effect was explained by material socio-economic factors, with employment emerging as a particularly important mediator, with little effect explained by psychosocial and behavioural factors. The results differed substantially from previous studies which suggested substantial mediation through psychosocial resources such as self-esteem, sense of control and stress but not through social support or relationship status. However, the differences between the results may be explained by limitations in the methodological approaches for mediation analysis present in all previous studies that could have led to bias, as well as differences in the study populations and the variables used to measure disability, mental health and socio-economic characteristics.

7.3.2 Mechanisms leading from disability acquisition to poor mental health

The findings of the analyses have highlighted the importance of socio-economic characteristics in shaping the mental health of people with disabilities, with both effect modification and mediation mechanisms implicated in the effect.

There is strong evidence from this thesis and other studies that people who are socio-economically advantaged prior to disability experience smaller mental health deterioration than those who experience disadvantage. Therefore, this suggests that socio-economic factors can provide a buffer protecting people who acquire a disability against mental health declines. I hypothesise that people who experience favourable social and economic conditions generally share a constellation of protective characteristics, attitudes or behaviours, reinforcing the idea of the interconnectedness of socio-economic factors in shaping how disability impacts on people's mental health.

There is also strong evidence supporting the hypothesis that material socio-economic characteristics subsequent to disability (and occurring as a result of disability acquisition) explain in part the effect of disability on mental health, with employment emerging as a particularly important explanatory factor. When further disentangling the mechanisms by which this occurred, there was evidence that the effect was mainly driven by interaction of the effect of disability and employment on mental health. This suggests that susceptibility to the mental health consequences of disability depends on exposure to contributing risk factors, such as unemployment, which can mitigate or exacerbate the effect of disability on mental health.

Not only do the results provide insight into the mechanisms leading to poor mental health and disability-related mental health inequalities, they also provide insight into the causes of poor mental health in the general population which may hold clues for advancing population mental health.

7.3.3 Policy implications

A better understanding of the mechanisms by which disability acquisition leads to poor mental health provides insight into policy recommendations, strategies and directions for improving the mental health of people with disabilities and advancing health equity, which is a matter of great political interest.

Addressing the social determinants of mental health

The findings of the research point to both upstream and downstream social determinants of disability-related mental health inequalities, and therefore require both upstream and downstream strategies to address them.

Socio-economic characteristics measured prior to disability were found to provide a buffer against the negative mental health effects of acquiring a disability, suggesting the need for strategies addressing upstream social determinants of mental health before people acquire a disability, therefore policies aiming to alleviate socio-economic

disadvantage both for people with disabilities and the general population. A social determinants of health approach to addressing disability-related mental health inequalities offers an opportunity to develop sustainable and equitable health outcomes in the population [329]. However, policy solutions to address the social determinants of health require a unified cross-sectoral approach, with interventions predominantly outside of the health sector.

The mediation results demonstrating the importance of material socio-economic factors in explaining the relationship between disability acquisition and mental health also suggest that social policy reforms that reduce socio-economic disadvantage among people who acquire a disability will improve mental health. This could be achieved through better social protection, but also through improved educational and employment opportunities for people with disabilities and access to affordable housing.

Improving employment opportunities for people with disabilities

The finding that employment is a key mediator of the relationship between disability acquisition and mental health highlights the need to also implement policy interventions to address specific downstream social determinants of health such as employment. Furthermore, the decomposition of the indirect effect suggested that the mediation resulted predominantly from interaction between disability and employment, whereby susceptibility to the mental health consequences of disability acquisition depended on exposure to socio-economic risk factors, such as unemployment. This implies that policies should target the differential susceptibility to poor mental health by equalising employment outcomes between people with and without disabilities.

Multilevel policy strategies need to target the causes of low employment rates for people with disabilities, including structural interventions such as investing in vocational and training opportunities, improving participation in, and outcomes of, disability employment services, subsidising programmes for businesses to hire people with disabilities, and strategies to get young people with disabilities into the labour force to improve people with disabilities' prospects of gaining employment. It is also important to maintain ongoing employment for people in the labour force who acquire a disability, improving the sustainability of employment by adapting the work environment such as improving workplace flexibility, educating employers about employment of people with disabilities and reasonable modifications, addressing discrimination in the workplace, and changing attitudes towards people with disabilities. Such policy changes, if effective, would improve the mental health of people with disabilities and have the potential to reduce current mental health inequalities by a third. Also of great relevance, though not

examined in this thesis, is the importance of meaningful, sustainable and high-quality employment for people with disabilities to promote mental health.

Finally, because employment is a pervasive social determinant of health, improving employment rates and employment conditions for people with disabilities will improve their mental health but is also likely to have flow-on effects to many other health outcomes.

Improving mental health systems, programmes and services

The findings of the thesis add to the comprehensive evidence base demonstrating the poor mental health experienced by people with disabilities and strengthen the evidence for a causal effect of disability on mental health.

Though the aim of the thesis was to better understand the socio-economic determinants of the poor mental health of people with disabilities rather than the impact of services, and therefore the key implications relate to addressing those social determinants of poor mental health, it is important for the health system to be resourced to respond to the needs of the population [329]. The large burden of poor mental health among the population of people with disabilities in Australia highlights the importance of improving mental health systems, programmes and services for people with disabilities, in order to address the mental health inequalities rather than perpetuate them.

The findings underscore the need to provide high-quality accessible and affordable mental health services for people with disabilities, and ensure availability of appropriate mental health information in accessible formats. Other strategies include improving mental health supports and programmes for people who acquire a disability and mental health promotion interventions specifically targeted to unemployed people with disabilities.

7.4 Directions for further research

This thesis has made important contributions to what is known about the mechanistic pathways leading from disability to poor mental health and has generated policy recommendations based on robust evidence. However, it does not completely solve the research problem stated in the introduction – the lack of evidence about the mechanisms driving the effect. Though employment has been identified as a key pathway explaining the effect, there are still uncertainties about other factors contributing to the effect, which present important avenues for future research. Furthermore, there remains uncertainty about *how* to implement policy interventions targeting known social determinants of

health on the pathways from disability acquisition to poor mental health to most effectively reduce current inequalities. Finally, the research in this thesis has also shed light on the need for better data and new methodological approaches to address the limitations of the current research.

7.4.1 Other mechanisms

The first mediation analysis estimated that a third of the effect of disability acquisition on mental health was explained by material factors. The subsequent analysis examined employment and income, quantifying the effect through each of these individually and estimated that 11% of the effect was explained by employment status and none through income. This implies that a large part of the indirect effect remains unexplained.

Further research is needed to quantify indirect effects through other distinct characteristics. Particularly notable for future analyses is the effect operating through financial hardship, identifying people who are living at the extreme edge of poverty and how this contributes to the impact of disability on mental health. Such research would be invaluable in clarifying the effect of poverty on the relationship between disability acquisition and mental health and provide insights that could inform policies targeting people and households with disabilities living in extreme poverty.

Another avenue for future research involves exploring other aspects of employment. The mediation analysis quantifying the indirect effect through employment examined a crude dichotomous variable comparing people who were in employment or not in employment. This decision was made because of the limitations of the statistical methods, which can only model the effect of binary or continuous variables. However, as was demonstrated in the analyses of effect modification, there were large differences in the magnitude of the effect of disability acquisition on mental health according to people's occupation prior to disability, with those in low skilled jobs experiencing even larger mental health declines than people who were unemployed. Therefore, quantifying the proportion of the effect operating through different characteristics of employment, particularly occupation type, may provide further policy-relevant insights into the influence of employment and employment characteristics in explaining the effect of disability acquisition on mental health.

Finally, qualitative studies are also necessary to complement the findings of this research, to attain a deeper understanding of the causes of the poor mental health experienced by people with disabilities, the impact of the interactions and interconnectedness of multiple axes of socio-economic inequalities, and the role of

personal factors, context, and environment in shaping the mental health of people with disabilities, in line with the ICF conceptualisation of disability and health.

7.4.2 Policy interventions to target known drivers of mental health inequalities

Although the findings of the research point strongly towards interventions targeting employment to reduce disability-related mental health inequalities, there remains a gap in the evidence about which policies to implement, and how to implement them, to most effectively improve mental health of people with disabilities.

There are several analytic approaches that could be used to assess the effect of policies on employment in addressing mental health inequalities. Firstly, this could be achieved through mathematical modelling to simulate the effect of hypothetical policy interventions on employment to guide policy decision-making. I propose that a pressing next step should be to use longitudinal data to model the effect of realistic population-level policy interventions, such as changes to employment rates for people with disabilities and employment conditions, in addressing mental health and disability-related mental health inequalities.

Similar analyses have been conducted to examine the effect of physical activity interventions on prevalence and inequalities in children's mental health problems [330] and obesity [331] and to examine the effect of interventions to improve school entry academic skills on socio-economic inequalities in educational achievement [332]. These studies used marginal structural models to compute the indirect effects through the mediator of interest, and then modify the value of the mediator for individuals in the sample to simulate the effect of a potential policy and estimate the effect on the outcome. This approach could be applied to simulate the effect of several realistic policy interventions on employment and compare and rank their effectiveness in improving the mental health of the general population and people with disabilities, and the effect on reducing disability-related mental health inequalities. Simulating the effect of realistic implementable policies on mental health may have more impact in terms of translation into policy reform than estimates of the proportion of the effect explained by a socio-economic characteristic.

Secondly, cross-national studies comparing the mental health of people with disabilities living in different countries could improve understanding of how the policy environment influences the effect of disability on mental health. However, it may be difficult to isolate the effect of specific policies from other differences between the countries, such as the wider policy landscape, the context and environment in which people live. Furthermore,

cross-national analyses require consistently measured, comparable measures of disability and mental health.

Finally, natural experiments provide a methodologically robust approach to generating causal estimates for understanding the health impacts of policy changes. Such approaches could involve whole population comparisons of mental health outcomes and inequalities before and after a policy change, comparisons between populations who were exposed and not exposed to a policy change, such as an employment programme implemented by State governments or the staggered implementation of the NDIS, or comparisons of population groups that were or were not eligible for an employment programme.

7.4.3 Improvements to mental health systems, programmes and services

The findings of this thesis underscore the need to provide high-quality mental health systems, programmes and services for people with disabilities. To complement the research of this thesis, it would be valuable to study the potential benefits of improving mental health systems, programmes and services, and what the effectiveness of different policies to achieve this, to implement evidence-based action to address the mental health needs of the population of people with disabilities.

7.4.4 Better data and statistical methods

Another key finding that emerged throughout this thesis was the need for better data and better methods to address the limitations of current research. Consistent, high-quality measures of disability as well as robust indicators of health and well-being are required for research and policy to enable better tracking of changes in the health of people with disabilities and health inequalities over time, monitor the effect of policies and interventions, and influence decisions such as resource allocation and service provision.

Data linkage provides an opportunity to replicate and extend this research using a data source subjected to different biases. Linked data has the potential to gather together high-quality data on disability, socio-economic characteristics and health outcomes for the same individual from different sources. With the increase in availability of digital health records, data linkage is particularly attractive for research on disability and health as it could provide whole population data rather than a small subsample of the population, maximising the external validity of results. Furthermore, it could bring together Census data and administrative data, which record disability and socio-economic characteristics, as well as other data sources such as hospital records or Medicare data which record more objective measures of mental health than recorded in self-reported surveys such as mental health service use and prescription of

pharmaceuticals. However, there are currently major barriers to accessing linked data for the whole population in Australia.

Another potential data source for research on disability and health in the future is the National Disability Data Asset, a longitudinal dataset incorporating datasets from multiple levels of government, which is currently being developed to address barriers and improve outcomes for Australians with disabilities [333].

Methodological developments are also necessary to address the limitations of the current research. One of the limitations of the causal mediation methods as they stand is their inability to estimate indirect effects through categorical mediators. Extensions to these methods to accommodate categorical mediators would be valuable to provide more flexibility in assessment of potential mediators of the effect of disability acquisition on mental health, such as the effect of employment characteristics.

It would also be valuable to develop methods to address the methodological limitations relating to information bias, which is particularly problematic because it is not possible to predict the direction and magnitude of bias caused by dependent differential measurement error. One such approach would be to develop a method to perform a quantitative bias analysis to estimate the impact of dependent differential measurement error on the results. Such methods would provide an important contribution to this research question and would have many applications in the wider field of social epidemiology.

7.5 Conclusion

In conclusion, this thesis has demonstrated that a broad range of socio-economic characteristics were found to be implicated in the mechanistic pathways leading from disability acquisition to poor mental health, with employment identified as a particularly important contributing factor in the effect.

The findings highlight the importance of the social determinants of health in generating disability-related mental health inequalities, suggesting that interventions should prioritise addressing the social determinants of health to reduce current mental health inequalities and improve the mental health of people with disabilities in Australia. Furthermore, the evidence that employment is a key mediator of the effect of disability acquisition on mental health indicates that policy strategies are needed to target the causes of low employment rates for people with disabilities. Implementation of effective evidence-based interventions to improve the social determinants of health, including

employment, for people with disabilities offers real possibilities for improving the mental health of people with disabilities as well as population mental health generally.

Though the need for further research remains, there is robust evidence on which to guide action. Given the increasing prevalence of disability with projected trends in population ageing and the changing disability policy landscape in Australia, this is a pivotal point in time to use robust evidence on modifiable determinants of health to contribute to policy strategies to reduce inequalities for people with disabilities. Collectively, the findings of this thesis suggest that efforts need to be made to prioritise action on the social determinants of health to reduce (or ideally eliminate) existing avoidable disability-related mental health inequalities and improve the mental health, well-being and lives of people with disabilities.

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