Predictors and health effects of smoking transitions in young adults

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Doctor of Philosophy (Medical Research)

Menzies Institute for Medical Research

University of Tasmania

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Declaration of originality

This thesis contains no material which has been accepted for a degree or diploma by the University or any other institution, except by way of background information duly acknowledged in the thesis, and to the best of my knowledge and belief no material previously published or written by any other person except where due acknowledgement is made in the text of the thesis, nor does the thesis contain any material that infringes copyright.

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The research associated with this thesis abides by the international and Australian codes on human and animal experimentation, the guidelines by the Australian Government's Office of the Gene Technology Regulator and the rulings of the Safety, Ethics and Institutional Biosafety Committees of the University.

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Abstract

Background: Young adults have the highest prevalence of current smoking and will have the greatest health benefits if they quit. Relatively few studies have focused specifically on this group. There is a need for high-quality data on the relationship between smoking and some factors that are either common in young adults (e.g. life-stage transitions) or known to be associated with lower cessation levels (e.g. post-cessation weight gain).

Aims: To 1) examine the impact of life-stage transitions and socioeconomic position (SEP) variation across the life course on (changing) smoking status; 2) quantify weight gain after smoking cessation and the difference in weight gain between quitters and continuing smokers; 3) explore the underlying mechanisms linking smoking cessation and weight gain; and 4) investigate the longitudinal relationship between change in smoking status and change in health-related quality of life (HRQoL) in young adults.

Methods: 1) For aim 1, 3 and 4, data were from the Childhood Determinants of Adult Health (CDAH) study, a 25-year follow-up of 8,498 children aged 8-15 years who participated in 1985 Australian Schools Health and Fitness Survey (ASHFS). Measurements included anthropometry, socio-demographic factors, smoking status, dietary behaviours, physical activity (PA), sedentary behaviours, and HRQoL.

2) A systematic review and meta-analysis was utilised to test the second aim. Five electronic databases were searched prior to January 2015. Population-based prospective cohort studies were included if they recorded the weight change of adult smokers from baseline (before quitting smoking) to follow-up (at least three months after cessation).

Results: The main findings were that the transition into relationship with a partner and entering parenthood were associated with beneficial changes in smoking behaviours, but these influenced young men and women differently. Exposure of low SEP for greater periods of time across the life course was associated with an increased risk of smoking in mid-adulthood. Parental smoking and a self-rated low

importance of not smoking at childhood appeared to be influential in mediating this relationship.

In the meta-analysis using data from 35 cohort studies including 63,403 quitters and 388,432 continuing smokers, we found that people who quit smoking gained an average of 4.1 kg weight over about five years, which was 2.6 kg greater than the gain in continuing smokers. In supporting analyses from the CDAH study, this post-cessation weight gain was not attenuated after adjustment for worsening dietary and PA behaviours. Relative to continuing smoking, quitting smoking was significantly associated with an improvement in physical HRQoL. No significant association was observed between changes in smoking status and change in mental HRQoL.

Conclusions: Partnering and parenting transitions and SEP trajectories across the life course predicted smoking status or changes in smoking status. Compared with continuing smoking, quitting smoking led to greater weight gain, which was not explained by changing dietary and PA behaviours, and a significant improvement in physical HRQoL. These analyses have provided novel information on predictors of smoking cessation and the associated health effects in young adults – a high priority group. The findings may help to promote smoking cessation and the maintenance of abstinence at the population and individual level.

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Statement of authorship

This thesis includes papers for which Jing Tian (JT) was not the sole author. JT was the first author in the research of each manuscript; however, she was assisted by the co-authors whose contributions are detailed below:

Chapter 4

Tian J, Venn A, Otahal P, Gall S. The association between quitting smoking and weight gain: a systematic review and meta-analysis of prospective cohort studies. Obes Rev, 2015; 16(10): 883-901.

JT designed the review protocol, carried out the literature search, contributed to data extraction, assessed the study quality, performed the data analysis, contributed to the results interpretation and wrote the manuscript.

AV designed the review protocol, assessed the study quality, helped with interpretation of the results and revised the manuscript.

PO contributed to data extraction, provided statistical support for meta-analysis and revised the manuscript.

SG designed the review protocol, carried out the literature search, helped with interpretation of the results and revised the manuscript.

Chapter 5

Tian J, Gall S, Smith K, Dwyer T, Venn A. Worsening dietary and physical activity behaviours do not readily explain why smokers gain weight after cessation: a cohort study in young adult. Nicotine Tob Res, 2016; pii: ntw196. [Epub ahead of print].

JT conceptualised the paper, conducted the data analysis, contributed to the results interpretation and wrote the manuscript.

SG assisted in conceptualising the paper, assisted with interpretation of the results and revision of the manuscript.

KS helped with interpretation of the results and revised the manuscript.

Statement of authorship

TD helped with interpretation of the results and revised the manuscript.

AV assisted in conceptualising the paper, assisted with analyses and interpretation of the results and revision of the manuscript.

Chapter 6

Tian J, Venn A, Blizzard L, Patton G, Dwyer T, Gall S. Smoking status and health-related quality of life: a longitudinal study in young adults. Qual Life Res, 2016; 25(3): 669-85.

JT conceptualised the paper, conducted the data analysis, contributed to the results interpretation and wrote the manuscript.

AV assisted in conceptualising the paper, assisted with interpretation of the results and revision of the manuscript.

LB advised on data analysis and interpretation, and revised the manuscript.

GP helped with interpretation of the results and revised the manuscript.

TD helped with interpretation of the results and revised the manuscript.

SG assisted in conceptualising the paper, assisted with analyses and interpretation of the results and revision of the manuscript.

Signed by first named supervisor, Professor Alison Venn

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Signed: ……Date: …….

List of Publications

Publications arising directly from the work described in this thesis

Chapter 4

Tian J, Venn A, Otahal P, Gall S. The association between quitting smoking and weight gain: a systemic review and meta-analysis of prospective cohort studies. Obes Rev, 2015; 16(10): 883-901.

Chapter 5

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Manuscripts published during candidature, but external to thesis material

Pan F, Laslett L, Tian J, Cicuttini F, Winzenberg T, Ding C, Jones G. Pain at sites outside the knee predicts knee cartilage volume loss in elderly people without knee osteoarthritis: A prospective study. Arthritis Care Res (Hoboken). 2016 Jul 7. doi: 10.1002/acr.22964. [Epub ahead of print].

Pan F, Tian J, Winzenberg T, Ding C, Jones G. Association between GDF5 rs143383 polymorphism and knee osteoarthritis: an updated meta-analysis based on 23,995 subjects. BMC Musculoskelet Disord. 2014 Dec 2;15:404. doi: 10.1186/1471-2474-15-404.

Conference presentations arising from this thesis

Oral presentations:

International conferences

1. Tian J, Gall S, Otahal P, Smith K, Dwyer T, Venn A. Smoking cessation and weight gain: are changing health behaviours explanatory? European Public Health Conference. Milan, Italy, 14-17th October 2015; Oceania Tobacco Control Conference. Perth, Australia, 20th-23rd October 2015.

2. Tian J, Gall S, Dwyer T, Venn A. Effects of partnering and parenting transitions on smoking continuity and change over 5 years in young Australians: the Childhood Determinants of Adult Health Study. European Public Health Conference. Milan, Italy, 14-17th October 2015; Oceania Tobacco Control Conference. Perth, Australia, 20th-23rd October 2015.

3. Tian J, Venn A, Blizzard L, Patton G, Dwyer T, Gall S. Change in smoking status and change in health-related quality of life in young adults. European Public Health Conference. Milan, Italy, 14-17th October 2015; Oceania Tobacco Control Conference. Perth, Australia, 20th-23rd October 2015.

National conferences

1. Tian J, Venn A, Blizzard L, Patton G, Dwyer T, Gall S. Change in smoking status and change in health-related quality of life in young adults. Population Health Congress. Hobart, Australia, 6-9th September 2015.

2. Tian J, Venn A, Otahal P, Gall S. The association between quitting smoking and weight gain: a systemic review and meta-analysis of prospective cohort studies. Australasian Epidemiological Association (AEA) Annual Meeting. Auckland, New Zealand, 8-10th October 2014.

3. Tian J, Otahal P, Blizzard L, Venn A. Risk of subsequent non-melanoma skin cancer in patients with a previous diagnosis. Australasian Epidemiological Association (AEA) Annual Meeting. Brisbane, Australia, 20-22nd October 2013.

Conference presentations

Local conference

1. Tian J, Seana Gall, Venn A. Weight gain after smoking cessation: changing health behaviours do not readily explain it. Three Minutes Thesis Competition - Tasmanian Health HDR Student Conference, Hobart, Australia, 4th June 2015.

Poster presentations:

National conferences

1. Tian J, Gall S, Smith K, Dwyer T, Venn A. Changing health behaviours do not readily explain why smokers gain weight after cessation: a cohort study in young adults. Population Health Congress. Hobart, Australia, 6-9th September 2015; Graduate Research Conference, Hobart, Australia, 3rd -4th Sep 2015.

2. Tian J, Venn A, Otahal P, Gall S. The association between quitting smoking and weight gain: a systemic review and meta-analysis of prospective cohort studies. Tasmanian Health HDR Student Conference, Hobart, Australia, 28th July 2014; Graduate Research Conference, Hobart, Australia, 4 -5th Sep 2014.

Conference presentations arising external to this thesis

Oral presentations:

1. Pan F, Aitken D, Tian J, Cicuttini F, Winzenberg T, Ding C, Jones G. Does "pain elsewhere" influence the association between knee structural pathology and knee pain? Annual European Congress of Rheumatology (EULAR). London, UK, 8th–11th Jun 2016.

2. Pan F, Ding C, Laslett L, Tian J, Winzenberg T, Cicuttini F, Jones G. Pain at multiple sites outside the knee predicts knee cartilage volume loss: a prospective study in older adults. Annual European Congress of Rheumatology (EULAR). Rome, Italy, 10th–13th Jun 2015; Population Health Congress. Hobart, Australia, 6th–9th Sep 2015.

Poster presentations and poster tour presentations:

1. Pan F, Blizzard L, Tian J, Cicuttini F, Winzenberg T, Ding C, Jones G. Does weight in the offspring of people with a total knee replacement for severe primary knee osteoarthritis have a more detrimental effect on knee cartilage and pain? a 10-year prospective study. Annual European Congress of Rheumatology (EULAR). London, UK, 8th–11th Jun 2016. (Poster tour presentation)

2. Pan F, Aitken D, Tian J, Cicuttini F, Winzenberg T, Ding C, Jones G. Does pain at other sites influence the association between knee pathology and knee pain? World Congress on Osteoarthritis. Amsterdam, Netherlands, 31st Mar-3rd Apr 2016.

3. Pan F, Ding C, Laslett L, Tian J, Winzenberg T, Cicuttini F, Jones G. Pain at multiple sites outside the knee predicts knee cartilage volume loss: a prospective study in older adults. Tasmanian Health Research Student Conference. Hobart, Australia, 4th June 2015; Graduate Research Conference, Hobart, Australia, 3rd–4th Sep 2015. Annual Scientific Meeting of Australia Rheumatology Association (ARA). Adelaide, Australia, 23th–26th May 2015.

Awards resulting from thesis material

2015 Poster Prize (AU$100): Best poster and best poster defence at the 5th Australia-China Biomedical Research Conference, Hobart Satellite Meeting for the poster entitled "Worsening dietary and physical activity behaviours do not readily explain why smokers gain weight after cessation: a cohort study in young adults"

2015 Australian Government Department of Health Scholarship (AU$1,952): to attend the 2015 Oceania Tobacco Control Conference (Perth) for three oral presentations "Smoking cessation and weight gain: are changing health behaviours explanatory?", "Effects of partnering and parenting transitions on smoking continuity and change over 5 years in young Australians: the Childhood Determinants of Adult Health Study" and "Change in smoking status and change in health-related quality of life in young adults"

2015 Australasian Epidemiological Association Student Award (AU$500): to attend the Population Health Congress (Hobart). I delivered an oral presentation entitled "Change in smoking status and change in health-related quality of life in young adults"

2015 University of Tasmania Conference and Research Travel Funding (AU$2031): to attend the European Public Health Conference (Milan, Italy). I gave three oral presentations "Smoking cessation and weight gain: are changing health behaviours explanatory?", "Effects of partnering and parenting transitions on smoking continuity and change over 5 years in young Australians: the Childhood Determinants of Adult Health Study" and "Change in smoking status and change in health-related quality of life in young adults"

2014 Australasian Epidemiological Association Student Award (AU$500): to attend the Australasian Epidemiological Association Annual Meeting (Auckland, New Zealand) for an oral presentation "The association between quitting smoking and weight gain: a systemic review and meta-analysis of prospective cohort studies"

Other items awarded during candidature

2016 Chinese Government Award for Outstanding Self-financed Students Aboard 2015 (AU$8000): This is a competitive award for outstanding academic achievement across different fields. Only 500 PhD students worldwide are honoured annually.

Scientific awards

2016 Menzies Institute for Medical Research Ten of the Best Awards 2015 (AU$1000): for outstanding academic performance of staff and students over the preceding 12 months. Only 10 were awarded annually.

2013 Tasmania Graduate Research Scholarship for current PhD study.

List of abbreviations

AIHW: Australian Institute of Health and Welfare

ARIA: Accessibility/Remoteness Index of Australia

ASCO: Australian Standard Classification of Occupations

ASHFS: Australian Schools Health and Fitness Survey

BMI: Body mass index

CBT: Cognitive-Behavioral Therapy

CDAH: Childhood Determinants of Adult Health

CI: Confidence Interval

CIDI: Composite International Diagnostic Interview

COPD: Chronic Obstructive Pulmonary Disease

CVD: Cardiovascular Disease

DALYs: Disability Adjusted Life Year

DGI: Dietary Guideline Index

DHHS: Department of Health and Human Services

FFQ: Food Frequency Questionnaire

FHQ: Food Habit Questionnaire

FVC: Forced Vital Capacity

GDP: Gross Domestic Product

HRQoL: Health-Related Quality of Life

IPAQ: International Physical Activity Questionnaire

IPW: Inverse Probability Weighting

IRSD: Index of Relative Socioeconomic Disadvantage

LTPA: Leisure Time Physical Activity

MCS: Mental Component Summary

MCSD: Minimal Clinical Significant Difference

MD: Mean Difference

MET: Metabolic Equivalent of Task

MI: Multiple Imputation

NDSHS: National Drug Strategy Household Survey

NNS: National Nutrition Survey

Abbreviations

NOQAS: Newcastle-Ottawa Quality Assessment Scale

NRT: Nicotine Replacement Therapy

NSDUH: National Survey on Drug Use and Health

OR: Odds Ratio

PA: Physical Activity

PCS: Physical Component Summary

RCT: Randomised Controlled Trial

RR: Risk Ratio / Relative Risk

SD: Standard Deviation

SEIFA: SocioEconomic Indexes For Areas

SEP: Socioeconomic Position

SF: Short Form

TV: Television

UK: United Kingdom

WHO: World Health Organization

Chapter 1

Introduction

Chapter 1 Introduction

1.1 Preface

This thesis presents research using two methods 1) a systematic review and meta-analysis and 2) original analyses using data from the Childhood Determinants of Adult Health (CDAH) study, a cohort study with 25 years follow-up of 8,498 children who participated in the 1985 Australian Schools Health and Fitness Survey (ASHFS) when aged 7 to 15 years. The CDAH study includes two waves of follow-up: one conducted during 2004-06 when participants were aged 26-36 years old and the other performed five years later in 2009-11.

Using data from repeated measures of lifestyle, physical characteristics and mental health collected since childhood, the study's long-term aim is to determine the contribution of childhood factors to the risk of developing cardiovascular disease (CVD), diabetes and mental health. In this thesis, the data from the CDAH study is used to explore the predictors and health effects of smoking transitions in young adults. Investigations of these relationships in young adulthood are important because this is a time when smoking prevalence peaks and progression from occasional to regular smoking often occurs. This is also a period when life-stage transitions often take place, such as establishing life-partnerships and having children, which may influence health behaviours like smoking. Almost all smoking-related health risks are avoidable if a person quits smoking in young adulthood, making it a priority to understand the drivers of smoking trajectories during this time.

This introductory chapter describes the epidemiology of smoking prevalence worldwide and in Australia, including its health effects; factors associated with trajectories of smoking, including drivers of cessation; and the specific objectives of this thesis.

1.2 Smoking prevalence

1.2.1 Worldwide

In 2013, the global prevalence of current smoking was 21% among persons aged 15 years and above, equivalent to 950 million male current smokers and 177 million female current smokers [1]. Substantial variation was observed by age, sex and country [2]. Smoking prevalence was higher in men than women (36% versus 7%), and was higher in high-income countries

(25%) than middle-income (21%) and low-income countries (16%) [1]. In men, the highest prevalence was seen at ages 30-34 years in developed countries and ages 45-49 years in developing countries. Among women, the highest prevalence is seen between the ages of 20 and 49 years in developed countries, while the highest prevalence occurs at older ages in developing countries, reaching the highest level between the ages of 50-54 [2].

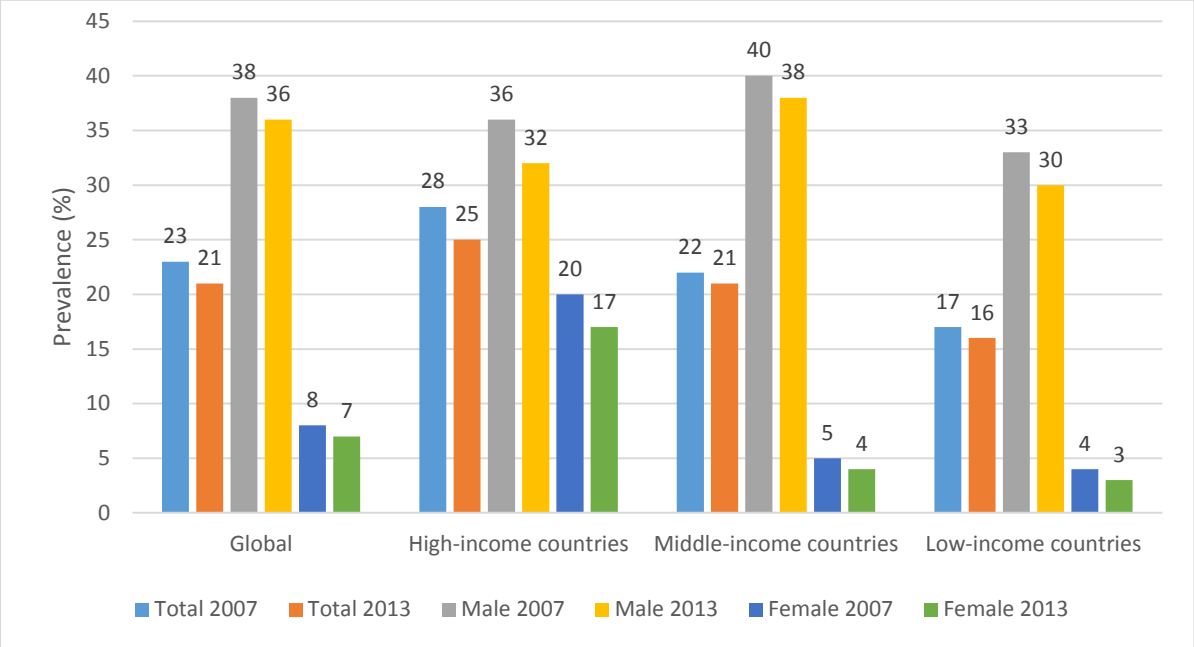


Figure 1-1 Age-standardised prevalence estimates for tobacco smoking among persons aged 15 years and over, globally, by sex, 2007 and 2013. Figure produced from data reported by World Health Organization [1]

As shown in **Figure 1-1**, there was a slight decline in smoking prevalence in recent years from 23% in 2007 to 21% in 2013; however, the total number of current smokers was stable between 2007 and 2013 due to an increase in the global population. Using a Bayesian hierarchical meta-regression modelling approach and national data about the prevalence of tobacco use from the World Health Organization (WHO) Comprehensive Information Systems for Tobacco Control, Ver Bilano and colleagues [3] reported that during 2000-10, the smoking prevalence among men fell in 125 out of 173 (72%) countries and among women fell in 155 out of 178 (87%) countries. In 2013, the World Health Assembly set a global target of a relative reduction in tobacco use by 30% among people aged 15 years or older from 2010 to 2025. If the current trends continue, only 37 countries (21%) will meet the target for men and less than half countries (88 countries [49%]) will meet the target for

women [3]. It is estimated that there will be 1.1 billion current tobacco smokers in 2025, which is similar to the number in 2013 [3].

1.2.2 Australia

In Australia, tobacco smoking was common in the middle of the 20th century, with more than three in four men and one in four women being regular smokers in 1945 [4]. Over the following decades, smoking rates fell dramatically as more people recognised the health concerns raised by research scientists [5-12] and medical authorities [13-18]. According to the latest report of the Australia-wide National Drug Strategy Household Survey (NDSHS), the prevalence of daily smoking was 12.8% among people aged 14 years or older in 2013 [19]. Women were less likely than men to have ever smoked. Overall, the highest daily smoking prevalence occurred at ages 25-29 years (16.1%) and 40-49 years (16.2%) respectively, but the distribution varied by sex. Among men, the highest daily smoking rate was seen in the age group of 40-49 years (17.9%), while for women, those aged 25-29 years were most likely to smoke daily (15.0%).

There are wide disparities in smoking within certain groups, such as those living in socioeconomic disadvantage. As shown in **Figure 1-2**, in 2013 the smoking prevalence among people living in area with high socioeconomic position (SEP) defined by socioeconomic indexes for areas (SEIFA) was 6.7% but among people living in areas with lower SEP was much higher at 19.9%. There was a decline in smoking prevalence in both groups from 1998 to 2013. However, the gap between low and high SEP groups widened from 8.6% in 1998 to 13.2% in 2013, and most importantly, it continuously grew from 2010 to 2013. There was, however, a 4.7% drop in smoking prevalence in the low SEP group between 2010 and 2013, the largest decline ever achieved, but there was also a 5.8% drop among the high SEP group. This issue will be addressed later in the thesis.

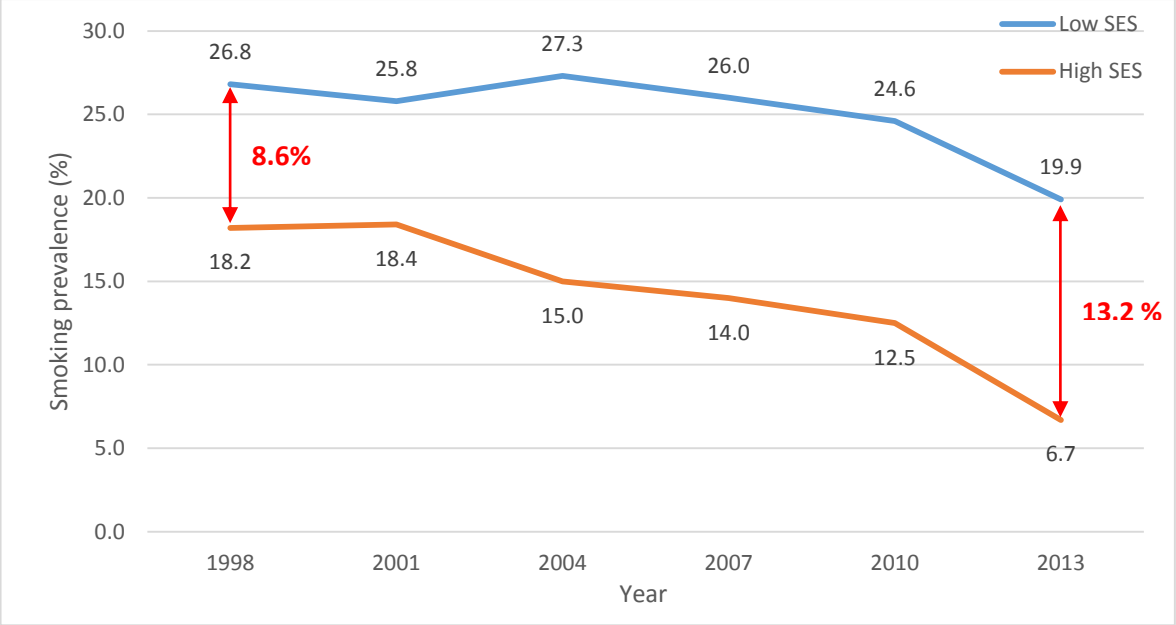


Figure 1-2 Smoking prevalence by SEIFA level in Australia, 1998-2013. Figure produced from data reported by Australian Institute of Health and Welfare National Drug Household Surveys 1998 [20], 2001 [21], 2004 [22], 2007 [23], 2010 [24], 2013 [19]

1.3 Smoking in young adults

There is a focus in this thesis on 'young adults'; however, the definition of a young adult varies widely in the literature. Some international age classifications refer to a young adult as a person aged between 19 and 24 years [25], with middle aged adults being people over the age of 40 years [26]. The participants in the CDAH study used in several parts of this thesis are aged in their mid-20s to late 30s and therefore fall outside these definitions. In the remainder of this thesis, the term young adult is used to define people in early adulthood before middle adulthood, effectively 16-41 years.

The process of smoking initiation also requires clarification. There is a sequence of stages which takes people from receptivity to dependence on smoking. As discussed by Flay (**Figure 1-3**) [27], initiation of smoking among children and adolescents can be divided into five primary stages: preparatory stage, trying stage, experimental stage, regular use, and addiction/dependent smoker. During the first stage (preparatory stage), children and adolescents form the attitudes and beliefs about smoking utility. Even though no actual smoking has taken place, smoking may be perceived as a way to become mature,

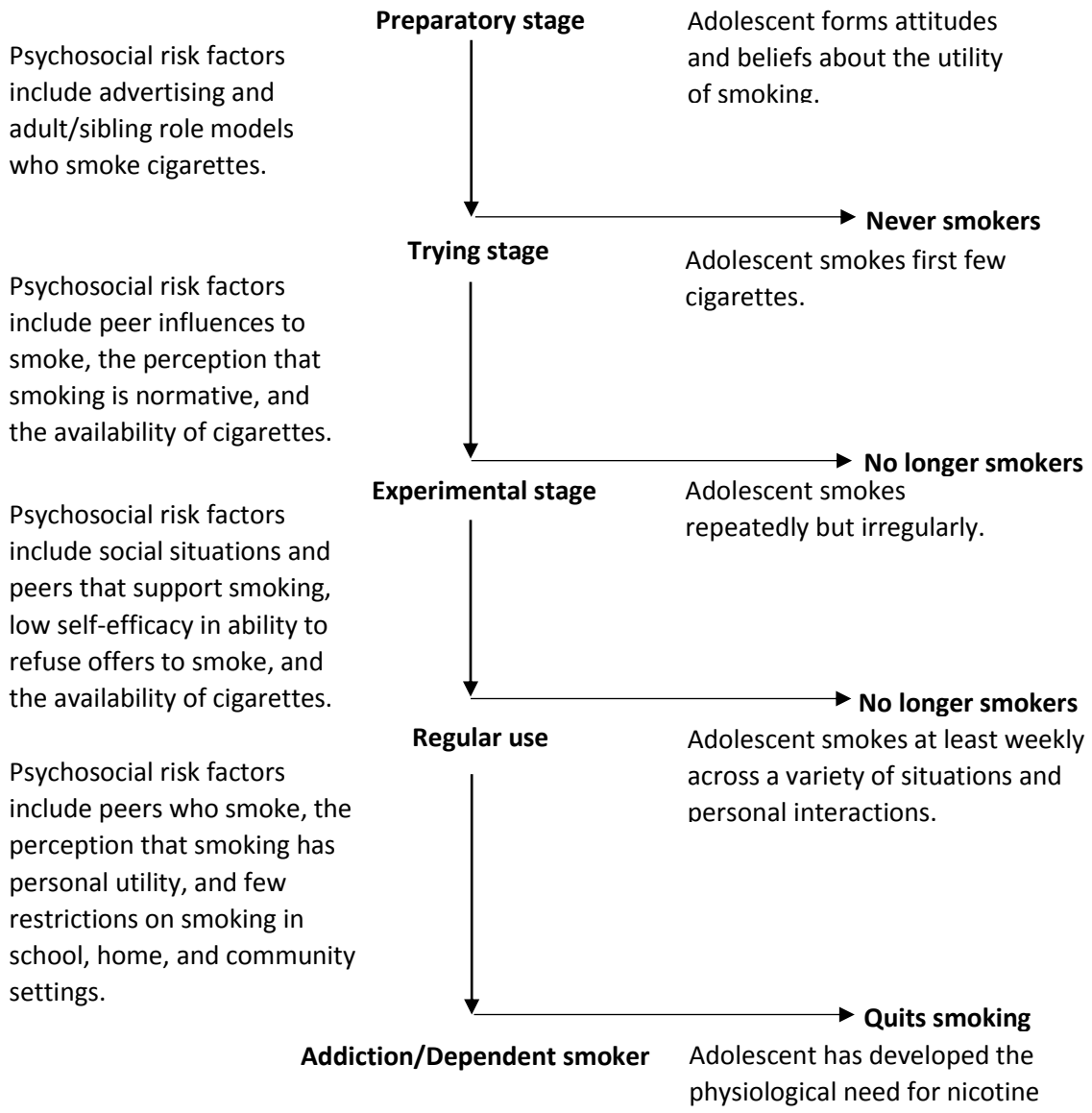


Figure 1-3 Developmental stages of smoking among children and adolescents; Figure reproduced from the 1994 Surgeon General's report of U.S. Department of Health and Human Services [28]

release stress, bond with peers, or show independence [29]. Children and adolescents smoke their first few cigarettes in the second stage (trying stage). This is reinforced if they have smoking peers and there may be transition to the next stage (experimental stage). At this stage, children and adolescents smoke repeatedly but irregularly. They generally smoke in a particular situation (such as at a party). In the fourth stage, smoking proceeds to a regular behaviour. Experimenters increase their tobacco use in frequency, usually on a daily or weekly base, and quantity, and smoke in varieties of situations. The final stage is marked by

psychological and physical dependence on nicotine. A person would experience nicotine tolerance and withdrawal symptoms if quitting smoking happens, and easily relapse to smoke if the person does quit.

There are a wide range of factors jointly influencing the decision to adopt or reject smoking among children and adolescents. The theory of triadic influence categorises these factors into three groups: intrapersonal, social, and environmental factors [30]. As illustrated in **Figure 1-4** [31], intrapersonal (intrinsic) factors cover cognitive functions, impulsivity, affective states thrill/sensation seeking, which affect self-efficacy and internal motivation to use tobacco; social (extrinsic) factors include family and peer influences that affect the perception of what constitutes normative behaviour; environmental factors include neighbourhood, cultural contexts, and general values, which influence the attitudes and evaluations of tobacco use [32]. The influence of different factors may vary at different smoking stages [33], but the evidence of stage-specific effects is weak [34].

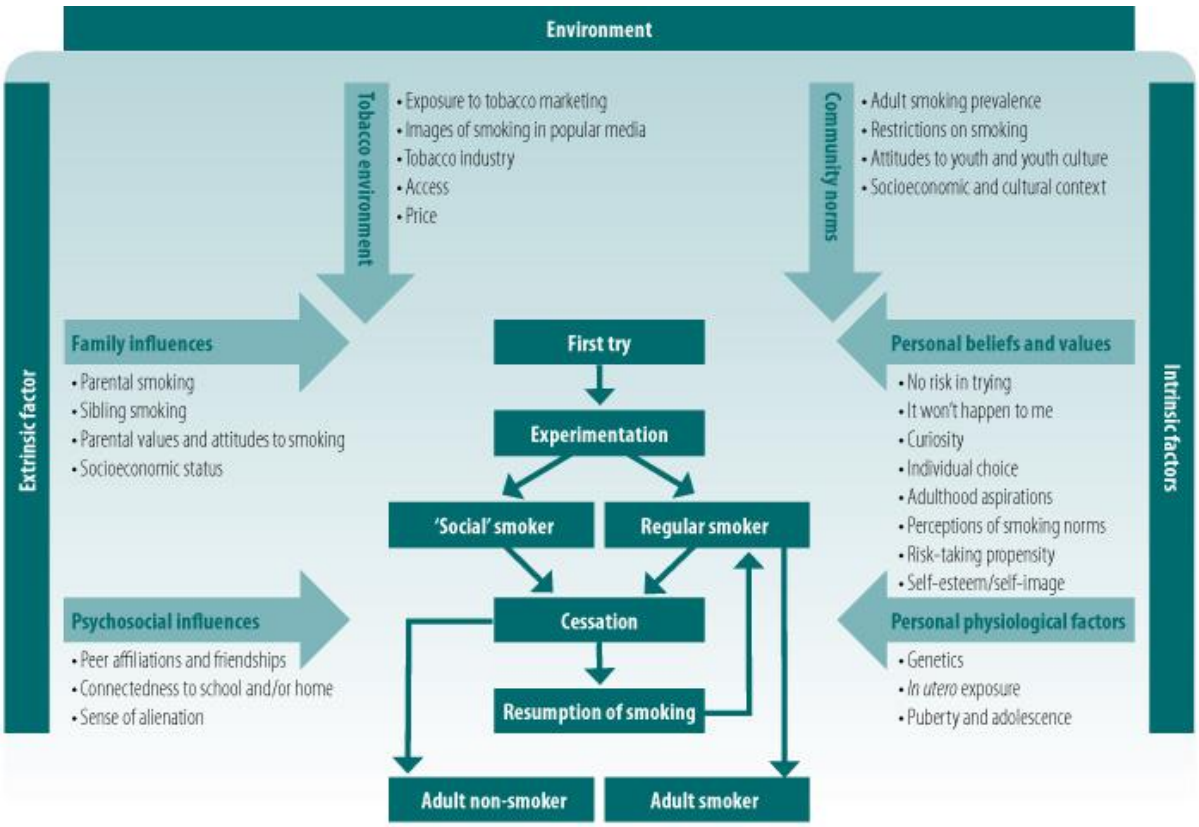


Figure 1-4 Factors influencing smoking initiation among children and adolescents; Figure reproduced from Scollo and Winstanley (with permission) [35]

Young adulthood is a critical time when changes in risk-taking behaviours such as smoking often occur [36,37]. According to a report of the Surgeon General, 88% of adult daily smokers tried their first cigarette before 18 years of age, and 99% of first use occurred by 26 years of age in the United States [38]. In the United Kingdom (UK), around 207,000 children started smoking each year [39], and about two-thirds of adult smokers started smoking before they were 18 years old, and over 80% before the age of 20 years [40]. Relative to other smokers, people who start smoking at an early age are more likely to smoke for longer and to die from a smoking induced disease [38].

1.3.1 Highest prevalence of cigarette smoking

Young adults have the highest prevalence of cigarette smoking among all age groups in developed countries [2]. For example, in 2012 in the United States, the highest prevalence of current cigarette smoking was in the age group of 18-25 years, which was 31.8% [41]. The prevalence was higher in males (36.6%) than females (27.1%), and this pattern existed for all racial/ethnic groups (see **Figure 1-5**). In 2010 in Australia, 22% adults aged 25-39 years were current smokers (25% for males and 20% for females). The prevalence in this age group was higher than any other age group [42].

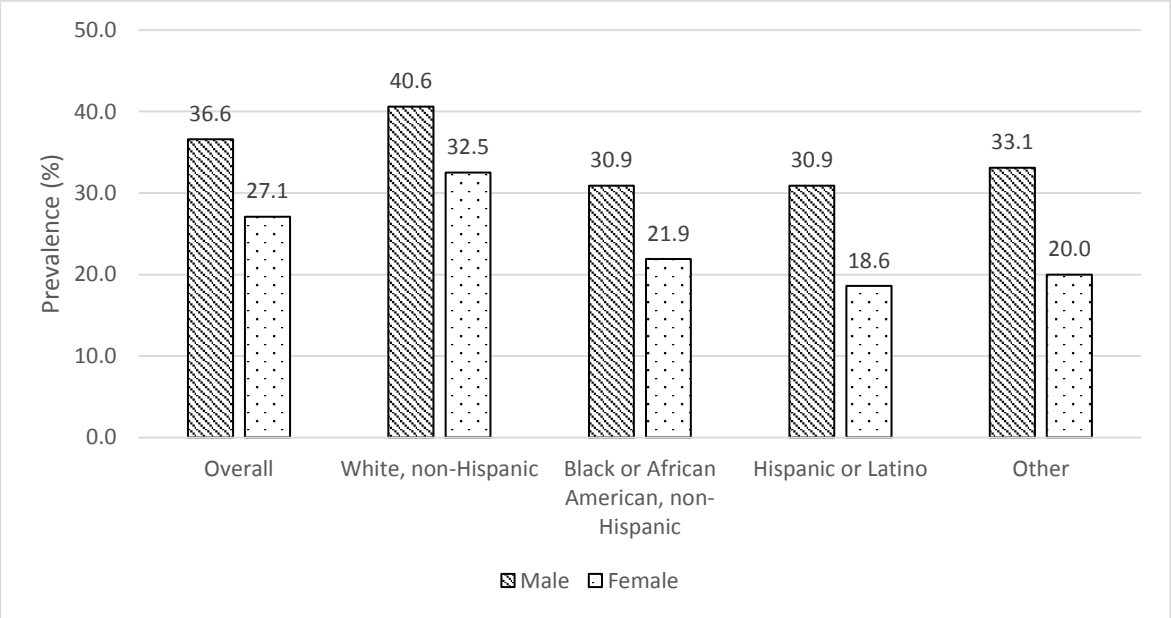


Figure 1-5 Prevalence of current cigarette smoking among young adults by gender and race/ethnicity; Figure produced from data reported by National Survey on Drug Use and Health 2012, United States [43]

1.3.2 Significant changes in smoking behaviours

Young adults experience significant changes in smoking behaviours in terms of initiation and quitting. As many as 25% of smokers take up smoking after they finish school but before the age of 24 in the United States and Canada [44,45]. With the enhancement of tobacco control efforts, people are delaying the age that they take up smoking [19], leading to an increased number of people who initiated smoking as a young adults and a decreased number that started before the age of 18 years (**Figure 1-6**). For example, in the 2013 United States National Survey on Drug Use and Health (NSDUH), it was projected that one million people began smoking at age 18 or older, which increased from 623,000 in 2002; meanwhile, the number of cigarette smokers who initiated prior to the age of 18 years was lower in 2013 than 2002 (1.0 million vs. 1.3 million) [46].

Progression from experimental to regular smoking often occurs in young adulthood [47]. Approximately 38% of current smokers aged 18-25 years reported progression to regular smoking after 18 years old in the United States in 2009 [48] compared with the estimate of 30% in 2007 [49]. In addition, any form of initiation of smoking is now rare after 25 years of age [50]. This is important because it means that if initiation does not occur by 25 years, then it will likely never occur. Therefore, young adulthood is identified as an important time window for marketing products by the tobacco industry [51] and prevention of uptake in the age group is a high priority as it could almost end the supply of new smokers.

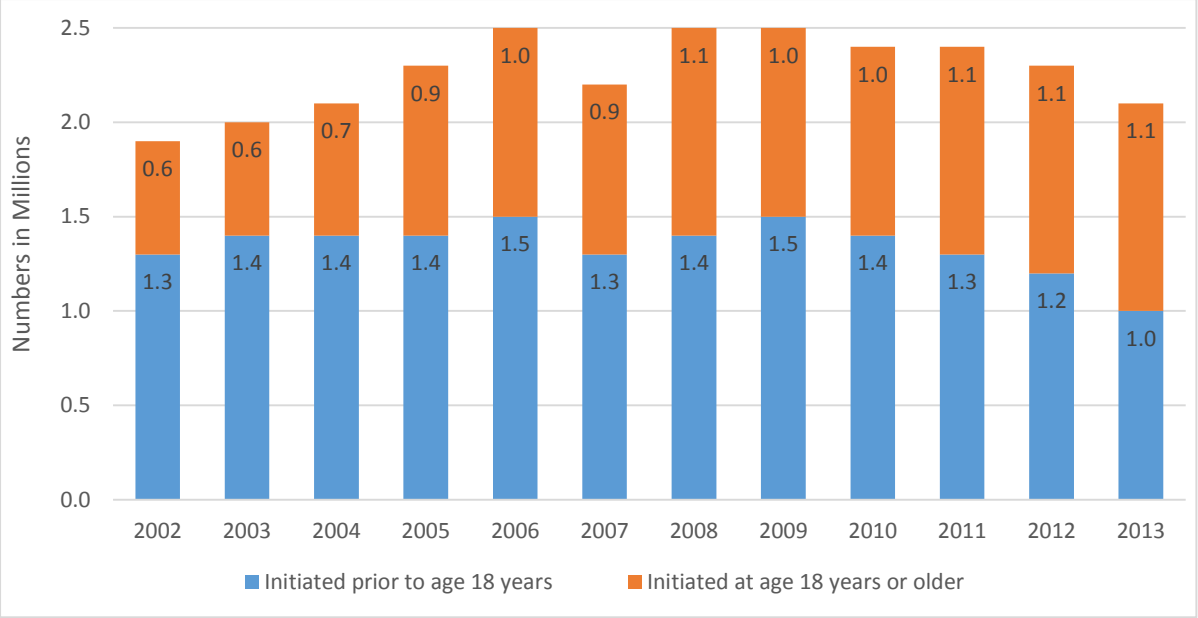


Figure 1-6 Past year cigarette initiates among people aged 12 years or older, by age at first use from 2002 to 2013; Figure produced from data reported by National Survey on Drug Use and Health 2013, United States [46]

1.3.3 Greater benefits of quitting smoking

Quitting smoking greatly benefits health, and the benefits are greater if the cessation occurs at a younger age. Compared with those who had never smoked, lifespan is shortened by 10 years among current smokers [52-55]. Of note, the survival curve for smokers who quit before the age of 35 years is nearly identical to that for people who have never smoked, indicating most risk can be avoided if cessation occurs in young adulthood [52-55]. It is therefore important to understand the drivers of smoking cessation among people in this age group so that efforts to increase cessation and reduce relapse can be enhanced.

1.4 Health consequences of smoking

1.4.1 Smoking and physical health

The tobacco epidemic including active smoking (direct tobacco smoking) and passive smoking (exposure to second-hand smoke) is one of the biggest public health issues the world has ever faced [56]. Around six million people die prematurely from smoking each year [57]. The six million deaths translate to a striking statistic: one death every five seconds. Over five million of these deaths are attributable to active smoking [56], while an additional 600,000 deaths are the result of being exposed to second-hand smoke [58]. According to the latest data from the WHO [59], 12% of mortality among adults aged 30 years and older was

attributed to tobacco worldwide, with the proportion of deaths higher among men than women. The burden of tobacco-related mortality is heaviest in low- and middle-income countries. It is projected that there will be a 9% decline in tobacco-attributable deaths in high-income countries during 2002-2030, while during the same period the number of deaths in low- and middle-income countries would double from 3.4 million to 6.8 million [57].

The adverse effects of smoking have been reported by several national and international agencies, such as the United States Surgeon General, the Royal College of Physicians of London, and the Monographs of the International Agency for Research into Cancer. Of these, the most regular series is from the Office of the United States Surgeon General, which has been focusing on various aspects of smoking since 1964. The repeated conclusion is that "Smoking is the single greatest cause of avoidable morbidity and mortality in the United States", and no reason has been found to reverse any earlier conclusions of causality. The report was updated in 2004 and 2014 with major changes being the inclusion of the health effects of passive smoking and recognition of the rising epidemic of smoking in women.

Tobacco use harms nearly every organ of the body, leading to many conditions and reduction in the general health of smokers [41]. **Figure 1-7** lists the health consequences causally linked to smoking, which was updated by the United States Surgeon General in 2014 [41]. Among adults aged 30 years and older, globally, tobacco smoking was responsible for 14% of all deaths from non-communicable diseases [59]. Among these diseases, 10% of all deaths from CVDs, 22% from various cancers and 36% from respiratory diseases were attributable to tobacco smoking [59].

Of the many diseases induced by smoking, the leading three causes of deaths are CVD, chronic obstructive pulmonary disease (COPD), and lung cancer [60]. For example, smoking increases the risk of coronary heart disease and stroke by about two to four folds, and the risk escalates with the amount of tobacco smoked [61]. Importantly, the cardiovascular risk is rapidly increased by even low levels of cigarette consumption [38]. In Australia, smoking is the greatest cause of COPD [55], accounting for 77% of male cases and 71% of female cases in 2004-5 [62]. Approximately half of current smokers who survive to their mid-70s develop mild to severe COPD [63]. Of cancers, lung cancer is the one that has been most thoroughly investigated with respect to smoking [64]. Up to 90% of lung cancer cases in prolonged

smokers are attributed to smoking [31]. Compared with never smokers, mortality from lung cancer is 16 times higher in smokers [65].

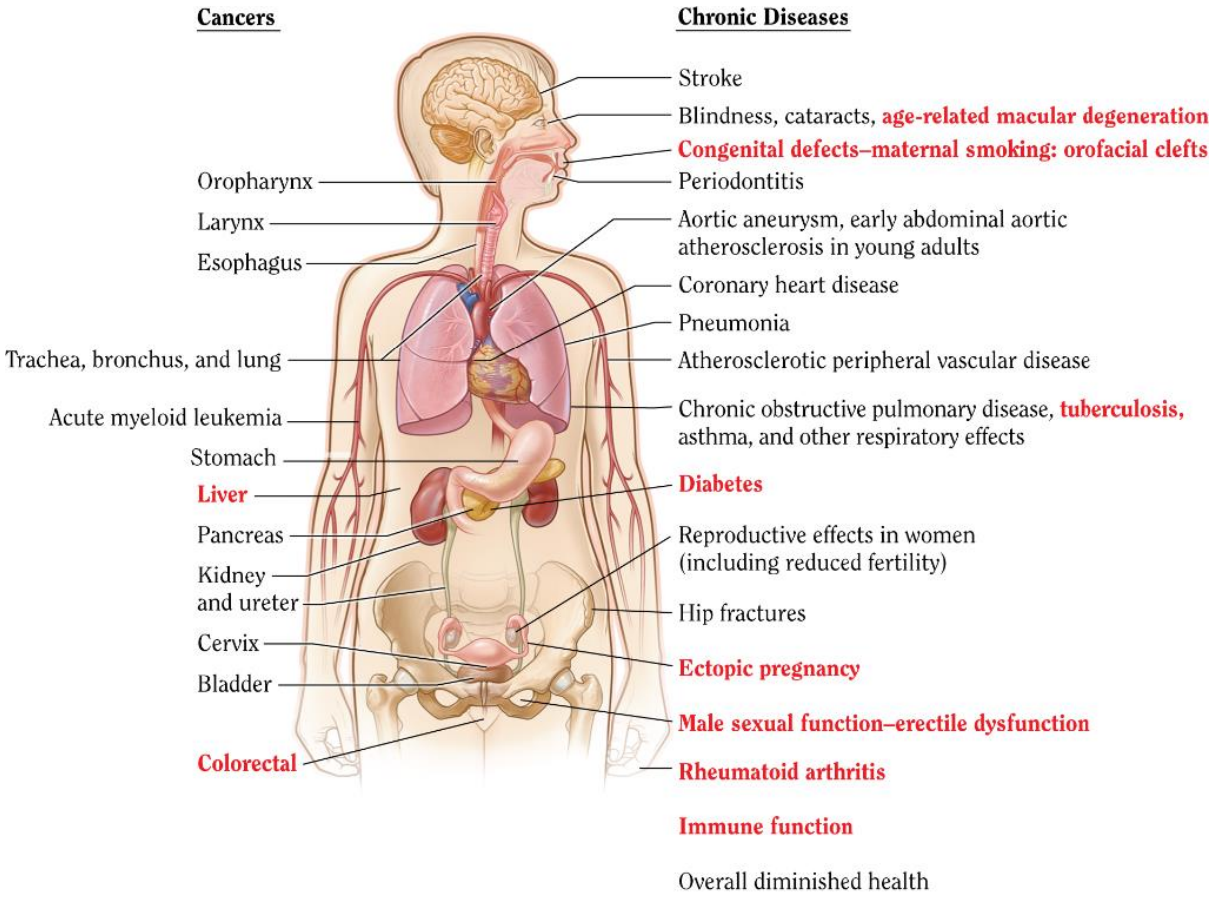


Figure 1-7 The health consequences causally linked to smoking; Each condition presented in red bold is a new disease that has been causally linked to smoking in the 2014 report of U.S. Department of Health and Human Services (USDHHS); from USDHHS 2014 (with permission) [41]

There have been several large, prospective and nationally representative studies worldwide investigating smoking and its relation to mortality in the 21st century [52-54,65,66]. In the United States, Jha and colleagues [52] collected data on smoking from 113,752 women and 88,496 men aged 25 to 79 years between 1997 and 2004 and linked these data to the National Death Index prior to 2006. They found that mortality from any cause among current smokers at baseline was about three times that of never smokers, and current smokers lost more than 10 years of life expectancy compared with never smokers. About 90% of the risk can be avoided if cessation occurs before the age of 40 years. The tripling of the relative risk (RR) of overall mortality and the reduction in life expectancy by almost a decade are consistent with the findings in other studies: a study of male British doctors [65], the Million

Women Study in the UK [53], a meta-analysis of five other contemporary cohort studies [67] and the Life Span Study in Japan [54].

The most recent available evidence on trends in mortality attributed to tobacco use is from China [66]. This country is and will continue to be a centre of global tobacco epidemic in the 21st century as it is the largest producer and consumer of cigarettes [68]. Over one-third of the world's cigarettes were consumed in China in 2014, more than the total amount of the next top 29 cigarette-consuming countries combined [69]. In 2010, about one million deaths were caused by tobacco use in China. The estimated number of deaths will double in 2030 and triple in 2050 unless there is widespread prevention of uptake and cessation [66]. Chen and colleagues [66] also pointed out the changing effects of tobacco use on male and female mortality were opposite, with the mortality attributable to tobacco use was increasing in men, but low, and decreasing among women.

1.4.2 Smoking and mental health

Apart from physical health, tobacco smoking is also closely related to mental health although the causal nature of the association is unclear. Cross sectional studies show that people who report mental health problems have higher rates of smoking [70-76], are more often heavier smokers [72,74] and have lower rates of quitting [73-75] than those without mental illness. For example, Lasser et al [73] reported that in an American national representative sample of adults, people with mental illness were about twice as likely to smoke as those without mental illness and they also had lower quit rates. Receiving treatment for mental health problems appears to significantly increase quit rates [77]. Quantitative and qualitative data has shown that people report that they smoke to stabilise their mood, for relaxation and enjoyment, and to alleviate feelings of stress, depression and anxiety [78-83]. Whether or not quitting smoking impairs or improves mental health or if relapse to smoking improves mental health are therefore of interest. Taylor and colleagues recently performed a comprehensive meta-analysis of longitudinal studies of healthy and clinical (including those diagnosed with physical and psychiatric disorders) adult populations to investigate changes in mental health after smoking cessation compared with continuing to smoke [84]. Based on data from 26 studies that satisfied their inclusion criteria, they concluded that smoking cessation was associated with improvements of several indicators of mental health

compared with continuing to smoke, including depression, anxiety, stress, mental health-related quality of life (HRQoL) and positive affect. The strength of the association was similar among the general population and those with psychiatric disorders. Furthermore, other investigators have reported that people who relapse to smoking after a quit attempt experience an increase in anxiety levels from baseline [85]. These longitudinal data provide strong evidence of an association between stopping smoking or cessation maintenance and improved mental health. Thus, worries about worsening mental health after cessation could be allayed.

In adolescents and young adults, the association between smoking and mental health has been highlighted in recent studies [86]. For example, in a USA cohort of college students, Wetter and colleagues [87] found that affect control by smoking was an important predictor of the transition from occasional to daily smoking. However, the directionality of this link is not entirely clear. Using data from a 4-wave longitudinal study in adolescents, Windle and Windle [88] found that depression was a predictor of future smoking after controlling for baseline smoking and smoking was a predictor of future depression after controlling for baseline depression. In addition, only one study from Australia has explored the association of smoking and mental health in adolescents or young adults, while with a cross-sectional design and small sample size (n=92) [89]. Future studies are needed to verify this result using longitudinal designs in larger samples.

1.4.3 Smoking and health-related quality of life

Health-related quality of life is a multi-dimensional concept that describes a subjective perception of physical and mental and social life. Utilisation of HRQoL in health research is important. This is because with the advancement of medical and public health services, better treatments of existing diseases occur and life expectancy is prolonged. Therefore, health assessment should not only focus on saving lives, but also improving their quality. Several instruments are available to measure HRQoL, such as the Medical Outcomes Study Short Forms (SF-12 and SF-36), the Health Utilities Index Mark 3 and EQ-5D. These tools have been infrequently used in tobacco-related research, especially in longitudinal studies, and could be included as an outcome to assess the impact of smoking on health, particularly in younger populations.

The relationship between smoking and HRQoL in the general population has been investigated in a number of cross-sectional studies [90-94]. Although these studies varied in the way HRQoL and smoking status were measured, the main message was consistent: on average HRQoL is poorer in smokers than non-smokers, and the strength of the association relates to the heaviness of smoking. Several longitudinal studies have been conducted examining the relationship between quitting smoking and changing HRQoL, producing mixed evidence [95-102]. For example, Piper et al found that compared with continuing smokers, quitters reported a significant improvement in HRQoL at the end of one and three years [96]. Using data from two Nurses' Health Study cohorts, Sarna et al. found both continuing smokers and quitters had significant declines in physical HRQoL and significant improvements in mental HRQoL over eight years follow-up [95]. Inconsistent findings may be explained by small sample size [99,100,102], short follow-up lengths [100,102], and specific groups targeted (i.e. university graduates [101], females [95], and participants from assisted cessation programmes [96-98,102]). No longitudinal data is available in young adults. A feature of many tobacco control campaigns is the use of graphic advertisements focused on the diseases caused by smoking. There is potentially a perception among younger smokers that these health effects are unlikely to occur for many decades and that their smoking will not yet be affecting their health [103,104]. It is possible that raising awareness among younger smokers of the effect that their smoking is having on their health through instruments measuring HRQoL could promote quit attempts.

1.4.4 Adverse health effects of smoking in young adults

Young smokers are less likely to suffer from diseases induced by smoking because they often take several decades to develop. However, smoking during young adulthood causes a range of immediate adverse health consequences, laying the foundation for developing serious diseases in later life [38]. As aforementioned, most of the risks are avoidable if people can quit smoking before the age of 35 years [52-55,65]. This is why examining the earlier effects of smoking on HRQoL and changes in smoking status on changing HRQoL in young adults is important and warranted. Of note, some of the following evidence is from studies of adolescents, therefore, the definition of young adult in this section is broader than previously defined.

1.4.4.1 Early signs of nicotine addiction

Defining nicotine dependence in young people is a topic of debate, with increasing recognition of the inappropriateness of using adult criteria (generally based on the premise that prolonged use is needed for establishing dependence) in young smokers. Recent research has also highlighted the qualitative difference in withdrawal symptoms experienced by adolescents and adults. Craving tobacco is the predominant symptom reported by young people during abstinence and withdrawal symptoms are generally minimal, while adults' nicotine dependence is characterised by emergent withdrawal symptoms [38]. Nonetheless, there is emerging evidence suggesting that nicotine dependence can be developed shortly after initiating smoking in young people, even at low levels of cigarette consumption [38,105].

1.4.4.2 Lung function, respiratory symptoms and diseases

Many studies of different populations have found that early tobacco use impairs lung growth and development [38]. In addition, a dose-response inverse relationship was reported between smoking and lung function reflected by forced expiratory volume in one second / forced vital capacity (FVC) and forced expiratory flow between 25% and 75% of the FVC among children and adolescents [106]. Compared with young non-smokers, young smokers' lung growth ceased earlier, they reported a lower maximal lung function, a briefer plateau phase, and presented a decline in lung function earlier [38]. Quitting smoking was associated with a smaller decline in lung function than continuing smoking [107].

Active smoking in children and adolescents is associated with higher frequency of respiratory symptoms [64,108], and the frequency of respiratory symptoms positively related to duration of smoking and the amount smoked [109-111]. Moreover, accumulating longitudinal data support the relationship of active smoking and the incidence, persistence and recurrence of asthma, wheeze and cough in adolescence and young adulthood [112,113], especially in girls [114,115]. For example, in Norway, Tollsfsen et al. [115] evaluated the incidence and course of wheeze and asthma in 2,399 adolescents, with data collected at baseline aged 13-15 years old and in follow-up at 17-19 years of age. They found that for subjects reporting no respiratory symptoms at baseline, the risk of developing wheeze at follow-up

was significantly greater in girls (odds ratio (OR): 2.8, 95% confidence interval (CI): 1.6, 4.9) than boys (OR: 1.8, 95% CI: 0.9-3.9).

1.4.4.3 Cardiovascular effects

Active and passive smoke exposure during adolescence and young adulthood lead to the early phases of cardiovascular injury, thereby increasing the risk of CVDs. Suggested mechanisms include endothelial injury and dysfunction, promotion of chronic inflammation, insulin resistance and promoting an atherogenic lipid profile [34]. Among them, atherosclerosis underlies much of cardiovascular mobility and mortality in adulthood [38]. Three studies have assessed the association between the presence and degree of atherosclerosis and cardiovascular risk factors including smoking in young people at autopsy, and found that smoking in adolescence and young adulthood contributed to atherosclerosis; this association was evident shortly after youth initiated smoking and readily observed in adulthood [116-118]. Three large longitudinal studies have examined the association of early exposure to tobacco and subclinical atherosclerosis later life, and all suggested a causal relationship and the response appears to be time and dose-dependent [119-121].

1.4.4.4 Other health problems

Tobacco use is also a risk factor for dental and musculoskeletal problems in young people [31]. Over half of periodontitis in young adults aged 19-30 years was associated with smoking [122]. There was also evidence that moderate smoking during young adulthood induced variations of saliva lipid pattern, the amount of which are important in maintaining oral cavity health [123]. A wealth of data has demonstrated the association of tobacco use to musculoskeletal problems in the elderly [124,125]. Recent research has also begun to link early tobacco use with unfavourable musculoskeletal phonotypes [126].

1.4.5 Economic costs of smoking

One way to measure the consequences of tobacco use on a society is to estimate its economic costs, which can include the costs of smoking-related illnesses, premature mortality and reduced productivity. Such estimates can be defined by the difference between healthcare or other costs that actually occur due to smoking and the costs with reduced levels of smoking [127]. The percentage of the total cost of smoking out of the gross

domestic product (GDP) is often used for cross-country comparisons. So far, most data on the economic burden of smoking are from developed countries. It was found that smoking places a high economic burden on the whole economy, reaching 1.4%-1.6% of GDP in the United States, 1.3%-2.2% of GDP in Canada, and 2.1%-3.4% of GDP in Australia [128]. The estimations are lower in developing countries compared with developed countries. Sun et al. [129] estimated that the total economic cost of smoking in 2000 in China was $5.0 billion, accounting for approximately 0.5% of China's GDP. A more recent study from China by Yang et al. [130] reported that the total economic cost of smoking in 2008 ($28.9 billion) was four times more than in 2000, which represented 0.7% of China's GDP. Similar proportions were documented in other developing countries such as India in 2004 (0.24%) [131], Vietnam in 2011 (0.97%) [132] and Thailand in 2009 (0.78%) [133]. The lower proportion of the total economic cost in national GDP in developing countries than developed countries may be explained by the earlier stage of tobacco epidemic, the long lag time between smoking and its adverse health effects, and limited access to and poor quality of medical care [127,134].

1.4.6 Health effects and economic costs of smoking in Australia

Smoking is the leading preventable cause of morbidity and mortality in Australia. It was responsible for 11.7% of Australia's deaths, that is 15,511 deaths in 2003 in Australia, with more than three-quarters accounted for by lung cancer, COPD and ischaemic heart disease [135]. In contrast to the estimated all-cause mortality attributable to smoking worldwide described in Section 1.4.1, this figure was up to two in three in a recent Australian study [55]. In this large-scale prospective study of 204,953 participants aged 45 years or over, Banks et al. [55] found that the life expectancy was 10 years shorter in current smokers than never smokers, the mortality attributable to smoking increased with increasing smoking intensity among current smokers, and the greater mortality diminished gradually with increasing time after cessation among former smokers. If quitting smoking is before the age of 45, no significant difference in mortality was evident between former smokers and never smokers.

There are two major studies of the costs of smoking to Australian society: the Australian Institute of Health and Welfare (AIHW) burden of disease study and Collins and Lapsley's studies of social costs [31]. The latest AIHW data was from 2003 and used the measure of disability adjusted life years (DALYs), reflecting the number of years of life lost due to ill

health, disability or early death [135]. Over 2.63 million DALYs were estimated lost due to disease and injury in 2003 in Australia, and of individual risk factors smoking accounted for the greatest proportion of DALYs lost (7.8% of total) [135]. Collins and Lapsley have estimated the economic costs of tobacco use to Australian society for several years, including 1988 [136], 1992 [137], 1998-99 [138] and 2004-05 [62]. They have also estimated the economic costs of tobacco use for three states in 1998-99 [139,140] and 2009-10 [141]. Their latest national report was based on 2004-05 data. According to this report [62], tobacco use was responsible for $31.5 billion (56.2% of total) in 2004-05, more than the total amount of alcohol (27.3%), illicit drugs (14.6%), alcohol and illicit drugs (1.9%) together. In addition, as noted by Collin and Lapsley, their approach to estimation was extremely conservative; the actual economic costs of smoking are likely to be higher [62].

1.5 Difficulties in quitting smoking and achieving long-term abstinence

The immediate and long-term benefits of quitting smoking for people at any age have been substantially explored in previous studies [41,52-55,65,66,142,143]. Quitting smoking is a difficult journey for most smokers. Mark Twain said "To cease smoking is the easiest thing I ever did; I ought to know because I've done it a thousand times", highlighting the difficulties and challenges to successful cessation. Indeed, numerous attempts and relapses have to be made prior to prolonged abstinence, and this may take several years [61,142]. For example, a recent Australian national survey of smokers aged 14 years or older found that 77% of the participants had tried to change their smoking behaviours in the previous 12 months, 29% reported trying to quit but not succeeding, 19% successfully quit smoking for over a month and 38% had attempted to reduce the amount of smoking per day [144]. Among adult smokers in the United States, nearly 70% reported that they wanted to quit completely in 2010, more than half said they had attempted to quit in the past year but only 6% had successfully quit [145].

Young adulthood is a prime time when people are either imbedding their smoking behaviour (as introduced in section 1.4) or quitting, making it an important period for preventing transition to regular smoking and also promoting cessation. Three reasons can be offered explaining this viewpoint. First, the proportion of young smokers who are interested in quitting is high, and most have actively engaged in cessation process [146].

Second, the estimated median age of cessation for people who started smoking as adolescents was in young adulthood, 33 years for males and 37 years for females [147]. Third, older adults (aged 35-64 years) were less likely to report quitting smoking successfully than young adults (aged 18-24 years) [148].

1.5.1 Reasons why it is difficult to quit

Tobacco products contain nicotine, which is a highly psychoactive ingredient. It is the nicotine in tobacco that causes people to become addicted to smoking and that is responsible for relapse because of withdrawal symptoms [149]. The report of the US Surgeon General in 1988 that solely focused on nicotine addiction concluded that: "1) cigarettes and other forms of tobacco are addictive; 2) nicotine is the drug in tobacco that causes addiction; 3) the pharmacological and behavioural processes that determine tobacco addiction are similar to those that determine addiction to drugs such as heroin or cocaine" [150].

1.6 Smoking cessation intervention strategies

Although most smokers quit unassisted [151,152], behavioural support, pharmacotherapies and a combination of these interventions have been shown to increase a person's success and help them to achieve long-term abstinence, especially for more dependent smokers [153-155]. Behavioural support interventions include self-help materials (e.g. brochures, books, videotapes and CDs), individual counselling, group quit courses, and cessation clinics in person or by telephone. Compared with no advice or usual care, behavioural support interventions are effective in increasing quit rates, although some only have a small effect [156]. For example, standard self-help materials alone only slightly increased quit rates compared to no intervention, but there was greater benefit for individually tailored self-help materials [154]. Nicotine replacement therapy (NRT), bupropion and varenicline have been approved for first-line pharmacotherapies for smoking cessation. All three were reported as effective in increasing smoking cessation relative to placebo or non-drug arms, with a pooled risk ratio (RR) of 1.60 (95% CI, 1.53 to 1.68) for abstinence for NRT, RR 1.62 (95% CI, 1.49 to 1.76) for bupropion, and RR 2.27 (95% CI, 2.02 to 2.55) for varenicline [156].

Combining behavioural support and pharmacotherapies increases the success of smoking cessation. For example, providing behavioural support for people using pharmacotherapy to

stop smoking increased the chance of success by 10% to 25% [157], and providing pharmacotherapy for people using usual care, brief advice or less intensive behavioural support increased the cessation success probability by 83% [158].

1.6.1 Factors that predict making a quit attempt and maintaining cessation

There are two major components of attempting to quit smoking: making a quit attempt and maintaining cessation. These two tasks are distinct, so the predictors are not necessarily equivalent [159]. Also, the factors affecting the success rate of quitting smoking vary from one person to another [160-183]. Overall, these can include [31]:

1. physiological factors (e.g. level of nicotine dependence, severity of withdrawal symptoms, experience of weight gain in previous quit attempts)
2. behavioural factors (e.g. duration of smoking, frequency of smoking, past attempts to quit)
3. social factors (e.g. living or working with smokers, having smoking friends, home or workplace subject to smoke free policies or seeing tobacco products displayed)
4. psychological or emotional/affective factors (e.g. stress, depression, anxiety, psychiatric disorders, fear of weight gain)
5. cognitive factors (e.g. knowledge, self-exempting beliefs, perceived disadvantages, motivation, self-efficacy)
6. barriers to access to interventions (e.g. living in rural area, affordable quitting medications and treatment programs)
7. other factors (e.g. marital status, have children living at home, education).

One recent systematic review provided comprehensive evidence on the determinants of trying to quit smoking and their success in general population among adults [181]. Eight studies from 17 articles were included. It found that in spite of considerable methodological heterogeneity across studies, population-based studies from several countries showed that past quit attempts and measures of motivation highly predicted quit attempts, whereas only nicotine dependence measures consistently predicted success or failure in these attempts. Occupation level was also shown to predict successful quitting but only two studies examined this. Other socio-demographic factors, such as age, gender, marital status and education level were not consistently associated with quit attempts or success.

Awareness of factors determining smoking cessation is of importance for identifying people at risk of relapse and to improve success in quitting. Identifying factors that predict successful cessation could be used to match smokers with a strategy that facilitates the success in quitting. In addition, this knowledge could enable to refine tobacco control polices and optimise health care resources. A better understanding of factors associated with successful cessation in well-designed cohort studies is one way that we can gain a better understanding of such factors and deliver on these aspirations.

In this thesis, some specific predictors and health effects of smoking cessation or continuation in young adults are examined. As shown in **Figure 1-8**, predictors include life-stage transitions [180] and SEP trajectories across the life course, and health effects covers weight gain after smoking cessation and HRQoL (introduced in the section of 1.4.3)., such as. These factors have been identified in some studies as being associated with changing smoking status. Some of these have been explored in a limited number of studies or studies that have substantial limitations. There is a lack of prospective evidence from 'real world' population-based studies and this weakens the case for causal relationships. As these factors are the main focus of this thesis, they are introduced in detail below.

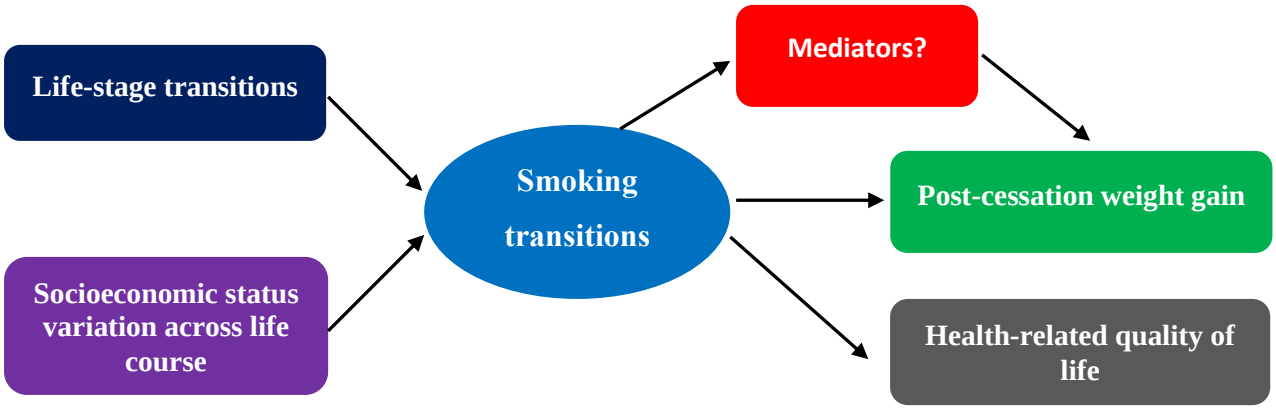


Figure 1-8 Framework of this thesis

1.6.1.1 Weight gain after smoking cessation

The relationship between smoking and body weight has been described for many years. Cross-sectional studies document that body weight is lower in smokers than non-smokers [184,185], and is higher in former smokers than both smokers and non-smokers [186]. Cohort studies also find that smokers gain weight after quitting [184,187,188]. Estimates of the magnitude of weight gain varied widely across studies. Travier et al. [188] found that over five years quitters gained twice as much weight as did continuing smokers; for men, the estimated annual weight gains per year were 0.41 kg for continuing smokers and 0.84 kg for quitters, with corresponding values of 0.36 kg and 0.85 kg for women. The amount of weight that smokers gain after quitting can be substantial over a longer period. Using data from the 2003-2012 National Health and Nutrition Examination Survey in the United State, Veldheer et al. [187] found that over a 10 year period, quitters gained an average weight of 8.4 kg as against 3.5 kg in continuing smokers. Heavy smoking, obesity, women, young age and being African American increased the risk of major weight gain after quitting [184,187,189].

Fear of weight gain discourages smokers from trying to quit, experience of weight gain is associated with relapse in former smokers, and expectation of weight control or weight loss relates to smoking initiation [190-195]. It is reported that about half of female and a quarter of male smokers do not try to quit because of their concerns about weight gain [193], and 52% female and 32% male former smokers reported relapse to smoking due to weight gain after cessation [194]. Some adolescents, especially girls, and young adults use smoking as a strategy to control or lose weight [191,192,195].

The mechanisms by which smoking cessation leads to weight gain are still not well understood and remain to be elucidated, but are likely to be because of more energy intake than energy expenditure for a period of time. Nicotine mediates most of the effects of smoking on weight by raising resting metabolic rate and suppressing appetite. Such weight-decreasing effects are removed after quitting smoking (absence of nicotine) so that resting metabolic rate (energy expenditure) decreases and appetite (energy intake) increases [196]. Some smokers substitute eating for the "hand to mouth" habit of smoking to cope with nicotine withdrawal symptoms, which may also contributes to an increase in energy intake [197]. Other possible explanations of post-cessation weight gain include increased preference of sweet food and decreased physical activity (PA) after quitting [179,198].

Several interventions to reduce weight gain after quitting smoking, including pharmacotherapies, exercise and dietary interventions, appear to achieve little success [199-202]. The latest systematic review and meta-analysis of interventions for preventing post-cessation weight gain was performed by Farley et al. in 2012 [202], concluding that 1) personalised weight management support may be effective in mitigating weight gain after cessation, but the supporting data was two few to be sure; 2) some pharmacotherapies, such as bupropion, fluoxetine, NRT and varenicline, and very low calorie diet limited weight gain during treatment, while this effect was not maintained after one year after quitting; 3) exercise interventions showed evidence of long-term success, but not in the short-term; 4) other interventions including weight management education only, and cognitive behavioural therapy to accept weight gain were not effective in weight reduction. Overall, no strong clinical recommendation can be made regarding how to effectively prevent post-cessation weight gain.

To address concerns about weight gain after quitting, it is important to provide accurate information on the amount of weight gain that might be expected after quitting. The most recently available and systematic estimate is from a meta-analysis of 62 randomised controlled trials (RCTs) of three first line treatments (NRT, bupropion, and verenicline) for smoking cessation, which reported that the average weight gain was 4-5 kg after 12 months of abstinence [203]. However, this result may not be generalisable to the general population of smokers [204]. This is because participants in cessation trials are usually heavier smokers and

more dependent on nicotine [205], which means that their likelihood of experiencing major weight gain after cessation may be greater than light smokers and those who are less dependent upon nicotine [184]. Furthermore, other studies have shown that people that engage in RCTs are more likely to have previously quit and relapsed [206], and may lack self-efficacy [207], with these potentially related to weight gain, and may therefore present a biased estimate of weight gain after cessation. In addition, this study only assessed the effects of quitting smoking on weight change within 12 months; therefore, the effects beyond 12 months are unclear. Also, people tend to gain weight as they age including quitters [208]; thus, a more accurate estimate should separate age-related weight gain from the weight gain attributable to smoking cessation. Fernandez and Chapman [204] pointed out a need of a meta-analysis of prospective population-based cohort studies to properly address this question. This is important because this information can then be communicated to smokers and may redirect efforts to understand and manage any weight gain that may occur after smoking cessation.

1.6.1.2 Impact of quitting on mental health

Abrupt discontinuation or decrease in tobacco use produces a group of withdrawal symptoms. Negative affect is a major component of withdrawal [209], including irritability, aggression, anxiety, difficulty concentrating, restlessness, impatience, depressed mood and insomnia [210], which peaks within the first week after quitting and lasts 2-4 weeks [211]. These symptoms occur in most smokers when they try to quit and are partly explained by nicotine's effects on the brain. Nicotine can promote the release of a variety of neurotransmitters, including dopamine, norepinephrine, serotonin, β-endorphin and GABA (γ-aminobutyric acid), thereby inducing pleasure, arousal, mood modulation, and a reduction in anxiety and tension [212]. Unsurprisingly, smoking for stress relief and enjoyment are commonly reported [78,79].

Despite smokers believing that smoking offers them mental health benefits, they might misattribute the ability of cigarettes to relieve nicotine withdrawal symptoms as a beneficial effect on mental health [84]. As discussed earlier, there is a strong relationship between smoking and poor mental health. People living with mental health problems have higher rates of smoking than the general population, and are also more likely to be heavier

smokers [213]. After quitting smoking, mental health has been reported to significantly improve in quitters relative to continuing smokers, reflected by several mood items, including anxiety, depression, mixed anxiety and depression, psychological quality of life, positive affect, and stress [84]. In addition, the effects and the strength of the association are similar for both the general population and those with mental health disorders [84]. However, as discussed above, this relationship is less clear among young adults and very few data is from Australia.

1.6.1.3 Life-stage transitions

A life-stage is a phase in life, through which people progress developmentally. Greece et al. proposed four major life transitions: leaving the parental home, occupying an instrumental role (e.g. attending college or university, or employment), marriage, and parenthood [214]. Life-stage transitions often accompany major changes in social networks, social roles, responsibilities and expectations, which have been proposed by a small number of studies to be relate to the adoption, maintenance and cessation of smoking. For example, in one longitudinal study of young women in Australia, moving out of the parents' home was associated with an increased risk of adopting smoking compared with those who were not living with their parents [215]. Marriage, being in a committed relationship or being a mother was significantly related to quitting, remaining an ex-smoker or not picking up smoking [180], whereas marital termination (e.g. through divorce or widowhood) increased daily cigarette consumption [216] and risk of relapsing or starting smoking [217]. Of note, all these associations were reported among young women or middle-older aged people. Therefore, the effects in young men are still unclear. A better understanding of how these transitions influence smoking might help with optimising and reinforcing tobacco control efforts to promote quitting and enhance abstinence from smoking.

1.6.1.4 Socioeconomic position

Socioeconomic position is the place that a person or group occupies in the structure of society[218]. There are several indicators of SEP, including markers such as education, income, occupation, housing tenure, car availability and neighbourhood deprivation[219,220]. Tobacco smoking disproportionately affects low SEP groups. As reported earlier, in most economically developed countries, people of higher SEP have lower smoking prevalence

than those in lower SEP groups, and are less likely to be heavy smokers and exposed to second-hand smoke [221-223]. A cumulative effect of disadvantage is also evident in these countries, with the smoking prevalence in people facing many forms of disadvantage five times of that in the most affluent[224,225].

In the past few decades, in countries with advanced tobacco control programs, the overall trend in smoking prevalence across most socio-demographic groups shows a downward trend, but the reductions are generally greater in the least socioeconomically disadvantaged than the most disadvantaged groups. This contributes to a widening disparity in the prevalence of smoking between SEP groups [31,226-228], which may keep rising with the trend toward greater socioeconomic inequality [229]. This disparity provides an opportunity for tobacco companies to sustain profits in declining markets, and highlights the importance of researching the impact of SEP on smoking. There has been a tendency in research of socioeconomic disparities in smoking to focus on the role of proximal (i.e. adult) or distal (i.e. childhood) SEP factors; however, this fails to account for the fact that SEP is potentially dynamic over a person life course. Different SEP trajectories over several life-stages (e.g. life-long disadvantage and upward mobility in SEP) may have different effects on smoking behaviours in adulthood and may have different underlying mediators that could be used to reduce inequalities in smoking. Various life course models have been proposed to test different hypotheses [230,231] regarding the effects of SEP across the life course on various health outcomes. It has also been recommended that multiple life course models should be examined in the same life course study. No study has investigated the determination of SEP variation over the life course on smoking behaviours. Furthermore, the mechanisms by which SEP across the life course may influence smoking behaviours have not been well examined.

1.7 Aims

This thesis aims to help fill the evidence gaps and provide novel information to extend current knowledge of the dynamic predictors and early health effects of smoking in young adults. The specific aims are listed below:

1. To prospectively examine the associations between partnering and parenting transitions and smoking continuity, cessation and relapse, and whether these effects differed between men and women (Chapter 2);
2. To test which life course model(s) best describe the association between SEP over the life course and smoking status at mid-adulthood (Chapter 3);
3. To examine the potential mediators linking SEP over the life course and later smoking status, such as parental smoking, attitudes toward smoking, intention to smoke and smoking experimentation in childhood and life-stage transitions in young adulthood (Chapter 3);
4. To quantify weight gain after smoking cessation and the difference in weight gain between quitters and continuing smokers using a systematic review and meta-analysis (Chapter 4);
5. To evaluate whether the greater weight gain after cessation in quitters than continuing smokers could be attributed to changes in several dietary and PA behaviours (Chapter 5);
6. To investigate the longitudinal relationship between change in smoking status and change in physical and mental HRQoL while considering a wide range of potential confounders (Chapter 6).

1.8 References

1. World Health Organization. *WHO report on the global tobacco epidemic, 2015: raising taxes on tobacco*. Geneva, Switzerland: 2015. http://www.who.int/tobacco/global_report/2015/en/. Accessed 19 January 2015.
2. Ng M, Freeman MK, Fleming TD, et al. Smoking prevalence and cigarette consumption in 187 countries, 1980-2012. *JAMA.* 2014;311(2):183-192. doi:10.1001/jama.2013.284692.
3. Bilano V, Gilmour S, Moffiet T, et al. Global trends and projections for tobacco use, 1990-2025: an analysis of smoking indicators from the WHO Comprehensive Information Systems for Tobacco Control. *Lancet.* 2015;385(9972):966-976. doi:10.1016/S0140-6736(15)60264-1.
4. Woodward SD. Trends in cigarette consumption in Australia. *Aust N Z J Med.* 1984;14(4):405-407.
5. Doll R, Hill AB. Smoking and carcinoma of the lung; preliminary report. *Br Med J.* 1950;2(4682):739-748.
6. Doll R, Hill AB. The mortality of doctors in relation to their smoking habits; a preliminary report. *Br Med J.* 1954;1(4877):1451-1455.
7. Doll R, Hill AB. Lung cancer and other causes of death in relation to smoking; a second report on the mortality of British doctors. *Br Med J.* 1956;2(5001):1071-1081.
8. Doll R, Hill AB. Mortality in Relation to Smoking: Ten Years' Observations of British Doctors. *Br Med J.* 1964;1(5396):1460-1467 CONCL.
9. Doll R, Peto R. Mortality in relation to smoking: 20 years' observations on male British doctors. *Br Med J.* 1976;2(6051):1525-1536.
10. Wynder EL. Laboratory contributions to the tobacco-cancer problem. *Br Med J.* 1959;1(5118):317-322.
11. Wynder EL, Graham EA. Tobacco smoking as a possible etiologic factor in bronchiogenic carcinoma; a study of 684 proved cases. *J Am Med Assoc.* 1950;143(4):329-336.
12. Wynder EL, Hoffmann D. *Tobacco and tobacco smoke: studies in experimental carcinogenesis.* New York: Academic Press; 1967.
13. Royal College of Physicians. *Smoking and health: a report of the Royal College of Physicians on smoking in relation to cancer of the lung and other diseases.* London: Pitman Medical Publishing Co Ltd; 1962.
14. Royal College of Physicians of London. *Smoking and health now: a new report and summary on smoking and its effects on health.* London: Pitman Medical; 1971.
15. Royal College of Physicians of London. *Smoking or health : the third report from the Royal College of Physicians of London.* Kent, England : Pitman Medical; 1977.
16. US Department of Health and Education and Welfare. *Smoking and health. Report of the Advisory Committee to the Surgeon General of the Public Health Service*. Rockville, Maryland: 1964. http://www.cdc.gov/tobacco/data_statistics/sgr/pre_1994/index.htm. Accessed 21 January 2016.
17. US Department of Health and Human Services. *The health consequences of smoking. A public health service review*. Rockville, Maryland: 1967. http://www.cdc.gov/tobacco/data_statistics/sgr/pre_1994/index.htm. Accessed 21 January 2016.

18. US Department of Health Education and Welfare. *The health consequences of smoking: a report of the Surgeon General*. Rockville, Maryland: 1972. http://www.cdc.gov/tobacco/data_statistics/sgr/pre_1994/index.htm. Accessed 21 January 2016.
19. Australian Institute of Health and Welfare. *National Drug Strategy Household Survey detailed report: 2013. Drug statistics series no. 28. Cat. no. PHE 183*. Canberra: 2014. http://www.aihw.gov.au/publication-detail/?id=60129549469&tab=3. Accessed 21 January 2016.
20. Adhikari P, Summerill A. *1998 National Drug Strategy Household Survey: detailed findings. Drug Statistics Series no. 6. Cat. no. PHE 27*. Canberra: 2000. http://www.aihw.gov.au/publication-detail/?id=6442467215. Accessed 27 June 2016.
21. Australian Institute of Health and Welfare. *2001 National Drug Strategy Household Survey: detailed findings. Drug statistics series no. 11. Cat. no. PHE 41*. Canberra: 2002. http://www.aihw.gov.au/publication-detail/?id=6442467418. Accessed 27 June 2016.
22. Australian Institute of Health and Welfare. *2004 National Drug Strategy Household Survey: detailed findings. Cat. no. PHE 66*. Canberra: 2005. http://www.aihw.gov.au/publication-detail/?id=6442467781. Accessed 27 June 2016.
23. Australian Institute of Health and Welfare. *2007 National Drug Strategy Household Survey: detailed findings. Drug statistics series no. 22. Cat. no. PHE 107*. Canberra: 2008. http://www.aihw.gov.au/publication-detail/?id=6442468195. Accessed 27 June 2016.
24. Australian Institute of Health and Welfare. *2010 National Drug Strategy Household Survey report. Drug statistics series no. 25. Cat. no. PHE 145*. Canberra: 2011. http://www.aihw.gov.au/publication-detail/?id=32212254712. Accessed 27 June 2016.
25. Department of International Economic and Scoial Affairs. *Provisional guidelines on standard international age classifications*. New York: Department of International Economic and Scoial Affairs; 1982. http://unstats.un.org/unsd/publication/SeriesM/SeriesM_74e.pdf. Accessed 17 May 2016.
26. Feldman RS. *Discovering the life span.* Boston: Pearson; 2015.
27. Flay BR. *Youth tobacco use: risks, patterns, and control.* New York: Oxford University Press; 1993.
28. U.S. Department of Health and Human Services. *Preventing Tobacco Use Among Young People: A Report of the Surgeon General*. Atlanta, Georgia: U.S. Department of Health and Human Services, Public Health Service, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 1994. https://www.cdc.gov/tobacco/data_statistics/sgr/1994/. Accessed 18 May 2016.
29. Perry CL, Murray DM, Klepp KI, Centers for Disease C. Predictors of adolescent smoking and implications for prevention. *MMWR Suppl.* 1987;36(4):41S-45S.
30. Flay BR, Petraitis J, Hu FB. *The theory of triadic influence: Preliminary evidence related to alcohol and tobacco use.* Washington, DC: U.S. Government Printing Office; 1995.

31. Scollo MM, Winstanley MH. *Tobacco in Australia: Facts and issues*. Melbourne: 2015. http://www.tobaccoinaustralia.org.au/. Accessed 21 January 2016.
32. Petraitis J, Flay BR, Miller TQ. Reviewing theories of adolescent substance use: organizing pieces in the puzzle. *Psychol Bull.* 1995;117(1):67-86.
33. Hassandra M, Vlachopoulos SP, Kosmidou E, Hatzigeorgiadis A, Goudas M, Theodorakis Y. Predicting students' intention to smoke by theory of planned behaviour variables and parental influences across school grade levels. *Psychol Health.* 2011;26(9):1241-1258. doi:10.1080/08870446.2011.605137.
34. U.S. Department of Health and Human Services. *How tobacco smoke causes disease: the biology and behavioral basis for smoking-attributable disease: a report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2010. http://www.cdc.gov/tobacco/data_statistics/sgr/2010/. Accessed 20 May 2016.
35. Winstanley M, Wood L, Letcher T, Greenhalgh E. Figure 5.2.1 Influences on uptake of smoking. In: Scollo MM, Winstanley MH, eds. *Tobacco in Australia: Facts and issues*. Melbourne: Cancer Council Victoria; 2014.
36. Backinger CL, Fagan P, Matthews E, Grana R. Adolescent and young adult tobacco prevention and cessation: current status and future directions. *Tob Control.* 2003;12 Suppl 4:IV46-53.
37. Lantz PM. Smoking on the rise among young adults: implications for research and policy. *Tob Control.* 2003;12 Suppl 1:i60-70.
38. U.S. Department of Health and Human Services. *Preventing tobacco use among youth and young adults: a report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2012. http://www.surgeongeneral.gov/library/reports/preventing-youth-tobacco-use/index.html. Accessed 27 March 2016.
39. Hopkinson NS, Lester-George A, Ormiston-Smith N, Cox A, Arnott D. Child uptake of smoking by area across the UK. *Thorax.* 2014;69(9):873-875. doi:10.1136/thoraxjnl-2013-204379.
40. Robinson S, Bugler C. *Smoking and drinking among adults, 2009: a report on the 2009 General Lifestyle Survey*. Newport, South Wales: Office for National Statistics; 2009. http://www.esds.ac.uk/doc/6737%5Cmrdoc%5Cpdf%5C6737report.pdf. Accessed 10 April 2016.
41. U.S. Department of Health and Human Services. *The health consequences of smoking—50 years of progress: a report of the Surgeon General*. Atlanta, GA: 2014. http://www.surgeongeneral.gov/library/reports/50-years-of-progress/. Accessed 30 January 2016.
42. World Health Organization. *WHO global report on trends in prevalence of tobacco smoking* Geneva, Switzerland: 2015. http://apps.who.int/iris/bitstream/10665/156262/1/9789241564922_eng.pdf. Accessed 12 April 2016.
43. Substance Abuse and Mental Health Services Administration. *Results from the 2012 National Survey on Drug Use and Health: Summary of National Findings*. Rockville, MD: Substance Abuse and Mental Health Services Administration; 2013.

http://www.samhsa.gov/data/sites/default/files/NSDUHresults2012/NSDUHresults2012.pdf. Accessed 20 June 2016.
44. Bernat DH, Klein EG, Forster JL. Smoking initiation during young adulthood: a longitudinal study of a population-based cohort. *J Adolesc Health.* 2012;51(5):497-502. doi:10.1016/j.jadohealth.2012.02.017.
45. O'Loughlin JL, Dugas EN, O'Loughlin EK, Karp I, Sylvestre MP. Incidence and determinants of cigarette smoking initiation in young adults. *J Adolesc Health.* 2014;54(1):26-32 e24. doi:10.1016/j.jadohealth.2013.07.009.
46. Substance Abuse and Mental Health Services Administration. *Results from the 2013 National Survey on Drug Use and Health: Summary of National Findings*. Rockville, MD: Substance Abuse and Mental Health Services Administration; 2014. http://www.samhsa.gov/data/sites/default/files/NSDUHresultsPDFWHTML2013/Web/NSDUHresults2013.pdf. Accessed 19 May 2016.
47. Hammond D. Smoking behaviour among young adults: beyond youth prevention. *Tob Control.* 2005;14(3):181-185. doi:10.1136/tc.2004.009621.
48. National Centre for Health Statistics. *National Health Interview Survey, 2009*. Atlanta, GA: U.S. Centres for Disease Control and Prevention; 2009. http://www.cdc.gov/nchs/nhis/nhis_2009_data_release.htm. Accessed 19 May 2016.
49. National Centre for Health Statistics. *National Health Institute Survey, 2007*. Atlanta, GA: U.S. Centres for Disease Control and Prevention; 2007. http://www.cdc.gov/nchs/nhis/nhis_2007_data_release.htm. Accessed 19 May 2016.
50. Edwards R, Carter K, Peace J, Blakely T. An examination of smoking initiation rates by age: results from a large longitudinal study in New Zealand. *Aust N Z J Public Health.* 2013;37(6):516-519.
51. Ling PM, Glantz SA. Why and how the tobacco industry sells cigarettes to young adults: evidence from industry documents. *Am J Public Health.* 2002;92(6):908-916.
52. Jha P, Ramasundarahettige C, Landsman V, et al. 21st-century hazards of smoking and benefits of cessation in the United States. *N Engl J Med.* 2013;368(4):341-350. doi:10.1056/NEJMsa1211128.
53. Pirie K, Peto R, Reeves GK, Green J, Beral V, Million Women Study C. The 21st century hazards of smoking and benefits of stopping: a prospective study of one million women in the UK. *Lancet.* 2013;381(9861):133-141. doi:10.1016/S0140-6736(12)61720-6.
54. Sakata R, McGale P, Grant EJ, Ozasa K, Peto R, Darby SC. Impact of smoking on mortality and life expectancy in Japanese smokers: a prospective cohort study. *BMJ.* 2012;345:e7093. doi:10.1136/bmj.e7093.
55. Banks E, Joshy G, Weber MF, et al. Tobacco smoking and all-cause mortality in a large Australian cohort study: findings from a mature epidemic with current low smoking prevalence. *BMC Med.* 2015;13:38. doi:10.1186/s12916-015-0281-z.
56. World Health Organization. *Tobacco fact sheet N°339*. Geneva, Switzerland: 2015. http://www.who.int/mediacentre/factsheets/fs339/en/. Accessed 22 January 2016.
57. Mathers CD, Loncar D. Projections of global mortality and burden of disease from 2002 to 2030. *PLoS Med.* 2006;3(11):e442. doi:10.1371/journal.pmed.0030442.
58. Oberg M, Jaakkola MS, Woodward A, Peruga A, Pruss-Ustun A. Worldwide burden of disease from exposure to second-hand smoke: a retrospective analysis of data from

192 countries. *Lancet.* 2011;377(9760):139-146. doi:10.1016/S0140-6736(10)61388-8.

59. World Health Organization. *WHO global report: mortality attributable to tobacco*. Geneva, Switzerland: 2012. http://www.who.int/tobacco/publications/surveillance/rep_mortality_attributable/en/. Accessed 22 January 2016.
60. Ezzati M, Lopez AD. Estimates of global mortality attributable to smoking in 2000. *Lancet.* 2003;362(9387):847-852. doi:10.1016/S0140-6736(03)14338-3.
61. US Department of Health and Human Services. *Reducing the health consequences of smoking: 25 years of progress. a report of the surgeon general*. Rockville, Maryland: 1989. https://profiles.nlm.nih.gov/ps/access/NNBBXS.pdf. Accessed 31 January 2016.
62. Collins DJ, Lapsley HM. *The costs of tobacco, alcohol and illicit drug abuse to Australian society in 2004/5*. Canberra: 2008. http://www.health.gov.au/internet/drugstrategy/publishing.nsf/Content/34F55AF632F67B70CA2573F60005D42B/$File/mono64.pdf. Accessed 29 January 2016.
63. Lundback B, Lindberg A, Lindstrom M, et al. Not 15 but 50% of smokers develop COPD?--Report from the Obstructive Lung Disease in Northern Sweden Studies. *Respir Med.* 2003;97(2):115-122.
64. US Department of Health and Human Services. *The health consequences of smoking: a report of the Surgeon General*. Atlanta, GA: US Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2004. http://www.cdc.gov/tobacco/data_statistics/sgr/2004/. Accessed 28 January 2016.
65. Doll R, Peto R, Boreham J, Sutherland I. Mortality in relation to smoking: 50 years' observations on male British doctors. *BMJ.* 2004;328(7455):1519. doi:10.1136/bmj.38142.554479.AE.
66. Chen Z, Peto R, Zhou M, et al. Contrasting male and female trends in tobacco-attributed mortality in China: evidence from successive nationwide prospective cohort studies. *Lancet.* 2015;386(10002):1447-1456. doi:10.1016/S0140-6736(15)00340-2.
67. Thun MJ, Carter BD, Feskanich D, et al. 50-year trends in smoking-related mortality in the United States. *N Engl J Med.* 2013;368(4):351-364. doi:10.1056/NEJMsa1211127.
68. Hu TW, Mao ZZ, Shi J, Chen WD. Tobacco taxation and its potential impact in China. 2008; http://www.worldlungfoundation.org/ht/a/GetDocumentAction/i/6556. Accessed 28 June, 2016.
69. Eriksen M, Mackay J, Schluger N, Gomeshtapeh FI, Drope J. *The tobacco atlas.* 5th ed. Atlanta: The American Cancer Society, Inc; 2015.
70. Glassman AH, Helzer JE, Covey LS, et al. Smoking, smoking cessation, and major depression. *JAMA.* 1990;264(12):1546-1549.
71. Hughes JR, Hatsukami DK, Mitchell JE, Dahlgren LA. Prevalence of smoking among psychiatric outpatients. *Am J Psychiatry.* 1986;143(8):993-997. doi:10.1176/ajp.143.8.993.
72. de Leon J, Dadvand M, Canuso C, White AO, Stanilla JK, Simpson GM. Schizophrenia and smoking: an epidemiological survey in a state hospital. *Am J Psychiatry.* 1995;152(3):453-455. doi:10.1176/ajp.152.3.453.

73. Lasser K, Boyd JW, Woolhandler S, Himmelstein DU, McCormick D, Bor DH. Smoking and mental illness: A population-based prevalence study. *JAMA.* 2000;284(20):2606-2610.
74. Hickman NJ, 3rd, Delucchi KL, Prochaska JJ. A population-based examination of cigarette smoking and mental illness in Black Americans. *Nicotine Tob Res.* 2010;12(11):1125-1132. doi:10.1093/ntr/ntq160.
75. Smith PH, Mazure CM, McKee SA. Smoking and mental illness in the U.S. population. *Tob Control.* 2014;23(e2):e147-153. doi:10.1136/tobaccocontrol-2013-051466.
76. Centers for Disease Control Prevention. Vital signs: current cigarette smoking among adults aged >/=18 years with mental illness - United States, 2009-2011. *MMWR Morb Mortal Wkly Rep.* 2013;62(5):81-87.
77. Cook BL, Wayne GF, Kafali EN, Liu Z, Shu C, Flores M. Trends in smoking among adults with mental illness and association between mental health treatment and smoking cessation. *JAMA.* 2014;311(2):172-182. doi:10.1001/jama.2013.284985.
78. McEwen A, West R, McRobbie H. Motives for smoking and their correlates in clients attending Stop Smoking treatment services. *Nicotine Tob Res.* 2008;10(5):843-850. doi:10.1080/14622200802027248.
79. Fidler JA, West R. Self-perceived smoking motives and their correlates in a general population sample. *Nicotine Tob Res.* 2009;11(10):1182-1188. doi:10.1093/ntr/ntp120.
80. Lerman C, Audrain J, Orleans CT, et al. Investigation of mechanisms linking depressed mood to nicotine dependence. *Addict Behav.* 1996;21(1):9-19.
81. Clancy N, Zwar N, Richmond R. Depression, smoking and smoking cessation: a qualitative study. *Fam Pract.* 2013;30(5):587-592. doi:10.1093/fampra/cmt032.
82. Twyman L, Bonevski B, Paul C, Bryant J. Perceived barriers to smoking cessation in selected vulnerable groups: a systematic review of the qualitative and quantitative literature. *BMJ Open.* 2014;4(12):e006414. doi:10.1136/bmjopen-2014-006414.
83. Thompson B, Thompson LA, Thompson J, Fredickson C, Bishop S. Heavy smokers: a qualitative analysis of attitudes and beliefs concerning cessation and continued smoking. *Nicotine Tob Res.* 2003;5(6):923-933. doi:10.1080/14622200310001615277.
84. Taylor G, McNeill A, Girling A, Farley A, Lindson-Hawley N, Aveyard P. Change in mental health after smoking cessation: systematic review and meta-analysis. *BMJ.* 2014;348:g1151. doi:10.1136/bmj.g1151.
85. McDermott MS, Marteau TM, Hollands GJ, Hankins M, Aveyard P. Change in anxiety following successful and unsuccessful attempts at smoking cessation: cohort study. *Br J Psychiatry.* 2013;202(1):62-67. doi:10.1192/bjp.bp.112.114389.
86. Kirst M, Mecredy G, Borland T, Chaiton M. Predictors of substance use among young adults transitioning away from high school: a narrative review. *Subst Use Misuse.* 2014;49(13):1795-1807. doi:10.3109/10826084.2014.933240.
87. Wetter DW, Kenford SL, Welsch SK, et al. Prevalence and predictors of transitions in smoking behavior among college students. *Health Psychol.* 2004;23(2):168-177. doi:10.1037/0278-6133.23.2.168.
88. Windle M, Windle RC. Depressive symptoms and cigarette smoking among middle adolescents: prospective associations and intrapersonal and interpersonal influences. *J Consult Clin Psychol.* 2001;69(2):215-226.

89. Colgan Y, Turnbull DA, Mikocka-Walus AA, Delfabbro P. Determinants of resilience to cigarette smoking among young Australians at risk: an exploratory study. *Tob Induc Dis.* 2010;8:7. doi:10.1186/1617-9625-8-7.
90. Bellido-Casado J, Martin-Escudero J, Duenas-Laita A, Mena-Martin FJ, Arzua-Mouronte D, Simal-Blanco F. The SF-36 Questionnaire as a measurement of health-related quality of life: assessing short- and medium-term effects of exposure to tobacco versus the known long-term effects. *Eur J Intern Med.* 2004;15(8):511-517. doi:10.1016/j.ejim.2004.06.015.
91. Wilson D, Parsons J, Wakefield M. The health-related quality-of-life of never smokers, ex-smokers, and light, moderate, and heavy smokers. *Prev Med.* 1999;29(3):139-144. doi:10.1006/pmed.1999.0523.
92. Mody RR, Smith MJ. Smoking status and health-related quality of life: as findings from the 2001 Behavioral Risk Factor Surveillance System data. *Am J Health Promot.* 2006;20(4):251-258.
93. McClave AK, Dube SR, Strine TW, Mokdad AH. Associations between health-related quality of life and smoking status among a large sample of U.S. adults. *Prev Med.* 2009;48(2):173-179. doi:10.1016/j.ypmed.2008.11.012.
94. Vogl M, Wenig CM, Leidl R, Pokhrel S. Smoking and health-related quality of life in English general population: implications for economic evaluations. *BMC Public Health.* 2012;12:203. doi:10.1186/1471-2458-12-203.
95. Sarna L, Bialous SA, Cooley ME, Jun HJ, Feskanich D. Impact of smoking and smoking cessation on health-related quality of life in women in the Nurses' Health Study. *Qual Life Res.* 2008;17(10):1217-1227. doi:10.1007/s11136-008-9404-8.
96. Piper ME, Kenford S, Fiore MC, Baker TB. Smoking cessation and quality of life: changes in life satisfaction over 3 years following a quit attempt. *Ann Behav Med.* 2012;43(2):262-270. doi:10.1007/s12160-011-9329-2.
97. Chen PC, Kuo RN, Lai CK, Tsai ST, Lee YC. The relationship between smoking status and health-related quality of life among smokers who participated in a 1-year smoking cessation programme in Taiwan: a cohort study using the EQ-5D. *BMJ Open.* 2015;5(5):e007249. doi:10.1136/bmjopen-2014-007249.
98. Hays JT, Croghan IT, Baker CL, Cappelleri JC, Bushmakin AG. Changes in health-related quality of life with smoking cessation treatment. *Eur J Public Health.* 2012;22(2):224-229. doi:10.1093/eurpub/ckq137.
99. Sales MP, Oliveira MI, Mattos IM, Viana CM, Pereira ED. The impact of smoking cessation on patient quality of life. *J Bras Pneumol.* 2009;35(5):436-441.
100. Erickson SR, Thomas LA, Blitz SG, Pontius LR. Smoking cessation: a pilot study of the effects on health-related quality of life and perceived work performance one week into the attempt. *Ann Pharmacother.* 2004;38(11):1805-1810. doi:10.1345/aph.1E194.
101. Guiterrez-Bedmar M, Segui-Gomez M, Gomez-Gracia E, Bes-Rastrollo M, Martinez-Gonzalez MA. Smoking status, changes in smoking status and health-related quality of life: findings from the SUN ("Seguimiento Universidad de Navarra") cohort. *Int J Environ Res Public Health.* 2009;6(1):310-320. doi:10.3390/ijerph6010310.
102. Zillich AJ, Ryan M, Adams A, Yeager B, Farris K. Effectiveness of a pharmacist-based smoking-cessation program and its impact on quality of life. *Pharmacotherapy.* 2002;22(6):759-765.

103. Romer D, Jamison P. *The role of perceived risk in starting and stopping smoking.* Thousand Oaks, CA: Sage Publications; 2001.
104. Slovic P. *Cigarette smokers: rational actors or rational fools?* Thousand Oaks, CA: Sage Publications; 2001.
105. DiFranza JR, Rigotti NA, McNeill AD, et al. Initial symptoms of nicotine dependence in adolescents. *Tob Control.* 2000;9(3):313-319.
106. Gold DR, Wang X, Wypij D, Speizer FE, Ware JH, Dockery DW. Effects of cigarette smoking on lung function in adolescent boys and girls. *N Engl J Med.* 1996;335(13):931-937. doi:10.1056/NEJM199609263351304.
107. Grol MH, Gerritsen J, Vonk JM, et al. Risk factors for growth and decline of lung function in asthmatic individuals up to age 42 years. A 30-year follow-up study. *Am J Respir Crit Care Med.* 1999;160(6):1830-1837. doi:10.1164/ajrccm.160.6.9812100.
108. US Department of Health and Human Services. *Preventing tobacco use among young people: a report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 1994. http://www.cdc.gov/tobacco/data_statistics/sgr/1994/index.htm. Accessed 20 May 2016.
109. Arday DR, Giovino GA, Schulman J, Nelson DE, Mowery P, Samet JM. Cigarette smoking and self-reported health problems among U.S. high school seniors, 1982-1989. *Am J Health Promot.* 1995;10(2):111-116.
110. Lam TH, Chung SF, Betson CL, Wong CM, Hedley AJ. Respiratory symptoms due to active and passive smoking in junior secondary school students in Hong Kong. *Int J Epidemiol.* 1998;27(1):41-48.
111. Sotir M, Yeatts K, Shy C. Presence of asthma risk factors and environmental exposures related to upper respiratory infection-triggered wheezing in middle school-age children. *Environ Health Perspect.* 2003;111(4):657-662.
112. Butland BK, Strachan DP. Asthma onset and relapse in adult life: the British 1958 birth cohort study. *Ann Allergy Asthma Immunol.* 2007;98(4):337-343. doi:10.1016/S1081-1206(10)60879-4.
113. Withers NJ, Low L, Holgate ST, Clough JB. The natural history of respiratory symptoms in a cohort of adolescents. *Am J Respir Crit Care Med.* 1998;158(2):352-357. doi:10.1164/ajrccm.158.2.9705079.
114. Vogelberg C, Hirsch T, Radon K, et al. Leisure time activity and new onset of wheezing during adolescence. *Eur Respir J.* 2007;30(4):672-676. doi:10.1183/09031936.00152906.
115. Tollefsen E, Langhammer A, Romundstad P, Bjermer L, Johnsen R, Holmen TL. Female gender is associated with higher incidence and more stable respiratory symptoms during adolescence. *Respir Med.* 2007;101(5):896-902. doi:10.1016/j.rmed.2006.09.022.
116. Berenson GS, Srinivasan SR, Bao W, Newman WP, 3rd, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. *N Engl J Med.* 1998;338(23):1650-1656. doi:10.1056/NEJM199806043382302.
117. Kadar A, Mozes G, Illyes G, et al. World Health organization (WHO) and the World Heart Federation (WHF) pathobiological determinants of atherosclerosis in youth study (WHO/WHF PBDAY Study) 1986-1996. Histomorphometry and histochemistry

of atherosclerotic lesions in coronary arteries and the aorta in a young population. *Nutr Metab Cardiovasc Dis.* 1999;9(5):220-227.
118. McGill HC, Jr., McMahan CA, Herderick EE, et al. Effects of coronary heart disease risk factors on atherosclerosis of selected regions of the aorta and right coronary artery. PDAY Research Group. Pathobiological Determinants of Atherosclerosis in Youth. *Arterioscler Thromb Vasc Biol.* 2000;20(3):836-845.
119. Loria CM, Liu K, Lewis CE, et al. Early adult risk factor levels and subsequent coronary artery calcification: the CARDIA Study. *J Am Coll Cardiol.* 2007;49(20):2013-2020. doi:10.1016/j.jacc.2007.03.009.
120. Juonala M, Jarvisalo MJ, Maki-Torkko N, Kahonen M, Viikari JS, Raitakari OT. Risk factors identified in childhood and decreased carotid artery elasticity in adulthood: the Cardiovascular Risk in Young Finns Study. *Circulation.* 2005;112(10):1486-1493. doi:10.1161/CIRCULATIONAHA.104.502161.
121. Gall S, Huynh QL, Magnussen CG, et al. Exposure to parental smoking in childhood or adolescence is associated with increased carotid intima-media thickness in young adults: evidence from the Cardiovascular Risk in Young Finns study and the Childhood Determinants of Adult Health Study. *Eur Heart J.* 2014;35(36):2484-2491. doi:10.1093/eurheartj/ehu049.
122. Haber J, Wattles J, Crowley M, Mandell R, Joshipura K, Kent RL. Evidence for cigarette smoking as a major risk factor for periodontitis. *J Periodontol.* 1993;64(1):16-23. doi:10.1902/jop.1993.64.1.16.
123. Palmerini CA, Saccardi C, Ferracci F, Arienti S. Lipid patterns in the saliva of smoking young adults. *Hum Exp Toxicol.* 2011;30(10):1482-1488. doi:10.1177/0960327111398672.
124. Law MR, Hackshaw AK. A meta-analysis of cigarette smoking, bone mineral density and risk of hip fracture: recognition of a major effect. *BMJ.* 1997;315(7112):841-846.
125. Shen GS, Li Y, Zhao G, et al. Cigarette smoking and risk of hip fracture in women: a meta-analysis of prospective cohort studies. *Injury.* 2015;46(7):1333-1340. doi:10.1016/j.injury.2015.04.008.
126. Taes Y, Lapauw B, Vanbillemont G, et al. Early smoking is associated with peak bone mass and prevalent fractures in young, healthy men. *J Bone Miner Res.* 2010;25(2):379-387. doi:10.1359/jbmr.090809.
127. World Health Organization. *Economics of tobacco toolkit: assessment of the economic costs of smoking*. Geneva, Switzerland: 2011. http://apps.who.int/iris/bitstream/10665/44596/1/9789241501576_eng.pdf. Accessed 28 January 2016.
128. Lightwood J, Collins D, Lapsley H, Novotny TE. *Tobacco control in developing countries.* Oxford, England: Oxford University Press; 2000.
129. Sung HY, Wang L, Jin S, Hu TW, Jiang Y. Economic burden of smoking in China, 2000. *Tob Control.* 2006;15 Suppl 1:i5-11. doi:10.1136/tc.2005.015412.
130. Yang L, Sung HY, Mao Z, Hu TW, Rao K. Economic costs attributable to smoking in China: update and an 8-year comparison, 2000-2008. *Tob Control.* 2011;20(4):266-272. doi:10.1136/tc.2010.042028.
131. John RM, Sung HY, Max W. Economic cost of tobacco use in India, 2004. *Tob Control.* 2009;18(2):138-143. doi:10.1136/tc.2008.027466.

132. Hoang Anh PT, Thu le T, Ross H, Quynh Anh N, Linh BN, Minh NT. Direct and indirect costs of smoking in Vietnam. *Tob Control.* 2016;25(1):96-100. doi:10.1136/tobaccocontrol-2014-051821.
133. Bundhamcharoen K, Aungkulanon S, Makka N, Shibuya K. Economic burden from smoking-related diseases in Thailand. *Tob Control.* 2015. doi:10.1136/tobaccocontrol-2015-052319.
134. World Bank. *Curbing the epidemic - governments and the economics of tobacco control*. Washington, D.C.: 1999. http://documents.worldbank.org/curated/en/1999/05/437174/curbing-epidemic-governments-economics-tobacco-control. Accessed 28 January 2016.
135. Begg S, Vos T, Barker B, Stevenson C, Stanley L, Lopez AD. *The burden of disease and injury in Australia 2003. PHE 82.* . Canberra: Welfare AIoHa; 2007. http://www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=6442459747. Accessed 29 January 2016.
136. Collins DJ, Lapsley HM. *Estimating the economic costs of drug abuse. National Campaign Against Drug Abuse Monograph Series No. 15*. Canberra: 1991. Accessed 29 January 2016.
137. Collins DJ, Lapsley HM. *The social costs of drug abuse in Australia in 1988 and 1992. National Drug Strategy Monograph Series No. 30.* Canberra: 1996. https://www.researchgate.net/publication/252250147_The_Social_Costs_of_Drug_Abuse_in_Australia_in_1988_and_1992. Accessed 29 January 2016.
138. Collins DJ, Lapsley HM. *Counting the cost: estimates of the social costs of drug abuse in Australia in 1998-9. National Drug Strategy Monograph Series No 49.* Canberra: 2002. http://drogfokuszpont.hu/wp-content/uploads/kokk_social_cost_australia_99.pdf. Accessed 29 January 2016.
139. Collins DJ, Lapsley HM. *Counting the costs of tobacco and the benefits of reducing smoking prevalence in Victoria*. Melbourne: Victorian Department of Human Services; 2006. https://www2.health.vic.gov.au/about/publications/factsheets/Counting%20the%20costs%20of%20tobacco%20and%20the%20benefits%20of%20reducing%20smoking%20prevalence%20in%20Victoria. Accessed 29 January 2016.
140. Collins DJ, Lapsley HM. *Counting the costs of tobacco and the benefits of reducing smoking prevalence in New South Wales*. Sydney: New South Wales Department of Health; 2005. Accessed 29 January 2016.
141. Collins DJ, Lapsley HM. *The social costs of smoking in Western Australia in 2009/10 and the social benefits of public policy measures to reduce smoking prevalence*. Perth: Cancer Council Western Australia; 2014. https://www.cancerwa.asn.au/resources/2014-10-28-Social-Costs-of-Smoking-Final.pdf. Accessed 29 January 2016.
142. US Department of Health and Human Services. *The health benefits of smoking cessation: a report of the Surgeon General* Atlanta, GA: 1990. http://profiles.nlm.nih.gov/NN/B/B/C/V/_/nnbbcv.pdf. Accessed 30 January 2016.
143. Mons U, Muezzinler A, Gellert C, et al. Impact of smoking and smoking cessation on cardiovascular events and mortality among older adults: meta-analysis of individual participant data from prospective cohort studies of the CHANCES consortium. *BMJ.* 2015;350:h1551. doi:10.1136/bmj.h1551.

144. Welfare AIoHa. *2010 National Drug Strategy Household Survey report. Drug statistics series no. 25. Cat. no. PHE 145.* Canberra: 2011. http://www.aihw.gov.au/WorkArea/DownloadAsset.aspx?id=10737421314. Accessed 31 January 2016.
145. Centers for Disease Control and Prevention. *Quitting smoking among adults—United States, 2001–2010. Morbidity and Mortality Weekly Report.* Atlanta, GA: 2011. http://www.cdc.gov/mmwr/pdf/wk/mm6044.pdf. Accessed 31 January 2016.
146. Gilpin EA, White VM, White MM, Pierce JP. Young adult smoking behavior: implications for future population health. *Am J Health Behav.* 2009;33(5):569-580.
147. Pierce JP, Gilpin E. How long will today's new adolescent smoker be addicted to cigarettes? *Am J Public Health.* 1996;86(2):253-256.
148. Messer K, Trinidad DR, Al-Delaimy WK, Pierce JP. Smoking cessation rates in the United States: a comparison of young adult and older smokers. *Am J Public Health.* 2008;98(2):317-322. doi:10.2105/AJPH.2007.112060.
149. Royal College of Physicians of London. *Nicotine addiction in Britain. A report of the Tobacco Advisory Group of the Royal College of Physicians*. London: 2000. http://shop.rcplondon.ac.uk/products/nicotine-addiction-in-britain?variant=6633984645. Accessed 31 January 2016.
150. US Department of Health and Human Services. *The health consequences of smoking: nicotine addiction: a report of the surgeon general. DHHS Publication No. (CDC) 88-8406*. Rockville, Maryland: 1988. https://profiles.nlm.nih.gov/ps/access/NNBBZD.pdf. Accessed 31 January 2016.
151. Edwards SA, Bondy SJ, Callaghan RC, Mann RE. Prevalence of unassisted quit attempts in population-based studies: a systematic review of the literature. *Addict Behav.* 2014;39(3):512-519. doi:10.1016/j.addbeh.2013.10.036.
152. Smith AL, Chapman S, Dunlop SM. What do we know about unassisted smoking cessation in Australia? A systematic review, 2005-2012. *Tob Control.* 2015;24(1):18-27. doi:10.1136/tobaccocontrol-2013-051019.
153. Stead LF, Hartmann-Boyce J, Perera R, Lancaster T. Telephone counselling for smoking cessation. *Cochrane Database Syst Rev.* 2013;8:CD002850. doi:10.1002/14651858.CD002850.pub3.
154. Hartmann-Boyce J, Lancaster T, Stead LF. Print-based self-help interventions for smoking cessation. *Cochrane Database Syst Rev.* 2014;6:CD001118. doi:10.1002/14651858.CD001118.pub3.
155. Cahill K, Stevens S, Perera R, Lancaster T. Pharmacological interventions for smoking cessation: an overview and network meta-analysis. *Cochrane Database Syst Rev.* 2013;5:CD009329. doi:10.1002/14651858.CD009329.pub2.
156. Patnode CD, Henderson JT, Thompson JH, Senger CA, Fortmann SP, Whitlock EP. *Behavioral Counseling and Pharmacotherapy Interventions for Tobacco Cessation in Adults, Including Pregnant Women: A Review of Reviews for the U.S. Preventive Services Task Force*. Rockville (MD)2015.
157. Stead LF, Koilpillai P, Lancaster T. Additional behavioural support as an adjunct to pharmacotherapy for smoking cessation. *Cochrane Database Syst Rev.* 2015;10:CD009670. doi:10.1002/14651858.CD009670.pub3.
158. Stead LF, Koilpillai P, Fanshawe TR, Lancaster T. Combined pharmacotherapy and behavioural interventions for smoking cessation. *Cochrane Database Syst Rev.* 2016;3:CD008286. doi:10.1002/14651858.CD008286.pub3.

159. West R, McEwen A, Bolling K, Owen L. Smoking cessation and smoking patterns in the general population: a 1-year follow-up. *Addiction.* 2001;96(6):891-902. doi:10.1080/09652140020051013.
160. Jampaklay A, Borland R, Yong HH, Sirirassamee B, Fotuhi O, Fong GT. Predictors of Successful Quitting among Thai Adult Smokers: Evidence from ITC-SEA (Thailand) Survey. *Int J Environ Res Public Health.* 2015;12(10):12095-12109. doi:10.3390/ijerph121012095.
161. Lund M. Social Inequality in Cigarette Consumption, Cigarette Dependence, and Intention to Quit among Norwegian Smokers. *Biomed Res Int.* 2015;2015:835080. doi:10.1155/2015/835080.
162. Hughes JR, Naud S, Fingar JR, Callas PW, Solomon LJ. Do environmental cues prompt attempts to stop smoking? A prospective natural history study. *Drug Alcohol Depend.* 2015;154:146-151. doi:10.1016/j.drugalcdep.2015.06.044.
163. Zvolensky MJ, Farris SG, Leventhal AM, Ditre JW, Schmidt NB. Emotional disorders and smoking: relations to quit attempts and cessation strategies among treatment-seeking smokers. *Addict Behav.* 2015;40:126-131. doi:10.1016/j.addbeh.2014.08.012.
164. Yong HH, Borland R, Thrasher JF, et al. Mediational pathways of the impact of cigarette warning labels on quit attempts. *Health Psychol.* 2014;33(11):1410-1420. doi:10.1037/hea0000056.
165. Hitchman SC, Fong GT, Zanna MP, Thrasher JF, Laux FL. The relation between number of smoking friends, and quit intentions, attempts, and success: findings from the International Tobacco Control (ITC) Four Country Survey. *Psychol Addict Behav.* 2014;28(4):1144-1152. doi:10.1037/a0036483.
166. Li L, Borland R, Fong GT, et al. Smoking-related thoughts and microbehaviours, and their predictive power for quitting. *Tob Control.* 2015;24(4):354-361. doi:10.1136/tobaccocontrol-2013-051384.
167. Yong LC, Luckhaupt SE, Li J, Calvert GM. Quit interest, quit attempt and recent cigarette smoking cessation in the US working population, 2010. *Occup Environ Med.* 2014;71(6):405-414. doi:10.1136/oemed-2013-101852.
168. Diemert LM, Bondy SJ, Brown KS, Manske S. Young adult smoking cessation: predictors of quit attempts and abstinence. *Am J Public Health.* 2013;103(3):449-453. doi:10.2105/AJPH.2012.300878.
169. Stockings E, Bowman J, McElwaine K, et al. Readiness to quit smoking and quit attempts among Australian mental health inpatients. *Nicotine Tob Res.* 2013;15(5):942-949. doi:10.1093/ntr/nts206.
170. Jardin BF, Carpenter MJ. Predictors of quit attempts and abstinence among smokers not currently interested in quitting. *Nicotine Tob Res.* 2012;14(10):1197-1204. doi:10.1093/ntr/nts015.
171. Caleyachetty A, Lewis S, McNeill A, Leonardi-Bee J. Struggling to make ends meet: exploring pathways to understand why smokers in financial difficulties are less likely to quit successfully. *Eur J Public Health.* 2012;22 Suppl 1:41-48. doi:10.1093/eurpub/ckr199.
172. Hagimoto A, Nakamura M, Morita T, Masui S, Oshima A. Smoking cessation patterns and predictors of quitting smoking among the Japanese general population: a 1-year follow-up study. *Addiction.* 2010;105(1):164-173. doi:10.1111/j.1360-0443.2009.02735.x.

173. Yang JJ, Song M, Yoon HS, et al. What Are the Major Determinants in the Success of Smoking Cessation: Results from the Health Examinees Study. *PLoS One.* 2015;10(12):e0143303. doi:10.1371/journal.pone.0143303.
174. Zhao L, Song Y, Xiao L, Palipudi K, Asma S. Factors influencing quit attempts among male daily smokers in China. *Prev Med.* 2015;81:361-366. doi:10.1016/j.ypmed.2015.09.020.
175. Tanihara S, Momose Y. Reasons for smoking cessation attempts among Japanese male smokers vary by nicotine dependence level: a cross-sectional study after the 2010 tobacco tax increase. *BMJ Open.* 2015;5(3):e006658. doi:10.1136/bmjopen-2014-006658.
176. Brown J, Beard E, Kotz D, Michie S, West R. Real-world effectiveness of e-cigarettes when used to aid smoking cessation: a cross-sectional population study. *Addiction.* 2014;109(9):1531-1540. doi:10.1111/add.12623.
177. Partos TR, Borland R, Yong HH, Hyland A, Cummings KM. The quitting rollercoaster: how recent quitting history affects future cessation outcomes (data from the International Tobacco Control 4-country cohort study). *Nicotine Tob Res.* 2013;15(9):1578-1587. doi:10.1093/ntr/ntt025.
178. Ghani WM, Razak IA, Yang YH, et al. Factors affecting commencement and cessation of smoking behaviour in Malaysian adults. *BMC Public Health.* 2012;12:207. doi:10.1186/1471-2458-12-207.
179. Filozof C, Fernandez Pinilla MC, Fernandez-Cruz A. Smoking cessation and weight gain. *Obes Rev.* 2004;5(2):95-103. doi:10.1111/j.1467-789X.2004.00131.x.
180. McDermott L, Dobson A, Owen N. Determinants of continuity and change over 10 years in young women's smoking. *Addiction.* 2009;104(3):478-487. doi:10.1111/j.1360-0443.2008.02452.x.
181. Vangeli E, Stapleton J, Smit ES, Borland R, West R. Predictors of attempts to stop smoking and their success in adult general population samples: a systematic review. *Addiction.* 2011;106(12):2110-2121. doi:10.1111/j.1360-0443.2011.03565.x.
182. Cengelli S, O'Loughlin J, Lauzon B, Cornuz J. A systematic review of longitudinal population-based studies on the predictors of smoking cessation in adolescent and young adult smokers. *Tob Control.* 2012;21(3):355-362. doi:10.1136/tc.2011.044149.
183. Hyland A, Borland R, Li Q, et al. Individual-level predictors of cessation behaviours among participants in the International Tobacco Control (ITC) Four Country Survey. *Tob Control.* 2006;15 Suppl 3:iii83-94. doi:10.1136/tc.2005.013516.
184. Williamson DF, Madans J, Anda RF, Kleinman JC, Giovino GA, Byers T. Smoking cessation and severity of weight gain in a national cohort. *N Engl J Med.* 1991;324(11):739-745. doi:10.1056/NEJM199103143241106.
185. Shimokata H, Muller DC, Andres R. Studies in the distribution of body fat. III. Effects of cigarette smoking. *JAMA.* 1989;261(8):1169-1173.
186. Klesges RC, Meyers AW, Klesges LM, La Vasque ME. Smoking, body weight, and their effects on smoking behavior: a comprehensive review of the literature. *Psychol Bull.* 1989;106(2):204-230.
187. Veldheer S, Yingst J, Zhu J, Foulds J. Ten-year weight gain in smokers who quit, smokers who continued smoking and never smokers in the United States, NHANES 2003-2012. *Int J Obes (Lond).* 2015;39(12):1727-1732. doi:10.1038/ijo.2015.127.

188. Travier N, Agudo A, May AM, et al. Longitudinal changes in weight in relation to smoking cessation in participants of the EPIC-PANACEA study. *Prev Med.* 2012;54(3-4):183-192. doi:10.1016/j.ypmed.2011.09.003.
189. Chiolero A, Faeh D, Paccaud F, Cornuz J. Consequences of smoking for body weight, body fat distribution, and insulin resistance. *Am J Clin Nutr.* 2008;87(4):801-809.
190. Flegal KM. The conundrum of smoking cessation and weight gain. *Prev Med.* 2012;54(3-4):193-194. doi:10.1016/j.ypmed.2012.01.015.
191. Camp DE, Klesges RC, Relyea G. The relationship between body weight concerns and adolescent smoking. *Health Psychol.* 1993;12(1):24-32.
192. French SA, Perry CL, Leon GR, Fulkerson JA. Weight concerns, dieting behavior, and smoking initiation among adolescents: a prospective study. *Am J Public Health.* 1994;84(11):1818-1820.
193. Levine MD, Bush T, Magnusson B, Cheng Y, Chen X. Smoking-related weight concerns and obesity: differences among normal weight, overweight, and obese smokers using a telephone tobacco quitline. *Nicotine Tob Res.* 2013;15(6):1136-1140. doi:10.1093/ntr/nts226.
194. Pisinger C, Jorgensen T. Weight concerns and smoking in a general population: the Inter99 study. *Prev Med.* 2007;44(4):283-289. doi:10.1016/j.ypmed.2006.11.014.
195. Wee CC, Rigotti NA, Davis RB, Phillips RS. Relationship between smoking and weight control efforts among adults in the united states. *Arch Intern Med.* 2001;161(4):546-550.
196. Audrain-McGovern J, Benowitz NL. Cigarette smoking, nicotine, and body weight. *Clin Pharmacol Ther.* 2011;90(1):164-168. doi:10.1038/clpt.2011.105.
197. Jo YH, Talmage DA, Role LW. Nicotinic receptor-mediated effects on appetite and food intake. *J Neurobiol.* 2002;53(4):618-632. doi:10.1002/neu.10147.
198. Rodin J. Weight change following smoking cessation: the role of food intake and exercise. *Addict Behav.* 1987;12(4):303-317.
199. Spring B, Howe D, Berendsen M, et al. Behavioral intervention to promote smoking cessation and prevent weight gain: a systematic review and meta-analysis. *Addiction.* 2009;104(9):1472-1486. doi:10.1111/j.1360-0443.2009.02610.x.
200. Parsons AC, Shraim M, Inglis J, Aveyard P, Hajek P. Interventions for preventing weight gain after smoking cessation. *Cochrane Database Syst Rev.* 2009(1):CD006219. doi:10.1002/14651858.CD006219.pub2.
201. Yang M, Bhowmik D, Wang X, Abughosh S. Does combination pharmacological intervention for smoking cessation prevent post-cessation weight gain? A systemic review. *Addict Behav.* 2013;38(3):1865-1875. doi:10.1016/j.addbeh.2012.11.007.
202. Farley AC, Hajek P, Lycett D, Aveyard P. Interventions for preventing weight gain after smoking cessation. *Cochrane Database Syst Rev.* 2012;1:CD006219. doi:10.1002/14651858.CD006219.pub3.
203. Aubin HJ, Farley A, Lycett D, Lahmek P, Aveyard P. Weight gain in smokers after quitting cigarettes: meta-analysis. *BMJ.* 2012;345:e4439. doi:10.1136/bmj.e4439.
204. Fernandez E, Chapman S. Quitting smoking and gaining weight: the odd couple. *BMJ.* 2012;345:e4544. doi:10.1136/bmj.e4544.
205. Fagerstrom KO, Kunze M, Schoberberger R, et al. Nicotine dependence versus smoking prevalence: comparisons among countries and categories of smokers. *Tob Control.* 1996;5(1):52-56.

206. Chapman S. Tar wars over smoking cessation. *BMJ.* 2011;343:d5008. doi:10.1136/bmj.d5008.
207. Pohl JM, Martinelli A, Antonakos C. Predictors of participation in a smoking cessation intervention group among low-income women. *Addict Behav.* 1998;23(5):699-704.
208. Klesges RC, Ward KD, Ray JW, Cutter G, Jacobs DR, Jr., Wagenknecht LE. The prospective relationships between smoking and weight in a young, biracial cohort: the Coronary Artery Risk Development in Young Adults Study. *J Consult Clin Psychol.* 1998;66(6):987-993.
209. Piper ME, Schlam TR, Cook JW, et al. Tobacco withdrawal components and their relations with cessation success. *Psychopharmacology (Berl).* 2011;216(4):569-578. doi:10.1007/s00213-011-2250-3.
210. Hughes JR. Effects of abstinence from tobacco: etiology, animal models, epidemiology, and significance: a subjective review. *Nicotine Tob Res.* 2007;9(3):329-339. doi:10.1080/14622200701188927.
211. Hughes JR. Effects of abstinence from tobacco: valid symptoms and time course. *Nicotine Tob Res.* 2007;9(3):315-327. doi:10.1080/14622200701188919.
212. Benowitz NL. Clinical pharmacology of nicotine: implications for understanding, preventing, and treating tobacco addiction. *Clin Pharmacol Ther.* 2008;83(4):531-541. doi:10.1038/clpt.2008.3.
213. Royal College of Physicians, Royal College of Psychiatrists. *Smoking and mental health*. London: 2013. https://cdn.shopify.com/s/files/1/0924/4392/files/smoking_and_mental_health_-_full_report_web.pdf?7537870595093585378. Accessed 15 February 2016.
214. Greene AL, Wheatley SM, Aldava JF. Stages on life's way: adolescents' im-plicit theories of the life course. *J Adolesc Res.* 1992;7(3):364-381.
215. McDermott L, Dobson A, Russell A. Changes in smoking behaviour among young women over life stage transitions. *Aust N Z J Public Health.* 2004;28(4):330-335.
216. Eng PM, Kawachi I, Fitzmaurice G, Rimm EB. Effects of marital transitions on changes in dietary and other health behaviours in US male health professionals. *J Epidemiol Community Health.* 2005;59(1):56-62. doi:10.1136/jech.2004.020073.
217. Lee S, Cho E, Grodstein F, Kawachi I, Hu FB, Colditz GA. Effects of marital transitions on changes in dietary and other health behaviours in US women. *Int J Epidemiol.* 2005;34(1):69-78. doi:10.1093/ije/dyh258.
218. Bradley RH, Corwyn RF. Socioeconomic status and child development. *Annual review of psychology.* 2002;53(1):371-399.
219. Galobardes B, Shaw M, Lawlor DA, Lynch JW, Davey Smith G. Indicators of socioeconomic position (part 1). *J Epidemiol Community Health.* 2006;60(1):7-12. doi:10.1136/jech.2004.023531.
220. Galobardes B, Shaw M, Lawlor DA, Lynch JW, Davey Smith G. Indicators of socioeconomic position (part 2). *J Epidemiol Community Health.* 2006;60(2):95-101. doi:10.1136/jech.2004.028092.
221. Hiscock R, Bauld L, Amos A, Fidler JA, Munafo M. Socioeconomic status and smoking: a review. *Ann N Y Acad Sci.* 2012;1248:107-123. doi:10.1111/j.1749-6632.2011.06202.x.
222. Gilman SE, Abrams DB, Buka SL. Socioeconomic status over the life course and stages of cigarette use: initiation, regular use, and cessation. *J Epidemiol Community Health.* 2003;57(10):802-808.

223. Jefferis B, Graham H, Manor O, Power C. Cigarette consumption and socio-economic circumstances in adolescence as predictors of adult smoking. *Addiction.* 2003;98(12):1765-1772.
224. Amos A, Bauld L, Hill S, Platt S, Robinson J. Tobacco control, inequalities in health and action at the local level in England. *London: Public Health Research Consortium.* 2011.
225. Sharma A, Lewis S, Szatkowski L. Insights into social disparities in smoking prevalence using Mosaic, a novel measure of socioeconomic status: an analysis using a large primary care dataset. *BMC Public Health.* 2010;10:755. doi:10.1186/1471-2458-10-755.
226. Kanjilal S, Gregg EW, Cheng YJ, et al. Socioeconomic status and trends in disparities in 4 major risk factors for cardiovascular disease among US adults, 1971-2002. *Arch Intern Med.* 2006;166(21):2348-2355. doi:10.1001/archinte.166.21.2348.
227. Peretti-Watel P, Constance J, Seror V, Beck F. Cigarettes and social differentiation in France: is tobacco use increasingly concentrated among the poor? *Addiction.* 2009;104(10):1718-1728. doi:10.1111/j.1360-0443.2009.02682.x.
228. Smith P, Frank J, Mustard C. Trends in educational inequalities in smoking and physical activity in Canada: 1974-2005. *J Epidemiol Community Health.* 2009;63(4):317-323. doi:10.1136/jech.2008.078204.
229. Organisation for Economic Co-operation and Development. *Divided we stand: why inequality keeps rising*. Paris, France: 2011. https://www.oecd.org/els/soc/49170768.pdf. Accessed 10 January 2016.
230. Mishra G, Nitsch D, Black S, De Stavola B, Kuh D, Hardy R. A structured approach to modelling the effects of binary exposure variables over the life course. *Int J Epidemiol.* 2009;38(2):528-537. doi:10.1093/ije/dyn229.
231. Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, Power C. Life course epidemiology. *J Epidemiol Community Health.* 2003;57(10):778-783.

Chapter 2

Effects of partnering and parenting transitions on changes in smoking status over 5 years in young Australians

Chapter 2. Effects of partnering and parenting transitions on smoking continuity over 5 years in young Australians

2.1 Preface

As briefly introduced in the previous section, young adulthood is a time for establishing life-partnerships and having children. It is also a critical period when smoking prevalence peaks and when progression from experimental to regular smoking often occurs. Few longitudinal studies have addressed the effects of partnering and parenting transitions on changing smoking status, especially in young men. This chapter aims to address this gap in the literature.

2.2 Introduction

Life-stage transitions are common among young adults as they complete education or training and enter work, and as their family, work, and financial responsibilities increase. Two important life-stage transitions are becoming partnered and having children. Over recent decades, transitions into marriage and parenthood have been occurring at later ages. In western countries, such as Australia, the median age at first marriage was 29.9 years for males and 28.3 for females,[1] and the average age of first time mothers was 28.6.[2] Life-stage transitions are often viewed as a time of "maturing out" and may be accompanied by major changes in social networks, social roles, responsibilities and expectations, which may impact positively or negatively on physical and mental health.[3,4]

The previous section established that cigarette smoking is a common unhealthy behaviour. It is the leading preventable cause of death and illness worldwide, with about half of current smokers dying prematurely from a tobacco-related disease, including various cancers, CVD, respiratory disease and other illness.[5] In spite of a low and decreasing smoking prevalence in Australia [6], the risks of smoking remain high, with up to two-thirds of deaths in current smokers attributed to smoking[7]. In Australia and other high income countries, young adulthood is a critical period when smoking prevalence peaks and when progression from experimental to regular smoking often occurs.[6,8]

Providing an in-depth exploration of the effects of partnering and parenting transitions on smoking continuity, cessation and relapse in this critical time may provide important insights

for health practitioners and policy makers that could optimise and reinforce their work to promote quitting and enhance sustained cessation; however, few longitudinal studies of these factors exist. In a longitudinal study of young women from Australia,[9] marriage, being in a committed relationship or being a mother significantly decreased the risk of continuing and resuming smoking but this study was unable to compare effects in men where the associations are unclear. Two longitudinal studies prospectively examined the effects of marital transitions on changes in health behaviours,[10,11] including cigarette smoking. However, participants of these two studies were middle-older aged, and were primarily registered female nurses or male health professionals with similar socioeconomic status so the results may not be generalisable to young adults and other socioeconomic groups. A further limitation of previous studies is inadequate control for confounding, with some potential confounders not considered, such as social support and psychiatric diagnoses, which are imbalanced in different family structures[12] and causally associated with the maintenance and relapse of smoking,[13-15].

In the current research, we aimed to prospectively examine the associations among men and women in terms of partnering and parenting transitions and smoking continuity, cessation and relapse in a population-based national cohort of young adults.

2.3 Methods

2.3.1 Design and participants

Participants were from the CDAH study. It is a follow-up of 8,498 participants from the 1985 ASHFS, which comprised a nationally representative sample of Australian school children aged 7–15 years.[16] A two-stage probability sampling framework was used to achieve a nationally representative sample. The first stage was the selection of schools (government, Catholic, and independent) with a probability proportional to size (n=109, 90.1% response rate), and the second stage was the random sampling of 10 boys and girls from each age strata within schools (n=8,498, 67.5% response rate).

During 2002–2004, 6,840 participants were traced and 5,170 agreed to take part in the CDAH Study. The first follow-up was conducted from 2004 to 2006 (CDAH-1, herein referred to as ‘baseline’) where 3,948 participants (aged 26-36 years) completed questionnaires and 2,410 of those attended one of 34 study clinics held around Australia for physical

measurements. Five years later in 2009–2011, the second follow-up (CDAH-2, herein referred to as 'follow-up') collected data from 2,815 participants aged 31–41 years via telephone, mail or online survey.

At ASHFS, the directors of education in each state granted approval, and consent was obtained from children and parents. At CDAH 1 and 2, the study protocol was approved by the Southern Tasmanian Health and Medical Ethics Committee. Written informed consent was obtained from participants.

The analyses for this study included participants who were ever smokers at baseline, had data on smoking at follow-up, marital and parental status at both baseline and follow-up, socio-demographic factors and other covariates at baseline (n=1,084, **Figure 2-1**). Compared with current or former smokers who were not included in the analyses due to any aforementioned reason, study participants were more often females, had a higher body mass index (BMI) and education level, were more likely to be employed as professionals or managers, were more often married or living as married and had children at baseline (Table S1). There was no statistically significant difference between the two groups in baseline age.

2.3.2 Marital status and partnering transitions

Participants reported their current marital status at baseline and follow-up. Marital status was categorised into three groups: single, married/living as married, and separated/divorced/widowed. Partnering transitions were classified as: not partnered (married/living as married) both times, became partnered, stayed partnered both times, and became separated/divorced/widowed. The group of not partnered both times was used as the reference group in analyses. People who became separated/divorced/widowed were compared to those who stayed partnered.

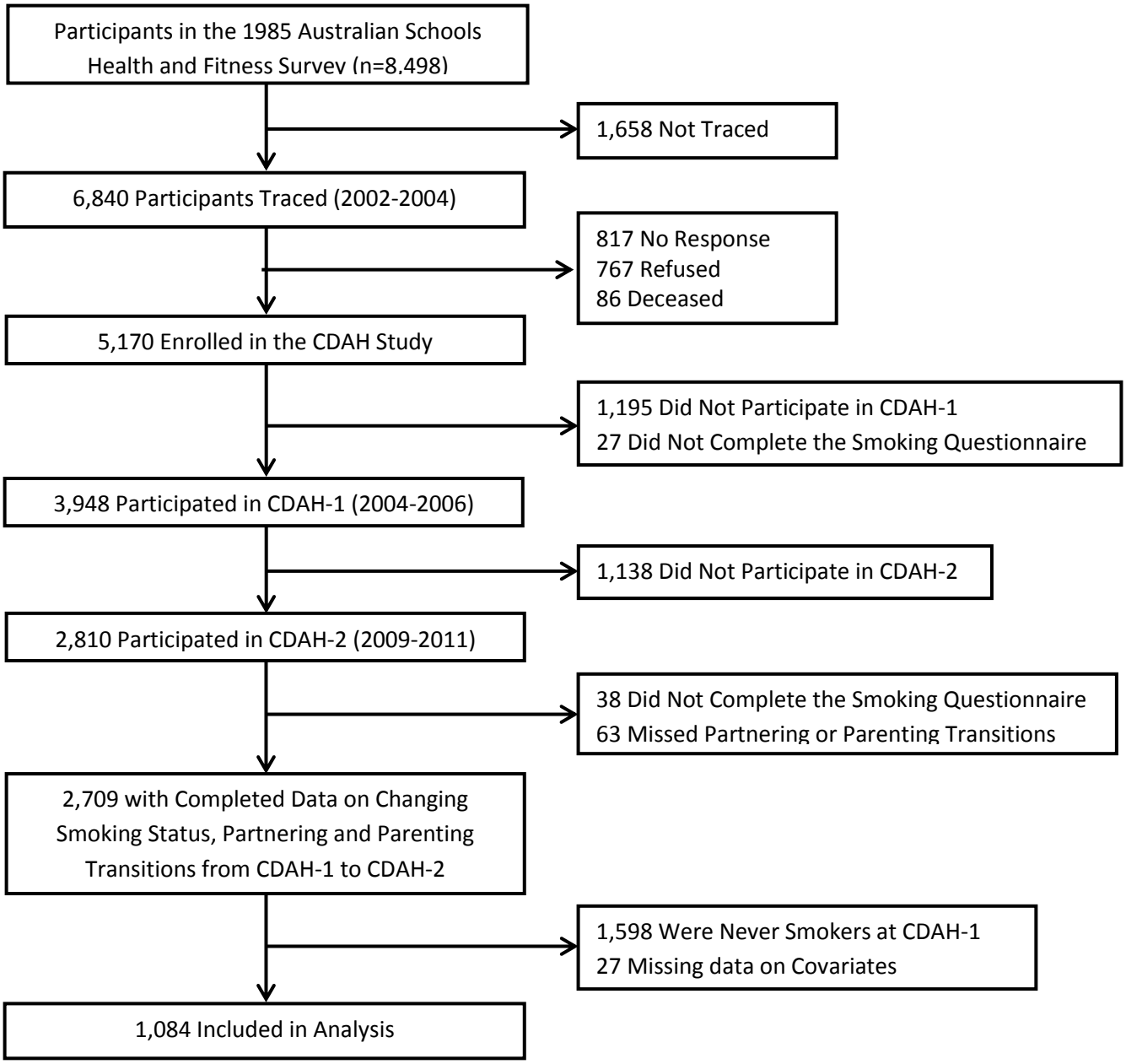


Figure 2-1 Flow chart of recruitment and retention of participants for Childhood Determinants of Adult Health study, Australia, 1985-2011.

2.3.3 Parental status and parenting transitions

At follow-up, participants reported how many biological children they had and the month and year of birth for each child. The date the participant completed the baseline questionnaire was used to determine whether each child had been born before or after the baseline assessment. Participants were then classified into four groups: no children both times, had first child born since baseline, had additional children born since baseline, and same number of children both times. If a participant had their first child plus additional children since baseline, they were classified as having a first child born since baseline. The group of no children both times was used as the reference group in analyses.

2.3.4 Smoking status assessment

Smoking status was defined according to the responses to two questions at baseline and follow-up. The first question asked "Over your lifetime, have you smoked at least 100 cigarettes, or a similar amount of tobacco?" Participants answering "yes" were classified as ever smokers, and those answering "no" as never smokers. Ever smokers were then asked the second question "How often do you now smoke cigarettes, cigars, pipes or any other tobacco products?" Participants who answered "not at all" were classified as former smokers, those who answered "daily" or "at least once a week" or "less than weekly" were classified as current smokers.

Analyses were restricted to ever smokers at baseline as the outcomes were quitting and resuming smoking during follow-up. Current smokers were dichotomised as continuing smokers and quitters. Former smokers were dichotomised into stable former smokers and resumed smokers.

2.3.5 Covariates

Socio-demographic information was self-reported at baseline, including age, sex, education and occupation. Accessibility/Remoteness Index of Australia (ARIA) classifications (residing in major city vs other) were assigned to participants based on the census collection district of their residential address. BMI was calculated from measured weight and height for most participants. A 15-item Index for Social Support assessed participants' perceptions and satisfaction with the social interaction available to them.[17] To control for poor health prior to beginning smoking, we used a question completed by participants in 1985 that asked "Is

your health usually?" with responses of "very good", "good", "average", "poor" and "very poor". Current psychological distress was determined from the mental component summary (MCS) measured by the SF-12.[18,19] Follow-up length was calculated from the dates the participant completed the baseline and follow-up questionnaires. Parenting transitions were considered as a covariate in the analyses of partnering transitions and vice versa.

2.3.6 Statistical analyses

Student t-tests and chi-square tests were used to compare differences in means and proportions, respectively. The log binomial regression model was used to estimate the associations of partnering and parenting transitions with quitting or resuming smoking.

Covariates were considered as potential confounders if they were causally related to the outcome, imbalanced between the exposure groups and caused a change of 10% or more in the effect estimate when included in a given regression model. Interactions between sex and partnering and parenting transitions on quitting or resuming smoking during follow-up were measured in multivariable models. We separated men and women for the analyses of quitting smoking because distinct sex differences were observed in the results ($p \leq 0.10$). The analyses of resuming smoking were not separated by sex as the p-values of interaction terms were >0.10.

Sensitivity analyses were conducted using inverse probability weighting (IPW) to examine the effects of loss to follow-up on the results [20]. IPW is a statistical technique for dealing with missing data. An additional probability model for non-missingness is constructed prior to the main analysis model using a binary regression method, for example logistic regression model. This model codes participants with missing values as 0 and observed as 1, any covariates potentially predictive of missingness are included in this model. The probability of being observed for each participant comes directly from the predicted values of the model. The inverse of this probability is then used as a weight in the main analysis model. Greater weight is given to people with a low probability of response. In this chapter, weights were determined by age, sex, education, marital status and whether having children.

All analyses were performed with STATA software, version 12.1 (Stata Corp, College Station, Texas 77845 USA). A two-tailed *P* value less than 0.05 was considered statistically significant.

2.4 Results

In total, 1,084 ever smokers (570 current smokers and 514 former smokers) at baseline were included in the analyses. **Table 2-1** shows their baseline socio-demographic and anthropometric characteristics. Overall, the mean age was 31.8 years, 40.4% were male, 27.8% were single and 68.3% were married or living as married; 46.1% did not have children. As compared with former smokers at baseline, current smokers tended to be younger (p=0.013) and more often male (p<0.001). They were less likely to have university education (p<0.001), to be employed as professionals/managers (p=0.003), to be married/living as married (p<0.001) and to have children (p<0.001). No significant difference was observed between the groups in baseline BMI.

Table 2-1 Baseline socio-demographic and anthropometric characteristics of participants in the Childhood Determinants of Adult Health Study, Australia, 2004–2006

Characteristic	Total (n = 1,084)	Former smokers (n=514)	Current smokers (n=570)
Age (years), Mean (SD)	31.8 (2.7)	32.0 (2.6)	31.6 (2.7)
Male, % (n)	40.4 (438)	33.6 (173)	46.5 (265)
BMI (kg/m^2), Mean (SD)*	25.2 (4.8)	25.1 (4.8)	25.2 (4.8)
Education, % (n)			
Any university education	30.0 (325)	35.8 (184)	24.7 (141)
Vocational training	32.9 (357)	33.3 (171)	32.6 (186)
High school only	37.1 (402)	30.9 (159)	42.6 (243)
Occupation*			
Professional or manager	46.3 (393)	50.1 (198)	43.0 (195)
Non-manual	21.9 (186)	22.0 (87)	21.8 (99)
Manual	18.0 (153)	12.9 (51)	22.5 (102)
Not in the workforce	13.8 (117)	14.9 (59)	12.8 (58)
Marital status, % (n)			
Single	27.8 (301)	19.1 (98)	35.6 (203)
Married/living as married	68.3 (740)	78.8 (405)	58.8 (335)
Separated/divorced/Widowed	4.0 (43)	2.1 (11)	5.6 (32)
No children, % (n)	46.1 (500)	39.3 (202)	52.3 (298)

BMI: body mass index; SD: standard divination.
* Sample size ranged from 849 to 1008.

During 5 years' follow-up, 233 out of 570 current smokers at baseline quit smoking and 337 continued; 58 out of 514 former smokers at baseline resumed smoking and 456 sustained cessation.

The risk of quitting smoking relative to continuing smoking, and resuming smoking relative to remaining quit, from baseline to follow-up by partnering transitions is documented in

Table 2-2 and **Figure 2-2**. There was an interaction between sex and partnering transitions in the analyses of quitting smoking (p=0.078). For current smokers at baseline, after adjustment for age, education, parenting transitions and follow-up length, the likelihood of quitting smoking was 184% higher for men and 50% higher for women who became partnered than those who were not partnered both times. A significant sex difference was observed in the effect of staying partnered on quitting smoking. Compared with those who were not partnered both times, men who stayed partnered reported a 112% greater probability of quitting smoking, while no significant difference was shown among women. Among former smokers at baseline, relative to those who were not partnered both times, the risk of resuming smoking was statistically significantly lower for those who became and stayed partnered.

Table 2-2 Relative risk (95% CI) of quitting smoking relative to continuing smoking, and resuming smoking relative to remaining quit, by partnering transitions

Partnering transitions	Number (%) with outcome	Unadjusted		Adjusted*	
		RR	95% CI	RR	95% CI
Current smokers at baseline - males (n=265)					
Not partnered both times	12/59 (20.3)	Ref.		Ref.	
Became partnered	31/55 (56.4)	2.77	1.59, 4.83	2.84	1.62, 4.98
Stayed partnered both times	59/141 (41.8)	2.06	1.20, 3.53	2.12	1.18, 3.80
Became separated/divorced/widowed	2/10 (20.0)	0.98	0.26, 3.75	1.01	0.26, 3.96
Current smokers at baseline - females (n=305)					
Not partnered both times	26/65 (40.0)	Ref.		Ref.	
Became partnered	32/56 (57.1)	1.43	0.98, 2.08	1.50	1.03, 2.18
Stayed partnered both times	64/161 (39.8)	0.99	0.70, 1.41†	1.13	0.80, 1.62
Became separated/divorced/widowed	7/23 (30.4)	0.76	0.38, 1.51	0.83	0.42, 1.62
Former smokers at baseline (n=514)					
Not partnered both times	11/49 (22.5)	Ref.		Ref.	
Became partnered	2/60 (3.3)	0.15	0.03, 0.64	0.14	0.03, 0.58
Stayed partnered both times	40/381 (10.5)	0.47	0.26, 0.85	0.51	0.27, 0.95
Became separated/divorced/widowed	5/24 (20.8)	0.93	0.36, 2.37	0.95	0.38, 2.40

Values in bold denote statistically significant results.
CI: confidence interval; RR: relative risk.
*Adjusted for baseline age, sex (only in resuming smoking analyses), education, parenting transitions and follow-up length.

Compared with those who stayed partnered, those who became separated/divorced/widowed from baseline to follow-up showed higher risk of continuing and resuming smoking, but these differences did not reach the statistical significance (data not shown).

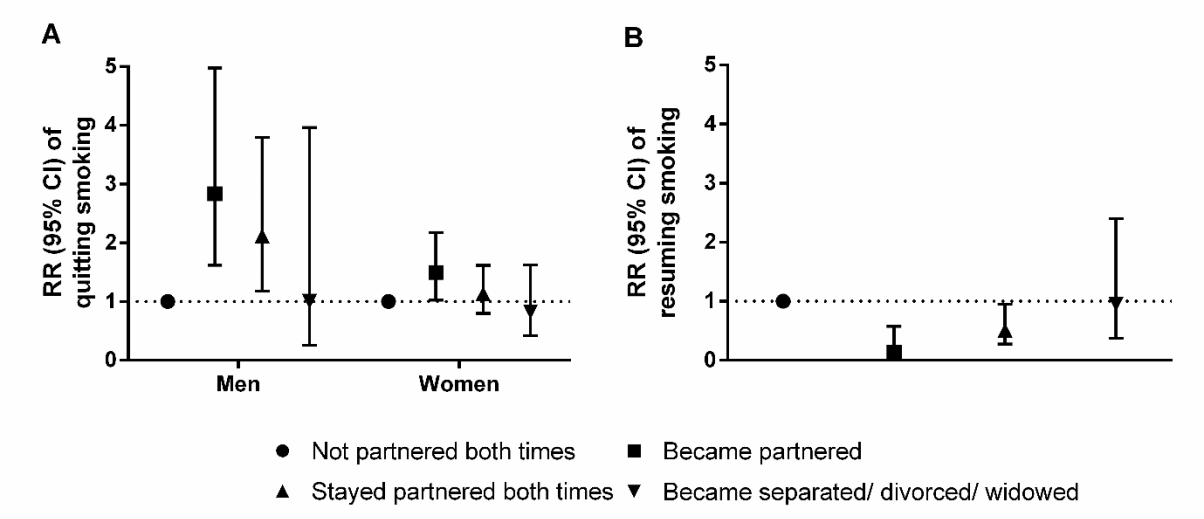


Figure 2-2 Relative risk (95% CI) of (A) quitting smoking relative to continuing smoking, and (B) resuming smoking relative to remaining quit, by partnering transitions. Adjusted for baseline age, sex (only in resuming smoking analyses), education, follow-up length

Among former smokers at baseline, no statistically significant difference was found between parenting transitions and whether resuming smoking from baseline to follow-up.

Table 2-3 and **Figure 2-3** presents the association of parenting transitions and continuity and relapse of smoking during follow-up. An interaction was present between sex and parenting transitions in the analyses of quitting smoking (p=0.072). Among female current smokers at baseline, compared with those who were childless both times, those who had a first child born since baseline were more likely to quit smoking in a multivariable model. Higher probabilities of quitting smoking were also evident when compared with women who had additional children since baseline (RR: 2.57, 95% CI: 1.57, 4.21) and women who had the same number of children at both time points (RR: 2.30, 95% CI: 1.62, 3.26).

Among former smokers at baseline, no statistically significant difference was found between parenting transitions and whether resuming smoking from baseline to follow-up.

Table 2-3 Relative risk (95% CI) of quitting smoking relative to continuing smoking, and resuming smoking relative to remaining quit, by parenting transitions

Parenting transitions	Number (%) with outcome	Unadjusted		Adjusted*	
		RR	95% CI	RR	95% CI
Current smokers at baseline - males (n=265)					
No children both times	36/98 (36.7)	Ref.		Ref.	
First child born since baseline	23/52 (44.2)	1.20	0.81, 1.80	1.05	0.68, 1.62
Additional children born since baseline	20/50 (40.0)	1.09	0.71, 1.67	0.97	0.58, 1.60
Same number of children both times	25/65 (38.5)	1.05	0.70, 1.57	0.96	0.60, 1.53
Current smokers at baseline - females (n=305)					
No children both times	46/103 (44.7)	Ref.		Ref.	
First child born since baseline	33/44 (75.0)	1.68	1.28, 2.21†	1.74	1.30, 2.33†
Additional children born since baseline	14/48 (29.2)	0.65	0.40, 1.07	0.68	0.41, 1.13
Same number of children both times	36/110 (32.7)	0.73	0.52, 1.03	0.76	0.53, 1.09
Former smokers at baseline (n=514)					
No children both times	15/104 (14.4)	Ref.		Ref.	
First child born since baseline	10/98 (10.2)	0.71	0.33, 1.50	0.68	0.31, 1.49
Additional children born since baseline	17/141 (12.1)	0.84	0.44, 1.60	1.00	0.47, 2.11
Same number of children both times	16/171 (9.4)	0.65	0.33, 1.26	0.82	0.38, 1.78

Values in bold denote statistically significant results.
CI: confidence interval; RR: relative risk.
* Adjusted for baseline age, sex (only in resuming smoking analyses), education, partnering transitions and follow-up length.
† Statistically significant difference compared with people who had additional children born since baseline and people who had the same of number of children.

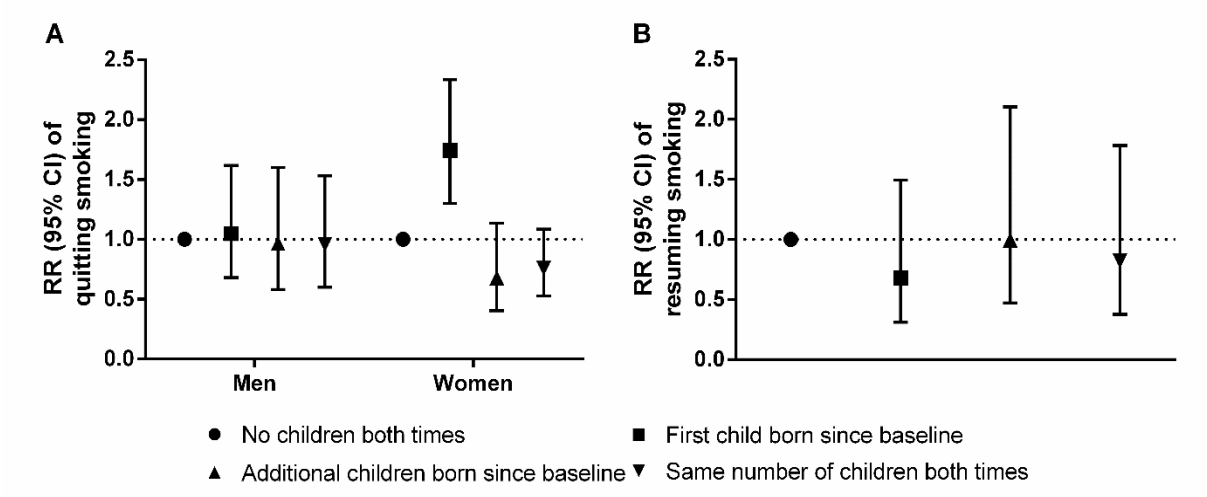


Figure 2-3 Relative risk (95% CI) of (A) quitting smoking relative to continuing smoking, and (B) resuming smoking relative to remaining quit, by parenting transitions. Adjusted for baseline age, sex (only in resuming smoking analyses), education, follow-up length and partnering transitions.

Sensitivity analyses conducted using IPW showed similar findings as the unweighted analyses (**Table 2-4** and **Table 2-5**).

Table 2-4 Weighted relative risk (95% CI) of quitting smoking relative to continuing smoking, and resuming smoking relative to remaining quit, by partnering transitions

Partnering transitions	Number (%) with outcome	Unadjusted		Adjusted*	
		RR	95% CI	RR	95% CI
Current smokers at baseline - male (n=265)					
Not partnered both times	12/59 (20.3)	Ref.		Ref.	
Became partnered	31/55 (56.4)	2.71	1.53, 4.81	2.81	1.57, 5.01
Stayed partnered both times	59/141 (41.8)	2.02	1.16, 3.52	2.09	1.15, 3.82
Became separated/divorced/widowed	2/10 (20.0)	0.99	0.26, 3.85	1.04	0.27, 4.06
Current smokers at baseline - female (n=305)					
Not partnered both times	26/65 (40.0)	Ref.		Ref.	
Became partnered	32/56 (57.1)	1.56	1.03, 2.36	1.63	1.08, 2.44
Stayed partnered both times	64/161 (39.8)	1.07	0.73, 1.58	1.23	0.85, 1.80
Became separated/divorced/widowed	7/23 (30.4)	0.82	0.39, 1.71	0.90	0.44, 1.86
Former smokers at baseline (n=514)					
Not partnered both times	11/49 (22.5)	Ref.		Ref.	
Became partnered	2/60 (3.3)	0.14	0.03, 0.62	0.13	0.03, 0.56
Stayed partnered both times	40/381 (10.5)	0.51	0.27, 0.95	0.56	0.30, 1.06
Became separated/divorced/widowed	5/24 (20.8)	0.94	0.35, 2.52	0.95	0.36, 2.51

Values in bold denote statistically significant results.
CI: confidence interval; RR: relative risk.
*Adjusted for baseline age, sex (only in resuming smoking analyses), education, parenting transitions and follow-up length.

Table 2-5 Weighted relative risk (95% CI) of quitting smoking relative to continuing smoking, and resuming smoking relative to remaining quit, by parenting transitions

Parenting transitions	Number (%) with outcome	Unadjusted		Adjusted*	
		RR	95% CI	RR	95% CI
Current smokers at baseline - male (n=265)					
No children both times	36/98 (36.7)	Ref.		Ref.	
First child born since baseline	23/52 (44.2)	1.22	0.81, 1.85	1.06	0.68, 1.66
Additional children born since baseline	20/50 (40.0)	1.16	0.75, 1.78	1.01	0.60, 1.71
Same number of children both times	25/65 (38.5)	1.05	0.69, 1.59	0.93	0.58, 1.50
Current smokers at baseline - female (n=305)					
No children both times	46/103 (44.7)	Ref.		Ref.	
First child born since baseline	33/44 (75.0)	1.69	1.28, 2.24†	1.72	1.29, 2.31†
Additional children born since baseline	14/48 (29.2)	0.66	0.40, 1.08	0.65	0.39, 1.10
Same number of children both times	36/110 (32.7)	0.72	0.51, 1.03	0.73	0.51, 1.05
Former smokers at baseline (n=514)					
No children both times	15/104 (14.4)	Ref.		Ref.	
First child born since baseline	10/98 (10.2)	0.61	0.28, 1.33	0.58	0.26, 1.27
Additional children born since baseline	17/141 (12.1)	0.89	0.46, 1.72	1.06	0.50, 2.26
Same number of children both times	16/171 (9.4)	0.62	0.32, 1.23	0.80	0.37, 1.69

Values in bold denote statistically significant results.
CI: confidence interval; RR: relative risk.
* Adjusted for baseline age, sex (only in resuming smoking analyses), education, partnering transitions and follow-up length.
† Statistically significant difference compared with people who had additional children born since baseline and people who had the same of number of children.

2.5 Discussion

In this longitudinal study of young Australian adults, we found becoming or staying partnered significantly increased the probability of quitting smoking and decreased the risk of resuming smoking during 5 years' follow-up. Some protective effects were stronger among men than women. Marriage or partnership termination was associated with higher risk of continuity and relapse of smoking, but these associations were not statistically significant and should be interpreted with caution. Regarding parenting transitions, we found compared with those staying childless, having a first child significantly increased the likelihood of quitting smoking among women, but not among men, whereas having additional children did not. To our knowledge, this is the first longitudinal study examining the associations between partnering and parenting transitions and continuity or relapse of smoking by sex in young adults.

Our finding that becoming partnered, relative to remaining not partnered, was associated with a higher probability of quitting smoking and a lower risk of resuming smoking concurs with previous longitudinal studies among young women[9,21] and middle aged or elderly women.[11] We observed greater benefits among young men. Men who stayed partnered were 112% more likely to quit smoking than their continuously not partnered peers. This is the first longitudinal population-based study on the relationship between partnering transitions and smoking cessation reported by young men. The health benefits of marriage or partnership on quitting smoking differed between sex, with greater benefits among young men than women, which may be explained by different family roles of men and women. Women have traditionally acted as the primary family caregivers,[22] and the social support husbands gain from their wives may be greater than the support wives gain from husbands.[23,24] Another possible explanation could be women's greater encouragement of regulatory health behaviours and prevention practices benefiting their spouses.[25] The lower prevalence of smoking among young women than men may also be a reason. According to the latest report from the AIHW, females were less likely to have smoked at any frequency than males,[26] and living with non-smoking partners has been reported to significantly increase smokers' quitting attempts[27] and success.[28]

Compared to staying married or partnered, a shift from married or partnered to being un-partnered through separation, divorce or becoming widowed increased, although not statistically significantly, the risks of continuity and relapse of smoking, which is consistent with previous reports.[10,11,29] In a cohort study of male health professionals aged 40-75[10] and a national panel survey[29] from United States, the break up of a marriage was associated with increased daily cigarette consumption relative to those that stayed married. Greater social support from spouses or partners[30,31] and the development of more concern with health behaviours are the two suggested reasons for the positive impacts of marriage or partnership[32] on changing smoking status. These may disappear after marriage or partnership dissolution and increase in stress,[33] which may lead to heavier consumption of cigarettes and continuity or relapse of smoking.

There were significant sex differences in the relationship between parental transitions and quitting smoking. Having a first child born since baseline significantly increased women's likelihood of quitting smoking relative to remaining childless, while no significant association was found among young men. To the best of our knowledge, this is the first longitudinal study determining the effects of transition into parenthood on smoking cessation in both men and women. This result is perhaps not surprising given the well-known health consequences of maternal smoking to babies and women themselves[34] and was supported by Tucker and colleagues' finding that the likelihood of making a quit attempt was greater among young women than men when moving into parenthood.[27] Given the detrimental effects of exposure of children to any parental smoking on vascular health[35-37] and lung function,[38,39] there seems to be a need for more emphasis on the harmful effects of paternal smoking as well.

So far, few prospective longitudinal studies have examined the relationship between the transition into motherhood and smoking cessation in young women[40] and the existing evidence has been conflicting[9,21,41,42] but mainly suggests a positive association except for certain groups, such as single[43,44] or younger mothers.[42] Women may choose to quit smoking during and after pregnancy primarily because of concerns about the baby's health,[45] thus providing a special widow of opportunity to promote smoking cessation. A review has demonstrated the efficacy of behavioural interventions assisting pregnant

smokers to quit.[46] However, in our study this protective effect was absent among young women who had additional children born during follow-up. This interesting finding was consistent with previous research that showed smoking cessation during and after pregnancy was inversely associated with parity, with multiparous women quitting smoking less frequently than primiparous women.[47,48] One possible explanation is that women who have an experience of smoking during a previous pregnancy and giving birth to a 'healthy' baby are less motivated to quit smoking in subsequent pregnancies.[49] It is also possible that women having additional children are more resistant to quitting smoking as a result of higher nicotine dependence and they may represent a group who were unable to quit after their first child was born. If they cannot quit smoking during their first pregnancy, it is less likely to happen in future pregnancies. This hypothesis was partly supported by our further analyses. We found that women having additional children were more likely to be daily smokers at baseline than those having a first child during follow-up (77.5% versus 56.6%, p=0.006).

The public health implications of these findings lie in the opportunities highlighted for future research to inform tobacco control initiatives and public health campaigns. These may include strengthening the messages regarding the importance and potentially beneficial influences of partners' smoking behaviour. This could include implementing programmes to help non-smokers support their partners to stop smoking and discourage relapse. Regarding younger parents there may be a need for more emphasis on multiparous smokers and young male smokers who transit into parenthood within maternity care and paediatric setting. In addition, there is an increasing number of people choosing to remain unpartnered and childless in society [50,51]. Few studies have been performed to investigate interventions to decrease the greater risk of continuing smoking in these groups. Examinations of why they are at greater risk and the mechanisms around social networks, peers and families would be useful.

Several limitations should be acknowledged in the interpretation of our results. First, approximately one-third of participants at baseline were lost to follow-up. Comparison between the follow-up group and those lost to follow-up on some socio-demographic characteristics revealed that non-respondents were more often males, less educated, less

employed as professionals or managers, less often married or living as married and less likely to have children at baseline. This may lead to an over- or under-estimation of the associations. Applying inverse probability weights to account for these differences produced similar results, suggesting this is not a major source of bias. Using self-reported smoking status, albeit through a standard questionnaire at baseline and follow-up, is the second possible limitation. The latest meta-analysis including 67 studies showed a trend of underestimation when comparing self-reported smoking status with smoking status determined through measures of cotinine in biological fluids.[52] Third, smoking status was divided only into three categories – never, former and current smokers. It would be useful to split current smokers into more groups according to smoking characteristics, such as frequency of smoking. This would allow an investigation of the impact of partnering and parenting transitions on moving from occasional to daily smoking and vice versa. Further research is needed in this area. Fourth, smoking trajectories during follow-up were unclear in that some participants may have quit and resumed smoking repeatedly. Success in prolonged smoking abstinence (≥ 6 months) tends to be low at only 3-5% after a given quit attempt,[53] therefore, it is likely that we had some misclassification of point prevalent and continuous abstainers. Fifth, people who had more than one child born during follow-up were categorised into the group of having first child born since baseline, which would underestimate the benefits of the initial transition into parenthood on smoking cessation.

Strengths of this study include its large sample size, population based cohort design, consideration of a range of potential confounders, having reasonable follow-up rate between CDAH 1 and 2, applying technique to account for possible bias due to loss to follow-up, and ability to explore the effects among young men and women separately. Furthermore, the mean age of our participants is very similar with the age people normally make partnering and parenting transitions.

2.6 Conclusion

In summary, transitions into relationships with a partner and parenthood are associated with beneficial changes in smoking behaviour, but they influence young men and women differently. The benefits of entering partnered relationships were greater among men than women, and the transition into parenthood was of greater benefit to women than men.

2.7 Postscript

The findings from this chapter showed the different effects of partnering and parenting transitions on continuity or relapse of smoking in young men and women. The determination of another important dynamic factor – SEP variation over life course on smoking behaviour and the underlying mechanisms are examined in Chapter 3.

2.8 References

1. Australian Bureau of Statistics. *Marriages and divorces, Australia, 2013 (ABS cat. No. 3310.0)*. Canberra: 2013. http://www.abs.gov.au/ausstats/abs@.nsf/Latestproducts/3310.0Main%20Features112013?opendocument&tabname=Summary&prodno=3310.0&issue=2013&num=&view=. Accessed 1 July 2015.
2. Australian Institute of Health and Welfare. *Australia's mothers and babies 2013—in brief. Perinatal statistics series no. 31. Cat no. PER 72.* Canberra: 2015. http://www.abs.gov.au/ausstats/abs@.nsf/Latestproducts/3301.0Main%20Features32013?opendocument&tabname=Summary&prodno=3301.0&issue=2013&num=&view=. Accessed 1 July 2015.
3. Raymore LA, Barber BL, Eccles JS. Leaving home, attending college, partnership and parenthood: The role of life transition events in leisure pattern stability from adolescence to young adulthood. *J Youth Adolesc.* 2001;30(2):197-223.
4. Wheaton B. Life transitions, role histories, and mental health. *Am Sociol Rev.* 1990:209-223.
5. World Health Organization. *Tobacco Fact Sheet No. 339*. Geneva: Media Centre WHO; 2013. http://www.who.int/mediacentre/factsheets/fs339/en/. Accessed 1 October 2015.
6. Australian Institute of Health and Welfare. *National Drug Strategy Household Survey 2013: data and references.* 2013. http://www.aihw.gov.au/alcohol-and-other-drugs/ndshs/2013/data-and-references/. Accessed 26 June 2015.
7. Banks E, Joshy G, Weber MF, et al. Tobacco smoking and all-cause mortality in a large Australian cohort study: findings from a mature epidemic with current low smoking prevalence. *BMC Med.* 2015;13:38. doi:10.1186/s12916-015-0281-z.
8. U.S. Department of Health and Human Services. *Preventing Tobacco Use Among Youth and Young Adults: A Report of the Surgeon General*. Atlanta, GA: U.S.: 2012. http://www.surgeongeneral.gov/library/reports/preventing-youth-tobacco-use/index.html. Accessed March 15 2016.
9. McDermott L, Dobson A, Owen N. Determinants of continuity and change over 10 years in young women's smoking. *Addiction.* 2009;104(3):478-487. doi:10.1111/j.1360-0443.2008.02452.xADD2452 [pii].
10. Eng PM, Kawachi I, Fitzmaurice G, Rimm EB. Effects of marital transitions on changes in dietary and other health behaviours in US male health professionals. *J Epidemiol Community Health.* 2005;59(1):56-62. doi:59/1/56 [pii]10.1136/jech.2004.020073.
11. Lee S, Cho E, Grodstein F, Kawachi I, Hu FB, Colditz GA. Effects of marital transitions on changes in dietary and other health behaviours in US women. *Int J Epidemiol.* 2005;34(1):69-78. doi:10.1093/ije/dyh258dyh258 [pii].
12. Shapiro A, Keyes CLM. Marital status and social well-being: are the married always better off? *Soc Indic Res.* 2008;88:329–346.
13. Diemert LM, Bondy SJ, Brown KS, Manske S. Young adult smoking cessation: predictors of quit attempts and abstinence. *Am J Public Health.* 2013;103(3):449-453. doi:10.2105/AJPH.2012.300878.
14. Vangeli E, Stapleton J, Smit ES, Borland R, West R. Predictors of attempts to stop smoking and their success in adult general population samples: a systematic review. *Addiction.* 2011;106(12):2110-2121. doi:10.1111/j.1360-0443.2011.03565.x.

15. Mendel JR, Berg CJ, Windle RC, Windle M. Predicting young adulthood smoking among adolescent smokers and nonsmokers. *Am J Health Behav.* 2012;36(4):542-554. doi:10.5993/AJHB.36.4.11.
16. Gall SL, Jose K, Smith K, Dwyer T, Venn A. The Childhood Determinants of Adult Health Study: a profile of a cohort study to examine the childhood influences on adult cardiovascular health. *Australas epidemiol.* 2009;16(1):35.
17. Henderson S, Duncan-Jones P, McAuley H, Ritchie K. The patient's primary group. *Br J Psychiatry.* 1978.
18. Ware Jr JE, Kosinski M, Keller SD. A 12-Item Short-Form Health Survey: construction of scales and preliminary tests of reliability and validity. *Med Care.* 1996;34(3):220-233.
19. Gill SC, Butterworth P, Rodgers B, Mackinnon A. Validity of the mental health component scale of the 12-item Short-Form Health Survey (MCS-12) as measure of common mental disorders in the general population. *Psychiatry Res.* 2007;152(1):63-71.
20. Mansournia MA, Altman DG. Inverse probability weighting. *BMJ.* 2016;352:i189. doi:10.1136/bmj.i189.
21. McDermott L, Dobson A, Russell A. Changes in smoking behaviour among young women over life stage transitions. *Aust N Z J Public Health.* 2004;28(4):330-335.
22. Lee C. Health, Stress and Coping among Women Caregivers: A Review. *J Health Psychol.* 1999;4(1):27-40. doi:10.1177/135910539900400104.
23. Umberson D, Chen MD, House JS, Hopkins K, Slaten E. The effect of social relationships on psychological well-being: Are men and women really so different? *Am Sociol Rev.* 1996:837-857.
24. House JS, Landis KR, Umberson D. Social relationships and health. *Science.* 1988;241(4865):540-545.
25. Umberson D. Family status and health behaviors: Social control as a dimension of social integration. *J Health Soc Behav.* 1987:306-319.
26. Australian Institute of Health and Welfare. *National Drug Strategy Household Survey detailed report: 2013. Drug statistics series no. 28. Cat. no. PHE 183*. Canberra: AIHW: 2014. http://www.aihw.gov.au/publication-detail/?id=60129549469. Accessed 8 April 2016.
27. Tucker JS, Ellickson PL, Orlando M, Klein DJ. Predictors of attempted quitting and cessation among young adult smokers. *Prev Med.* 2005;41(2):554-561. doi:10.1016/j.ypmed.2004.12.002.
28. Manchon Walsh P, Carrillo P, Flores G, Masuet C, Morchon S, Ramon JM. Effects of partner smoking status and gender on long term abstinence rates of patients receiving smoking cessation treatment. *Addict Behav.* 2007;32(1):128-136. doi:10.1016/j.addbeh.2006.03.027.
29. Umberson D. Gender, marital status and the social control of health behavior. *Soc Sci Med.* 1992;34(8):907-917.
30. Waldron I, Lye D. Family roles and smoking. *Am J Prev Med.* 1989;5(3):136-141.
31. Mermelstein R, Cohen S, Lichtenstein E, Baer JS, Kamarck T. Social support and smoking cessation and maintenance. *J Consult Clin Psychol.* 1986;54(4):447-453.
32. Backett KC, Davison C. Lifecourse and lifestyle: the social and cultural location of health behaviours. *Soc Sci Med.* 1995;40(5):629-638.

33. McDermott LJ, Dobson AJ, Owen N. From partying to parenthood: young women's perceptions of cigarette smoking across life transitions. *Health Educ Res.* 2006;21(3):428-439. doi:cyl041 [pii]10.1093/her/cyl041.
34. US Department of Health and Human Services. *The health consequences of smoking—50 years of progress. A report of the Surgeon General*. Atlanta, GA: 2014. http://www.surgeongeneral.gov/library/reports/50-years-of-progress/. Accessed 8 April 2016.
35. Gall S, Huynh QL, Magnussen CG, et al. Exposure to parental smoking in childhood or adolescence is associated with increased carotid intima-media thickness in young adults: evidence from the Cardiovascular Risk in Young Finns study and the Childhood Determinants of Adult Health Study. *Eur Heart J.* 2014;35(36):2484-2491. doi:10.1093/eurheartj/ehu049.
36. West HW, Juonala M, Gall SL, et al. Exposure to parental smoking in childhood is associated with increased risk of carotid atherosclerotic plaque in adulthood: the Cardiovascular Risk in Young Finns Study. *Circulation.* 2015;131(14):1239-1246. doi:10.1161/CIRCULATIONAHA.114.013485.
37. Juonala M, Magnussen CG, Venn A, et al. Parental smoking in childhood and brachial artery flow-mediated dilatation in young adults: the Cardiovascular Risk in Young Finns study and the Childhood Determinants of Adult Health study. *Arterioscler Thromb Vasc Biol.* 2012;32(4):1024-1031. doi:10.1161/ATVBAHA.111.243261.
38. Svanes C, Omenaas E, Jarvis D, Chinn S, Gulsvik A, Burney P. Parental smoking in childhood and adult obstructive lung disease: results from the European Community Respiratory Health Survey. *Thorax.* 2004;59(4):295-302.
39. David GL, Koh WP, Lee HP, Yu MC, London SJ. Childhood exposure to environmental tobacco smoke and chronic respiratory symptoms in non-smoking adults: the Singapore Chinese Health Study. *Thorax.* 2005;60(12):1052-1058. doi:10.1136/thx.2005.042960.
40. Cengelli S, O'Loughlin J, Lauzon B, Cornuz J. A systematic review of longitudinal population-based studies on the predictors of smoking cessation in adolescent and young adult smokers. *Tob Control.* 2012;21(3):355-362. doi:10.1136/tc.2011.044149.
41. Chen PH, White HR, Pandina RJ. Predictors of smoking cessation from adolescence into young adulthood. *Addict Behav.* 2001;26(4):517-529.
42. Tucker JS, Ellickson PL, Klein DJ. Smoking cessation during the transition from adolescence to young adulthood. *Nicotine Tob Res.* 2002;4(3):321-332. doi:10.1080/14622200210142698.
43. Baron R, Mannien J, de Jonge A, et al. Socio-demographic and lifestyle-related characteristics associated with self-reported any, daily and occasional smoking during pregnancy. *PLoS One.* 2013;8(9):e74197. doi:10.1371/journal.pone.0074197.
44. Siahpush M, Shaikh RA, Tibbits M, Huang TT, Singh GK. The association of lone-motherhood with smoking cessation and relapse: prospective results from an Australian national study. *Int J Environ Res Public Health.* 2013;10(7):2906-2919. doi:10.3390/ijerph10072906.
45. Orleans CT, Barker DC, Kaufman NJ, Marx JF. Helping pregnant smokers quit: meeting the challenge in the next decade. *Tob Control.* 2000;9 Suppl 3:III6-11.
46. Patnode CD, Henderson JT, Thompson JH, Senger CA, Fortmann SP, Whitlock EP. Behavioral Counseling and Pharmacotherapy Interventions for Tobacco Cessation in

Adults, Including Pregnant Women: A Review of Reviews for the US Preventive Services Task Force. *Ann Intern Med.* 2015;163(8):608-621.
47. Lu Y, Tong S, Oldenburg B. Determinants of smoking and cessation during and after pregnancy. *Health Promot Int.* 2001;16(4):355-365.
48. Palma S, Perez-Iglesias R, Pardo-Crespo R, Llorca J, Mariscal M, Delgado-Rodriguez M. Smoking among pregnant women in Cantabria (Spain): trend and determinants of smoking cessation. *BMC Public Health.* 2007;7:65. doi:10.1186/1471-2458-7-65.
49. Giglia RC, Binns CW, Alfonso HS. Which women stop smoking during pregnancy and the effect on breastfeeding duration. *BMC Public Health.* 2006;6:195. doi:10.1186/1471-2458-6-195.
50. Rowland D, Merlo R. The prevalence of childlessness in Australia. *People and Place.* 2000;8(2):21.
51. Boddington B, Didham R. Increases in childlessness in New Zealand. *Journal of Population Research.* 2009;26(2):131-151.
52. Connor Gorber S, Schofield-Hurwitz S, Hardt J, Levasseur G, Tremblay M. The accuracy of self-reported smoking: a systematic review of the relationship between self-reported and cotinine-assessed smoking status. *Nicotine Tob Res.* 2009;11(1):12-24. doi:10.1093/ntr/ntn010.
53. Hughes JR, Keely J, Naud S. Shape of the relapse curve and long-term abstinence among untreated smokers. *Addiction.* 2004;99(1):29-38.

Table S1 Comparison of baseline characteristics of participants and non-participants*

Characteristic	Participants (n = 1,084)	Non-participants (n = 783)	P-value
Age (years), Mean (SD)	31.8 (2.7)	32.1 (2.7)	0.050
Male, % (n)	40.4 (438)	50.2 (400)	0.000
Body mass index (kg/m^2), Mean (SD)	25.2 (4.8)	25.9 (7.9)	0.027
Education, % (n)			0.001
Any university education	30.0 (325)	22.4 (174)	
Vocational training	32.9 (357)	38.6 (300)	
High school only	37.1 (402)	39.1 (304)	
Occupation			0.011
Professional or manager	46.3 (393)	42.5 (183)	
Non-manual	21.9 (186)	19.0 (82)	
Manual	18.0 (153)	26.0 (112)	
Not in the workforce	13.8 (117)	12.5 (54)	
Marital status, % (n)			0.031
Single	27.8 (301)	31.2 (241)	
Married/living as married	68.3 (740)	63.0 (487)	
Separated/divorced/Widowed	4.0 (43)	5.8 (45)	
None children, % (n)	46.1 (500)	55.4 (98)	0.022

SD, standard deviation.

* Sample size varied because of missing data (range 849-1,084 for participants, and 177-783 for non-participants).

Chapter 3

Socioeconomic position over the life course and smoking status in mid-adulthood: results from a 25-year follow-up study

Chapter 3 Socioeconomic position over the life course and smoking status in mid-adulthood: results from a 25-year follow-up study

3.1 Preface

Tobacco smoking disproportionately affects low SEP groups, but its effect over the life course from childhood is unclear. Various theoretical life course models have been proposed and ideally, multiple SEP measures and multiple life course models should be examined in one study sample. However, no study has investigated which life course model best describes the association of SEP over the life course and smoking status in mid-adulthood. This chapter aims to address this gap and further investigate the underlying mechanisms linking these two factors.

3.2 Introduction

As discussed in detail in the Introduction, tobacco smoking (including second-hand smoke) is one of the biggest threats to public health the world has ever faced, killing approximately six million people each year. Nearly half of smokers worldwide will die prematurely from tobacco-related diseases, including cancer of several organs, CVD, respiratory disease and other conditions [1]. It is the second leading risk factor for global disease burden and injury, accounting for 6.3% of global DALYs in 2010 [2].

SEP describes the place that a person or group occupies in the structure of society [3]. There are several indicators of SEP, including education, income, occupation, housing tenure, car availability and neighbourhood deprivation [4,5]. Tobacco smoking disproportionately affects low SEP groups. In most countries at the final stage of the tobacco epidemic [6], such as Australia, New Zealand, the UK, the USA, Canada and many countries in the European Union, people in low SEPs have higher prevalence of smoking than high SEP groups, and are more likely to be heavy smokers and exposed to second-hand smoke [7-9]. Childhood socioeconomic disadvantage is positively associated with adulthood smoking [10]. Tobacco smoking varies between different socioeconomic trajectories from childhood to adulthood. Using data from 1,103 young adults (mean age 28.9 years) in France, Bowes and colleagues found that people whose SEP declined from childhood to adulthood were two times likely to smoke compared with those with stable high trajectory [11]. A cumulative effect of disadvantage is also evident in these countries, with the smoking prevalence in people

facing multiple disadvantages (defined by multiple criteria) five times that in the most affluent [12,13].

In the past few decades, there has been an overall downward trend in smoking prevalence across most demographic groups in countries with advanced tobacco control programs [14,15]. However, the declines are generally greater in less disadvantaged groups. This contributes to a widening disparity in smoking between various SEP groups [14,16-18], which will keep rising with the trend toward greater socioeconomic inequality [19]. This disparity provides an opportunity for tobacco companies to sustain sales in declining markets. Their efforts targeting low SEP communities have been reported [20].

Understanding whether SEP at different life stages differentially impacts later smoking and the underlying mechanisms is important as it may help to develop policies to reduce the high prevalence in low SEP groups. Various theoretical life course models have been proposed to test different hypotheses about the relationship between SEP and smoking [21,22]. A critical period model refers to a limited time window when an exposure has exclusively adverse or protective effects on outcome. There is no influence outside the specified time period. In a sensitive period model, an exposure would have stronger effects on outcome at one time period than at other times. The accumulation of risk model hypothesises that an exposure impacts an outcome equally and accumulatively over the life course. The longer a person stays in a high-risk category, the greater the adverse influences on health. Compared with the aforementioned life course models, a social mobility model has been less strictly defined. Two commonly explored hypotheses are inter-generational and intra-generational mobility. An inter-generational mobility model hypothesises that all downtrend changes are equally harmful to the outcome and all upward shifts are equally beneficial. These changes in exposure, for example SEP, are between parents and offspring. The intra-generational mobility model assumes that any downwards change in SEP in adulthood would be harmful to the outcome and any upwards mobility in adulthood would be beneficial, independent of childhood social background.

Recently, Pollitt and colleagues systematically reviewed papers that examined the associations between life course SEP and cardiovascular risk factors including smoking [23]. They suggested that future life course studies should use data collected from childhood and

multiple SEP measures and multiple life course models in one sample [23]. To our knowledge, no study has investigated how the different life course models might describe the association between SEP at different life stages and smoking status in later life. Understanding ways to reduce tobacco use in socioeconomically disadvantaged groups has been named as a research priority for tobacco control in Australia [24].

Of importance to the role of SEP in smoking behavior is the reasons for the association. It has been documented that children from socioeconomically disadvantaged families have greater exposure to parental smoking [10,25], favourable attitudes toward smoking [26], higher intention to smoke [27] and early smoking experimentation [28]. In turn, these factors are associated with increased risk of future smoking [10,29-34]. However, only limited longitudinal research has explored the extent to which these factors might account for the SEP differences in later smoking. In particular, using data of a birth cohort of 1,265 New Zealand children with 25-year follow-up, Fergusson et al [10] found that parental and peer smoking accounted for over 15% of the relationship between childhood socioeconomic disadvantage and adult smoking; however, this study did not take into account the impacts of adult SEP, which is significantly associated with smoking in adulthood [7]. Far too little attention has been paid to attitudes toward smoking, intention to smoke and smoking experimentation in childhood. In addition, researchers have indicated that people of low SEP may have different family and relationship trajectories than those of high SEP [35,36]. As presented in the previous chapter, these transitions were related to changes in smoking status. So far, however, no study has examined whether the relationship of SEP trajectories with smoking in adulthood is mediated by family transitions.

We aimed to address the gaps by performing the current study in an Australian national cohort. We also examined the relationship of life course trajectories of SEP and nicotine dependence in mid-adulthood and whether exposure to parental smoking, attitudes toward smoking, intention to smoke and early smoking experimentation in childhood, and transition into a relationship with a partner and entering parenthood could mediate the relationship between SEP over the life course and later smoking status.

3.3 Methods

3.3.1 Participants

Sampling procedures have been presented in the section of 2.3.1. In this chapter, ASHFS, CDAH 1 and 2 also refer to childhood, CDAH follow-up 1 and 2.

3.3.2 SEP assessments over the life course

The study used SEP data at three time points: in childhood, at CDAH follow-up 1 (age 26-36 years) and at CDAH follow-up 2 (age 31-41 years). Three indicators of SEP assessed at each time point were used: occupation, education and area-level disadvantage. It seemed unwieldy to include all results in the main text, so only occupation was reported in the main result and the other two were reported in the sensitivity analyses. Furthermore, compared with occupation, education cannot move downwards. This limits the possibility of testing the social mobility model using education. SEIFA, an area-level measure encompassing aspects of occupation, education and employment, was used as a secondary measure and has been less often used than occupation in the literature.

3.3.2.1 Occupation and education assessment

Baseline SEP data on occupation and education was retrospectively reported by participants at CDAH follow-up 1. For each parent separately, participants reported the main occupation of their father/mother and the highest level of education achieved by their father/mother (or other male/female who lived with them and was like a father/mother to them) for most of the time when they were growing up until the age of 12 years. Similar measurement has been used in several other epidemiology studies [38-40]. The levels of occupation and education for whichever parent had the highest were used as two indicators of childhood SEP.

The Australian Standard Classification of Occupations (ASCO) was used to assess the occupation level at three time points [41]. ASCO classifies occupation into nine levels, ranging from 1 = manager or administrator to 9 = labourer or related worker. Participants not in the labour force were treated as a separate group. These ten groups were then regrouped to form two categories: non-manual (managers, professionals and white collar) and manual (blue collar and not in labour force) group. The term manual was used for convenience as

people not in labour force were included into this group. However, their proportions were not high at baseline (1.3%), CDAH follow-up 1 (12.3%) and 2 (13.3%).

Educational attainment was measured according to the responses to the question about the highest level of education achieved by participants or their parents. Two categories were created from ten levels responses: with post-school qualification (any university degree or trade/vocational training) and without post-school qualification group (year 12 or less). Similar measures have been used in other epidemiological studies [14,42].

3.3.2.2 Area-level disadvantage

Socioeconomic indexes for areas (SEIFA) is a product developed by the Australian Bureau of Statistics that ranks residential area in Australia based on socioeconomic advantage and disadvantage and consists of four indexes [43]. The Index of Relative Socioeconomic Disadvantage (IRSD) was the most commonly used and we used it in this study. IRSD focuses on relative disadvantage and is derived from variables such as income, educational attainment, housing tenure and car availability. Participants were assigned to a score based on census collection area of their residence place. A low score indicates a high proportion of relatively disadvantaged people in an area and a high score indicates a relative lack of disadvantage in general. In order to limit the number of alternative pathways over life course, IRSD was dichotomised into high disadvantaged and less disadvantaged group based on the median value.

3.3.3 Smoking status assessment

Assessment of smoking status at CDAH follow-up 2 has been described in the section of 2.3.4.

3.3.4 Nicotine dependence assessment

Nicotine dependence of current smokers at CDAH follow-up 2 was assessed using the Fagerstrom Test for Nictoine Dependence [44]. This test includes six questions, with the summed score ranging from 1 to 10. Higher score indicates highly dependent on nicotine.

3.3.5 Mediating factors

Several variables at baseline and from the CDAH study follow-up 1 and follow-up 2 were selected to test their roles in mediating the relationship between SEP disadvantage and smoking in mid-adulthood.

Children aged 9-15 years completed questionnaires in small groups with a study data collector. Children under 9 years of age were deemed too young to complete the questionnaires reliably. Parental smoking was reported as “none”, “one or two” parents smoking. Information on smoking experimentation was collected using a question “Have you ever smoked even part of a cigarette?”. Children could respond “no”, “yes, a few puffs”, “yes, I have smoked fewer than 10 cigarettes in my life” and “yes, I have smoked more than 10 cigarettes in my life”. The latter three categories were collapsed into one group as with childhood smoking experimentation. Other data collected included the importance of being a non-smoker and intention to smoke.

Current marital status and whether having children at CDAH follow-up 1 and 2, and partnering and parenting transitions from CDAH follow-up 1 and 2were classified in the same way as described in the previous chapter (sections of 2.3.3 and 2.3.4).

3.3.6 Statistical analysis

Means with standard deviations (SDs) and numbers with proportions were used to describe the socio-demographic and smoking status at CDAH follow-up 2 and SEP characteristics for each life course model. The log multinomial model, which estimated RRs and 95% CIs with multiple attributes [45], was used to estimate the smoking status in mid-adulthood by each life course model with never smokers as the reference category. We did not separate men and women for analyses as tests of interaction revealed no evidence of significant difference.

A structured modelling approach was used to test which life course model(s) best fitted the data [21]. This framework compares a set of nested models – each corresponding to the sensitive/critical period, accumulation, social mobility and “no effects” hypotheses – to a saturated model. The saturated model included SEP at three time points and two and three-way interactions. Theoretically, this model should provide the maximum model fit. The sensitive period model assumes the effects of SEP varying over the life course. It was modelled by simultaneously including the SEP indicator at three time points. By contrast,

the critical period model assumes SEP only has an effect within a single time period and models as many possible scenarios as there are time points of SEP measurement. The accumulation model was tested by summing the number of times that a person has occupied a disadvantaged SEP across the early life span to form an overall score ranging from 0 to 3, which was then used as the exposure either continuous or categorical in log multinomial models. As introduced earlier, the social mobility model is more complex compared with the sensitive, critical period or accumulation models. Models specifications and constrains are described in detail in **Appendix 3 Table S1**.

Likelihood ratio tests were used to examine whether the fit of each nested model was as good as the fully saturated model. A large *P*-value (>0.10) indicates no evidence of statistically significant difference between the tested nested model and the fully saturated model; therefore, the tested nested model could provide an adequate description of the association of SEP and smoking status in mid-adulthood, unless the *P*-value for the no effect model was >0.10, which means there is no association between SEP at any time point and the smoking status in mid-adulthood. Two or more nested life course models might fit the data similarly as the fully saturated model.

The association between SEP across three life stages and nicotine dependence in mid-adulthood was examined using linear regression to obtain beta coefficients and 95% CIs from the best fitting life course model(s).

In analyses exploring whether the relationship between disadvantaged SEP and higher risk of being a smoker could be mediated by parental smoking, self-rated importance of being a non-smoker, intention to smoke in the following year and smoking experimentation during childhood, and partnering and parenting transitions from CDAH follow-up 1 to 2. These variables were added to the best-fitting life course model(s) according to *a priori* causal knowledge and univariable analyses. Variables finally entered into model(s) were significantly associated with smoking status at CDAH follow-up 2 and occupation level at baseline or CDAH follow-up 2. The percent excess risk explained by the tested mediator was obtained by a ratio where the numerator included the difference in RRs between models before (RRu) and after adding the possible mediators (RRa), and the denominator included the unadjusted excess risk (% excess risk explained = (RRu – RRa)/(RRu – 1) * 100) [46,47].

Approximately 20% of participants were missing one or more potential mediators' information. Therefore, multiple imputation (MI) by chained equations was used [48], with the number of imputation being 20 [49].

The following sensitivity analyses were conducted to test the robustness of our results. First, we considered the effect of loss to follow-up using combined MI and IPW [50], and examined the differences in effect size between weighted and unweighted results. Baseline age, sex and school type were used to impute data and the following factors at baseline were used to determine the weights: height (cm), weight (kg), arm girth (cm), waist girth (cm), hip girth (cm), sit and reach (cm), sit-ups (number), standing long jump (cm), time spent in 1.6 km run (minutes: seconds), time spent in 50 m run (seconds), area-based SEP, school enjoyment, school assessed and self-reported scholastic ability [30,37]. These variables were chose partly because they were nearly complete by all participants at baseline but principally because they were associated with health status, which significantly related to the pattern of missingness. The second sensitivity analysis was conducted to determine whether defining SEP based on education and area-level disadvantage changed the findings. The third sensitivity analysis was performed by categorising parental smoking and smoking experimentation at 9-15 years into four groups.

All analyses were performed with STATA software, version 12.1 (Stata Corp, College Station, Texas 77845 USA).

3.4 Results

Of the 2,879 participants who at CDAH follow-up 1 provided the main occupation of their parents until they were aged 12, 41 missed data on their own main occupation at CDAH follow-up 1 and 1,341 were lost to follow-up at CDAH follow-up 2 (**Figure 3-1**). Those missing information on smoking at CDAH follow-up 2 (n=5) were further excluded, leaving 1,492 participants in the final analyses. The characteristics of this sample at CDAH follow-up 2 is shown in **Table 3-1.** The mean age at CDAH follow-up 2 was 36.5 years. Most were females and never smokers.

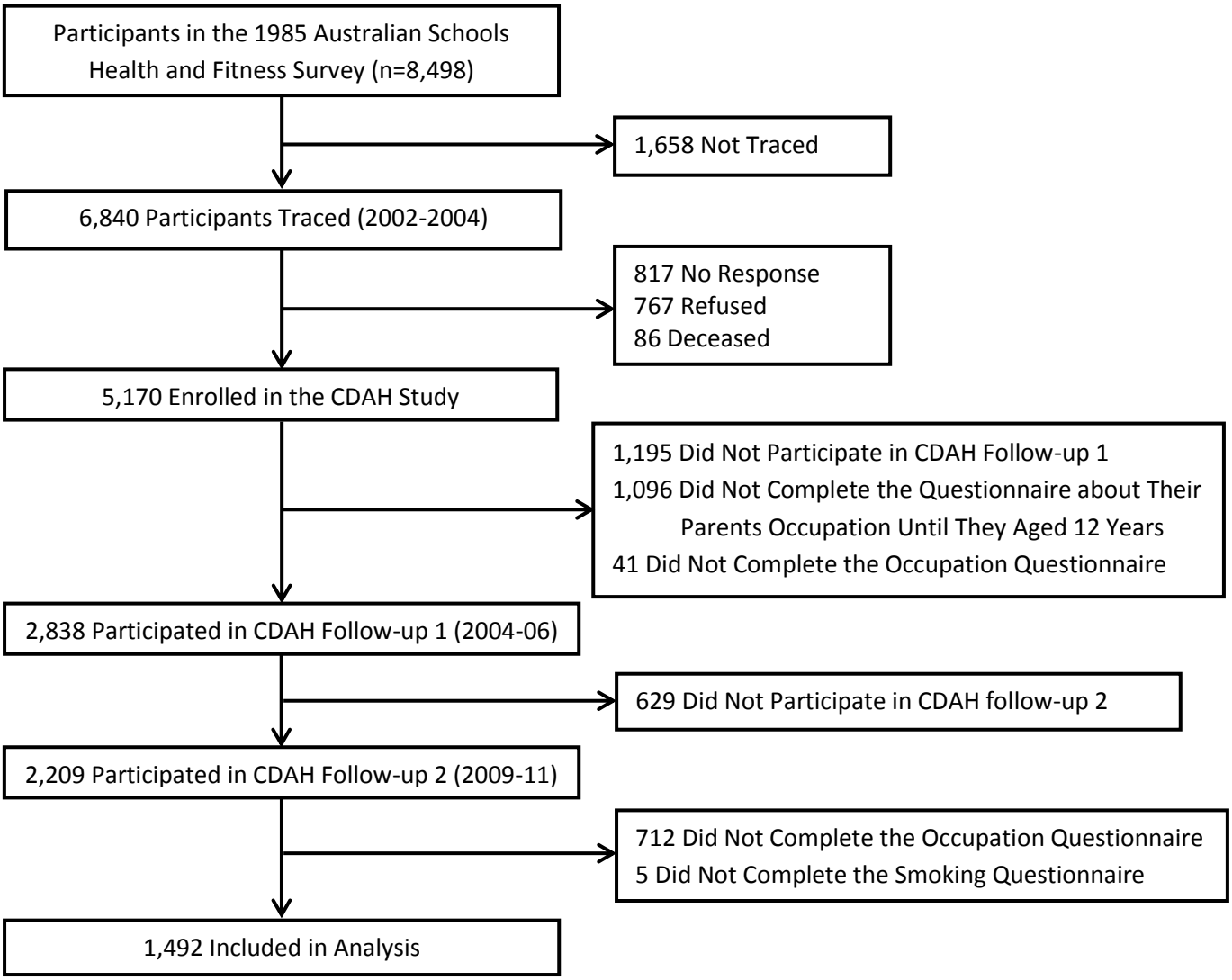


Figure 3-1 Flow chart of recruitment and retention of participants for Childhood Determinants of Adult Health Study, Australia, 1985-2011.

Table 3-1 Characteristics of the sample at CDAH follow-up 2*

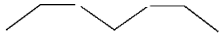
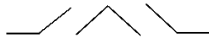
Characteristics	Total (n=1,492)
Age (years), Mean (SD)	36.5 (2.6)
Males, % (n)	36.7 (547)
Married or living as married, % (n)	81.4 (1213)
Education, % (n)	
Any university education	50.5 (749)
Vocational training	29.7 (440)
High school only	19.8 (294)
Weight status†, % (n)	
Normal (<25)	50.3 (708)
Overweight (25-29.9)	32.9 (463)
Obese (≥30)	16.8 (237)
Smoking status, % (n)	
Never smokers	60.7 (905)
Former smokers	25.2 (376)
Current smokers	14.1 (211)

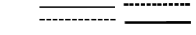
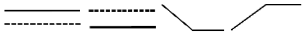
* Sample size varied because of missing data (range, 1,408-1,490).
† Defined by body mass index.

Using baseline characteristics, compared with those lost to follow-up, those who participated in the follow-up study were more often female and less likely to be overweight or obese. No evidence of statistically significant difference was observed in age, Australian-born, health status, highest level of either parent's occupation and education, and area-based SEP (**Appendix 3 Table S2**).

The distribution of non-manual or manual occupation at each time point, the accumulated experience of manual occupation and social mobility across the three time points are presented in **Table 3-2**. About half the participants (47.2%) were categorised in manual occupation at least one time across the early life span. Occupation level at each of the three time points was significantly associated with smoking status at CDAH follow-up 2.

Table 3-2 Summary statistics of the sample by each life course model*

Life course model			Trajectory	Total (n=1,492)	Never smokers (n=905)	Former smokers (n=376)	Current smokers (n=211)
Saturated model, % (n)							
Baseline	CDAH follow-up 1	CDAH follow-up 2					
0	0	0		52.8 (787)	55.0 (498)	53.5 (201)	41.7 (88)
1	0	0		12.5 (187)	13.6 (123)	10.4 (39)	11.9 (25)
0	1	0		7.0 (104)	6.2 (56)	7.2 (27)	10.0 (21)
0	0	1		6.4 (96)	5.5 (50)	9.3 (35)	5.2 (11)
1	1	0		2.6 (38)	2.1 (19)	3.2 (12)	3.3 (7)
1	0	1		2.5 (37)	2.1 (19)	2.4 (9)	4.3 (9)
0	1	1		10.3 (154)	10.3 (93)	9.3 (35)	12.3 (26)
1	1	1		6.0 (89)	5.2 (47)	4.8 (18)	11.4 (24)
P-value						0.001	
Individual time period (sensitive/ critical period model)							
Baseline, % (n)							
Non-manual				76.5 (1,141)	77.0 (697)	79.3 (298)	69.2 (146)
Manual				23.5 (351)	23.0 (208)	20.7 (78)	30.8 (65)
P-value						0.018	
CDAH follow-up 1, % (n)							
Non-manual				74.2 (1,107)	76.2 (690)	75.5 (284)	63.0 (133)
Manual				25.8 (385)	23.8 (215)	24.5 (92)	37.0 (78)
P-value						<0.001	
CDAH follow-up 2, % (n)							
Non-manual				74.8 (1,116)	76.9 (696)	74.2 (279)	66.8 (141)
Manual				25.2 (376)	23.1 (209)	25.8 (97)	33.2 (70)
P-value						0.009	
Accumulation model: number of times manual, % (n)							
0 time manual				52.8 (787)	55.0 (498)	53.5 (201)	41.7 (88)
1 time manual				25.9 (387)	25.3 (229)	26.9 (101)	27.0 (57)
2 times manual				15.4 (229)	14.5 (131)	14.9 (56)	19.9 (42)
3 times manual				6.0 (89)	5.2 (47)	4.8 (18)	11.4 (24)
P-value						0.001	
Social mobility model, % (n)							

Inter-generational mobility †					
Stable (non-)manual		58.7 (876)	60.2 (545)	58.2 (219)	53.1 (112)
Moving downwards		23.7 (354)	22.0 (199)	25.8 (97)	27.5 (58)
Moving upwards		17.6 (262)	17.8 (161)	16.0 (60)	19.4 (41)
P-value				0.232	
Intra-generational (adult) mobility †					
Stable (non-)manual		81.6 (1,217)	84.1 (761)	77.9 (293)	77.3 (163)
Moving downwards		8.9 (133)	7.6 (69)	11.7 (44)	9.5 (20)
Moving up wards		9.5 (142)	8.3 (75)	10.4 (39)	13.3 (28)
P-value				0.020	

* Some summed proportions not 100% due to rounding off; non-manual occupation level denoted by 0 and manual occupation level denoted by 1.

† The inter-generational mobility model hypothesises that all downtrend changes are equally harmful to the outcome and all upward shifts are equally beneficial. The intra-generational mobility model assumes that any downwards change in SEP in adulthood would be harmful to the outcome and any upwards mobility in adulthood would be beneficial, independent of childhood social background.

Table 3-3 describes the results of fitting the life course models. The *P*-value for the likelihood ratio test between the no effect model and the saturated model was less than 0.10, indicating no effect model did not provide an adequate description of the relationship between SEP across the three time points and smoking status at CDAH follow-up 2. The sensitive period model and the accumulation model both provided similar fit as the saturated model (both *P*-values > 0.10). The sensitive period model showed that, compared with non-manual group, those with either parent having a manual occupation during their childhood had 41% higher risk of being current smokers at follow-up 2 and those having a manual occupation themselves at follow-up 1 had 54% higher risk. The accumulation model showed that, compared to those consistently in non-manual SEP across the three time points, those having greater accumulated exposure to manual SEP had a significantly higher risk of being current smokers at CDAH follow-up 2, with 33% risk increase per time point in manual SEP. This suggests that SEP at all three time points are important in the determination of smoking status in mid-adulthood, but childhood and young adulthood may be particularly important.

Table 3-4 documents the relationship between SEP over the life course and nicotine dependence in mid-adulthood for the best fitting life course models. No significant association was observed in the sensitive period model. In the accumulation model, the duration of exposure to manual SEP was positively associated with nicotine dependence using either continuous or categorical summed score.

As there is an overlap of ages between participants at the CDAH follow-up 1 (aged 26-36 years) and at the CDAH follow-up 2 (aged 31-41 years), further analyses were performed to explore whether the significant association at CDAH follow-up 1 in the sensitive period model was due to an age effect. **Figure 3-2** shows the interaction between age and occupation level at CDAH follow-up 1 when participants were aged 26-36 years. Relative to never-smokers, the risk of being current smokers for those in manual SEP versus those in non-manual SEP decreased with age increased, while the risk of being former smokers increased. Therefore, the significant association observed at age 26-36 years in the sensitive period model could potentially be an age effect. The impact of manual occupation on smoking status at middle age may be greater in young adulthood than mid-adulthood.

Table 3-3 Relative risks (95% CIs) and likelihood ratio test results for CDAH follow-up 2 smoking status by each life course model*

Life course model	Smoking status		Model fit and comparison to saturated model
	Former smokers	Current smokers	
	RR (95% CI)	RR (95% CI)	*P*-value†
No effect model	-	-	0.001
Sensitive period model			0.187
Manual, baseline	0.81 (0.65, 1.01)	1.41 (1.07, 1.84)	
Manual, CDAH follow-up 1	0.88 (0.69, 1.13)	1.54 (1.12, 2.11)	
Manual, CDAH follow-up 2	1.10 (0.87, 1.40)	1.11 (0.80, 1.54)	
Critical period model			
Manual, baseline	0.81 (0.65, 1.00)	1.51 (1.15, 1.98)	0.012
Manual, CDAH follow-up 1	0.92 (0.75, 1.12)	1.70 (1.32, 2.19)	0.058
Manual, CDAH follow-up 2	1.01 (0.83, 1.23)	1.50 (1.15, 1.95)	0.009
Accumulation model			0.219
Linear	0.94 (0.85, 1.04)	1.33 (1.18, 1.50)	
Categorical 0 time manual	1	1	
1 time manual	0.98 (0.80, 1.21)	1.36 (1.00, 1.86)	
2 times manual	0.91 (0.71, 1.18)	1.71 (1.22, 2.40)	
3 times manual	0.76 (0.49, 1.17)	2.48 (1.67, 3.70)	
Social mobility model			
Inter-generational mobility			0.001
Stable (non-)manual	1	1	
Moving downwards	1.08 (0.88, 1.32)	1.30 (0.97, 1.75)	
Moving upwards	0.86 (0.67, 1.11)	1.29 (0.92, 1.80)	
Intra-generational mobility			0.007
Stable (non-)manual	1	1	
Moving downwards	1.35 (1.04, 1.75)	1.17 (0.76, 1.80)	
Moving up wards	1.14 (0.86, 1.51)	1.49 (1.04, 2.13)	

RR: relative risk, CI: confidence interval.

* All models were adjusted for age and sex at CDAH follow-up 2.

† Bold *P*-values indicate the tested life course model fit the data as good as the saturated model.

Bold RRs (95% CIs) indicate statistically significant results from the best fitting life course models.

Table 3-4 β coefficients (95% CIs) for nicotine dependence at CDAH follow-up 2 for the best fitting life course models

Life course model	Nicotine dependence continuous β (95% CI)*	P-values
Sensitive period model		
Manual, baseline	0.56 (-0.29, 1.40)	0.197
Manual, CDAH follow-up 1	0.77 (-0.19, 1.73)	0.113
Manual, CDAH follow-up 2	0.57 (-0.39, 1.53)	0.242
Accumulation model		
Linear	0.70 (0.30, 1.09)	0.001
Categorical, 0 time manual	0	
1 time manual	1.29 (0.29, 2.30)	0.012
2 times manual	1.13 (0.07, 2.19)	0.037
3 times manual	2.13 (0.91, 3.34)	0.001

CI: confidence interval.
* Adjusted for age and sex at CDAH follow-up 2, n=144.
Bold β coefficients (95% CIs) indicate statistically significant result.

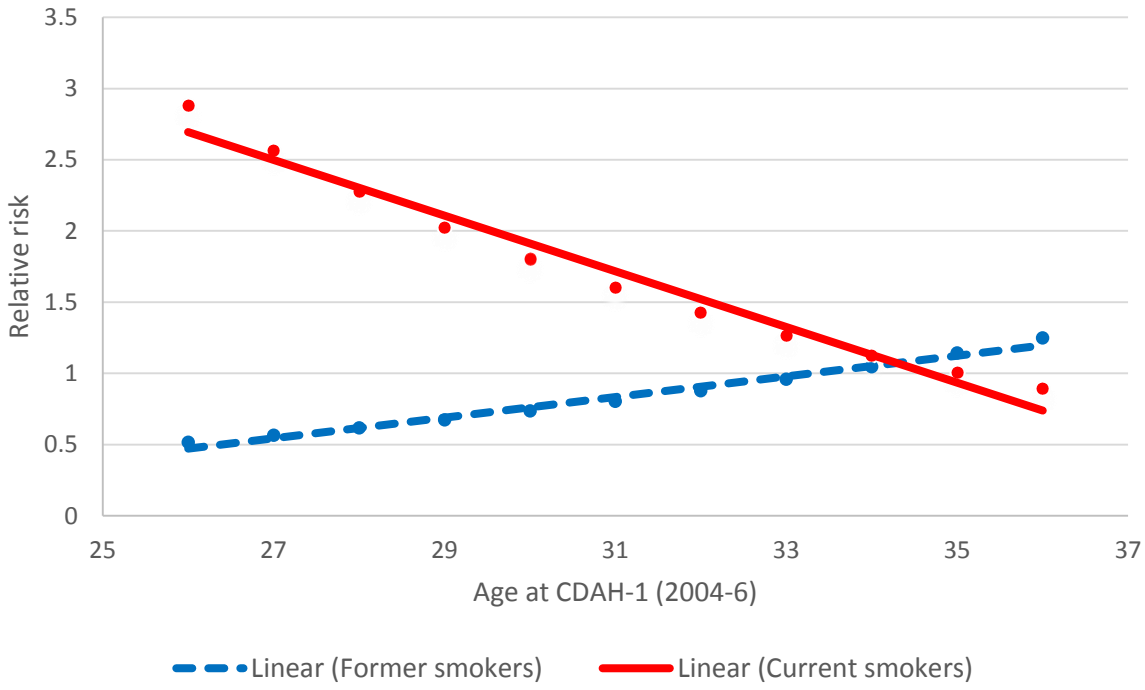


Figure 3-2 Interaction between age and occupation level (non-manual or manual) at CDAH follow-up 1 in 2004-6 when participants were aged 26-36 years.

We investigated factors associated with smoking status at CDAH follow-up 2 and the main occupation level of either parent at baseline or participants themselves at CDAH follow-up 2 using univariable analyses (**Appendix 3 Table S3 and S4**). Being exposed to parental smoking in childhood, self-reported lower importance of being a non-smoker, childhood smoking experimentation, intention to smoke in the following year at baseline and partnering or parenting transitions during CDAH follow-up 1 and 2 were all significantly associated with being a current smoker. Factors associated with baseline or CDAH follow-up 2 SEP included exposure to parental smoking, the importance to be a non-smoker and partnering or parenting transitions. As shown in **Table 3-5**, in the sensitive period model adjustment for exposure to parental smoking and self-rated importance to be a non-smoker accounted for 32% of the excess risk associated with low SEP in childhood on current smoking in mid-adulthood. In the accumulation model, these two factors explained 15% of the excess risk associated with greater exposure to manual SEP. Taking account of partnering and parenting transitions from CDAH follow-up 1 to 2 did not change the results in the sensitive period and accumulation model.

Sensitivity analyses conducted by re-analysing the data using MI and IPW produced similar patterns of results as the unweighted analyses (**Appendix 3 Table S5 and S6**). The changes in the magnitude of statistically significant estimates were within 18.8% of those from unweighted analyses. Findings using education to define SEP were similar to the original results (**Appendix 3 Table S7 and S8**). The results differed appreciably when we defined SEP according to area-level disadvantage IRSD (**Appendix 3 Table S9 and S10**). We found that the sensitive period model provided similar fit as the saturated model but not the accumulation model. In the sensitive period model, relative to never smokers, the risk of being a former smoker was significantly lower in those living in high disadvantaged area than those living in less disadvantaged area. In addition, young and mid-adulthood were sensitive periods that determine the risk of being a current smoker rather than childhood observed from SEP determined by occupation and education. Sensitivity analyses categorising parental smoking and smoking experimentation at 9-15 years into four groups produced similar results to the original one (data not shown).

Table 3-5 Sensitive period model for being current smokers at CDAH follow-up 2 adjusted for age, sex at CDAH follow-up 2 and potential mediators*

Life course model	Model 1 Age and sex	Model 2 Model 1 + Parental smoking status	Model 3 Model 2 + The importance of being a non-smoker	Model 4 Model 1 + Partnering and parenting transitions from CDAH follow-up 1 to 2
Sensitive period model				
Former smokers at CDAH follow-up 2				
Manual, baseline	0.81 (0.65, 1.01)	0.80 (0.64, 1.00)	0.79 (0.63, 0.98)	0.81 (0.65, 1.01)
Manual, CDAH follow-up 1	0.88 (0.69, 1.13)	0.89 (0.70, 1.14)	0.85 (0.66, 1.10)	0.87 (0.68, 1.12)
Manual, CDAH follow-up 2	1.10 (0.87, 1.40)	1.10 (0.86, 1.40)	1.09 (0.85, 1.39)	1.10 (0.86, 1.39)
Current smokers at CDAH follow-up 2				
Manual, baseline	1.41 (1.07, 1.84)	1.35 (1.04, 1.77)	1.28 (0.98, 1.68)	1.41 (1.07, 1.84)
Manual, CDAH follow-up 1	1.54 (1.12, 2.11)	1.61 (1.17, 2.22)	1.56 (1.12, 2.17)	1.54 (1.11, 2.13)
Manual, CDAH follow-up 2	1.11 (0.80, 1.54)	1.07 (0.77, 1.48)	1.05 (0.75, 1.47)	1.12(0.81, 1.55)
Accumulation model				
Linear				
Former smokers at CDAH follow-up 2	0.94 (0.85, 1.04)	0.94 (0.85, 1.04)	0.92 (0.83, 1.01)	0.93 (0.84, 1.03)
Current smokers at CDAH follow-up 2	1.33 (1.18, 1.50)	1.32 (1.18, 1.49)	1.28 (1.14, 1.44)	1.33 (1.18, 1.50)

* Statistics presented are relative risks (95% confidence intervals).

3.5 Discussion

In this longitudinal study of Australian adults, we found that the sensitive period model and the accumulation model best described the associations between SEP across the early life span and smoking status in mid-adulthood. The associations were such that the risk of being a current smoker was statistically higher in those exposed to low SEP in childhood and early adult life, and those exposed over a greater number of periods. In the sensitive period and accumulation model, the associations were attenuated slightly to moderately after taking into account exposure to parental smoking and the self-reported importance to be a non-smoker at baseline, suggesting that these factors may be important mediators. To the best of our knowledge, this was the first study to examine longitudinal relationships of SEP and smoking status using a counterfactual framework to distinguish between a series of theoretical life course models.

The sensitive period model was supported by our data. Being exposed to low SEP at childhood and early adulthood increased the risk of being a current smoker in mid-adulthood when SEP at all three life stages were mutually adjusted. There is considerable evidence showing that smoking in adulthood is influenced by childhood and adulthood socioeconomic disadvantage [10,23,51,52]. For example, according to Kestila et al. [51], the odds of being a daily smoker among young adults whose parents had the lowest educational attainment were about five times those of their peers in the highest education category. However, not all studies supported this point. For example, Poulton et al. [53] showed that young adults' tobacco dependence was not linked to low childhood SEP.

Relative to those having a more advantaged SEP at childhood, the observed higher risk of being a current smoker in mid-adulthood for SEP disadvantaged children was found to be partially mediated via parental smoking exposure and rating being a non-smoker of lower importance even after adjustment for adulthood SEP. This finding is consistent with findings of past studies by Paul et al. [30] and Fergusson et al. [10], which concluded that current smoking in adulthood was predicted by exposure to parental smoking, which accounted for over 25% of the relationship between childhood social background and later smoking [10].

In the sensitive period model, we also observed an age effect at CDAH follow-up 1 when participants were aged 26-36 years. Relative to never smokers, the risk of being current

smokers in mid-adulthood for those in low SEP versus their peers in high SEP decreased as participants got older. In contrast, the probability of being former smokers was increased. This data provides insight into the trend of disparity in smoking prevalence between various SEP groups with age and indicates that the gap between high and low SEP groups narrowed with age. It suggests that young adults should be recognised as an important group for future work to reduce socioeconomic inequality in tobacco use.

Apart from the sensitive period model, our findings also supported the accumulation model, with people who accumulated more time in disadvantaged SEP from childhood to mid-adulthood being significantly more likely to be a current smoker. This is supported by Smith et al's study [54] which assessed the influence of SEP over three life stages on risk factors of CVDs including smoking among 5,766 men, and revealed a positive graded association between the number of time periods belonging to manual occupation and the proportion that were current smokers. Similar results have also been reported among women [55,56]. One study by Lawlor et al. [56] reported that women in manual position in both childhood and adulthood had a 75% higher odds of being a current smoker as compared to those who consistently stayed in non-manual social class at both time points.

Previous evidence on the validity of using area-based SEP measures as proxies of individual-level indicators has been conflicting [57-60]. In this chapter, we also found inconsistent results of using socioeconomic indicators at the individual and area levels. One of the possible reasons for the difference is the different constructs of area and individual-level socioeconomic measures [57]. Using data from three big population-based epidemiologic studies, Diez and colleagues reported that although area and individual-level indicators were associated, but far from perfectly correlated and provided complementary information on living circumstances [61]. Presence of contextual area effects may be another reason to explain discrepancies between area- and individual-based estimates of socioeconomic differences in smoking [57].This involves mechanisms through which contextual effects of area on smoking could be meditated, including greater likelihood of being exposed to smoking and tobacco advertising [7].

Some limitations should be considered in the interpretation of our findings. First, self-report could result in the misclassification of smoking status; however, the tendency to under-

report compared with smoking confirmed by cotinine measurement may lead to an underestimation of the effect of SEP on current smoking [62]. Second, two indices of childhood SEP (occupation and education) were collected retrospectively at CDAH follow-up 1, which may have resulted in a measurement error compared with the measurement of adult SEP. Such recall was found to be only moderately correct over five decades [63]. Given a relatively short time period (14-24 years) in our study, the recall bias should be smaller but is still likely to underestimate the real effects. The third potential limitation was loss to follow-up. Some significant differences were observed between those who participated in the follow-up study and those who did not. Therefore, our sample was not strictly representative of the general population, limiting the generalisability of our results. Applying combined MI and IPW to account for these differences demonstrated similar results, suggesting that this is not a major source of bias. Fourth, dichotomising SEP is very simplified. We could not explore that whether there is a gradient of effects across socioeconomic levels; however, the number of life course trajectories would increase greatly if we classified SEP into more groups, correspondingly decreasing the number of people in each group. Fifth, we did not know the duration of exposure; therefore, the identification of the accumulation model in this study does not refer to the exact length of exposure to low or high SEP. Sixth, the results obtained from SEP defined by area-level disadvantage were different from those from occupation and education, indicating the effects of various SEP indicators on smoking may not be interchangeable [64]. The last limitation related to the measure of mediating variables at baseline. For example, there was a widespread age range (9-15 years) at the time of entry into the study. This is important as recall and the impact of parental smoking could be very different at 9 and 15 years of age, and the meaning of being a never smoker or having experimented with smoking is different for a 9 versus 15 years old (e.g. having tried smoking may be unusual at 9 years old, but probably almost a norm for 15 year olds).

The strengths of the current study include its large national sample, the 25 years follow-up period, the use of a novel methodology and the efforts to explore the underlying mechanisms. Although several studies have examined the association of SEP and smoking status using a life course approach, none of them has tested multiple life course models in the same sample. As concluded by Pollitt and colleagues in a systematic review [23], analyses

using data followed from childhood to adulthood, multiple SEP measures and multiple life course designs within the same sample offer the best approach to test which theories best describe the association between life course SEP and the outcome.

3.6 Conclusion

To conclude, low SEP was associated with an increased risk of being a current smoker in adults aged in their 30s. The accumulation model and the sensitive period fitted the relationship between SEP across the early life span and smoking status in mid-adulthood as well as the saturated model. This suggests that the risk was greatest among those who were exposed to low SEP for longer and those exposed during childhood and young adulthood. The association seemed to be partly mediated through exposure to parental smoking and the self-reported importance to be a non-smoker in childhood. Our findings provide a more detailed understanding of the development of socioeconomic inequalities in smoking up to mid-adulthood. However, how to effectively reduce these disparities is a very big challenge for health professionals and policy makers. Some suggested methods and the public health implications in detail of our findings are presented in chapter 7. More research is needed to provide an optimal way to discourage taking up smoking, encourage quitting and promote sustained cessation, especially among low SEP groups.

3.7 Postscript

This chapter showed the determination of SEP variations across the early life span on smoking status in mid-adulthood. The following three chapters investigate the health effects of changing smoking status in young adulthood.

3.8 References

1. World Health Organization. *Tobacco. Fact sheet N°339. Updated 6 July 2015.* Genneva: 2015. Accessed 11 November 2015.
2. Lim SS, Vos T, Flaxman AD, et al. A comparative risk assessment of burden of disease and injury attributable to 67 risk factors and risk factor clusters in 21 regions, 1990-2010: a systematic analysis for the Global Burden of Disease Study 2010. *Lancet.* 2012;380(9859):2224-2260. doi:10.1016/S0140-6736(12)61766-8.
3. Bradley RH, Corwyn RF. Socioeconomic status and child development. *Annual review of psychology.* 2002;53(1):371-399.
4. Galobardes B, Shaw M, Lawlor DA, Lynch JW, Davey Smith G. Indicators of socioeconomic position (part 1). *J Epidemiol Community Health.* 2006;60(1):7-12. doi:10.1136/jech.2004.023531.
5. Galobardes B, Shaw M, Lawlor DA, Lynch JW, Davey Smith G. Indicators of socioeconomic position (part 2). *J Epidemiol Community Health.* 2006;60(2):95-101. doi:10.1136/jech.2004.028092.
6. Thun M, Peto R, Boreham J, Lopez AD. Stages of the cigarette epidemic on entering its second century. *Tobacco control.* 2012;21(2):96-101.
7. Hiscock R, Bauld L, Amos A, Fidler JA, Munafo M. Socioeconomic status and smoking: a review. *Ann N Y Acad Sci.* 2012;1248:107-123. doi:10.1111/j.1749-6632.2011.06202.x.
8. Gilman SE, Abrams DB, Buka SL. Socioeconomic status over the life course and stages of cigarette use: initiation, regular use, and cessation. *J Epidemiol Community Health.* 2003;57(10):802-808.
9. Jefferis B, Graham H, Manor O, Power C. Cigarette consumption and socio-economic circumstances in adolescence as predictors of adult smoking. *Addiction.* 2003;98(12):1765-1772.
10. Fergusson DM, Horwood LJ, Boden JM, Jenkin G. Childhood social disadvantage and smoking in adulthood: results of a 25-year longitudinal study. *Addiction.* 2007;102(3):475-482. doi:10.1111/j.1360-0443.2006.01729.x.
11. Bowes L, Chollet A, Fombonne E, Galera C, Melchior M. Lifecourse SEP and tobacco and cannabis use. *Eur J Public Health.* 2013;23(2):322-327. doi:10.1093/eurpub/cks065.
12. Amos A, Bauld L, Hill S, Platt S, Robinson J. Tobacco control, inequalities in health and action at the local level in England. *London: Public Health Research Consortium.* 2011.
13. Sharma A, Lewis S, Szatkowski L. Insights into social disparities in smoking prevalence using Mosaic, a novel measure of socioeconomic status: an analysis using a large primary care dataset. *BMC Public Health.* 2010;10:755. doi:10.1186/1471-2458-10-755.
14. Scollo MM, Winstanley MH. *Tobacco in Australia: Facts and issues. Melbourne: Cancer Council Victoria; 2015. Available from* www.TobaccoInAustralia.org.au. Accessed.
15. U.S. Department of Health and Human Services. *The health consequences of Smoking—50 Years of Progress: A Report of the Surgeon General*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion,

Office on Smoking and Health; 2014. http://www.cdc.gov/tobacco/data_statistics/sgr/50th-anniversary/index.htm. Accessed 2 July 2016.
16. Kanjilal S, Gregg EW, Cheng YJ, et al. Socioeconomic status and trends in disparities in 4 major risk factors for cardiovascular disease among US adults, 1971-2002. *Arch Intern Med.* 2006;166(21):2348-2355. doi:10.1001/archinte.166.21.2348.
17. Peretti‐Watel P, Constance J, Seror V, Beck F. Cigarettes and social differentiation in France: is tobacco use increasingly concentrated among the poor? *Addiction.* 2009;104(10):1718-1728.
18. Smith P, Frank J, Mustard C. Trends in educational inequalities in smoking and physical activity in Canada: 1974-2005. *J Epidemiol Community Health.* 2009;63(4):317-323. doi:10.1136/jech.2008.078204.
19. OECD. *Divided we stand: why inequality keeps rising.* . 2011. Accessed.
20. Lee JG, Henriksen L, Rose SW, Moreland-Russell S, Ribisl KM. A Systematic Review of Neighborhood Disparities in Point-of-Sale Tobacco Marketing. *Am J Public Health.* 2015;105(9):e8-18. doi:10.2105/AJPH.2015.302777.
21. Mishra G, Nitsch D, Black S, De Stavola B, Kuh D, Hardy R. A structured approach to modelling the effects of binary exposure variables over the life course. *Int J Epidemiol.* 2009;38(2):528-537. doi:10.1093/ije/dyn229.
22. Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, Power C. Life course epidemiology. *J Epidemiol Community Health.* 2003;57(10):778-783.
23. Pollitt RA, Rose KM, Kaufman JS. Evaluating the evidence for models of life course socioeconomic factors and cardiovascular outcomes: a systematic review. *BMC Public Health.* 2005;5:7. doi:10.1186/1471-2458-5-7.
24. The Australian National Preventive Health Agency. *A priority-driven research agenda for tobacco control in Australia: final report*. Canberra: The Australian National Preventive Health Agency; 2013. https://health.gov.au/internet/anpha/publishing.nsf/Content/A27B1B3A44327688CA257B96000165E9/$File/Web%20resolution_a%20priority%20reasearch%20agency%20tobacco%20control.pdf. Accessed 5 August 2013.
25. Australian Bureau of Statistics. *Marriages and divorces, Australia, 2013 (ABS cat. No. 3310.0)*. Canberra: 2013. http://www.abs.gov.au/ausstats/abs@.nsf/Latestproducts/3310.0Main%20Features112013?opendocument&tabname=Summary&prodno=3310.0&issue=2013&num=&view=. Accessed 1 July 2015.
26. de Vries H. Socio-economic differences in smoking: Dutch adolescents' beliefs and behaviour. *Soc Sci Med.* 1995;41(3):419-424.
27. Cremers HP, Oenema A, Mercken L, Candel M, de Vries H. Explaining socio-economic differences in intention to smoke among primary school children. *BMC Public Health.* 2014;14:191. doi:10.1186/1471-2458-14-191.
28. Green MJ, Leyland AH, Sweeting H, Benzeval M. Socioeconomic position and early adolescent smoking development: evidence from the British Youth Panel Survey (1994-2008). *Tob Control.* 2016;25(2):203-210. doi:10.1136/tobaccocontrol-2014-051630.
29. Cremers HP, Mercken L, de Vries H, Oenema A. A longitudinal study on determinants of the intention to start smoking among Non-smoking boys and girls of high and low

socioeconomic status. *BMC Public Health.* 2015;15:648. doi:10.1186/s12889-015-1917-9.
30. Paul SL, Blizzard L, Patton GC, Dwyer T, Venn A. Parental smoking and smoking experimentation in childhood increase the risk of being a smoker 20 years later: the Childhood Determinants of Adult Health Study. *Addiction.* 2008;103(5):846-853. doi:10.1111/j.1360-0443.2008.02196.x.
31. Tjora T, Hetland J, Aaro LE, Overland S. Distal and proximal family predictors of adolescents' smoking initiation and development: a longitudinal latent curve model analysis. *BMC Public Health.* 2011;11:911. doi:10.1186/1471-2458-11-911.
32. Wakefield M, Kloska DD, O'Malley PM, et al. The role of smoking intentions in predicting future smoking among youth: findings from Monitoring the Future data. *Addiction.* 2004;99(7):914-922. doi:10.1111/j.1360-0443.2004.00742.x.
33. Choi WS, Pierce JP, Gilpin EA, Farkas AJ, Berry CC. Which adolescent experimenters progress to established smoking in the United States. *Am J Prev Med.* 1997;13(5):385-391.
34. Qing Y, Termsirikulchai L, Vatanasomboon P, Sujirarat D, Tanasugarn C, Kengganpanich M. Factors related to tobacco use among middle school students in China. *Southeast Asian J Trop Med Public Health.* 2011;42(5):1249-1261.
35. Harley C, Mortimer JT. Markers of transition to adulthood, socioeconomic status of origin, and trajectories of health. *Ann N Y Acad Sci.* 1999;896:367-369.
36. Magadi MA, Agwanda AO. Determinants of transitions to first sexual intercourse, marriage and pregnancy among female adolescents: evidence from South Nyanza, Kenya. *J Biosoc Sci.* 2009;41(3):409-427. doi:10.1017/S0021932008003210.
37. Gall SL, Jose K, Smith K, Dwyer T, Venn A. The Childhood Determinants of Adult Health Study: a profile of a cohort study to examine the childhood influences on adult cardiovascular health. *Australasian Epidemiologist.* 2009;16(1):35.
38. Lidfeldt J, Li TY, Hu FB, Manson JE, Kawachi I. A prospective study of childhood and adult socioeconomic status and incidence of type 2 diabetes in women. *Am J Epidemiol.* 2007;165(8):882-889. doi:10.1093/aje/kwk078.
39. Lynch JW, Kaplan GA, Cohen RD, et al. Childhood and adult socioeconomic status as predictors of mortality in Finland. *Lancet.* 1994;343(8896):524-527.
40. Power C, Graham H, Due P, et al. The contribution of childhood and adult socioeconomic position to adult obesity and smoking behaviour: an international comparison. *Int J Epidemiol.* 2005;34(2):335-344. doi:10.1093/ije/dyh394.
41. Statistics ABo. *Australian standard classification of occupations.* Australian Government Publishing Service Canberra; 1997.
42. Stringhini S, Batty GD, Bovet P, et al. Association of lifecourse socioeconomic status with chronic inflammation and type 2 diabetes risk: the Whitehall II prospective cohort study. *PLoS Med.* 2013;10(7):e1001479. doi:10.1371/journal.pmed.1001479.
43. Pink B. *Socio-economic indexes for areas (SEIFA)–Technical paper*. Canberra: (ABS) ABoS; 2011. Accessed.
44. Heatherton TF, Kozlowski LT, Frecker RC, Fagerstrom KO. The Fagerstrom Test for Nicotine Dependence: a revision of the Fagerstrom Tolerance Questionnaire. *Br J Addict.* 1991;86(9):1119-1127.
45. Blizzard L, Hosmer D. The log multinomial regression model for nominal outcomes with more than two attributes. *Biometrical Journal.* 2007;49(6):889-902.

46. Richiardi L, Bellocco R, Zugna D. Mediation analysis in epidemiology: methods, interpretation and bias. *Int J Epidemiol.* 2013;42(5):1511-1519. doi:10.1093/ije/dyt127.
47. Kaufman JS, Maclehose RF, Kaufman S. A further critique of the analytic strategy of adjusting for covariates to identify biologic mediation. *Epidemiol Perspect Innov.* 2004;1(1):4. doi:10.1186/1742-5573-1-4.
48. Azur MJ, Stuart EA, Frangakis C, Leaf PJ. Multiple imputation by chained equations: what is it and how does it work? *Int J Methods Psychiatr Res.* 2011;20(1):40-49. doi:10.1002/mpr.329.
49. Graham JW, Olchowski AE, Gilreath TD. How many imputations are really needed? Some practical clarifications of multiple imputation theory. *Prev Sci.* 2007;8(3):206-213. doi:10.1007/s11121-007-0070-9.
50. Seaman SR, White IR, Copas AJ, Li L. Combining multiple imputation and inverse-probability weighting. *Biometrics.* 2012;68(1):129-137. doi:10.1111/j.1541-0420.2011.01666.x.
51. Kestila L, Koskinen S, Martelin T, et al. Influence of parental education, childhood adversities, and current living conditions on daily smoking in early adulthood. *Eur J Public Health.* 2006;16(6):617-626. doi:10.1093/eurpub/ckl054.
52. Lacey RE, Cable N, Stafford M, Bartley M, Pikhart H. Childhood socio-economic position and adult smoking: are childhood psychosocial factors important? Evidence from a British birth cohort. *Eur J Public Health.* 2011;21(6):725-731. doi:10.1093/eurpub/ckq179.
53. Poulton R, Caspi A, Milne BJ, et al. Association between children's experience of socioeconomic disadvantage and adult health: a life-course study. *Lancet.* 2002;360(9346):1640-1645. doi:10.1016/S0140-6736(02)11602-3.
54. Smith GD, Hart C, Blane D, Gillis C, Hawthorne V. Lifetime socioeconomic position and mortality: prospective observational study. *BMJ.* 1997;314(7080):547-552.
55. Heslop P, Smith GD, Macleod J, Hart C. The socioeconomic position of employed women, risk factors and mortality. *Soc Sci Med.* 2001;53(4):477-485.
56. Lawlor DA, Ebrahim S, Davey Smith G, British women's h, health s. Socioeconomic position in childhood and adulthood and insulin resistance: cross sectional survey using data from British women's heart and health study. *BMJ.* 2002;325(7368):805.
57. Diez Roux AV, Merkin SS, Hannan P, Jacobs DR, Kiefe CI. Area characteristics, individual-level socioeconomic indicators, and smoking in young adults: the coronary artery disease risk development in young adults study. *Am J Epidemiol.* 2003;157(4):315-326.
58. Greenwald HP, Polissar NL, Borgatta EF, McCorkle R. Detecting survival effects of socioeconomic status: problems in the use of aggregate measures. *J Clin Epidemiol.* 1994;47(8):903-909.
59. Geronimus AT, Bound J, Neidert LJ. On the validity of using census geocode characteristics to proxy individual socioeconomic characteristics. *Journal of the American Statistical Association.* 1996;91(434):529-537.
60. Soobader M-j, LeClere FB, Hadden W, Maury B. Using aggregate geographic data to proxy individual socioeconomic status: does size matter? *American Journal of Public Health.* 2001;91(4):632.

61. Diez-Roux AV, Kiefe CI, Jacobs DR, Jr., et al. Area characteristics and individual-level socioeconomic position indicators in three population-based epidemiologic studies. *Ann Epidemiol.* 2001;11(6):395-405.
62. Connor Gorber S, Schofield-Hurwitz S, Hardt J, Levasseur G, Tremblay M. The accuracy of self-reported smoking: a systematic review of the relationship between self-reported and cotinine-assessed smoking status. *Nicotine Tob Res.* 2009;11(1):12-24. doi:10.1093/ntr/ntn010.
63. Batty GD, Lawlor DA, Macintyre S, Clark H, Leon DA. Accuracy of adults' recall of childhood social class: findings from the Aberdeen children of the 1950s study. *J Epidemiol Community Health.* 2005;59(10):898-903. doi:10.1136/jech.2004.030932.
64. Laaksonen M, Rahkonen O, Karvonen S, Lahelma E. Socioeconomic status and smoking: analysing inequalities with multiple indicators. *Eur J Public Health.* 2005;15(3):262-269. doi:10.1093/eurpub/cki115.

3.9 Appendix 3

Table S1 Model specification and constraints for life course models

	Life course model specification	Constraints
Saturated model	$\alpha + b_1S_1 + b_2S_2 + b_3S_3 + \theta_{12}S_1S_2 + \theta_{13}S_1S_3 + \theta_{23}S_2S_3 + \theta_{123}S_1S_2S_3$	
No effect model	α	
Sensitive period model	$\alpha + b_1S_1 + b_2S_2 + b_3S_3$	$b1 \neq b2 \neq b3$, $\theta_{12} = \theta_{13} = \theta_{23} = \theta_{123} = 0$
Critical period model		
Baseline, childhood	$\alpha + b_1S_1$	$b2 = b3 = 0$, $\theta_{12} = \theta_{13} = \theta_{23} = \theta_{123} = 0$
CDAH follow-up 1, early adulthood	$\alpha + b_2S_2$	$b1 = b3 = 0$, $\theta_{12} = \theta_{13} = \theta_{23} = \theta_{123} = 0$
CDAH follow-up 2, middle adulthood	$\alpha + b_3S_3$	$b1 = b2 = 0$, $\theta_{12} = \theta_{13} = \theta_{23} = \theta_{123} = 0$
Accumulation model	$\alpha + bS1 + bS2 + bS3$	$b1 = b2 = b3 = b$, $\theta_{12} = \theta_{13} = \theta_{23} = \theta_{123} = 0$
Social mobility models		
Inter-generational mobility	$\alpha + b_1S_1 + b_2S_2 + b_3S_3 + \theta_{12}S_1S_2 + \theta_{23}S_2S_3$	$b2 = (b1 + b3)$, $\theta_{12} = \theta_{23} = -b2$, $\theta_{13} = \theta_{123} = 0$
Intra-generational mobility	$\alpha + b_2S_2 + b_3S_3 + \theta_{23}S_2S_3$	$\theta_{23} = -(b2 + b3)$, $b1 = \theta_{12} = \theta_{13} = \theta_{123} = 0$

Si are the binary indicators of socioeconomic position (SEP) at time i, with i=1, 2, 3; Si=0 refers to advantaged SEP at time i while Si=1 refers to disadvantaged SEP at time i.

Table S2 Comparison of baseline characteristics of participants and non-participants*

Characteristics	Participants (n=1,492)	Non-participants (n=1,387)	P-value
Age (years), Mean (SD)	11.1 (2.5)	11.1 (2.5)	0.419
Males, % (n)	36.7 (547)	53.3 (739)	<0.001
Body mass index, Mean (SD)	18.1 (2.7)	18.2 (2.8)	0.160
Australian-born, % (n)	93.5 (1,115)	94.0 (1,007)	0.582
Weight status†, % (n)			0.035
Normal	92.1 (1,373)	89.3 (1,239)	
Overweight	7.0 (105)	9.3 (129)	
Obese	0.9 (13)	1.4 (19)	
Health status, % (n)			0.950
Very good	37.1 (443)	36.0 (387)	
Good	43.8 (523)	44.5 (479)	
Average/poor/very poor	19.2 (229)	19.5 (210)	
Highest level of either parent's occupation			0.198
Managers and professionals	58.6 (874)	55.0 (763)	
White collar	17.9 (267)	18.5 (257)	
Blue collar	22.2 (331)	25.2 (350)	
Not in labour force	1.3 (20)	1.2 (17)	
Highest level of either parent's education			0.068
Any university education	30.1 (439)	26.2 (355)	
Vocational training	32.1 (468)	34.1 (463)	
High school only	37.8 (552)	39.7 (539)	
Area-based SEP in 1985, % (n)			0.502
High	26.1 (307)	26.3 (277)	
Mid-high	28.3 (333)	28.9 (305)	
Mid-low	38.6 (453)	36.3 (383)	
Low	7.0 (82)	8.4 (89)	

* Sample size varied because of missing data (range 1,175-1,492 for participants, range 1,054-1,387 for non-participants).

† Defined by body mass index.

Table S3. Univariable analyses about factors associated with smoking status at CDAH follow-up 2

Variables	Total	Never smokers	Former smokers	Current smokrs	*P*-value
Childhood factors					
Parental smoking status in 1985	2,302	1,296	646	360	<0.001
None	1,324 (57.5)	806 (62.2)	367 (56.8)	151 (41.9)	
Father	311 (13.5)	147 (11.3)	90 (14.0)	74 (20.6)	
Mother	243 (10.6)	113 (8.7)	74 (11.5)	56 (15.6)	
Both	424 (18.4)	230 (17.8)	115 (17.8)	79 (21.9)	
The importance to be a non-smoker	2,310	1,299	648	363	<0.001
Very important	1,829 (79.2)	1,107 (85.2)	462 (71.3)	260 (71.6)	
Of some important	267 (11.6)	118 (9.1)	99 (15.3)	50 (13.8)	
Of little important	101 (4.4)	33 (2.5)	50 (7.7)	18 (5.0)	
Not important	113 (4.9)	41 (3.2)	37 (5.7)	35 (9.6)	
Smoking experimentation	2,307	1,297	647	363	<0.001
No	1,288 (55.9)	876 (67.5)	263 (40.7)	149 (41.1)	
Yes	1,019 (44.2)	421 (32.5)	384 (59.4)	214 (59.0)	
Intention to smoke in 1985 the following year	2,307	1,297	646	364	<0.001
Yes	60 (2.6)	11 (0.9)	26 (4.0)	23 (6.3)	
No	1,858 (80.5)	1,164 (89.8)	452 (70.0)	242 (66.5)	
Don’t know	389 (16.9)	122 (9.4)	168 (26.0)	99 (27.2)	
Adulthood factors					
Partnering transitions	2,762	1,575	767	420	<0.001
Not partnered both time points	394 (14.3)	205 (13.0)	86 (11.2)	103 (24.5)	
Became partnered	413 (15.0)	230 (14.6)	132 (17.2)	51 (12.1)	
Stayed partnered	1,821 (65.9)	1,073 (68.1)	518 (67.5)	230 (54.8)	
Became separated/divorced/widowed	134 (4.9)	67 (4.3)	31 (4.0)	36 (8.6)	
Parenting transitions	2,897	1,639	800	458	<0.001
No children both time points	934 (32.2)	506 (30.9)	241 (30.1)	187 (40.8)	
First child born since CDAH follow-up 1	609 (21.0)	401 (24.5)	155 (19.4)	53 (11.6)	
Additional children since CDAH follow-up 1	571 (19.7)	311 (19.0)	176 (22.0)	84 (18.3)	
Same number of children	783 (27.0)	421 (25.7)	228 (28.5)	134 (29.3)	

Table S4. Univariable analyses about factors associated with non-manual or manual occupation level at childhood or CDAH follow-up 2

Variables	Total	Non-manual	Manual	P-value
Childhood factors				
Parental smoking status in 1985	2,257	1,654	603	0.001
None	1,327 (58.8)	1,013 (61.3)	314 (52.1)	
Father	291 (12.9)	198 (12.0)	93 (15.4)	
Mother	211 (9.4)	153 (9.3)	58 (9.6)	
Both	428 (19.0)	290 (17.5)	138 (22.9)	
The importance to be a non-smoker	2,266	1,663	603	0.153
Very important	1,792 (79.1)	1,321 (79.4)	471 (78.1)	
Of some important	266 (11.7)	191 (11.5)	75 (12.4)	
Of little important	102 (4.5)	81 (4.9)	21 (3.5)	
Not important	106 (4.7)	70 (4.2)	36 (6.0)	
Smoking experimentation	2,266			0.305
No	1,284 (56.7)	953 (57.3)	331 (54.9)	
Yes	982 (43.3)	710 (42.7)	272 (45.1)	
Intention to smoke in 1985 the following year	2,266			0.385
Yes	58 (2.6)	41 (2.5)	17 (2.8)	
No	1,839 (81.2)	1,361 (81.8)	478 (79.3)	
Don’t know	369 (16.3)	261 (15.7)	108 (17.9)	
Adulthood factors				
Partnering transitions	1,675	1,240	435	0.063
Not partnered both time points	238 (14.2)	182 (14.7)	56 (12.9)	
Became partnered	232 (13.9)	186 (15.0)	46 (10.6)	
Stayed partnered	1,133 (67.6)	818 (66.0)	315 (72.4)	
Became separated/divorced/widowed	72 (4.3)	54 (4.4)	18 (4.1)	
Parenting transitions	1,681	1,250	431	<0.001
No children both time points	511 (30.4)	420 (33.6)	91 (21.1)	
First child born since CDAH follow-up 1	373 (22.2)	285 (22.8)	88 (20.4)	
Additional children since CDAH follow-up 1	337 (20.1)	215 (17.2)	122 (28.3)	
Same number of children	460 (27.4)	330 (26.4)	130 (30.2)	

Table S5 Relative risks (95% CIs) and likelihood ratio test results for CDAH follow-up 2 smoking status by each life course model, after applying multiple imputation and inverse probability weighting*

Life course model	Smoking status	
	Former smokers	Current smokers
	RR (95% CI)	RR (95% CI)
No effect model	-	-
Sensitive period model		
Manual, baseline	0.85 (0.65, 1.13)	1.44 (1.02, 2.01)
Manual, CDAH follow-up 1	0.77 (0.57, 1.03)	1.66 (1.07, 2.57)
Manual, CDAH follow-up 2	1.27 (0.96, 1.68)	1.04 (0.68, 1.60)
Critical period model		
Manual, baseline	0.84 (0.64, 1.11)	1.53 (1.08, 2.15)
Manual, CDAH follow-up 1	0.86 (0.66, 1.12)	1.77 (1.28, 2.45)
Manual, CDAH follow-up 2	1.09 (0.84, 1.40)	1.47 (1.06, 2.04)
Accumulation model		
Linear	0.95 (0.83, 1.09)	1.34 (1.16, 1.56)
Social mobility model		
Inter-generational mobility	0.92 (0.80, 1.05)	1.18 (0.98, 1.43)
Intra-generational mobility	0.94 (0.80, 1.11)	1.27 (1.02, 1.57)

RR: relative risk, CI: confidence interval.

* All models were adjusted for age and sex at CDAH follow-up 2.

Bold RRs (95% CIs) indicate statistically significant results from the best fitting life course models.

Table S6 Sensitive period model for being current smokers at CDAH follow-up 2 adjusted for age, sex at CDAH follow-up 2 and potential mediators, after applying multiple imputation and inverse probability weighting *

Life course model	Model 1 Age and sex	Model 2 Model 1 + Parental smoking status	Model 3 Model 2 + The importance of being a non-smoker	Model 4 Model 1 + partnering and parenting transitions from CDAH follow-up 1 to 2
Sensitive period model				
Former smokers at CDAH follow-up 2				
Manual, baseline	0.96 (0.75, 1.24)	0.95 (0.73, 1.22)	0.94 (0.72, 1.21)	0.94 (0.73, 1.21)
Manual, CDAH follow-up 1	0.78 (0.59, 1.04)	0.79 (0.60, 1.05)	0.78 (0.58, 1.04)	0.79 (0.59, 1.06)
Manual, CDAH follow-up 2	1.18 (0.90, 1.55)	1.18 (0.89, 1.55)	1.17 (0.89, 1.55)	1.15 (0.87, 1.52)
Current smokers at CDAH follow-up 2				
Manual, baseline	1.41 (1.04, 1.91)	1.33 (0.99, 1.80)	1.27 (0.92, 1.76)	1.42 (1.05, 1.94)
Manual, CDAH follow-up 1	1.51 (0.99, 2.32)	1.59 (1.03, 2.45)	1.56 (1.00, 2.44)	1.36 (0.83, 2.25)
Manual, CDAH follow-up 2	1.19 (0.77, 1.82)	1.14 (0.74, 1.78)	1.14 (0.73, 1.79)	1.24 (0.77, 1.98)
Accumulation model				
Linear				
Former smokers at CDAH follow-up 2	0.96 (0.86, 1.09)	0.96 (0.86, 1.09)	0.95 (0.85, 1.08)	0.96 (0.85, 1.08)
Current smokers at CDAH follow-up 2	1.36 (1.19, 1.55)	1.34 (1.17, 1.53)	1.32 (1.15, 1.52)	1.33 (1.15, 1.54)

* Statistics presented are relative risks (95% confidence intervals).

Table S7 Likelihood ratio test results for CDAH follow-up 2 smoking status by each life course model, socioeconomic position determined by education

Life course model	Model fit and comparison to saturated model
	P-value*
No effect model	<0.001
Sensitive period model	0.211
Critical period model	
Without post-school qualification, baseline	<0.001
Without post-school qualification, CDAH follow-up 1	0.088
Without post-school qualification, CDAH follow-up 2	0.001
Accumulation model	0.771

* Bold *P*-values indicate that the tested life course model(s) adequately fits data as the saturated model, n=2,109.

Table S8 Relative risks (95% CIs) for smoking status at CDAH follow-up 2 for the best fitting life course models, socioeconomic position determined by education*

Life course model	Smoking status (n=2,109)†	
	Former smokers RR (95% CI)	Current smokers RR (95% CI)
Sensitive period model		
Without post-school qualification, baseline	0.90 (0.77, 1.05)	1.30 (1.05, 1.61)
Without post-school qualification, CDAH follow-up 1	1.31 (1.02, 1.68)	1.91 (1.35, 2.71)
Without post-school qualification, CDAH follow-up 2	0.93 (0.70, 1.23)	1.18 (0.83, 1.68)
Accumulation model		
Linear	1.04 (0.98, 1.11)	1.45 (1.33, 1.58)
Categorical, 0 time without post-school qualification	1	1
1 time without post-school qualification	0.96 (0.81, 1.15)	1.38 (1.05, 1.83)
2 times without post-school qualification	1.33 (1.09, 1.62)	2.61 (1.97, 3.46)
3 times without post-school qualification	1.02 (0.81, 1.29)	2.88 (2.17, 3.82)

RR: relative risk; CI: confidence interval.

* All models adjusted for age and sex at CDAH follow-up 2, n=2,109.

† Relative to never smokers.

Bold indicates statistically significant results.

Table S9 Likelihood ratio test results for CDAH follow-up 2 smoking status by each life course model, socioeconomic position determined by area-level disadvantage

Life course model	Model fit and comparison to saturated model
	P-value*
No effect model	<0.001
Sensitive period model	0.493
Critical period model	
Manual, baseline 1985	<0.001
Manual, follow-up 1 2004-6	<0.001
Manual, follow-up 2 2009-11	<0.001
Accumulation model	<0.001
Social mobility model	
Inter-generational mobility	<0.001
Intra-generational mobility	<0.001

* Bold *P*-values indicate that the tested life course model(s) adequately fits data as the saturated model, n=2,049.

Table S10 Relative risks (95% CIs) for smoking status at CDAH follow-up 2 for the best fitting life course model, socioeconomic position determined by area-level disadvantage*

Life course model	Smoking status (n=2,109)†	
	Former smokers RR (95% CI)	Current smokers RR (95% CI)
Sensitive period model		
High disadvantaged, baseline	0.71 (0.62, 0.83)	0.99 (0.81, 1.21)
High disadvantaged, CDAH follow-up 1	1.01 (0.86, 1.19)	1.41 (1.10, 1.80)
High disadvantaged, CDAH follow-up 2	1.09 (0.92, 1.28)	1.40 (1.10, 1.80)

RR: relative risk; CI: confidence interval.

* All models adjusted for age and sex at CDAH follow-up 2, n=2,049

† Relative to never smokers.

Bold indicates statistically significant results.

Chapter 4

The association between quitting smoking and weight gain: a systematic review and meta-analysis of prospective cohort studies

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Chapter 4 The association between quitting smoking and weight gain: a systematic review and meta-analysis of prospective cohort studies

4.1 Preface

Fear of weight gain is a common reason smokers do not try to quit, and weight gain after cessation increases the risk of relapse. Providing an accurate estimate of post-cessation weight gain in smokers is therefore important; however, the estimated magnitude of the weight gain varies widely in the literature. An editorial was recently published in the British Medical Journal in which the authors called for a meta-analysis of population-based cohort studies to settle this question, prompting the study presented in this chapter. The following text of this chapter has been published in the journal Obesity Reviews.

4.2 Introduction

As note earlier, cigarette smoking remains a considerable risk to public health being responsible for nearly 6 million deaths worldwide every year, with half of current smokers estimated to eventually die of a tobacco-related disease [1]. Quitting smoking substantially reduces these health risks [2,3], but is difficult to achieve. Although the vast majority of adult daily smokers report they would like to quit, less than half report attempting to quit [4] and less than 5% of unaided quit attempts result in prolonged abstinence [5]. The reasons for this are many, but one commonly cited reason is fear of weight gain, particularly among females [6,7] and those who are obese [8]. Cooper and colleagues reported that worries about weight gain discourage half of female and a quarter of male smokers from trying to quit smoking [9]. Pisinger et al. reported that 52% of women and 32% of men relapse due to weight gain after quitting [10]. Perceptions about smoking and weight are also associated with the uptake of smoking, with some authors reporting smoking as a strategy to control or lose weight among adolescents [11], especially in girls [12], younger adults [13] and smokers who experienced weight gain in the previous quit attempt [14].

The fear regarding cessation and weight gain is not unfounded. In a large comprehensive narrative review based on 41 prospective studies completed over 25 years ago, the authors showed that those who quit smoking gained an average of 2.9 kg [15]. However, it was not clear what method was used to combine estimates. Authors for a recent meta-analysis including 62 RCTs concluded that smoking cessation was associated with a mean weight gain

of 4-5 kg after 12 months of abstinence [16]. Although this might seem like unequivocal evidence that smoking cessation leads to weight gain, the study prompted considerable discussion about the generalisability of the findings to all smokers [17]. This is because participants in RCTs of smoking cessation treatments are usually not representative of the general population of smokers [18]. Those who seek help to quit smoking tend to be more dependent on nicotine [19], have previously quit and relapsed [20] and may lack self-efficacy [21] compared to those who do not seek help to quit. Importantly, these characteristics may also be associated with weight gain potentially resulted in a biased estimate. Indeed, up to three quarters of successful ex-smokers quit smoking unaided [20,22]. The authors for the meta-analysis of RCTs reported weight gain only among those who quit smoking with no reference to those who continued to smoke. As we know that, on average, adults gain weight irrespective of smoking status as they age [23,24], it is important to understand the difference in weight gain for those who quit relative to those who continue to smoke.

Population-based studies examining change in smoking status and change in weight longitudinally may provide a more generalisable estimate of weight gain after cessation [17]; however, no meta-analysis has been undertaken, nor has a meta-analysis of the difference in weight gain between quitters and continuing smokers been conducted. Therefore, our aim was to conduct a systematic review and meta-analysis of prospective cohort studies that examined associations between smoking cessation and change in measures of body size, including weight, BMI and waist circumference. Given the evidence from RCTs that weight gain was higher in groups with higher nicotine dependence, and smokers in RCTs were more likely to have this characteristic, we hypothesised that the magnitude of weight gain following smoking cessation in population-based observational study participants would be smaller than the weight gain in RCT participants selected to test pharmacological interventions.

4.3 Methods

We reported this meta-analysis in accordance with PRISMA[25] and MOOSE guidelines [26]. The published protocol is available on the database of the international prospective register of systematic reviews (PROSPERO, registration number: CRD42014010076).

4.3.1 Search strategy

A systematic hand literature search was performed using the following electronic databases: Medline, EMBASE, Web of Science, SCOPUS and CINAHL for articles published prior to January 2015. Search filters designed by Scottish Intercollegiate Guidelines Network for observational studies were incorporated in the electronic database search strategies [27]. Search terms were taken from each database's vocabulary tool where available. No language restriction was enforced. The search strategy was implemented by the research team and an expert librarian. The detailed search terms in each database can be found in **Appendix 4-1**. In an attempt to identify all relevant studies, citation lists and the bibliographies of review articles, monographs, and the studies included were also scrutinized. Discrepancies in the outcome of the scrutinizing procedure between two investigators were addressed by consensus after discussion.

4.3.2 Inclusion and exclusion criteria

Studies were included if they satisfied the following criteria:

1) Population: participants were adult smokers from population-based cohort studies;
2) Exposure: the exposure of interest was smoking cessation. Available data on those who quit smoking and those who continued smoking during follow-up;
3) Outcome: the outcome of interest was change in body size. Measurement of body size before quitting and at least three months after quitting;
4) Study design: prospective cohort studies;
5) Sufficient data: for continuous endpoints; sample size, mean and SD of change in weight, BMI or waist circumference, or data from which these could be calculated. For categorical endpoints; the number of quitters and continuing smokers in each category of body size change, or sufficient data to calculate these for at least one follow-up time point.

We excluded reviews, non-human studies, and studies without sufficient data.

4.3.3 Selection of studies

Two reviewers (JT and SG) independently identified the eligible papers. The initial screening assessed the titles and abstracts and was set to be relatively broad to retain as many relevant studies as possible. A full text review of potentially eligible papers identified from the initial screening by both of the reviewers was then undertaken. When published

information was insufficient, the corresponding author was contacted to obtain further information. If we failed to contact an author, that study was excluded from the review. If multiple articles were on the same study sample with the same exposure and outcome, the most recent publication or the one with the largest sample size was retained. Endnote X4 (http://www.endnote.com) was used to manage the located records. Discrepancies were resolved by discussion between the two reviewers, with a third (AV) providing input for those where a decision could not be made.

4.3.4 Data collection process

Data were extracted by two independent authors (JT and PO) using a standardised record form. Discrepancies were resolved by consensus among four authors (JT, PO, SG and AV). The following data were recorded: name of the first author, year of publication, country, sample size, age range of the study population, duration of follow-up, numbers in exposure categories during follow-up, crude and adjusted mean and SD of weight, BMI and waist circumference change, and/or crude and adjusted RRs or ORs of weight gain according to smoking status. When available, we also extracted the following: gender split of the study population, baseline body size, baseline difference of body size between quitters and continuing smokers, amount of cigarette consumption, and measurement methods for exposure and outcome. Corresponding authors were contacted for further information when the information was insufficient.

4.3.5 Assessment of study methodological quality

Two investigators (JT and AV) independently appraised the methodological quality of included studies and resolved any differences. The assessment was based on the Newcastle-Ottawa Quality Assessment Scale (NOQAS) [28], which is a validated scale for non-randomized studies in meta-analysis. Modifications were conducted to accommodate the topic of this review (**Appendix 4-2**). There were a total of 7 items within three categories in the adapted NOQAS scale: three for selection of participants and measurement of exposure, two for comparability of cohorts on the basis of the design or analysis, and two for outcomes assessment and adequacy of follow-up. We deemed studies with a rating of less than four as low quality. The quality of studies were considered in sensitivity analyses rather than as weightings in the main analyses [26].

4.3.6 Statistical methods

4.3.6.1 Data extraction and imputation

The summary measure was the change in body size over time, which included weight, BMI and waist circumference, stratified by smoking status defined as quitting or continuing smoking. All weights were converted to kilograms and circumferences to centimeters. Continuous outcome measures were the mean and SD of absolute changes in these measures of body size over the follow-up period. Categorical measures of change in weight were also extracted, including the number of participants in each weight change category. A variety of cut-off points to define weight gain were used across the studies (see 'results' for details).

For studies that provided the mean and SD of weight/BMI/waist circumference at baseline and follow-up rather than the change, we calculated the mean change; subtracting the follow-up mean from the baseline mean and associated SD of these changes using the recommended formulae [29]. For studies that reported CIs and P-values from t-test or F-test but not the actual SD, the SD was calculated from the table of t-distribution or F-distribution with corresponding degrees of freedom [29].

4.3.6.2 Meta-analysis method

Given the observational nature of the studies and high heterogeneity between effect sizes, random-effects models were used to calculate pooled mean differences (MDs) and RRs. For continuous outcomes, a pooled MD in weight/BMI/waist circumference and 95% CI between those who quit smoking and those who continued smoking were computed. For categorical outcomes, we estimated pooled RRs for each weight gain category (see 'results') according to smoking status. For all analyses, a two-tailed p-value of less than 0.05 was considered statistically significant.

4.3.6.3 Assessment of heterogeneity

Heterogeneity assumption was assessed using the Chi-square test based *Q*-statistic and I^2 statistics [30-32], which reflect the extent of heterogeneity across studies and inter-study heterogeneity, respectively. Random effects meta-regression analyses were performed to explore the possible sources of heterogeneity and their effects on the results.

4.3.6.4 Assessment of publication bias

Publication bias was evaluated using the visual inspection of a funnel plot and further assessed by the Eggers test [32,33]. An asymmetric plot suggested a possible publication bias, and updated estimates of the pooled effect size were assessed using Duval and Tweedie's trim and fill method. When the p-value equaled or was less than 0.10, significant publication bias was considered.

4.3.6.5 Subgroup analyses

Given the possibility that change in weight could be confounded by other characteristics that were different between those who quit and those who continued to smoke, the pooled estimates were calculated separately for studies with crude and adjusted results. When data were sufficient and appropriate, subgroup analyses by representativeness of the cohort, smoking status measurement, sex, outcome assessment, adequacy of follow-up, geographic region, and baseline cigarette consumption were performed.

4.3.6.6 Sensitivity analyses

We evaluated the robustness of our findings via several methods. The first method was by removing studies that are liable to present a risk of bias (studies with less than four stars in the quality assessment) and compared the pooled estimates with and without the excluded studies. Second, we performed the Duval and Tweedie nonparametric "trim and fill" procedure to consider whether the hypothetical "missing" studies affect our result [34]. Third, to assess possible bias in assumptions made during estimation of SDs we removed studies for which SDs for change in weight or BMI were not reported.

Statistical analyses were performed using R 3.1.1 (R Core Team (2014). R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. URL http://www.R-project.org/.).

4.4 Results

4.4.1 Study selection

A total of 6,733 papers were retrieved from an initial search in five electronic databases (**Figure 4-1**). After exclusion of duplicates (n = 1,804), the titles and abstracts of 4,929 records were initially reviewed, with 147 potentially relevant papers undergoing full text review. Of these articles, 94 were excluded, leaving 53 papers for systematic review. Of

these, only 30 could be included with other studies excluded due to insufficient (n = 9) and overlapping (n = 14) data. We also identified six papers by reviewing the reference lists of review articles, monographs, and included papers in the systematic review [35-40]. In total, we identified 45 studies eligible for inclusion with 36 of these included in the meta-analysis.

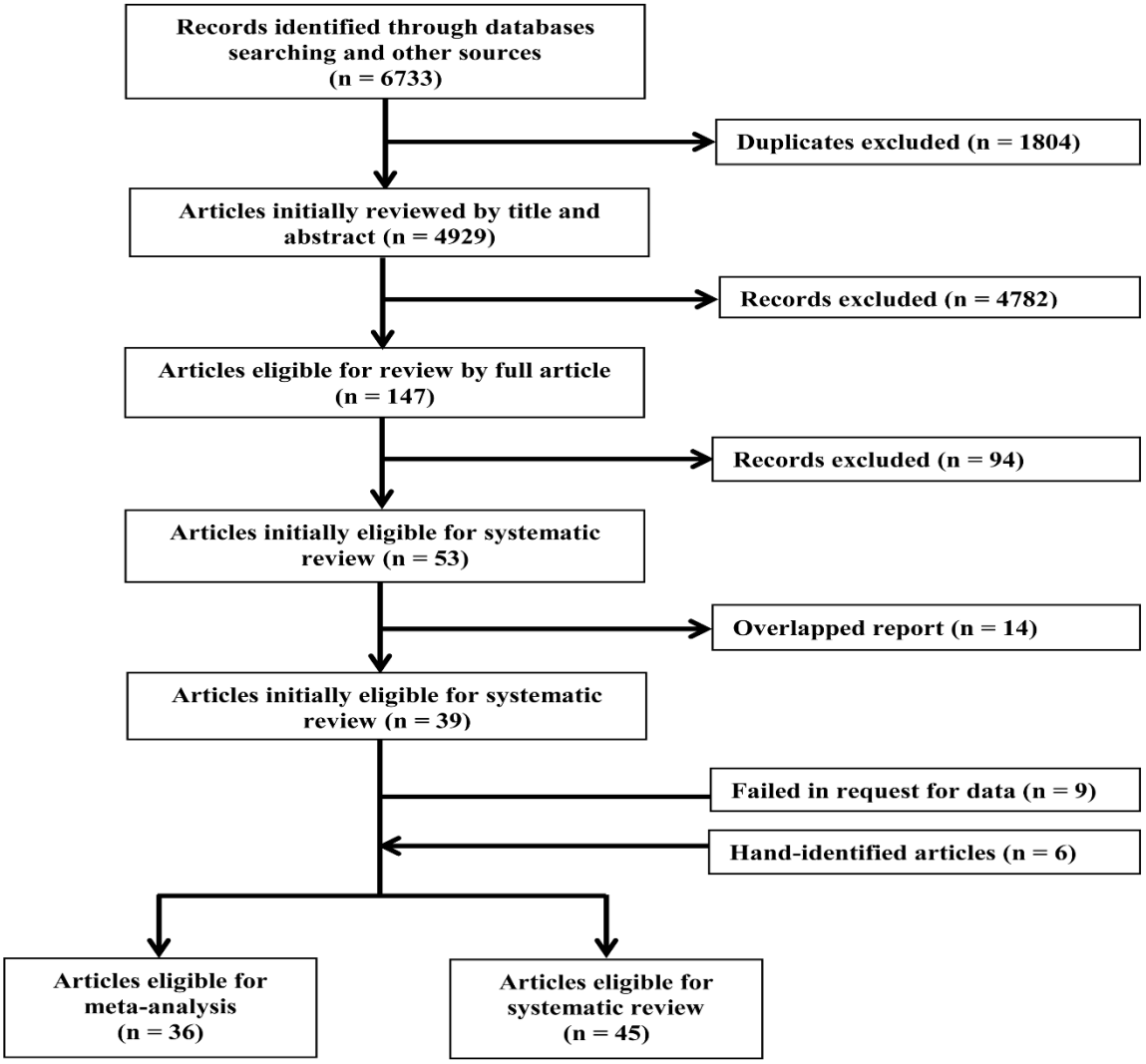

Figure 4-1 Flow chart of articles identified in search and included in meta-analysis

Two papers reported different indicators of weight change from the same study: one [41] reported weight (kg) change and the other [42] reported BMI change. We treated these as one study with two measures of body size. Of the nine papers without sufficient data for meta-analysis, one paper reported the same indicators of weight change from two different cohorts [43]. We treated this as two different studies. Therefore, the total number of eligible studies was 45, with 35 studies included in the meta-analysis.

4.4.2 Outcome categories

Of the 35 studies, there were 24, 15, and two reporting continuous changes in weight, BMI and waist circumference, respectively. For weight change, the majority of studies (n = 14) reported only crude changes, with the remainder reporting only an adjusted result (n = 6) or both crude and adjusted results (n = 4). For studies of change in BMI, ten studies reported crude BMI change only, three reported adjusted results only and two reported both crude and adjusted results.

Various categorical outcomes were reported for weight change across the studies (n = 12, **Appendix 4-3**). For absolute weight change (n = 10), the most frequent categories were weight gain of at least 3 kg (n = 2), 5 kg (n = 4) and 10 kg (n = 3). Other categories reported were weight gain of at least 0.91 kg (n = 1), l kg (n = 1), 2.25 kg (n = 1), 2.3 kg (n = 1), more than 4 kg (n = 1), at least 10 lb (about 4.5 kg, n = 1), more than 8 kg (n = 1), at least 20 lb (about 9.1 kg, n = 1) and 11.3 kg (n = 1). To facilitate meta-analysis, we created the following subgroups: weight gain of at least 1 kg (n = 2), 2.5 kg (n = 4), 5.0 kg (n = 6) and 10.0 kg (n = 6) with studies reporting cut offs within a 20% range of these included in each subgroup. For relative weight change (n = 2), one study reported weight change of more than 4% and one reported change of at least of 5%. Similarly, we pooled these two studies together. No study reported categorical BMI or waist circumference change.

4.4.3 Extraction and imputation

Four SDs of weight change were calculated from F-distribution (n = 1) [44] t-distribution (n = 1) [45] and baseline and follow-up values (n = 2) [46,47]. Five SDs of BMI change were calculated from F-distribution (n = 2) [44,48], t-distribution (n = 1) [45] and baseline and follow-up BMI (n = 2) [49,50]. No imputation was performed for waist circumference change. Ten studies reported effects stratified by sex [24,39,44,47,48,51-55]; weighted mean and SDs were calculated from these studies to estimate overall effect.

4.4.4 Study characteristics

Characteristics of the 35 identified studies are summarized in **Table 4-1**. The follow-up length ranged from 1 to 16 years (mean 5.2 years). The earliest study was published in 1975, and the latest was published in 2013. The number of participants per study ranged from 111 to 300,767, with a total of 451,835 participants across studies. Similar numbers of studies

had sufficient data for meta-analysis in males (n = 25) and females (n = 21). Five studies analysed the male and female data together. Most studies were conducted in North America (n = 13) or Europe (n = 11) with the remainder from Asia (n = 8), Australia (n = 2), and Africa (n = 1).

Table 4-1 Detailed characteristics of included studies in meta-analysis

ID	Author	Year	Country	Study name	Target population	Sample size (Q/CS)	Sex	Maximum FU(year)	Outcome (change)
1	Gordon [56]	1975	USA	The Framingham Heart Study	Adult population of Framingham, Massachusetts	58/464	M	6	Weight
2	Bosse [35]	1980	USA	Normative Aging Study	Males, predominantly white, middle-class	237/468	M	5	Weight
3	Friedman [57]	1980	USA	Kaiser-Permanente multiphasic check-up	Voluntary subscribers	2738/6810	M/F	1.5	Weight
4	Williamson [54]	1991	USA	National health and Nutrition Examination Survey and Follow-up Study	Non-institutionalized civilian population	959/1885	M/F	9.9	Weight
5	Noppa [41] Lissner [42]	1980 1992	Sweden	Prospective Population Study of Women	Middle-aged female residents	72/454	F	6	Weight and BMI
6	Swan [40]	1995	USA	Two surveys of the National Academy of Sciences-National Research Council Twin Registry	Adult male twins born between 1917 and 1927	2179/1569	M	16	Weight
7	Hodge [48]	1996	Mauritius	---	Adults	227/815	M/F	5	BMI
8	Kawachi [46]	1996	USA	The Nurses' Health Study	Registered nurses	1276/5148	F	2	Weight
9	Burnette [36]	1998	USA	Healthy Women Study	Premenopausal women with a driver's license	26/85	F	4.8	Weight
10	Bartholomew [51]	1998	Australia	Busselton Population Health Surveys	All adult residents listed on the electoral roll	235/526	M/F	6	Weight
11	Klesges [23]	1998	USA	Coronary Artery Risk Development in Young Adults Study(CARDIA)	Permanent young residents	156/744	M/F	7	Weight

ID	Author	Year	Country	Study name	Target population	Sample size (Q/CS)	Sex	Maximum FU(year)	Outcome (change)
12	Froom [38]	1999	Israel	CORDIS	Male factory workers	65/392	M	2.6	BMI
13	Burke [44]	2000	USA	San Antonio Heart Study(SAHS)	Randomly selected from low-, middle- and high-income neighbourhoods in San Antonio	293/445	M/F	8	Weight and BMI
14	Goya Wannamethee [58]	2001	Britain	The British Regional Heart Study	General practice registers	567/1980	M	5	Weight
15	Lee [59]	2001	Korea	---	Male healthy workers aged 25-50 years	708/5372	M	4	Weight
16	Janzon [37]	2004	Sweden	---	Female residents	388/1162	F	9	Weight
17	Brown [60]	2005	Australia	The Australian Longitudinal Study on Women's Health (ALSWH)	Middle-age women	286/1063	F	5	Weight
18	Chinn [39]	2005	Europe	The European Community Respiratory Health Survey	Residents	555/1604	M/F	Annual change in 9 years	Weight and BMI
19	John [61]	2006	German	Transitions in Alcohol Consumption and Smoking(TACOS)	Residents of Lubeck city	77/549	M/F (C)	3	BMI
20	Sneve [55]	2008	Norway	Tromsø study	Birth cohort and residents	395/1279	M/F	7	BMI
21	Song [62]	2008	South Korea	Korean National Health System Study	Male civil servants	27700/273067	M	2	BMI
22	Byung [63]	2009	South Korea	---	Male residents of Seoul or Kyung-gi province	496/1292	M	2.9	Weight

ID	Author	Year	Country	Study name	Target population	Sample size (Q/CS)	Sex	Maximum FU(year)	Outcome (change)
23	Munafò [50]	2009	UK	The Caerphilly Prospective Study	Men residents from the town of Caerphilly and adjoining villages	137/506	M	14	BMI
24	Reas [52]	2009	Norway	OsLof Study	Adult residents	361/368	M/F	11	Weight and BMI
25	Basterra-Gortari [53]	2010	Spain	The SUN study	University graduates	614/1509	M/F	4.2	Weight
26	Holz [64]	2010	Germany	The World Health Organization MONICA Project (Monitoring of Trends and Determinants in Cardiovascular Disease), the 3rd Augsburg survey	People living in southern Germany(Augsburg city, and the counties of Augsburg and Aichach-Friedberg)	214/452	M/F	9.6	Weight, BMI and waist circumference
27	Kawada [49]	2010	Japan	---	Company workers	59/1006	M	1	BMI
28	Suwazono [65]	2010	Japan	---	Steel company workers	445/2403	M	3	Weight and BMI
29	Yeh [66]	2010	USA	The Atherosclerosis Risk in Communities(ARIC) Study	Middle-aged adults	380/2018	M/F (C)	3	Weight and waist circumference
30	Yoon [45]	2010	South Korea	---	Hospital visitors who had a complete preventive medical evaluation	226/950	M	1.6	Weight and BMI
31	Hansson [67]	2011	Sweden	Stockholm Public Health Cohort Study	Residents of Stockholm County	284/729	M	5	Weight
32	Luo [68]	2012	USA	The Women's Health Initiative (WHI)	Postmenopausal women	2054/5335	F	3	Weight
33	Oba [47]	2012	Japan	The Japan Public Health Centre-Based	Residents of 11 public health centre areas	2242/13136	M/F	5	Weight and BMI

ID	Author	Year	Country	Study name	Target population	Sample size (Q/CS)	Sex	Maximum FU(year)	Outcome (change)
				Prospective Study(JPHC)					
34	Travier [24]	2012	10 European countries	European Prospective Investigation into Cancer and Nutrition-Physical Activity, Nutrition, Alcohol, Cessation of smoking, Eating out of home And obesity (EPIC-PANACEA)	General population in 23 centres from 10 European countries	15664/51721	M/F	1	Weight
35	Clair [69]	2013	USA	Framingham Offspring Study	Offspring cohort of Framingham Heart Study	1030/1126	M/F (C)	4	Weight

CS: continuing smokers; F: participants are only female; FU: follow-up; M: participants are only male; M/F: participants include both male and female, but the data were analysed separately; M/F(C): participants include both male and female, and the data was analysed as a whole; Q, quitters.

4.4.5 Quality assessment

The results of methodological quality assessment for each individual study are summarized in **Appendix 4-4**. Most studies had truly or somewhat representative samples for analysis (n = 26). At follow-up, no study bio-verified the smoking status, and eight studies collected the smoking data from structured interview. Over half of studies (n = 19) controlled for both age and sex on the basis of design or analysis, and there were eight studies that controlled for socio-economic status or illness. Regarding the assessment of outcome, most studies collected the data from objective measurements (n = 22). No study reported complete follow-up of participants. Nine studies had follow-up of more than 80% (thus reducing the likelihood of bias) or compared those with and without follow-up, or discussed the effect of loss to follow-up in the limitations. Twenty-one studies were scored medium to high quality on the modified Newcastle-Ottawa scale, and 14 had a rating of less than four out of a possible score of 7, suggesting a higher risk of bias. One study published as a brief report scored only one point due to insufficient information [63].

4.4.6 Association between smoking cessation and change in continuous measures of weight, BMI and waist circumference

Of the eligible studies identified, the vast majority (n = 43) reported data on changes in continuous measures of body size except two [59,68]. Of the eligible studies included in meta-analysis (n = 35), 31 studies reported that those who quit smoking gained significantly more weight, BMI or waist circumference than those who continued smoking; however, three studies [48,57,69] showed no significant difference in weight or BMI gain between these two groups. Notably, two of these three studies used imputed or collapsed data: one [69] required estimation of overall weight gain across two groups (people who did and did not develop diabetes during follow-up) and collapsing of weight change in quitters across two time periods: recent (≤ 4 years) and long-term (> 4 years). The authors stated that recent quitters gained more weight than continuing smokers and long term quitters, with no statistically significant difference in weight gain between long-term quitters and continuing smokers. The other paper also reported a statistically significant difference of BMI [48] gain but only in men. There was one study [58] that recorded more weight gain in quitters than any other smoking group including continuing smokers group but did not state whether it was a statistically significant difference.

Of the ten studies excluded due to insufficient data for meta-analysis [43,53,70-76], one reported significantly greater weight gain in quitters relative to continuing smokers [70]. Six studies [43,53,71-73] used stable never smokers as the reference group, and reported significant greater weight gain in quitters and significant weight loss in continuing smokers. Based on this information, we could conclude that there was a significant difference in weight gain between quitters and continuing smokers; however, the significant difference disappeared after considering change in BMI as a potential confounder in one study [72]. There were some studies [74-76] that described the magnitude of weight gain in quitters and continuing smokers but did not report whether it was significant.

No studies reported that those who quit smoking lost weight compared to those that continued smoking.

4.4.6.1 Effects of smoking cessation on weight change

Of the 33 studies with data on changes in continuous measures of weight, 24 had sufficient data to be included in the meta-analysis. Of the remaining nine studies, four reported significantly greater weight gain in quitters than continuing smokers [43,53,70], one reported no statistical difference between these two groups [57], while the remaining four only gave the magnitude of weight gain without statement of whether the difference was significantly different [58,74-76].

Figure 4-2 shows the pooled effects of quitting smoking on continuous weight change in quitters and continuing smokers. We were able to pool crude data from 18 studies (n = 26,313 quitters and n = 82,962 continuing smokers) and adjusted data from 10 studies (n = 18,606 quitters and n = 62,936 continuing smokers). The unadjusted average of weight gain was 3.41 kg for quitters and 1.39 kg for continuing smokers over an average of 5.73 years follow up. The pooled magnitude of weight gain was slightly higher in studies with adjustment, with quitters gaining 4.1 kg and continuing smokers gaining 1.47 kg over an average of 5.15 years follow up. Compared with those who continued to smoke, those who quit smoking had significantly greater crude weight gain during follow-up (MD: 1.76 kg; 95% CI: 1.47 to 2.05; $p < 0.001$). Analyses using adjusted estimates of weight change suggested a stronger effect (MD: 2.61 kg, 95% CI: 1.61to 3.60; $p < 0.001$). The test for heterogeneity was

statistically different for both crude ($p < 0.001$; $I^2 = 97\%$) and adjusted ($p < 0.001$; $I^2 = 98\%$) estimates, indicating substantial inter-study variation.

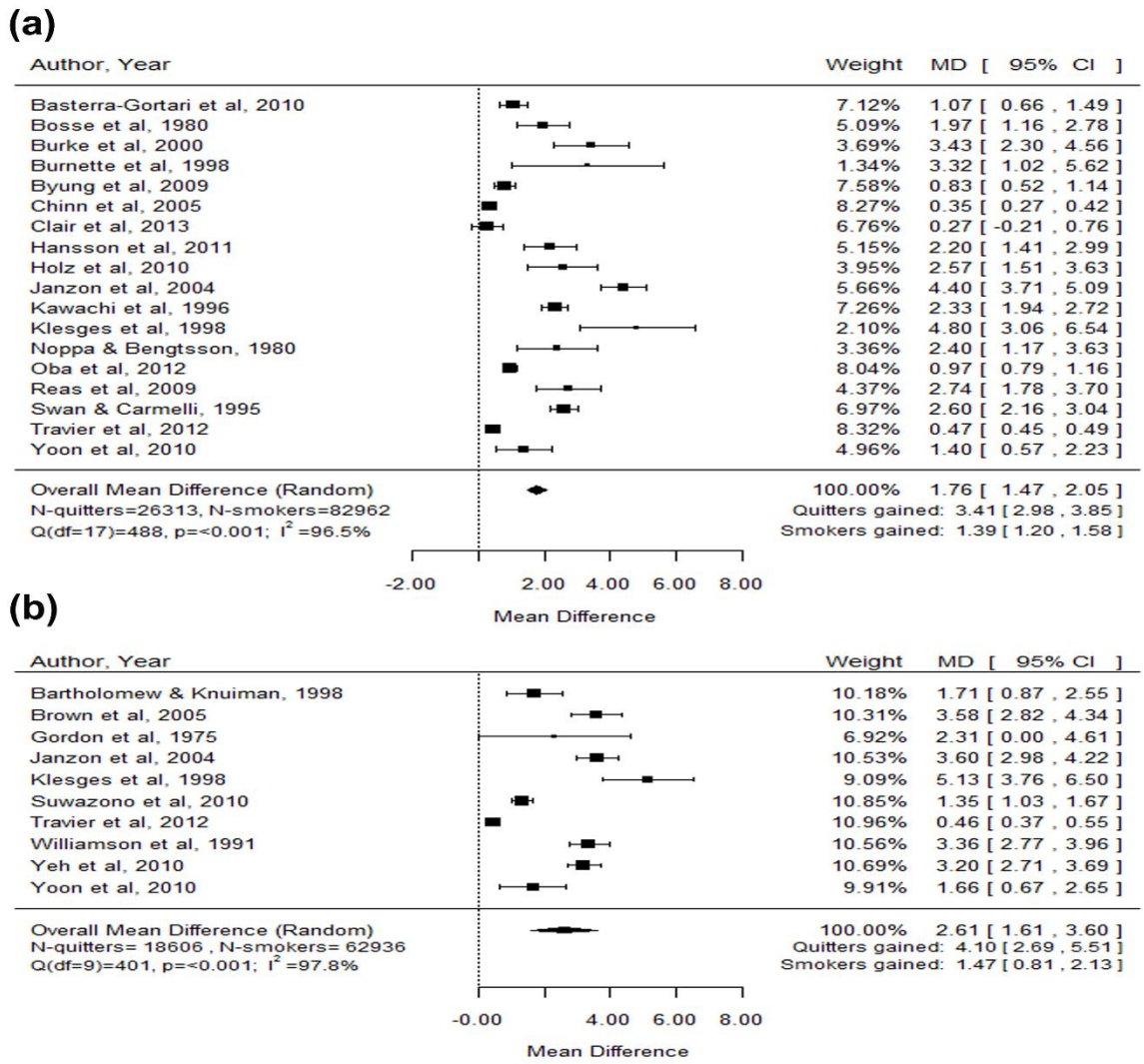

Figure 4-2 Association between smoking cessation and change in absolute weight (kg) from baseline to longest follow-up in quitters and continuing smokers. (a) Crude result; (b) Adjusted result.

4.4.6.2 Effects of smoking cessation on BMI change

There were 17 studies where changes in continuous measures of BMI were examined, 15 had sufficient data for meta-analysis (**Figure 4-3**). The remaining two studies reported quitters had greater increase in BMI than continuing smokers [71,73].

Twelve studies with 32,190 quitters and 294,201 continuing smokers reported crude changes in BMI and five studies of 1,160 quitters and 4,548 continuing smokers reported adjusted changes in BMI. The unadjusted average of BMI gain was 1.09 kg/m^2 for quitters

and 0.39 kg/m^2 for continuing smokers over an average of 4.99 years follow up. A similar average of BMI gain was found in studies with adjustment, with 1.14 kg/m^2 BMI gain in quitters and 0.44 kg/m^2 BMI gain in continuing smokers during 4.84 years follow up. In crude analyses, those who quit smoking had significantly greater BMI gain overtime (MD: 0.60 kg/m^2; 95% CI: 0.48 to 0.72; p < 0.001). The effect was slightly greater when adjusted MDs in BMI were considered (MD: 0.63 kg/m^2; 95% CI: 0.46 to 0.80; p < 0.001). There was evidence of statistical heterogeneity across studies presenting crude results (p < 0.001; I^2 = 96%), and weaker evidence in studies reporting adjusted estimates (p = 0.137; I^2 = 43%).

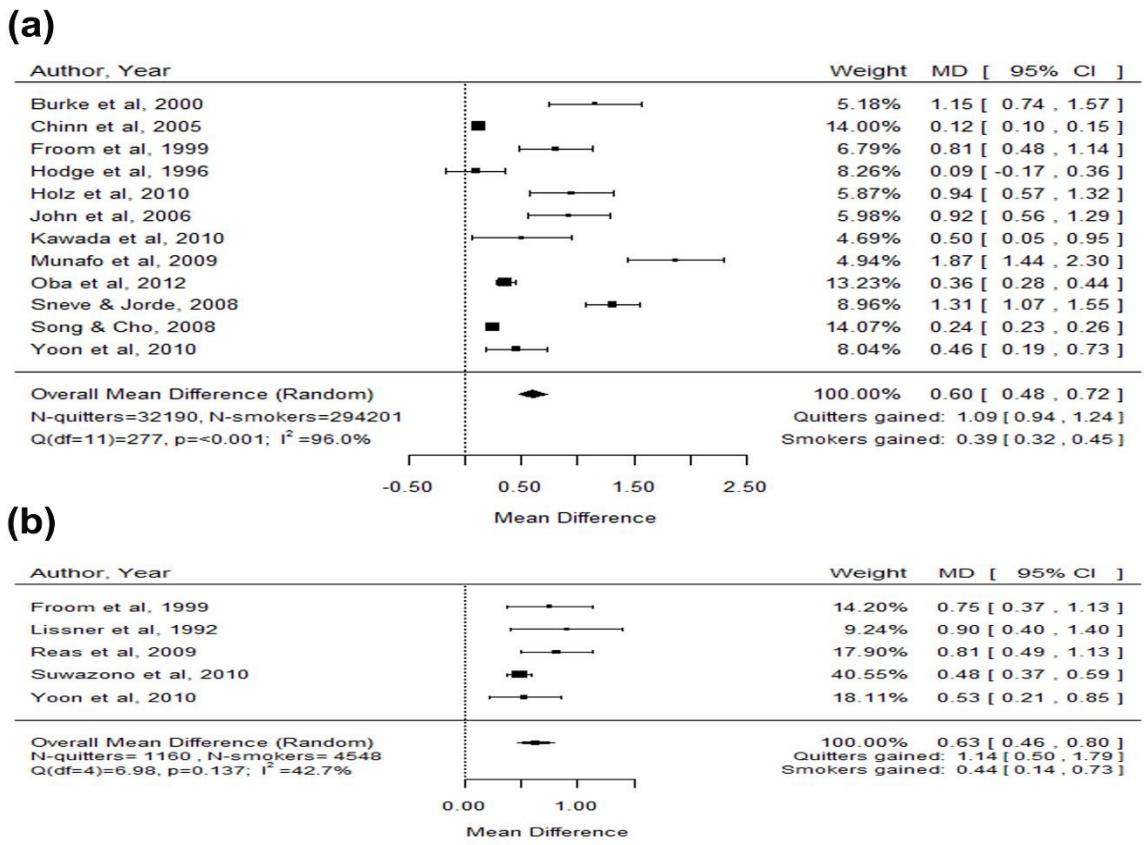

Figure 4-3 Association between smoking cessation and change in BMI (kg/m^2) from baseline to longest follow-up in quitters and continuing smokers. (a) Crude result; (b) Adjusted result.

4.4.6.3 Effects of smoking cessation on waist circumference change

Among the four studies with continuous measures of change in waist circumference for those who quit and those who continued smoking, only two studies had the required data for meta-analysis with one reporting a crude result and the other an adjusted result. As per our protocol we did not combine these for analysis. Both studies reported increases in waist circumference among quitters (crude Mean (SD): 9.11 (7.34) cm; adjusted Mean (SD): 3.20

(6.46) cm) and continuing smokers (crude Mean (SD): 6.93 (6.91) cm; adjusted Mean (SD): 0.60 (5.73) cm). However, the increases were greater in quitters than continuing smokers: crude MD 2.18 cm; 95% CI: 1.01 to 3.35 [64] and adjusted MD 2.60 cm; 95% CI: 1.97 to 3.23 [66]. Of the two studies with insufficient data, one reported greater waist circumference gain in quitters than in continuing smokers [71], while the association was unclear in the other [72].

4.4.7 Association between smoking cessation and changes in weight category

4.4.7.1 Effects of smoking cessation on absolute weight gain of at least 1 kg

Two studies among 945 quitters and 5,840 continuing smokers reported categorical weight gain of at least 1 kg, with follow-up ranging from 4 to 5 years (mean 4.5 years). Compared with continuing to smoke, quitting smoking was associated with a higher risk of gaining weight of 1 kg or more (RR: 1.26; 95% CI: 1.08 to 1.47; p = 0.003; **Table 4-2**). There was significant heterogeneity between studies (p = 0.033; I^2 = 78%).

4.4.7.2 Effects of smoking cessation on absolute weight gain of at least 2.5 kg

Four studies reported categorical weight gain of at least 2.5 kg, with follow-up ranging from 5 to 16 years (mean 9.0 years). Quitting smoking was associated with gaining weight over time, with 45% of those who quit smoking (n = 5,666) and 24% of those who continued to smoke (n = 17,653) gaining at least 2.5 kg during follow-up. Quitters had a 49% higher risk of gaining weight of 2.5 kg or more compared with continuing smokers (RR: 1.49; 95% CI: 1.31 to 1.70; p < 0.001; **Table 4-2**). The studies demonstrated significant heterogeneity (p < 0.001; I^2 = 90%).

4.4.7.3 Effects of smoking cessation on absolute weight gain of at least 5 kg

Six studies of 6,014 quitters and 19,778 continuing smokers reported comparisons of categorical weight gain of at least 5 kg, with follow-up ranging from 1.5 to 7 years (mean 4.4 years). Those who quit smoking were significantly more likely to gain at least 5 kg during follow-up than those who continued to smoke (RR: 1.84; 95% CI: 1.53 to 2.21; p < 0.001). There was significant heterogeneity observed across studies (p < 0.001; I^2 = 91%; **Table 4-2**).

4.4.7.4 Effects of smoking cessation on absolute weight gain of at least 10 kg

Results for studies that examined weight gain of 10 kg or more (n = 6,390 quitters and 12,525 continuing smokers) were consistent with the previously described results, although associations were somewhat stronger. The follow-up length ranged from 1.5 to 16 years

(mean 7.6 years). Those who quit smoking had a two-fold risk of weight gain of at least 10 kg over follow-up (RR: 2.14; 95% CI: 1.78 to 2.57; $p < 0.001$). Significant heterogeneity between these studies was observed ($p = 0.015$; $I^2 = 64\%$; **Table 4-2**).

4.4.7.5 Effects of smoking cessation on relative weight gain of 5%

Two studies including 851 quitters and 2,709 continuing smokers reported an outcome of relative weight gain of more than 5%. Among these studies, 50% of quitters and 30% of continuing smokers gained over 5% of their baseline weight equating to a pooled RR of 1.70 (95% CI: 1.56 to 1.86; $p < 0.001$). There was no evidence of heterogeneity between these studies ($p = 0.652$; $I^2 = 0\%$; **Table 4-2**).

Table 4-2 Association between smoking status and change in weight category from baseline to longest follow-up in quitters and continuing smokers

Group	Study No.	Quitters			Continuing smokers			P^H	I^2 (%)	RR (95% CI)
		Events	Total	%	Events	Total	%			
Absolute weight change: ≥ 1kg	2	667	945	70.6	3526	5840	60.4	0.033	78.1	**1.26 (1.08, 1.47)**
Absolute weight change: ≥ 2.5kg	4	2534	5666	44.7	4252	17653	24.1	< 0.001	89.8	**1.49 (1.31, 1.70)**
Absolute weight change: ≥ 5kg	6	1592	6014	26.5	3113	19778	15.7	< 0.001	91.1	**1.84 (1.53, 2.21)**
Absolute weight change: ≥ 10kg	6	725	6390	11.3	709	12525	5.7	0.015	64.4	**2.14 (1.78, 2.57)**
Relative weight change: ≥ 5%	2	428	851	50.3	812	2709	30.0	0.652	0.0	**1.70 (1.56, 1.86)**

Bold denotes statistically significant result

CI: confidence interval; P^H: P-value of heterogeneity; RR: risk ratio.

4.4.8 Subgroup analyses

The results of subgroup analyses are summarized in **Table 4-3** and **Table 4-4**. We were unable to examine the effect of cigarette consumption before quitting on the magnitude of body size change. Three studies had the relevant information but with disparate measurement of cigarette consumption and outcomes [41,55,56]. The difference of weight or BMI gain between groups was bigger in studies with cohorts that were truly or somewhat representative of their source populations, with smoking status obtained from structured interview at follow-up, in females, and in studies with moderate to good quality, even though these did not reach statistical significance. There was no evidence of significant difference between different weight or BMI measurements. The effect of loss to follow-up on weight or BMI gain was not consistent. Among studies of weight change, studies with loss to follow-up less than 20%, or compared those with follow-up and those lost, or discussed the effect of loss to follow-up in the limitations had greater effect sizes than remained studies; however, among studies of BMI change, the pooled effect sizes were smaller in studies with the aforementioned characteristics. Geographic region was found to affect the result with weight gain considerably greater in studies from North America compared to those from Asia.

4.4.9 Meta-regression analyses

To explore possible sources of heterogeneity, we performed meta-regression analyses examining the following explanatory variables: study region, method of outcome measurement, follow-up length, proportion of males and baseline measures of age, weight or BMI. We also considered whether differences in baseline measures of age, weight or BMI between quitters and continuing smokers had an effect. Owing to the requirement for at least 10 studies to reliably perform meta-regression [29], these analyses were only performed for the continuous outcomes of weight (crude, n = 18 studies and adjusted, n = 10 studies), and BMI change (crude only, n = 12 studies). Follow-up length was found to be a source of heterogeneity for change in continuous measures of weight and BMI (**Figure 4-4**).

4.4.10 Publication bias

Visual inspection of the Begg funnel plot and Egger's test suggested publication bias among the studies reporting change in continuous measures of weight (crude: $p < 0.001$) and BMI (crude: $p < 0.001$; adjusted: $p = 0.020$) (**Figure 4-5**).

Table 4-3 Subgroup analysis for continuing weight change in quitters and continuing smokers

Change in weight		Study No.	Quitters	CS	P^H	I^2 (%)	MD (95% CI)	$P^{subgroup\ difference}$
Crude								
Representativeness	Truly or somewhat	11	20255	70900	< 0.001	96.4	**1.86 (1.52, 2.20)**	0.104
	Selected group, or no description	7	6058	12062	< 0.001	93.2	**1.49 (0.82, 2.16)**	
Smoking status	From structured interview	2	433	822	0.670	0.0	**2.61 (1.85, 3.37)**	0.097
	Written self-report or no description	16	25880	82140	< 0.001	96.7	**1.68 (1.38, 1.98)**	
Sex	Male	12	12183	37437	< 0.001	95.0	**1.39 (1.08, 1.70)**	0.418
	Female	11	12944	43655	< 0.001	96.1	**1.74 (1.29, 2.19)**	
Outcome	Objectively measured	10	3197	7490	< 0.001	96.1	**2.39 (1.35, 3.43)**	0.170
	Self-reported, or no description	8	23116	75472	< 0.001	97.3	**1.58 (1.04, 2.13)**	
Lost to follow-up	≤ 20% or with comparison of FU and "dropout"	5	2447	9112	<0.001	98.4	**2.78 (1.08, 4.47)**	0.202
	> 20% and with description of those lost	8	20367	69203	<0.001	93.1	**1.37 (0.91, 1.83)**	
	> 20% and no description, or no statement	5	3499	4647	<0.001	91.8	**1.88 (0.98, 2.79)**	
Geographic region	Asia	3	2964	15378	0.418	0.0	**0.95 (0.79, 1.11)**	**0.005**
	Europe	8	18152	57999	< 0.001	96.6	**1.60 (1.25, 1.94)**	
	North America	7	5197	9585	< 0.001	92.1	**2.48 (1.54, 3.42)**	
Adjusted								
Representativeness	Truly or somewhat	9	18380	61986	< 0.001	98.0	**2.71 (1.64, 3.78)**	0.544
	Selected group, or no description	1	226	950	1.000	NA	**1.66 (0.67, 2.65)**	
Smoking status	From structured interview	2	1148	3903	0.678	0.0	**3.27 (2.89, 3.65)**	0.407
	Written self-report or no description	8	17458	59033	< 0.001	97.0	**2.41 (1.40, 3.42)**	
Sex	Male	6	7136	23311	< 0.001	92.9	**1.64 (0.86, 2.42)**	0.357
	Female	5	10934	36863	< 0.001	98.0	**2.52 (0.65, 4.39)**	
Outcome	Objectively measured	7	2276	8134	< 0.001	92.6	**2.70 (1.68, 3.72)**	0.780
	Self-reported, or no description	3	16330	54802	< 0.001	98.9	**2.39 (0.11, 4.68)**	
Lost to follow-up	≤ 20% or with comparison of FU and "dropout"	3	830	2969	0.116	53.6	**3.89 (3.17, 4.61)**	0.085
	> 20% and with description of those lost	5	17492	58553	<0.001	98.2	**2.00 (0.83, 3.18)**	
	> 20% and no description, or no statement	2	284	1414	0.614	0.0	**1.76 (0.85, 2.67)**	

Change in weight		Study No.	Quitters	CS	P^H	I^2 (%)	MD (95% CI)	$P^{subgroup\ difference}$
Geographic region	Asia	2	671	3353	0.553	0.0	**1.38 (1.07, 1.68)**	0.431
	Australia	2	521	1589	0.001	90.5	**2.65 (0.82, 4.49)**	
	Europe	2	16052	52883	< 0.001	99.0	2.01 (-1.06, 5.09)	
	North America	4	1362	5111	0.055	60.6	**3.52 (2.81, 4.23)**	

Bold denotes statistically significant result.

CI, confidence interval; CS, continuing smokers; FU, follow-up; MD, mean difference; NA, not applicable; P^H, P-value of heterogeneity.

Table 4-4 Subgroup analysis for continuing BMI change in quitters and continuing smokers

Change in BMI		Study No.	Quitters	CS	P^H	I^2 (%)	MD (95% CI)	$P^{subgroup\ difference}$
Crude								
Representativeness	Truly or somewhat	10	31905	292245	< 0.001	96.7	**0.62 (0.49, 0.75)**	0.419
	Selected group, or no description	2	285	1956	0.881	0.0	**0.47 (0.24, 0.70)**	
Smoking status	From structured interview	3	429	1713	< 0.001	95.9	0.91 (-0.04, 1.86)	0.142
	Written self-report or no description	9	31761	292488	< 0.001	96.2	**0.54 (0.42, 0.66)**	
Sex	Male	11	31032	290399	< 0.001	95.0	**0.52 (0.40, 0.64)**	0.812
	Female	6	1081	3253	< 0.001	94.1	**0.58 (0.07, 1.08)**	
Outcome	Objectively measured	10	29871	280516	< 0.001	96.4	**0.62 (0.49, 0.75)**	0.588
	Self-reported, or no description	2	2319	13685	0.003	88.4	**0.61 (0.06, 1.16)**	
Lost to FU	≤ 20% or with comparison of FU and "dropout"	1	226	950	1.000	NA	**0.46 (0.19, 0.73)**	0.737
	> 20% and with description of those lost	7	30650	289047	< 0.001	90.7	**0.63 (0.44, 0.81)**	
	> 20% and no description, or no statement	4	1314	4204	< 0.001	98.1	**0.83 (0.09, 1.57)**	
Geographic region	Asia	5	30292	288551	< 0.001	82.1	**0.40 (0.26, 0.53)**	**0.038**
	Europe	5	1378	4390	< 0.001	97.9	**1.02 (0.30, 1.75)**	
	Africa	1	227	815	NA	NA	0.09 (-0.17, -0.36)	
	North America	1	293	445	NA	NA	**1.15 (0.74, 1.57)**	
Adjusted								
Representativeness	Truly or somewhat	4	934	3598	0.073	57.0	**0.68 (0.45, 0.90)**	0.578
	Selected group, or no description	1	226	950	1.000	NA	**0.53 (0.21, 0.85)**	
Smoking status	From structured interview	3	489	1195	0.896	0.0	**0.81 (0.59, 1.03)**	**0.010**
	Written self-report or no description	2	671	3353	0.783	0.0	**0.49 (0.38, 0.59)**	
Sex	Male	4	933	3924	0.104	51.3	**0.62 (0.42, 0.82)**	0.400
	Female	2	227	624	0.554	0.0	**0.79 (0.44, 1.14)**	
Outcome	Objectively measured	4	799	4180	0.255	26.1	**0.57 (0.41, 0.72)**	0.219
	Self-reported, or no description	1	361	368	NA	NA	**0.81 (0.49, 1.13)**	
Lost to FU	≤ 20% or with comparison of FU and "dropout"	2	587	1318	0.217	34.4	**0.67 (0.39, 0.95)**	0.483
	> 20% and with description of those lost	2	510	2795	0.184	43.3	**0.55 (0.32, 0.78)**	

Change in BMI		Study No.	Quitters	CS	P^H	I^2 (%)	MD (95% CI)	$P^{subgroup\ difference}$
	> 20% and no description, or no statement	1	63	435	1.000	NA	**0.90 (0.40, 1.40)**	
Geographic region	Asia	3	736	3745	0.409	0.0	**0.51 (0.41, 0.61)**	**0.024**
	Europe	2	424	803	0.773	0.0	**0.84 (0.57, 1.11)**	

Bold denotes statistically significant result.

BMI: body mass index; CI: confidence interval; CS: continuing smokers; FU: follow-up; MD: mean difference; NA: not applicable; P^H: P-value of heterogeneity.

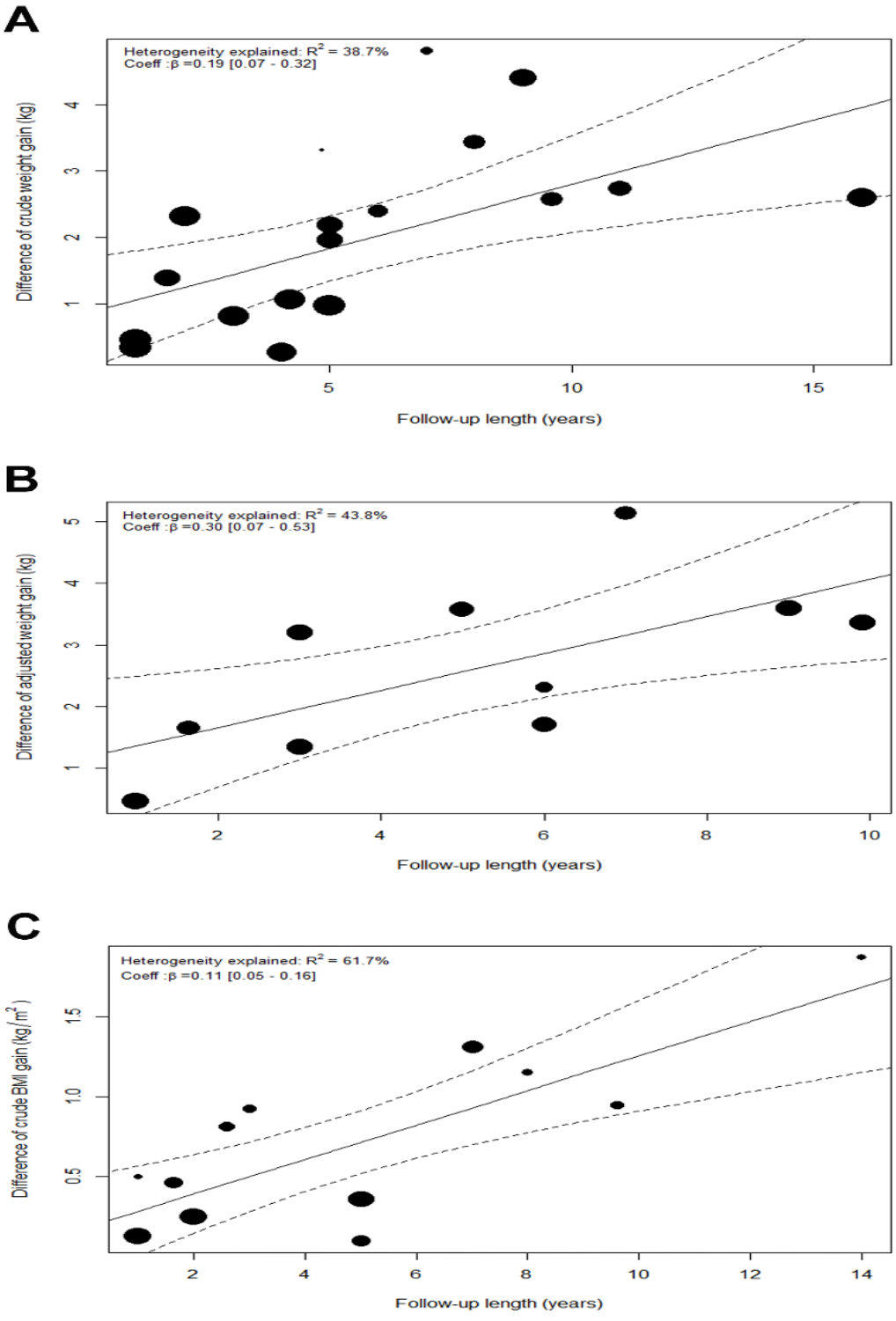

Figure 4-4 Bubble plot of estimated effects of follow-up length. (A) Crude difference in weight change (kg); (B) Adjusted difference in weight change (kg); (C) Crude difference in BMI change (kg/m^2). The size of the bubbles indicates the random effects weight of each study in the meta-analysis. The trend line indicates the degree to which the weight/BMI increases with the duration of follow-up increases.

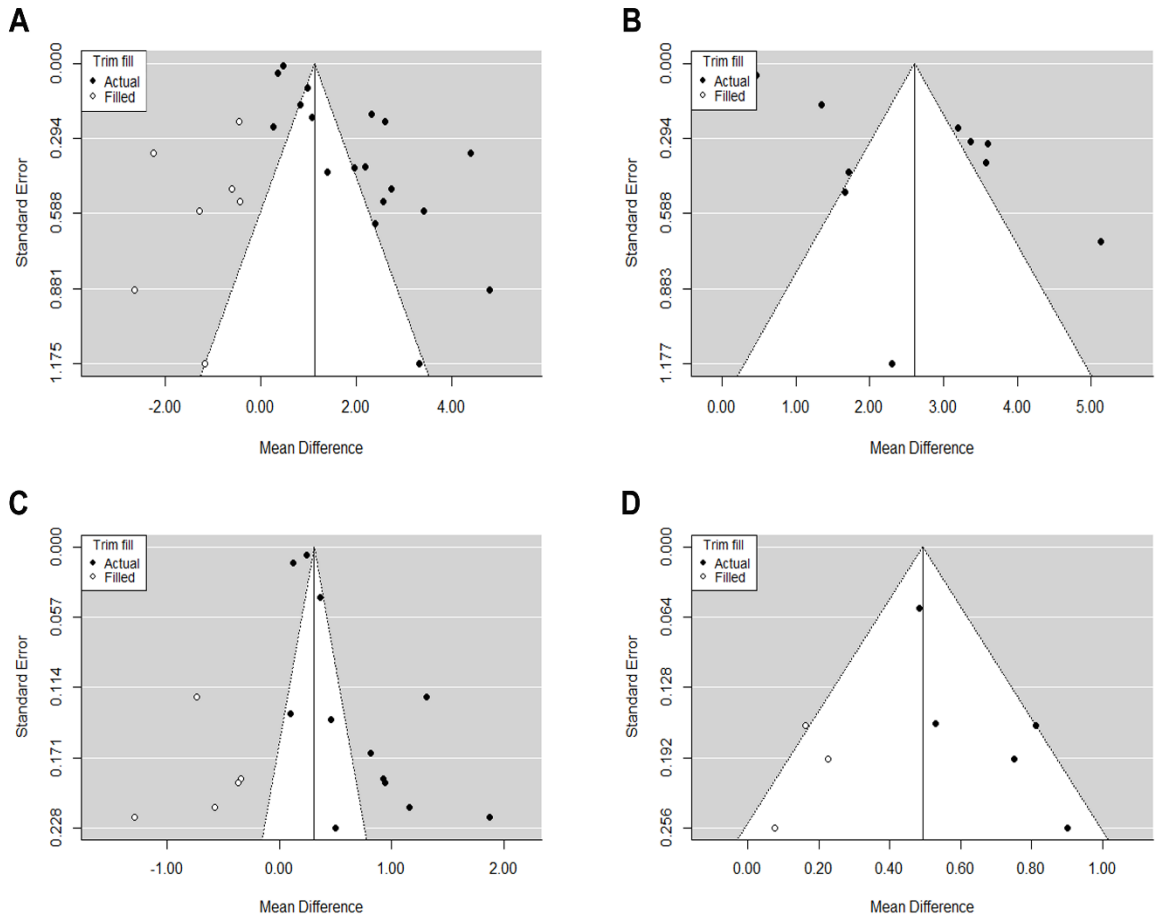

Figure 4-5 Funnel plots with trim and fill. (A) Crude difference in weight change (kg); (B) Adjusted difference in weight change (kg); (C) Crude difference in BMI change (kg/m2); (D) Adjusted difference in BMI change (kg/m^2); Trim and Fill method estimated no missing studies for adjusted difference in weight change.

4.4.11 Sensitivity analyses

As specified *a priori*, we performed sensitivity analyses to examine the effect of study quality and estimation of study SDs. Removing studies with low score on the Newcastle-Ottawa Scale did not greatly affect the results (crude weight change: MD 1.67 kg; 95% CI: 1.33 to 2.01; no study was removed for the analysis of adjusted weight change; crude BMI change: MD 0.76 kg/m$^{2;}$ 95% CI: 0.25 to 1.27; no study was removed for the analysis of adjusted BMI change) , nor did removing those studies for which we estimated the SD (crude weight change: MD 1.69 kg; 95% CI: 1.38 to 2.00; adjusted weight change: MD 2.71 kg; 95% CI: 1.64 to 3.78; crude BMI change: MD 0.52 kg/m$^{2;}$ 95% CI: 0.40 to 0.64; adjusted BMI change: MD 0.67 kg/m$^{2;}$ 95% CI: 0.45 to 0.90). We also undertook a sensitivity analysis using the 'trim and fill' method [34]. If we were to take the 'trim and fill method' as correct then the types of studies potentially missing were those where the effect was null or even reversed. This suggested a somewhat attenuated but still statistically significant effect size (crude weight change: MD 1.13 kg; 95% CI: 0.86 to 1.40; $p < 0.001$; crude BMI change: MD0.31 kg/m^2; 95% CI: 0.19 to 0.43; $p < 0.001$; adjusted BMI change: MD 0.49 kg/m^2; 95% CI: 0.32 to 0.67; $p < 0.001$; **Figure 4-5**).

4.5 Discussion

In this first systematic review and meta-analysis of the association between smoking cessation and weight gain in prospective cohort studies, we found that quitting smoking was associated with mean weight gain of approximately 4.1 kg or 1.1 kg/m^2 BMI units over an average of five years. The pooled adjusted estimate of MD in weight gain between quitters and continuing smokers was 2.6 kg or 0.6 kg/m^2 BMI units. The greatest difference in weight gain was evident in those studies with the longest follow-up and those conducted in North America.

Our meta-analysis has several strengths. We had a large sample size and it is likely that the participants in these studies are more similar to the general population than participants in the meta-analysis of RCTs [16], enabling good generalisability. Further, we were able to include a large proportion of the identified studies in the meta-analysis. In addition, compared with RCTs, cohort studies had longer follow-up time, and it was then possible to assess the effects of quitting smoking on weight change beyond 12 months. Lastly, in

addition to change in weight, other anthropometric measures were considered, including change in BMI and waist circumference.

Several limitations of this review should also be acknowledged. First, it is possible that there has been misclassification of quitting smoking in the included studies; only one [23] bio-verified smoking status at baseline and over half of included studies collected smoking information from self-administered questionnaire at follow-up. However, subgroup analysis according to the ascertainment of smoking status indicated no significant difference, and comparison of self-reported smoking status with results from biochemical validation suggests high levels of sensitivity (87%) and specificity (89%), especially for observational studies and reports by adults [77]. Second, it is not clear how many quitters were continuously or intermittently abstinent during the follow-up. As discussed below, this may have led to an underestimate of the effect of smoking cessation on weight gain. Third, no study reported or adjusted for the use of smoking cessation treatments, such as NRT, which might influence the weight change at least in the short term. However, we know that use of these aids is not common among those trying to quit [22,78]. Fourth, heterogeneity between studies is a potential problem in the interpretation of our results, with follow-up length as a substantial source of heterogeneity. Fifth, significant publication bias was observed. This may be because about a quarter of studies (10 out of 45) did not have sufficient data for our analysis despite attempting to contact authors. Sensitivity analysis using the 'trim and fill' method [34] suggests that this has not greatly affected our results, as the result was of a similar magnitude and significance.

The finding of weight gain of 4.1 kg was higher than the 2.9 kg reported in the comprehensive review done over 25 year ago [15] and somewhat similar to the 4.7 kg reported in the meta-analysis of RCTs [16]; *however, when taking into consideration the longer average follow up length of studies in the current meta-analysis (5 years versus 1 year), the magnitude of total post cessation weight gain in population-based observational studies may be smaller than that in RCTs as people including quitters tend to gain weight as they grow older [17]*. This finding supported our main hypothesis. It is possible that we underestimated the effect size as we likely included both point prevalent and continuous abstainers. A previous study [79] suggested that the estimate of post cessation weight gain in

continuous abstainers (5.90 kg) was about twice that of point prevalent abstainers (3.04 kg) after 1 year follow-up. However, the nature of prospective cohort studies makes it difficult to accurately distinguish point prevalent from continuous abstainers. Moreover, our subgroup analyses suggested that the difference in weight gain was generally greater in studies with better measurement of exposure and outcome, and with lower rate of loss to follow-up. Therefore, it is very likely that the weight gain would be greater if the study is perfectly performed.

A trend toward a larger difference of weight and BMI gain was observed among women than men, although it did not reach statistical significance, possibly suggesting a greater metabolic impact of smoking in women than men. This is supported by animal research showing that the effects of nicotine on body weight and eating behavior were greater in female than male rats [80,81]. The sex differences we observed might also be explained by different clustering of weight-related PA and dietary behaviors between men and women [82,83].

The magnitude of the difference in weight gain was significantly different between geographic regions, with studies conducted in North America showing a greater difference than those in Asia. Weight gain occurs because of an interaction of multiple factors at the level of the individual, the community and the population, all of which may differ between regions, including PA, dietary behaviors, culture and traditions, public policy and genetic factors. Previous evidence has shown that baseline BMI was positively related to weight gain after cessation [84]. As the prevalence of overweight and obesity is highest in North America (61% for overweight or obesity and 27% for obesity) and lowest in Asia (22% for overweight or obesity and 5% for obesity) [85], this may partly explain the regional difference seen here. The other possible explanation is the higher proportion of women in studies of North American (about 50%) than Asian (< 10%) origin. Weight gain attributable to smoking cessation differs between ethnic groups with greater weight gain in blacks than whites, and in Mexican Americans than non-Hispanic whites, which could be due to genetic factors but also the individual and community factors cited above [23,44,54]. Unfortunately, no study has examined the difference between Asians and Americans.

We found that follow-up length was positively related to difference in weight gain after smoking cessation, suggesting that the slope of weight gain in quitters was steeper than continuing smokers. This finding was supported by a study with repeated measures of weight within 5 years (not included in our review because of its trial design) [86]. In this study, among both male and female sustained quitters, about 60% of weight gain after quitting smoking occurred during the first year and the remaining 40% spread evenly over the remaining 4 years of follow-up. In addition, both weight gain (kg) and percent weight gain was significantly higher among sustained quitters than in continuing smokers ($p < 0.0001$). However, other reports indicated that the excess weight gain after quitting smoking may be transient and probably occurs in the first years after abstinence [24,53,87]. At present relatively little is known about why the magnitude of weight gain difference was larger in studies with a longer follow-up. Further research to investigate the long-term effects of smoking cessation on weight gain needs to replicate and elucidate how smoking cessation affects the weight gain in the long term.

Our results showed that people who continued smoking increased their waist circumference over follow-up, but the increase was greater in those who quit smoking, with a MD of 2.6 cm between these two groups. Waist circumference is an indicator of visceral adipose tissue, which is associated with an increased risk of metabolic syndrome, diabetes and CVDs [88]. Evidence has suggested that smoking contributes to greater accumulation of visceral fat, and that women are more likely to be affected than men [89], but changes after smoking cessation are unclear and, due to the unavailability of data, we could not perform a subgroup analysis according to sex.

It is important to consider its impact on weight-concerned smokers attempting to quit as there are no effective approaches to prevent weight gain after cessation [90-92]. Personalised weight management support may be effective in mitigating post-cessation weight gain, but the data are too few to be sure [90]. At this stage, more efforts should focus on reducing smoking-related weight concerns and encouraging weight concerned smokers to sustain abstinence. Evidence from trials suggested that cognitive-behavioral therapy (CBT) may be an effective approach to reduce weight concerns [93], and the combination of CBT and bupropion therapy could enhance the abstinence for weight concerned smokers [94].

Although the results were fairly consistent between crude and adjusted estimates, there was suboptimal control for covariates in most studies. Relatively few studies adjusted for baseline weight or BMI [23,24,37,66], socioeconomic factors [23,24,37,45,66], alcohol consumption [23,24,37,45,65], PA [23,24,37,45,60,65,66], energy intake [23,24,60], illness [37,45] and duration of follow-up [37,66]. No studies considered mental health, which is known to have significant associations with weight change [95,96] and smoking cessation [97,98]. This is important because, as noted previously, the existing results from numerous trials have not been sufficient to make clinical recommendations regarding the prevention of weight gain after cessation. Studies with careful consideration of such covariates could indicate the mechanisms for weight gain after cessation and therefore ways that it can be prevented. Better-designed observational studies and smaller well-controlled clinical trials are needed to address this gap in knowledge.

Changes in body weight occur when energy intake exceeds energy expenditure over a period of time. People expend energy through resting metabolic rate, PA and the thermic effects of food. The mechanisms of post-cessation weight gain remain poorly understood but are possibly mediated through increasing energy intake and decreasing resting metabolic rate [99,100]. Significant increases of daily energy intake have been observed shortly after smoking cessation in some studies [101-103] but not all [104]. Scarce data is available on the long-term changes in energy intake after smoking cessation. Even so, strict dieting while quitting is not recommended because it might impede quit attempts and induce relapse [105]. Similarly, not all studies support a link between decreases in nicotine concentrations after smoking cessation and reductions in resting metabolic rate [101,106,107]. The reduction in resting metabolic rate following cessation are reported to range from 4% to 16%, accounting for less than 40% of weight gain associated with smoking cessation [100].

Weight gain after quitting smoking does not appear to be easily explained by changes in energy intake and alcohol consumption [46,50,108], but the magnitude of weight gain has been reported to be somewhat lower among quitters who maintained or increased their PA compared with quitters who either decreased their PA [46,108] or remained sedentary [109]. Notably, the amount of weight gain following smoking cessation appears influenced not only by nicotine intake but also by the level of PA that a smoker engages in while smoking

[110]. Perkins and colleagues [111] found that the magnitude of excess energy expenditure attributable to nicotine was more than twice as great during light PA than during rest. Consequently, two smokers with the same smoking histories, daily PA and caloric intakes might have very different amounts of weight gain [100].

4.6 Conclusions

Individuals who quit smoking gained, on average, approximately 4.1 kg or 1.1 kg/m^2 BMI units over about five years. The MD in weight gain between quitters and continuing smokers was 2.6 kg or 0.6 kg/m^2 BMI units. Better designed observational studies and smaller well controlled clinical trials are needed to determine what is associated with greater weight gain in quitters than continuing smokers.

4.7 Postscript

The results of this chapter are potentially relevant to general practitioners and other health professionals that assist people with smoking. Given the well-documented health benefits of quitting smoking, clinicians should inform smokers about the likelihood of weight gain, and implement strategies to help smokers minimise weight gain. However, it is still unclear what inventions work effectively to limit or even prevent the post-cessation weight gain. The next chapter explores whether changing health behaviours could explain the greater weight gain in quitters than continuing smokers in order to shed light on the ways it may be prevented.

4.8 References

1. World Health Organization. Tobacco, fact sheet N°339. 2013; http://www.who.int/mediacentre/factsheets/fs339/en/index.html. Accessed 8 July, 2014.
2. Jha P, Ramasundarahettige C, Landsman V, et al. 21st-century hazards of smoking and benefits of cessation in the United States. *N Engl J Med.* 2013;368(4):341-350. doi:10.1056/NEJMsa1211128.
3. Pirie K, Peto R, Reeves GK, Green J, Beral V, Million Women Study C. The 21st century hazards of smoking and benefits of stopping: a prospective study of one million women in the UK. *Lancet.* 2013;381(9861):133-141. doi:10.1016/S0140-6736(12)61720-6.
4. U.S. Department of Health and Human Services. *The Health Consequences of Smoking: 50 Years of Progress. A Report of the Surgeon General*. Atlanta, GA: Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2014. http://www.surgeongeneral.gov/library/reports/50-years-of-progress/. Accessed 15 December 2014.
5. Hughes JR, Keely J, Naud S. Shape of the relapse curve and long-term abstinence among untreated smokers. *Addiction.* 2004;99(1):29-38.
6. Pomerleau CS, Zucker AN, Stewart AJ. Characterizing concerns about post-cessation weight gain: results from a national survey of women smokers. *Nicotine Tob Res.* 2001;3(1):51-60. doi:10.1080/14622200020032105.
7. Levine MD, Perkins KA, Marcus MD. The characteristics of women smokers concerned about postcessation weight gain. *Addict Behav.* 2001;26(5):749-756.
8. Levine MD, Bush T, Magnusson B, Cheng Y, Chen X. Smoking-related weight concerns and obesity: differences among normal weight, overweight, and obese smokers using a telephone tobacco quitline. *Nicotine Tob Res.* 2013;15(6):1136-1140. doi:10.1093/ntr/nts226.
9. Cooper TV, Dundon M, Hoffman BM, Stoever CJ. General and smoking cessation related weight concerns in veterans. *Addict Behav.* 2006;31(4):722-725.
10. Pisinger C, Jorgensen T. Weight concerns and smoking in a general population: the Inter99 study. *Prev Med.* 2007;44(4):283-289. doi:10.1016/j.ypmed.2006.11.014.
11. Camp DE, Klesges RC, Relyea G. The relationship between body weight concerns and adolescent smoking. *Health Psychol.* 1993;12(1):24-32.
12. French SA, Perry CL, Leon GR, Fulkerson JA. Weight concerns, dieting behavior, and smoking initiation among adolescents: a prospective study. *Am J Public Health.* 1994;84(11):1818-1820.
13. Wee CC, Rigotti NA, Davis RB, Phillips RS. Relationship between smoking and weight control efforts among adults in the united states. *Arch Intern Med.* 2001;161(4):546-550.
14. Weekley CK, 3rd, Klesges RC, Reylea G. Smoking as a weight-control strategy and its relationship to smoking status. *Addict Behav.* 1992;17(3):259-271.

15. Klesges RC, Meyers AW, Klesges LM, La Vasque ME. Smoking, body weight, and their effects on smoking behavior: a comprehensive review of the literature. *Psychol Bull.* 1989;106(2):204-230.
16. Aubin HJ, Farley A, Lycett D, Lahmek P, Aveyard P. Weight gain in smokers after quitting cigarettes: meta-analysis. *BMJ.* 2012;345:e4439. doi:10.1136/bmj.e4439.
17. Fernandez E, Chapman S. Quitting smoking and gaining weight: the odd couple. *BMJ.* 2012;345:e4544. doi:10.1136/bmj.e4544.
18. Le Strat Y, Rehm J, Le Foll B. How generalisable to community samples are clinical trial results for treatment of nicotine dependence: a comparison of common eligibility criteria with respondents of a large representative general population survey. *Tob Control.* 2011;20(5):338-343. doi:10.1136/tc.2010.038703.
19. Fagerstrom KO, Kunze M, Schoberberger R, et al. Nicotine dependence versus smoking prevalence: comparisons among countries and categories of smokers. *Tob Control.* 1996;5(1):52-56.
20. Chapman S. Tar wars over smoking cessation. *BMJ.* 2011;343:d5008. doi:10.1136/bmj.d5008.
21. Pohl JM, Martinelli A, Antonakos C. Predictors of participation in a smoking cessation intervention group among low-income women. *Addict Behav.* 1998;23(5):699-704.
22. Chapman S, MacKenzie R. The global research neglect of unassisted smoking cessation: causes and consequences. *PLoS Med.* 2010;7(2):e1000216. doi:10.1371/journal.pmed.1000216.
23. Klesges RC, Ward KD, Ray JW, Jacobs Jr DR, Cutter G, Wagenknecht LE. The prospective relationships between smoking and weight in a young, biracial cohort: The Coronary Artery Risk Development in Young Adults study. *Journal of Consulting and Clinical Psychology.* 1998;66(6):987-993.
24. Travier N, Agudo A, May AM, et al. Longitudinal changes in weight in relation to smoking cessation in participants of the EPIC-PANACEA study. *Preventive Medicine.* 2012;54(3-4):183-192.
25. Moher D, Liberati A, Tetzlaff J, Altman DG. Preferred reporting items for systematic reviews and meta-analyses: the PRISMA statement. *BMJ.* 2009;339:b2535. doi:10.1136/bmj.b2535.
26. Stroup DF, Berlin JA, Morton SC, et al. Meta-analysis of observational studies in epidemiology: a proposal for reporting. Meta-analysis Of Observational Studies in Epidemiology (MOOSE) group. *JAMA.* 2000;283(15):2008-2012.
27. Scottish Intercollegiate Guidelines Network. Searching filters. 2013; http://www.sign.ac.uk/methodology/filters.html. Accessed 4 April 2013.
28. Wells G, Shea B, O'Connell D, et al. The Newcastle-Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in meta-analyses. http://www.ohri.ca/programs/clinical_epidemiology/oxford.asp. Accessed 23 Feburary, 2013.
29. Higgins JPT, Green S, eds. *Cochrane Handbook for Systematic Reviews of Interventions Version 5.1.0.* The Cochrane Colllaboration; 2011.
30. Higgins JP. Commentary: Heterogeneity in meta-analysis should be expected and appropriately quantified. *Int J Epidemiol.* 2008;37(5):1158-1160. doi:10.1093/ije/dyn204.
31. Higgins JP, Thompson SG, Deeks JJ, Altman DG. Measuring inconsistency in meta-analyses. *BMJ.* 2003;327(7414):557-560.

32. Egger M, Davey Smith G, Schneider M, Minder C. Bias in meta-analysis detected by a simple, graphical test. *BMJ.* 1997;315(7109):629-634.
33. Begg CB, Mazumdar M. Operating characteristics of a rank correlation test for publication bias. *Biometrics.* 1994;50(4):1088-1101.
34. Duval S, Tweedie R. Trim and fill: A simple funnel-plot-based method of testing and adjusting for publication bias in meta-analysis. *Biometrics.* 2000;56(2):455-463.
35. Bosse R, Garvey AJ, Costa PT, Jr. Predictors of weight change following smoking cessation. *Int J Addict.* 1980;15(7):969-991.
36. Burnette MM, Meilahn E, Wing RR, Kuller LH. Smoking cessation, weight gain, and changes in cardiovascular risk factors during menopause: the Healthy Women Study. *Am J Public Health.* 1998;88(1):93-96.
37. Janzon E, Hedblad B, Berglund G, Engstrom G. Changes in blood pressure and body weight following smoking cessation in women. *J Intern Med.* 2004;255(2):266-272.
38. Froom P, Kristal-Boneh E, Melamed S, Gofer D, Benbassat J, Ribak J. Smoking cessation and body mass index of occupationally active men: the Israeli CORDIS Study. *Am J Public Health.* 1999;89(5):718-722.
39. Chinn S, Jarvis D, Melotti R, et al. Smoking cessation, lung function, and weight gain: a follow-up study. *Lancet.* 2005;365(9471):1629-1635; discussion 1600-1621.
40. Swan GE, Carmelli D. Characteristics associated with excessive weight gain after smoking cessation in men. *Am J Public Health.* 1995;85(1):73-77.
41. Noppa H, Bengtsson C. Obesity in relation to smoking: a population study of women in Goteborg, Sweden. *Prev Med.* 1980;9(4):534-543.
42. Lissner L, Bengtsson C, Lapidus L, Bjorkelund C. Smoking initiation and cessation in relation to body fat distribution based on data from a study of Swedish women. *American Journal of Public Health.* 1992;82(2):273-275.
43. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and long-term weight gain in women and men. *N Engl J Med.* 2011;364(25):2392-2404. doi:10.1056/NEJMoa1014296.
44. Burke JP, Hazuda HP, Stern MP. Rising trend in obesity in Mexican Americans and non-Hispanic whites: is it due to cigarette smoking cessation? *International Journal of Obesity.* 2000;24(12):1689-1694. doi:10.1038/sj.ijo.0801439.
45. Yoon C, Goh E, Park SM, Cho B. Effects of smoking cessation and weight gain on cardiovascular disease risk factors in Asian male population. *Atherosclerosis.* 2010;208(1):275-279. doi:10.1016/j.atherosclerosis.2009.07.024.
46. Kawachi I, Troisi RJ, Rotnitzky AG, Coakley EH, Colditz GA. Can physical activity minimize weight gain in women after smoking cessation? *Am J Public Health.* 1996;86(7):999-1004.
47. Oba S, Noda M, Waki K, et al. Smoking cessation increases short-term risk of type 2 diabetes irrespective of weight gain: the Japan Public Health Center-Based Prospective Study. *PLoS ONE.* 2012;7(2):e17061.
48. Hodge AM, Dowse GK, Gareeboo H, Tuomilehto J, Alberti K, Zimmet PZ. Incidence, increasing prevalence, and predictors of change in obesity and fat distribution over 5 years in the rapidly developing population of Mauritius. *International Journal of Obesity.* 1996;20(2):137-146.
49. Kawada T, Otsuka T, Inagaki H, et al. Association of smoking status, insulin resistance, body mass index, and metabolic syndrome in workers: A 1-year follow-up study. *Obes Res Clin Pract.* 2010;4(3):e163-246.

50. Munafo MR, Tilling K, Ben-Shlomo Y. Smoking status and body mass index: A longitudinal study. *Nicotine and Tobacco Research.* 2009;11(6):765-771.
51. Bartholomew HC, Knuiman MW. Longitudinal analysis of the effect of smoking cessation on cardiovascular risk factors in a community sample: The Busselton study. *Journal of Cardiovascular Risk.* 1998;5(4):263-271.
52. Reas DL, Nygård JF, Sørensen T. Do quitters have anything to lose? Changes in body mass index for daily, never, and former smokers over an 11-year period (1990-2001). *Scandinavian Journal of Public Health.* 2009;37(7):774-777.
53. Basterra-Gortari FJ, Forga L, Bes-Rastrollo M, Toledo E, Martinez JA, Martinez-Gonzalez MA. Effect of smoking on body weight: longitudinal analysis of the SUN cohort. *Revista Espanola de Cardiologia.* 2010;63(1):20-27.
54. Williamson DF, Madans J, Anda RF, Kleinman JC, Giovino GA, Byers T. Smoking cessation and severity of weight gain in a national cohort. *New England Journal of Medicine.* 1991;324(11):739-745.
55. Sneve M, Jorde R. Cross-sectional study on the relationship between body mass index and smoking, and longitudinal changes in body mass index in relation to change in smoking status: The Tromsø Study. *Scandinavian Journal of Public Health.* 2008;36(4):397-407.
56. Gordon T, Kannel WB, Dawber TR, McGee D. Changes associated with quitting cigarette smoking: the Framingham Study. *American Heart Journal.* 1975;90(3):322-328.
57. Friedman GD, Siegelaub AB. Changes after quitting cigarette smoking. *Circulation.* 1980;61(4):716-723.
58. Goya Wannamethee S, Gerald Shaper A, Perry IJ. Smoking as a modifiable risk factor for type 2 diabetes in middle-aged men. *Diabetes Care.* 2001;24(9):1590-1595.
59. Lee DH, Ha MH, Kim JR, Jacobs Jr DR. Effects of smoking cessation on changes in blood pressure and incidence of hypertension a 4-year follow-up study. *Hypertension.* 2001;37(2 I):194-198.
60. Brown WJ, Williams L, Ford JH, Ball K, Dobson AJ. Identifying the energy gap: Magnitude and determinants of 5-year weight gain in midage women. *Obesity research.* 2005;13(8):1431-1441. doi:10.1038/oby.2005.173.
61. John U, Meyer C, Rumpf HJ, Hapke U, Schumann A. Predictors of increased body mass index following cessation of smoking. *American Journal on Addictions.* 2006;15(2):192-197.
62. Song YM, Cho HJ. Risk of stroke and myocardial infarction after reduction or cessation of cigarette smoking: A cohort study in korean men. *Stroke.* 2008;39(9):2432-2438.
63. Byung JK, Bum SK, Ki CS, Jin HK, Man HL, Jung RP. Association of smoking status, weight change, and incident metabolic syndrome in men: A 3-year follow-up study. *Diabetes Care.* 2009;32(7):1314-1316.
64. Holz T, Thorand B, Doring A, Schneider A, Meisinger C, Koenig W. Markers of Inflammation and Weight Change in Middle-Aged Adults: Results From the Prospective MONICA/KORA S3/F3 Study. *Obesity.* 2010;18(12):2347-2353. doi:10.1038/oby.2010.73.
65. Suwazono Y, Dochi M, Oishi M, Tanaka K, Morimoto H, Sakata K. Longitudinal Effect of Smoking Cessation on Physical and Laboratory Findings. *Am J Prev Med.* 2010;38(2):192-200.

66. Yeh H-C, Duncan BB, Schmidt MI, Wang N-Y, Brancati FL. Smoking, smoking cessation, and risk for type 2 diabetes mellitus: a cohort study. *Annals of Internal Medicine.* 2010;152(1):10-17. doi:10.7326/0003-4819-152-1-201001050-00005.
67. Hansson J, Galanti MR, Magnusson C, Hergens MP. Weight gain and incident obesity among male snus users. *BMC Public Health.* 2011;11. doi:10.1186/1471-2458-11-371.
68. Luo J, Rossouw J, Tong E, et al. Smoking cessation, weight gain, and risk of type 2 diabetes mellitus among postmenopausal women. *Arch Intern Med.* 2012;172(5):2012.
69. Clair C, Rigotti NA, Porneala B, et al. Association of smoking cessation and weight change with cardiovascular disease among adults with and without diabetes. *JAMA - Journal of the American Medical Association.* 2013;309(10):1014-1021.
70. Gritz ER, Carr CR, Marcus AC. Unaided smoking cessation: great American smokeout and new year's day quitters. *J Psychosoc Oncol.* 1989;6(3-4):217-234.
71. Kahn HS, Tatham LM, Rodriguez C, Calle EE, Thun MJ, Heath Jr CW. Stable behaviors associated with adults' 10-year change in body mass index and likelihood of gain at the waist. *American Journal of Public Health.* 1997;87(5):747-754.
72. Koh-Banerjee P, Chu N-F, Spiegelman D, et al. Prospective study of the association of changes in dietary intake, physical activity, alcohol consumption, and smoking with 9-y gain in waist circumference among 16 587 US men. *American Journal of Clinical Nutrition.* 2003;78(4):719-727.
73. Lund Haheim L, Lund Larsen PG, Sogaard AJ, Holme I. Risk factors associated with body mass index increase in men at 28 years follow-up. *Qjm.* 2006;99(10):665-671.
74. Lund-Larsen PG, Tretli S. Changes in smoking habits and body weight after a three-year period--the cardiovascular disease study in Finnmark. *J Chronic Dis.* 1982;35(10):773-780.
75. Rissanen AM, Heliovaara M, Knekt P, Reunanen A, Aromaa A. Determinants of weight gain and overweight in adult Finns. *European Journal of Clinical Nutrition.* 1991;45(9):419-430.
76. Rodu B, Stegmayr B, Nasic S, Cole P, Asplund K. The influence of smoking and smokeless tobacco use on weight amongst men. *J Intern Med.* 2004;255(1):102-107.
77. Patrick DL, Cheadle A, Thompson DC, Diehr P, Koepsell T, Kinne S. The validity of self-reported smoking: a review and meta-analysis. *Am J Public Health.* 1994;84(7):1086-1093.
78. Shiffman S, Brockwell SE, Pillitteri JL, Gitchell JG. Use of smoking-cessation treatments in the United States. *Am J Prev Med.* 2008;34(2):102-111. doi:10.1016/j.amepre.2007.09.033.
79. Klesges RC, Winders SE, Meyers AW, et al. How much weight gain occurs following smoking cessation? A comparison of weight gain using both continuous and point prevalence abstinence. *J Consult Clin Psychol.* 1997;65(2):286-291.
80. Grunberg NE, Popp KA, Winders SE. Effects of nicotine on body weight in rats with access to "junk" foods. *Psychopharmacology (Berl).* 1988;94(4):536-539.
81. Grunberg NE, Winders SE, Popp KA. Sex differences in nicotine's effects on consummatory behavior and body weight in rats. *Psychopharmacology (Berl).* 1987;91(2):221-225.

82. Hall SM, McGee R, Tunstall C, Duffy J, Benowitz N. Changes in Food Intake and Activity After Quitting Smoking. *Journal of Consulting and Clinical Psychology.* 1989;57(1):81-86.
83. Auer R, Vittinghoff E, Kiefe C, et al. Change in physical activity after smoking cessation: the Coronary Artery Risk Development in Young Adults (CARDIA) study. *Addiction.* 2014;109(7):1172-1183. doi:10.1111/add.12561.
84. Lycett D, Munafo M, Johnstone E, Murphy M, Aveyard P. Associations between weight change over 8 years and baseline body mass index in a cohort of continuing and quitting smokers. *Addiction.* 2011;106(1):188-196.
85. World Health Organization. Global Health Observatory (GHO) data, overweight and obesity. 2014; http://www.who.int/gho/ncd/risk_factors/overweight_text/en/. Accessed 23 April, 2015.
86. O'Hara P, Connett JE, Lee WW, Nides M, Murray R, Wise R. Early and late weight gain following smoking cessation in the Lung Health Study. *Am J Epidemiol.* 1998;148(9):821-830.
87. Flegal KM. The conundrum of smoking cessation and weight gain. *Prev Med.* 2012;54(3-4):193-194. doi:10.1016/j.ypmed.2012.01.015.
88. Han TS, Sattar N, Lean M. ABC of obesity. Assessment of obesity and its clinical implications. *BMJ.* 2006;333(7570):695-698.
89. Chiolero A, Faeh D, Paccaud F, Cornuz J. Consequences of smoking for body weight, body fat distribution, and insulin resistance. *Am J Clin Nutr.* 2008;87(4):801-809.
90. Farley AC, Hajek P, Lycett D, Aveyard P. Interventions for preventing weight gain after smoking cessation. *Cochrane Database Syst Rev.* 2012;1:CD006219. doi:10.1002/14651858.CD006219.pub3.
91. Yang M, Bhowmik D, Wang X, Abughosh S. Does combination pharmacological intervention for smoking cessation prevent post-cessation weight gain? A systemic review. *Addict Behav.* 2013;38(3):1865-1875. doi:10.1016/j.addbeh.2012.11.007.
92. Parsons AC, Shraim M, Inglis J, Aveyard P, Hajek P. Interventions for preventing weight gain after smoking cessation. *Cochrane Database Syst Rev.* 2009(1):CD006219. doi:10.1002/14651858.CD006219.pub2.
93. Perkins KA, Marcus MD, Levine MD, et al. Cognitive-behavioral therapy to reduce weight concerns improves smoking cessation outcome in weight-concerned women. *J Consult Clin Psychol.* 2001;69(4):604-613.
94. Levine MD, Perkins KA, Kalarchian MA, et al. Bupropion and cognitive behavioral therapy for weight-concerned women smokers. *Arch Intern Med.* 2010;170(6):543-550. doi:10.1001/archinternmed.2010.33.
95. Pan A, Kawachi I, Luo N, et al. Changes in Body Weight and Health-Related Quality of Life: 2 Cohorts of US Women. *Am J Epidemiol.* 2014;180(3):254-262. doi:10.1093/aje/kwu136.
96. Verkleij SP, Adriaanse MC, Wendel-Vos GC, Schuit AJ. Longitudinal relation between weight change and quality of life in a community-based population: a prospective cohort study. *Eur J Public Health.* 2013;23(2):285-290. doi:10.1093/eurpub/cks058.
97. Taylor G, McNeill A, Girling A, Farley A, Lindson-Hawley N, Aveyard P. Change in mental health after smoking cessation: systematic review and meta-analysis. *BMJ.* 2014;348:g1151. doi:10.1136/bmj.g1151.

98. Lasser K, Boyd JW, Woolhandler S, Himmelstein DU, McCormick D, Bor DH. Smoking and mental illness: A population-based prevalence study. *JAMA.* 2000;284(20):2606-2610.
99. Audrain-McGovern J, Benowitz NL. Cigarette smoking, nicotine, and body weight. *Clin Pharmacol Ther.* 2011;90(1):164-168. doi:10.1038/clpt.2011.105.
100. Filozof C, Fernandez Pinilla MC, Fernandez-Cruz A. Smoking cessation and weight gain. *Obes Rev.* 2004;5(2):95-103. doi:10.1111/j.1467-789X.2004.00131.x.
101. Stamford BA, Matter S, Fell RD, Papanek P. Effects of smoking cessation on weight gain, metabolic rate, caloric consumption, and blood lipids. *Am J Clin Nutr.* 1986;43(4):486-494.
102. Allen SS, Brintnell DM, Hatsukami D, Reich B. Energy intake and physical activity during short-term smoking cessation in postmenopausal women. *Addict Behav.* 2004;29(5):947-951.
103. Levine MD, Cheng Y, Kalarchian MA, Perkins KA, Marcus MD. Dietary intake after smoking cessation among weight-concerned women smokers. *Psychol Addict Behav.* 2012;26(4):969-973. doi:10.1037/a0028948.
104. Allen SS, Hatsukami D, Christianson D, Brown S. Energy intake and energy expenditure during the menstrual cycle in short-term smoking cessation. *Addict Behav.* 2000;25(4):559-572.
105. Cheskin LJ, Hess JM, Henningfield J, Gorelick DA. Calorie restriction increases cigarette use in adult smokers. *Psychopharmacology (Berl).* 2005;179(2):430-436. doi:10.1007/s00213-004-2037-x.
106. Hofstetter A, Schutz Y, Jequier E, Wahren J. Increased 24-hour energy expenditure in cigarette smokers. *N Engl J Med.* 1986;314(2):79-82. doi:10.1056/NEJM198601093140204.
107. Moffatt RJ, Owens SG. Cessation from cigarette smoking: changes in body weight, body composition, resting metabolism, and energy consumption. *Metabolism.* 1991;40(5):465-470.
108. Gennuso KP, Thraen-Borowski KM, Schlam TR, et al. Smokers' physical activity and weight gain one year after a successful versus unsuccessful quit attempt. *Prev Med.* 2014;67:189-192. doi:10.1016/j.ypmed.2014.07.040.
109. Prod'hom S, Locatelli I, Giraudon K, et al. Predictors of weight change in sedentary smokers receiving a standard smoking cessation intervention. *Nicotine Tob Res.* 2013;15(5):910-916. doi:10.1093/ntr/nts217.
110. Walker JF, Collins LC, Rowell PP, Goldsmith LJ, Moffatt RJ, Stamford BA. The effect of smoking on energy expenditure and plasma catecholamine and nicotine levels during light physical activity. *Nicotine Tob Res.* 1999;1(4):365-370.
111. Perkins KA, Epstein LH, Marks BL, Stiller RL, Jacob RG. The effect of nicotine on energy expenditure during light physical activity. *N Engl J Med.* 1989;320(14):898-903. doi:10.1056/NEJM198904063201404.

4.9 Appendix 4-1: Search strategy

Databases and vocabulary tools

The following databases and vocabulary tools were used to identify publications of interest:

- Medline with Full Text: MeSH
- EMBASE: EMTREE function
- CINAHL: CINAHL headings
- SCOPUS: no vocabulary tools available
- Web of Science: no vocabulary tools available

Search terms

Search terms were taken from each of the databases' vocabulary tools where available. The SCOPUS and Web of Science do not have such vocabulary tools, thus the terms that were found in the other databases were also used in these two databases. Initially, key words generated by the authors were entered in each database's vocabulary tool (where available) in order to generate appropriate search terms. These were: "smoking cessation", "quitting smoking", "tobacco use cessation", "smoking reduction", and "tobacco use reduction" (terms related to the exposure); "body weight change", "weight gain", "weight loss ", "weight change", and "change in waist circumference", "change in body mass index", and "change in waist-hip ratio" (terms related to the outcome); and "cohort studies", "case-control studies", "longitudinal studies", "follow-up studies", "prospective studies", "retrospective studies", and "observational studies" (terms related to the study design). Once the appropriate search terms were established, they were grouped together according to the PICOS (population, intervention, control group, outcome, and study design) framework and entered into the search field of each database.

Refining searches

Searches in the above databases were limited (where it is allowed in the database) to human subjects. In order to capture variations as comprehensive as possible, truncated terms were used where appropriate such as for "study or studies", "cessation or cessations", "reducing or reduction". Wildcard terms were used where appropriate for words with different spelling (e.g. to search for behaviour or behaviour, the term "behavi$r" will be used).

Medline (MeSH terms):

1. Smoking cessation$1.tw.
2. Smoking abstinence.tw.
3. Exp "tobacco use cessation"/
4. Tobacco use cessation$1.tw.
5. Smoking abstination.tw
6. Quit$ smoking.tw.
7. Stop$ smoking.tw.
8. Smoking dehabituation.tw
9. "Nicotine cessation$1".tw.
10. "Nicotine abstinence".tw.
11. "Nicotine abstination".tw.
12. Abstinence from tobacco.tw.
13. Abstinence from smoking.tw.
14. Abstinence from nicotine.tw.
15. Smoking reduc$.tw.
16. "Tobacco use reduc$".tw.
17. Reduc$ smoking.tw.
18. Modified smoking.tw.
19. Modified tobacco consumption.tw.
20. Modification of cig$.tw.
21. Modification of smoking.tw.
22. Cig$ reduc$. tw
23. Reduc$ cig$.tw
24. Reduction in cig$.tw
25. Reduc$ tobacco consumption.tw.
26. Harm reduc$.
27. Tobacco consumption.tw.
28. Or/1-27
29. Exp body weight changes/
30. Body weight change$1.tw.

31. *Body size/
32. *Body weight/
33. Exp waist circumference/
34. Exp body mass index/
35. Exp skinfold thickness
36. Exp waist-hip ratio/
37. Weight.tw.
38. Body size$1.tw.
39. Body weight$1.tw.
40. Waist circumference$1.tw.
41. Waist-hip ratio$1.tw.
42. Skinfold thickness$.tw.
43. Body mass index.tw.
44. Bmi.tw.
45. Quetelet$ index.tw.
46. Or/31-45
47. (Chang$ or increas$ or reduc$ or gain$ or decreas$ or los$).tw.
48. 46 and 47
49. or/29,30,48
50. Epidemiologic studies/
51. Exp case control studies/
52. Exp cohort studies/
53. Case control.tw.
54. (Cohort adj (study or studies)).tw.
55. Cohort analy$.tw.
56. (Follow up adj (study or studies)).tw.
57. (Observational adj (study or studies)).tw.
58. Longitudinal.tw.
59. Retrospective.tw.
60. Or/50-59
61. 28 and 49 and 60

#1-28: searching for exposure

#29-49: searching for outcome

#50-60: SIGN searching filter for observational studies in Medline via Ovid

EMBASE (EMTREE function)

1. Exp smoking cessation/
2. Exp smoking abstinence/
3. Smoking cessation$1.tw.
4. Smoking dehabituation.tw.
5. Quit$ smoking.tw
6. Stop$ smoking.tw.
7. Smoking abstinence.tw.
8. "Tobacco use cessation$1".tw.
9. Nicotine cessation$1.tw.
10. Nicotine abstinence$1.tw.
11. Nicotine abstination.tw.
12. Abstinence from tobacco.tw.
13. Abstinence from smoking.tw.
14. Abstinence from nicotine.tw.
15. Smoking reduc$.tw
16. "Tobacco use reduc$".tw.
17. Reduc$ smoking.tw.
18. Modification of smoking.tw.
19. Modified smokng.tw.
20. Modified tobacco consumption.tw.
21. Modification of cig$.tw.
22. Reduc$ tobacco consumption.tw.
23. Cigar$ reduc$.tw.
24. Reduc$ cigar$.tw.
25. Harm reduc$.tw
26. Tobacco consumption.tw.
27. Or/1-26
28. Exp weight change/
29. Exp weight reduction/
30. Weight reduction program?.tw.

31. Emaciation.tw.
32. Weight watching.tw.
33. Exp weight gain/
34. Weight lifting.tw.
35. Exp weight fluctuation/
36. Weight fluctuation?.tw.
37. Exp body weight/
38. Weight.tw.
39. Exp body size/
40. Body size.tw.
41. Exp waist circumference/
42. Waist circumference$1.tw.
43. Exp waist hip ratio/
44. (Waist adj hip ratio).tw.
45. Exp skinfold thickness/
46. (Skinfold thickness or skinfold measurement).tw.
47. Exp body mass
48. (Body mass index or BMI or Quetelet index).tw.
49. Exp weight height ratio/
50. (Weight adj height ratio).tw.
51. Or/37-50
52. (Chang$ or increas$ or gain$ or decreas$ or reduc$ or los$).tw.
53. 51 and 52
54. Or/28-36,53
55. Clinical study/
56. Case control study
57. Family study/
58. Longitudinal study/
59. Retrospective study/
60. Prospective study/
61. Randomized controlled trials/

62. 60 not 61
63. Cohort analysis/
64. (Cohort adj (study or studies)).mp.
65. (Case control adj (study or studies)).tw.
66. (Follow up adj (study or studies)).tw.
67. (Observational adj (study or studies)).tw.
68. (Epidemiologic$ adj (study or studies)).tw.
69. (Cross sectional adj (study or studies)).tw.
70. Or/55-59,62-69
71. 27 and 54 and 70

#1-27: searching for exposure

#28-54: searching for outcome

#55-70: SIGN searching filter for observational studies in EMBASE via Ovid

CINAHL via EBSCO

1. MH "Smoking Cessation"
2. MH "Smoking Cessation Programs"
3. "smoking cessation"
4. (MH "Smoking+")
5. "tobacco consumption"
6. "tobacco use"
7. (MH "nicotine") or "nicotine"
8. 4 or 5 or 6 or 7
9. Quit* or stop* or abstinence or dehabituation or reduc* or decreas* or gain* or increas* or modif*
10. 8 and 9
11. 1 or 2 or 3 or 10
12. (MH "Body Weight Changes+")
13. TI "weight"
14. AB "weight"
15. (MH "Body Weight")
16. TI "body weight"
17. AB "body weight"
18. (MH "Waist Circumference")
19. TI "Waist Circumference*"
20. AB "Waist Circumference*"
21. (MH "Waist-Hip Ratio")
22. TI "Waist-Hip Ratio*"
23. AB "Waist-Hip Ratio*"
24. (MH "Body Mass Index")
25. TI "Body Mass Index*" or "BMI"
26. AB "Body Mass Index*" or "BMI"
27. (MH "Body Size")
28. TI "Body Size*"
29. AB "Body Size*"

30. 13 or 14 or 15 or 16 or 17 or 18 or 19 or 20 or 21 or 22 or 23 or 24 or 25 or 26 or 27 or 28 or 29
31. Chang* or increas* or gain* or decreas* or reduc* or los*
32. 30 and 31
33. 12 or 32
34. (MH "Prospective Studies+")
35. TI prospective stud* OR AB prospective stud*
36. (MH "Case Control Studies+")
37. TI case control stud* OR AU case control stud*
38. (MH "Correlational Studies")
39. TI correlational stud* OR AB correlational stud*
40. TI cohort stud* OR AB cohort stud*
41. (MH "Nonexperimental Studies+")
42. TI longitudinal stud* OR AB longitudinal stud*
43. TI observational stud* OR AB observational stud*
44. 34 or 35 or 36 or 37 or 38 or 39 or 40 or 41 or 42 or 43
45. 11 and 33 and 44

#1-11: searching for exposure

#12-33: searching for outcome

#34-44: searching filter for observational studies

Scopus

Because there is no controlled vocabulary search terms for Scopus, all the following searching terms were performed in the **article title, abstract and keywords** to strike a balance between sensitivity and precision. Boolean 'OR' operator were used to join the synonyms, related terms and variant spellings. Then, different sets of terms were joined together with Boolean 'AND' operator. In order to capture variations as comprehensive as possible, truncation and wildcards were be utilized in the searching procedure.

- **Intervention** (In this review intervention is viewed as exposure):

  #1 ("smoking cessation" or "smoking abstinence" or "tobacco use cessation" or "nicotine cessation" or "nicotine abstinence" or "abstinence from smoking" or "abstinence from tobacco" or "abstinence from nicotine" or "quit* smoking" or "stop* smoking" or "smoking reduc*" or "tobacco use reduc*" or "modified smoking" or "modified tobacco consumption" or "modification of cig*" or "modification of smoking")

- **Outcome**:

  #2 ("weight" or "body weight" or "body size" or "body mass index*" or "BMI" or "waist-hip ratio*" or "waist hip ratio" or "waist circumference*" or "quetelet index*")

  #3 (chang* or increas* or gain* or decreas* or reduc* or los*)

  #4 #2 and #3

- **Study design**:

  #5 ("epidemiologic* stud*" or "cohort stud*" or "case control stud*" or "case-control stud*" or "longitudinal stud*" or "follow up stud*" or "follow-up stud*" or "prospective stud*" or "retrospective stud*" or "observational stud*")

- #1 and #4 and #5
- **Limiter:** human

Web of Science

- **Intervention** (In this review, intervention will be viewed as the exposure):

  #1 Topic=(smoking cessation) OR Topic=(smoking abstinence) OR Topic=(tobacco use cessation) OR Topic=(nicotine cessation) OR Topic=(nicotine abstinence) OR Topic=(abstinence from smoking) OR Topic=(abstinence from tobacco) OR Topic=(abstinence from nicotine) OR Topic=(quit* smoking) OR Topic=(stop* smoking) OR Topic=(smoking reduc*) OR Topic=(tobacco use reduc*) OR Topic=(modified smoking) OR Topic=(modified tobacco consumption) OR Topic=(modification of cig*) OR Topic=(modification of smoking)

- **Outcome**:

  #2 Topic=(weight) OR Topic=(body weight) OR Topic=(body size) OR Topic=(body mass index*) OR Topic=(BMI) OR Topic=(waist-hip ratio*) OR Topic=(waist hip ratio) OR Topic=(waist circumference*) OR Topic=(quetelet index*)

  #3 (chang* or increas* or gain* or decreas* or reduc* or los*)

  #4 #2 and #3

- **Study design**:

  #5 Topic=(epidemiologic* stud*) OR Topic=(cohort stud*) OR Topic=(case control stud*) OR Topic=(case-control stud*) OR Topic=(longitudinal stud*) OR Topic=(follow up stud*) OR Topic=(follow-up stud*) OR Topic=(prospective stud*) OR Topic=(retrospective stud*) OR Topic=(observational stud*)

- #1 and #4 and #5

4.10 Appendix 4-2: Adapted version of Newcastle-Ottawa quality assessment scale for cohort studies

Identification details:

Study ID: **Reviewer:**

Data: **Author(year):**

Journal of Reference:

Newcastle-Ottawa quality assessment scale cohort studies adapted version				
		Star awarded system	**Star awarded**	**Star**
Studies selection criteria				
1) Representativeness of the exposed cohort (maximum 1 star)	a) truly representative of the average _____ (describe) in the community	*		
	b) somewhat representative of the average _____ in the community	*		
	c) selected group of users eg nurses, volunteers	(no star)		
	d) no description of the derivation of the cohort	(no star)		
2) Selection of the non-exposed cohort (maximum 1 star)	a) drawn from the same community as the exposed cohort	*		
	b) drawn from a different source	(no star)		
	c) no description of the derivation of the non-exposed cohort	(no star)		
3) Ascertainment of exposure (maximum 1 star)	a) bio-verified smoking status	*		
	b) structured interview	*		
	c) written self-report	(no star)		
	d) no description	(no star)		
Comparability				
1) Comparability of cohorts on the basis of the design or analysis (maximum of 2 star)	a) study controls for age and sex	*		
	b) study controls for additional factors: SES or illness	*		
Studies outcome criteria				
1) Assessment of outcome (maximum 1 star)[#]	a) independent measurement	*		
	b) record linkage	*		
	c) self-report	(no star)		
	d) no description	(no star)		
2) Adequacy of follow up of cohorts (maximum 1 star)[&]	a) complete follow up - all subjects accounted for	*		
	b) subjects lost to follow up unlikely to introduce bias - small number lost > 80% (select an adequate %) follow up, or description provided of those lost)	*		
	c) follow up rate < 80% (select an adequate %) and no description of those lost	(no star)		
	d) no statement	(no star)		
			Final Score	

[#] 0.5* is given if the self-reported outcome was highly correlated with the measured one (correlation is more than 0.95); [&] 0.5* is given if lost to follow-up > 20% and with description of those lost.

4.11 Appendix 4-3: Summary of studies with categorical results

ID	Author (year), country	Sex	Race	Weight measure methods	Maximum F-U(year)	Outcome categories	Other information
Absolute weight change							
1	Bosse (1980), USA	M	Predominantly white	M	5	< -0.91kg; ≥ -0.91 to ≤ 0.91kg; > 0.91kg	None
2	Noppa (1980), Sweden	F	NA	M	5	≤ -10.0kg; > -9.9 to ≤ -5.0kg; > -4.9 to ≤ -0.1kg; > 0.0 to ≤ 4.9kg; > 5.0 to ≤ 9.9kg; ≥10.0kg;	None
3	Friendman (1980), USA	M/F	White	M	1.5	≥10.0lb; ≥20.0lb;	Quitters and CS were grouped into < 1 pack/day and ≥ 1 pack/day.
4	Williaamson (1991), USA	M/F	White (≥80%), Black, and other	M	7	≤3.0kg; >3.0 to ≤8.0kg; >8.0 to ≤13.0kg; >13.0kg	Quitters were grouped into recent (< 1 year) and sustained (≥ 1year).
5	Swan (1994), USA	M	NA	SR	16	<-2.3kg; ≥-2.3 to <2.3kg; ≥2.3 to <11.3kg; ≥11.3kg	None
6	Klesges (1998), USA	M/F (C)	Black White	M M	7 7	≥5.0kg; ≥10.0kg ≥5.0kg; ≥10.0kg	None None
7	Lee (2001), South Korea	M	NA	M	4	≥1kg; >4kg	None
8	Brown (2005), Australia	F	NA	SR	5	< -2.25kg; ≥ -2.25 to <2.25kg; ≥2.25 to <5kg; ≥5 to <10kg; ≥10kg	None
9	Luo (2012), USA	F	NA	M	3	≥5kg	None
10	Oba (2012), Japan	M/F	NA	SR	5	≥3kg	None
Relative weight change							

11	Wannamethee (2001), England, Wales and Scotland	M	NA	M at screening, SR at F-U	5	Loss of ≥4%; Within 4%; >4% to ≤10%; >10%	None
12	Hansson (2011), Sweden	M	NA	SR	5	≥5%	None

4.12 Appendix 4-1: Summary of quality assessment

ID	First author	Year	Total scores	Selection			Comparability		Outcome	
				Repre. of the exp. cohort	Repre. of the unexp. cohort	Exposure ascertain.	Cont. of age & sex	Cont. of SES or illness	Outcome assessment	Adequacy of F-U
1	Gordon	1975	4	*	*	0	*	0	*	0
2	Bosse	1980	3	0	*	0	*	0	*	0
3	Friendman	1980	3	*	*	0	*	0	0	0
4	Williaamson	1991	6.5	*	*	*	*	*	*	0.5*
5	Lissner	1992	6	*	*	*	*	0	*	*
6	Swan	1994	2	0	*	0	*	0	0	0
7	Kawachi	1996	2.5	0	*	0	0	0	0.5*	*
8	Hodge	1996	5	*	*	*	0	0	*	*
9	Burnette	1997	4.5	*	*	0	*	0	*	0.5*
10	Bartholomew	1998	4.5	*	*	0	*	0	*	0.5*
11	Klesges	1998	6	*	*	0	*	*	*	*
12	Froom	1999	4.5	*	*	*	0	0	*	0.5*
13	Burke	2000	3.5	*	*	0	0	0	*	0.5*
14	Wannamethee	2001	4	*	*	*	0	0	0.5*	0.5*
15	Lee	2001	2.5	0	*	0	0	0	*	0.5*
16	Janzon	2004	6	*	*	0	*	*	*	*
17	Brown	2005	4	*	*	0	*	0	0	*
18	Chinn	2005	4	*	*	0	0	0	*	*
19	John	2006	2.5	*	*	0	0	0	0	0.5*

ID	First author	Year	Total scores	Selection			Comparability		Outcome	
				Repre. of the exp. cohort	Repre. of the unexp. cohort	Exposure ascertain.	Cont. of age & sex	Cont. of SES or illness	Outcome assessment	Adequacy of F-U
20	Song	2008	3.5	*	*	0	0	0	*	0.5*
21	Sneve	2008	4	*	*	0	0	0	*	*
22	Byung	2009	1	0	*	0	0	0	0	0
23	Munafo	2009	7	*	*	*	*	*	*	*
24	Reas	2009	4	*	*	*	*	0	0	0
25	Yoon	2010	4	0	*	0	*	*	*	0
26	Suwazono	2010	4.5	*	*	0	*	0	*	0.5*
27	Holz	2010	3.5	*	*	0	0	0	*	0.5*
28	Yeh	2010	6	*	*	*	*	*	0.5*	0.5*
29	Basterra-Gortari	2010	5	0	*	0	*	*	0.5*	0.5*
30	Kawada	2010	2.5	0	*	0	0	0	*	0.5*
31	Hansson	2011	4.5	*	*	0	*		0	0.5*
32	Oba	2012	3	*	*	0	0	0	0.5*	0.5*
33	Travier	2012	5	*	*	0	*	*	0.5*	0.5*
34	Luo	2012	3.5	*	*	0	0	0	*	0.5*
35	Clair	2013	2.5	0	*	0	0	0	*	0.5*

Chapter 5

Worsening dietary and physical activity behaviours do not readily explain why smokers gain weight after cessation: a cohort study in young adults

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Chapter 5 Worsening dietary and physical activity behaviours do not readily explain why smokers gain weight after cessation: a cohort study in young adults

5.1 Preface

As described in the previous chapter, fear of weight gain often discourages smokers from trying to quit but guidance on ways to most effectively avoid this weight gain is lacking. It is important to identify what causes post-cessation weight gain and the ways it may be prevented. The current study aims to explore the effects of several changing dietary and PA behaviours on the relationship between smoking cessation and weight gain in a cohort of young Australian smokers.

5.2 Introduction

Smoking and overweight or obesity are well-documented risk factors for many diseases, including CVDs. The changing population prevalence of these risk factors, however, are moving in opposite directions, with smoking declining and overweight increasing [1-3]. As described in the previous chapter, quantitative analyses have shown that, on average, smokers weigh less than non-smokers, and quitters weigh more than continuing smokers [3,4]. A recent meta-analysis of RCTs found that those quitting smoking gained an average of 4-5kg after 12 months of abstinence, with most of the weight gain occurring in the first three months of quitting [5]. The estimate from a meta-analysis of prospective cohort studies was 4.1kg over an average of five years, which is 2.6kg greater than the gain in continuing smokers [6]. Therefore, it is unsurprising that fear of weight gain is commonly cited by smokers as a reason for not quitting, especially for weight-concerned groups like women [7,8] and obese smokers [9] even though the health benefits of quitting far outweigh the health risks associated with weight gain [10,11].

Several systematic reviews of interventions that aimed to prevent weight gain after smoking cessation, including pharmacotherapies, exercise and dietary interventions showed little success with no strong clinical recommendation available to smokers who want to quit [5,12-15]. It would be beneficial to identify factors that can explain or modify weight gain following smoking cessation but the literature is sparse or inconclusive.

Time spent in sedentary behaviours, such as sitting or television (TV) viewing, is believed to be one of the factors underlying the globally increasing prevalence of overweight and obesity [16]. Sitting and TV viewing time have been found to be positively associated with adiposity or weight gain in many populations [17-19]; however, no study has tested their roles in post-cessation weight gain among those who quit smoking. There are two longitudinal studies investigating the relationship between weight gain and concurrent change of PA level among quitters and continuing smokers. One of these studies focused only on leisure time PA (LTPA) rather than total PA [20] while the sample in the other study [21] was limited to participants in RCTs of smoking cessation treatments and they are usually not representative of smokers in the general population [22].

In terms of dietary factors, there is evidence that quitters have a desire to have something in their mouths to substitute for cigarettes which may result in changes in diet, such as an increase in sugar [23,24], fat [23,25,26] and overall daily calorie intake [23,26,27]. Few studies have measured the effects of changing dietary behaviours after quitting smoking [28]. Dietary behaviours, which reflect the ways people eat (for example, consumption of fruit and vegetable, discretionary foods and takeaway food, and breakfast skipping) and their diet quality are closely associated with weight fluctuation [29-33]. Compared with the public health messages based on energy intake, those addressing dietary behaviours may be easier for people to understand. For example, a recommendation to limit takeaway food consumption is easier to follow than a recommendation to restrict energy intake (which would require knowledge of the energy content of all foods consumed). No reported study has assessed the effects of changing dietary behaviours on the magnitude of weight gain after smoking cessation.

The aim of this study was to evaluate whether the greater weight gain after cessation in quitters than continuing smokers could be attributed to changes in several dietary and PA behaviours in a cohort of young adults.

5.3 Methods

5.3.1 Participants

Sampling procedures have been presented in the section of 2.3.1.

5.3.2 Smoking status assessment

Assessment of smoking status has been described in the section of 2.3.4.Weekly and less than weekly smokers were defined as occasional smokers. Daily smokers were asked to report the number of cigarettes smoked per day and recall the age at which they started smoking daily. Duration of smoking in years was calculated using this age subtracted from the age at which they completed the questionnaire and pack-years of smoking was calculated by multiplying the number of packs of cigarettes smoked per day by the duration of smoking in years. People who were daily smokers at baseline but former smokers at follow-up were also asked to report the age at which they stopped daily smoking and the total number of times they had tried to quit daily smoking.

The main exposure in this longitudinal analysis was quitting smoking between baseline and follow-up, therefore participants were restricted to current smokers at baseline and categorised as continuing smokers (current smokers at baseline and follow-up) or quitters (current smokers at baseline and former smokers at follow-up).

5.3.3 Anthropometric measurements

At baseline, weight and height were objectively measured at study clinics for most participants (n=2,410) by trained clinic staff. A subsample of these participants also self-reported their weight and height before measurements were taken to assess the accuracy of self-reported values. The difference between clinic and self-reported weight and height was used to calculate a correction factor from a linear regression model [35]. Participants who did not visit a study clinic (n=1,557) self-reported their weight and height, and the correction factor was applied to adjust for error. For the anthropometric measurements, participants wore light clothing without shoes. All measurements were made by trained staff. Body weight was measured using a Heine portable scale (Heine, Dover, NH, USA) and recorded to the nearest 0.1kg. Height was measured using a portable Leicester stadiometer (Invicta, Leicester, UK) and recorded to the nearest 0.1cm. BMI (kg/m^2) was calculated from height and weight.

Weight was self-reported at follow-up. Adjusted weight values were calculated using the correction factor applied at baseline [35]. BMI was calculated using adjusted height at baseline and adjusted weight at follow-up.

5.3.4 Dietary assessment

Dietary behaviours were assessed using a meal pattern chart, a 127-item food frequency questionnaire (FFQ) and a food habit questionnaire (FHQ) at baseline and follow-up. Dietary behaviours considered as potential mediators of weight change were changes in "discretionary" foods, fruit and vegetable, dietary guideline index (DGI) score, takeaway food, breakfast skipping and alcohol consumption.

A meal pattern chart for the previous day was completed and the day of the week recorded by participants. The chart divided the day into hourly intervals from 0600 to 2300 and 2300 to 0600 was combined. For each time interval, participants were asked to choose one of four responses to the question "Did you eat anything?": "no", "a snack", "a small meal" or "a large meal". Examples of each meal type were given. Breakfast was defined as eating a snack, small meal, or large meal from 0600 to 0900 [36]. A similar method of assessing meal patterns has been used in a previous study [37].

The FFQ included 127 items and participants reported how often each item was consumed in the previous 12 months, using a 9-point scale from "never or less than once a month" to "six or more times per day". Daily equivalents were calculated for each FFQ item, assuming one serving was consumed at each eating occasion, as described elsewhere [30]. The FFQ was a modified version of the one which was used in the 1995 National Nutrition Survey (NNS) [38-41]. It was based on an existing FFQ developed for Australian populations [42].

Foods that do not fit into five core food groups (fruit, vegetables, dairy, breads and cereals, lean meats) are considered "discretionary" foods. They are typically high in fat, salt and sugars and provide very few essential nutrients [43]. Examples of discretionary foods include ice cream, savoury pastry, pizza, hot chips, etc. Examples in detail were listed in our previous publication [30]. For analysis the takeaway food items (hamburgers, pizza, hot chips, fried fish and savoury pastry) were excluded from the discretionary foods variables so that they could be distinguished separately. Daily alcohol consumption in grams was estimated

from the usual frequency reported in the FFQ of 10 common alcoholic beverages multiplied by the average alcohol concentration of each beverage.

The FHQ included questions on takeaway food and usual fruit and vegetable consumption. Participants were asked to answer "How many times per week would you usually eat hot takeaway meals (e.g. pizza, burgers, fried or roast chicken, Chinese/Indian/Thai takeaway)" from choosing one of five responses ranging from "I don't eat takeaway" to "6-7 meals per week". For analysis, the answers were dichotomised to less than twice per week or twice a week or more as we have shown that eating takeaway food twice a week or more was associated with abdominal obesity [30]. Four categories were created to examine change in takeaway food consumption during follow-up: twice a week at neither baseline or follow-up, twice a week or more at baseline only, twice a week or more at follow-up only, twice a week or more at both baseline and follow-up. Takeaway food consumption from the short question has been validated in a previous study [30].

Self-reported daily fruit and vegetable consumption was measured using two short questions "how many servings of fruit/vegetables (excluding potatoes) do you usually eat each day". Examples of serving sizes were given and possible response options included "I don't eat this food", "1 serving or less", "2-3 servings", "4-5 servings" or "6 or more servings". We combined these to get an overall estimate of daily fruit and vegetable consumption. Short questions have been used in previous studies [39,44] and have been shown to be valid measures for fruit and vegetable intake [45].

Information from the FFQ and FHQ was used to assess diet quality using a DGI based on the Dietary Guidelines for Australian Adults [46] and the Australian Guide to Healthy eating [43]. The score included 15 components and each component was scored from 0 to 10, with 10 indicating that a participant was meeting the requirement or had an optimal intake. For example, in regard to fruit intake, 2 servings/day was the recommended amount and scored 10 points, 1 serving/day scored 5 points and no consumption of fruit scored 0 point. The total sum of DGI score ranged from 0 to 150. A higher score denoted better compliance with the dietary guidelines. The mean score was around 100 in an Australian NNS [39], but no recommended score is currently available for the general population. If people have a score of 100 and the potential range is 0-150, then they are meeting two-thirds of the dietary

guidelines. More detailed information about the scores is presented elsewhere [39]. This score has been shown to be a valid measure of diet quality [39,47].

5.3.5 Self-reported PA assessment

Self-reported PA was measured using the long version of the International Physical Activity Questionnaire (IPAQ-L) [48]. Participants were asked to report the total time (mins) and frequency (times/week) of occupational, domestic, commuting and LTPA during the past week. Minutes/week spent in each domain were calculated by multiplying frequency by duration. Time spent doing PA in each domain was summed to provide an estimate of total minutes of PA. Time spent sitting was reported for a typical weekday and weekend day. To determine the average daily sitting time (minutes/day), time spent sitting on weekdays and weekend days were summed and divided by seven. Daily TV viewing time (hours/week) in the past week was estimated from self-reported total time spent watching TV, digital video disks, or videocassettes by participants in relation to weekdays and weekend days as described in detail elsewhere [49].

5.3.6 Pedometer-determined PA

Participants wore a Yamax Digiwalker pedometer (SW-200) for 7 consecutive days and recorded total steps at the end of each day, daily start time and daily end time. Daily records were excluded if the pedometer was worn for less than 8 hours or >60,000 steps were reported. Mean daily steps were calculated for participants with a minimum of four valid days of readings. In general, pedometers have been shown to strongly correlate with concurrent accelerometer measures (γ=0.86) and observed time spent in activity (γ=0.82) [50].

5.3.7 Other covariates

Socio-demographic characteristics were self-reported at baseline including age, sex, marital status (married or living as married versus other), education (high school only, vocational training, any university education) and occupation (not in the labour force, manual, non-manual, and professional or manager). Follow-up length and baseline BMI were also considered as potential confounders in the analyses.

5.3.8 Statistical analyses

Analyses were restricted to participants who were not pregnant and who had completed 1) both baseline and follow-up smoking questionnaires and 2) baseline dietary questionnaires

and the IPAQ-L. Approximately half the current smokers (49%) were missing one or more dietary or PA behaviours at follow-up. Therefore MI by chained equations was used [51]. The number of imputations was 40 [52]. Changes in dietary and PA behaviours were generated based on collected information at baseline and imputed data at follow-up.

Means with SDs and numbers with proportions were used to describe the socio-demographic characteristics, dietary and PA behaviours of the participants according to whether or not they quit smoking from baseline to follow-up. Comparisons between the two groups were performed using t-tests for continuous variables and chi-square tests for categorical variables. Linear regression models were used to assess the association between smoking cessation and weight change. In analyses that explored whether changes in dietary and PA behaviours could explain the post-cessation weight gain, a base model was initially fitted, adjusting for socio-demographic characteristics (age, sex, and education level), follow-up length and baseline BMI. A second model adjusted for change in dietary factors and a third model adjusted for change in PA behaviours. Change in dietary and PA variables were entered into the base model one at a time. A fourth model included both changes in dietary and PA variables. Potential confounding factors kept in the base model were variables which were associated with the outcome and were not mediators between the exposure and the outcome, and which resulted in a >10% change in the coefficient of the principal study factor when added in the model.

Sensitivity analyses excluding participants with imputed data were performed to examine the influence of missing data on results. The analysis was also repeated among participants with pedometer-measured PA.

A two-tailed P value less than 0.05 was considered statistically significant. All analyses were performed with STATA software, version 12.1 (Stata Corp, College Station, Texas 77845 USA).

5.4 Results

Of the 785 participants who were current smokers at baseline, 274 were lost to follow-up and we excluded pregnant women at baseline or follow-up (n=6), those who were missing weight or BMI change data (n=28), and those who were missing baseline dietary or PA data

(n=196). This left 281 participants. 124 of them quit smoking during the 5-year follow-up. The anthropometric and socio-demographic characteristics of participants are shown in **Table 5-1**. The age ranged from 26 to 36 years for both continuing smokers and quitters. Compared with continuing smokers, quitters were more often female, employed as professionals or managers and smoked weekly or less than weekly, smoked less cigarettes per day and had a lighter exposure to tobacco, with some differences of borderline statistical significance. There were no statistically significant differences between the two groups in age, marital status, education level, weight, BMI, weight status and duration of smoking at baseline.

Table 5-1 Socio-demographic and anthropometric characteristics of continuing smokers and quitters in the Childhood Determinants of Adult Health Study, Australia, 2004-2006*

Characteristic	Continuing smokers (n=157)	Quitters (n=124)	P-value
Age (years)	31.3±2.4	31.6±2.7	0.487
Males sex (%)	54.1	43.6	0.078
Married or living as married (%)	66.2	58.9	0.204
Education (%)			0.150
Any university education	24.8	35.5	
Vocational training	35.7	29.8	
High school only	39.5	34.7	
Occupation (%)†			0.053
Professional or manager	41.5	53.2	
Nonmanual	19.1	22.6	
Manual	27.6	15.3	
Not in the labour force	11.8	8.9	
Weight (kg)	79.1±16.0	76.4±17.8	0.188
BMI (kg/m^2)	26.1±4.7	25.4±4.5	0.222
Weight status (%)			0.302
Normal (< 25)	46.5	51.6	
Overweight (25 – 29.9)	35.7	37.1	
Obese (≥ 30)	17.8	11.3	
Change in weight (kg)	**2.3±7.4**	**4.4±7.3**	**0.019**
Change in BMI (kg/m^2)	**0.7±2.5**	**1.4±2.6**	**0.021**
Frequency of smoking			**0.001**
Less than weekly	**10.8**	**26.6**	
Weekly	**15.9**	**17.7**	
Daily	**73.3**	**55.7**	
Number of cigarettes/day†	**13.4±7.3**	**11.1±7.2**	**0.045**
Smoking duration (years)†	14.2±4.1	13.5±5.0	0.305
Pack-years†	9.9±6.7	8.1±6.8	0.078

Bold denotes statistically significant result.
BMI: body mass index; SD: standard deviation.
* Limited to participants with full information of smoking status, age, sex, change in weight, change in BMI, dietary and physical activity variables at baseline; Mean±SD except for percentages; P-values determined by t test or person χ^2 test (where appropriate).
† Sample size ranged 174-276.

During five years follow-up, continuing smokers gained an average of 2.3kg (SD: 7.4) weight and 0.7kg/m^2 (SD: 2.5) BMI. Quitters gained an average of 4.4kg (SD: 7.2) weight and 1.4kg/m^2 (SD: 2.5) BMI. The amount of post-cessation weight gain was moderately increased after taking into account baseline frequency of smoking. It was largely unchanged after adjustment for cigarettes smoked per day, duration of smoking and pack-years among daily smokers at baseline (**Appendix 5 Table S1**). No significant difference was observed in post-cessation weight gain related to time since quitting and number of previous quit attempts (**Appendix 5 Table S2**).

Compared with the general population of 25-34 years old Australians, a higher proportion of our sample (from whom the 281 current smokers were drawn) were married/living as married (69.9% versus 56.8%) [53] and were university-educated (48.3% versus 35.1%) [54], and a lower proportion currently smoked (20.5% versus 29.8%) [55]. The proportion classified as being overweight or obese (BMI ≥ 25) was very similar (48.1% versus 46.5%) [55].

Participants lost to follow-up were more likely to be single and less educated. There were no statistically significant differences in age, weight status and occupation level at baseline between smokers who participated in the follow-up and those who did not (data not shown).

Table 5-2 presents the dietary behaviours at baseline, follow-up, and their changes during follow-up. At baseline, quitters reported a higher DGI score (98.6 versus 93.8, P=0.031) and less daily alcohol consumption (11.0 versus 15.6 g/day, P=0.008) than continuing smokers. No other statistically significant differences were observed in baseline dietary behaviours. Similar differences were found at follow-up (DGI score: 103.5 versus 96.6, P=0.008; alcohol consumption: 8.8 versus 13.0 g/day, P=0.018) and quitters also consumed less discretionary foods, more fruit and vegetables, less often skipped breakfast and less often consumed takeaway food at least two times per week than continuing smokers; however, these differences did not reach statistical significance. There were no significant differences in changing dietary behaviours between quitters and continuing smokers.

Table 5-2 Dietary behaviours at baseline, follow-up and changes during follow-up, for continuing smokers and quitters*

Dietary behaviours	Continuing smokers (n=157)	Quitters (n=124)	P-value
Servings of 'discretionary' foods /day			
Baseline	4.3±2.6	3.8±2.3	0.082
Follow-up	4.0±3.3	3.4±2.5	0.101
Change from baseline to follow-up†	-0.3±2.8	-0.4±2.4	0.888
Servings of fruit and vegetables/day			
Baseline	3.6±1.5	3.6±1.7	0.808
Follow-up	3.6±1.6	3.9±1.8	0.127
Change from baseline to follow-up†	-0.0±1.6	0.2±1.5	0.162
Diet Guideline Index score			
Baseline	**93.8±19.3**	**98.6±17.3**	**0.031**
Follow-up	**96.6**±21.0	**103.5±21.1**	**0.008**
Change from baseline to follow-up†	2.8±19.5	4.9±19.4	0.386
Alcohol consumption (grams/day)			
Baseline	**15.6±20.3**	**11.0±9.6**	**0.020**
Follow-up	**13.0±17.3**	**8.8±10.9**	**0.018**
Change from baseline to follow-up†	-2.6±22.0	-2.2±11.2	0.867
Consuming takeaway food (≥2/wk) (%)			
Baseline	33.1	32.3	0.878
Follow-up	32.6	22.2	0.094
Change from baseline to follow-up			
Neither baseline or follow-up	54.8	59.6	0.158
Baseline only	12.6	18.1	
Follow-up only	12.1	8.1	
Both baseline and follow-up	20.5	14.1	
Skipping breakfast (%)			
Baseline	38.9	39.5	0.910
Follow-up	41.7	31.3	0.126
Change from baseline to follow-up			
Neither baseline or follow-up	39.3	44.4	0.190
Baseline only	19.0	24.2	
Follow-up only	21.8	16.0	
Both baseline and follow-up	19.9	15.3	

Bold denotes statistically significant result.
SD: standard deviation.
* Mean±SD except for percentages; P-values determined by t test or person χ^2 test (where appropriate).
† Calculated using follow-up values minus baseline ones.

Table 5-3 describes the PA behaviours at baseline, follow-up, and their changes from baseline to follow-up. No statistically significant difference was observed between quitters and continuing smokers at baseline, follow-up or in their changes during follow-up. Overall, the PA behaviours in quitters tended to become healthier compared with those among continuing smokers, except time spent in sitting. Quitters reported more time spent in sitting than continuing smokers at both baseline and follow-up.

Table 5-4 documents the results for the linear regression analyses of smoking cessation on weight change. Before adjustment, quitters gained an average of 2.09kg greater weight than continuing smokers. This association was largely unchanged after adjustment for baseline age, sex, BMI, education level and follow-up length. Further adjustment for change in each dietary and PA behaviour slightly altered the estimate, with changes in β coefficients ranging from -0.08% to 15.46%. In the final fully adjusted model, the mean weight gain was 2.32kg greater in quitters than continuing smokers, and the overall change in β coefficient was 20.33%. Factors included in the fully adjusted model were baseline age, sex, BMI, education level, follow-up length, changes in dietary behaviours (discretionary foods, fruit and vegetable, DGI score, consuming takeaway food, skipping breakfast and alcohol), change in LTPA and sitting time.

When BMI replaced weight as the outcome, the effects of changes in dietary and PA behaviours on the magnitude of BMI change after quitting smoking were similar to the change in weight. The results are summarized in the **Appendix 5 Table S3.**

In sensitivity analyses, similar results were observed after excluding persons with imputed data (**Appendix 5 Table S4, S5 and S6**). Change in pedometer-measured PA was available for 52 continuing smokers and 58 quitters over follow-up (**Appendix 5 Table S7**). When the analysis was repeated using pedometer-measured PA, the change in β coefficient was similar in magnitude to that found with change in total PA measured by the IPAQ-L.

Table 5-3 Physical activity behaviours at baseline, follow-up and changes during follow-up, for continuing smokers and quitters*

Physical activity behaviours	Continuing smokers (n=157)		Quitters (n=124)		P-value
	Mean	SD	Mean	SD	
Total PA (minutes/week)					
Baseline	889.9	541.9	796.2	526.8	0.146
Follow-up	786.1	731.8	744.9	639.6	0.600
Change from baseline to follow-up†	-103.8	774.8	-51.3	719.3	0.542
Total LTPA (minutes/week)					
Baseline	134.3	170.8	135.3	151.3	0.956
Follow-up	132.4	238.9	175.6	232.4	0.107
Change from baseline to follow-up†	-1.8	241.4	40.3	245.1	0.128
Sitting time (minutes/day)					
Baseline	324.4	175.2	340.7	154.4	0.417
Follow-up	330.5	200.2	351.8	181.3	0.336
Change from baseline to follow-up†	6.0	209.5	11.1	206.9	0.835
TV viewing time (hours/day)					
Baseline	2.4	1.4	2.1	1.8	0.088
Follow-up	2.3	2.0	2.0	1.7	0.131
Change from baseline to follow-up†	-0.1	2.1	-0.1	2.0	0.986

Bold denotes statistically significant result.
LTPA: leisure time physical activity; PA: physical activity; SD: standard deviation; TV: television.
* P-values determined by t test.
† Calculated using follow-up values minus baseline ones.

Table 5-4 Effects of changes in dietary and physical activity behaviours on the magnitude of weight (kg) gain after quitting smoking during follow-up, compared with continuing smoking

	Models	β	95% CI	Change in β*
	Unadjusted	2.09	0.35, 3.83	
	Model 1†	1.93	0.18, 3.67	
Changing dietary behaviours	Model 1 + changing discretionary foods consumption	1.95	0.21, 3.69	1.11%
	Model 1 + changing fruit and vegetable consumption	1.94	0.18, 3.69	0.46%
	Model 1 + changing diet guideline index score	1.99	0.24, 3.74	3.28%
	Model 1 + changing alcohol consumption	1.99	0.23, 3.75	3.23%
	Model 1 + change in eating takeaway food	2.00	0.25, 3.76	3.83%
	Model 1 + change in skipping breakfast	1.93	0.19, 3.66	-0.07%
	Model 1 + changing all dietary behaviours	2.10	0.31, 3.88	8.66%
Changing PA behaviours	Model 1 + changing total PA	1.98	0.24, 3.72	2.70%
	Model 1 + changing LTPA	2.23	0.48, 3.97	15.46%
	Model 1 + changing sitting time	1.93	0.18, 3.67	-0.08%
	Model 1 + changing TV viewing time	1.93	0.18, 3.68	0.22%
	Model 1 + changing LTPA and sitting time	2.23	0.48, 3.97	15.61%
Changing dietary and PA behaviours	Model 1 + changing all dietary behaviours, LTPA and sitting time	2.32	0.54, 4.10	20.33%

* Relative to Model 1.

† Adjusted for age, sex, BMI, education and follow-up length.

BMI: body mass index; CI: confidence interval; PA: physical activity; LTPA: leisure time physical activity; TV: television.

5.5 Discussion

We found that compared with continuing smoking, smoking cessation was associated with an excess weight gain of 1.9 kg in young adults. Unexpectedly, this weight gain was not substantially attenuated after adjustment for changes in dietary and PA behaviours, implying that the effects of smoking cessation on weight may not be mediated by these lifestyle factors. Indeed, we observed a greater trend towards healthier behaviours among quitters than continuing smokers, with quitters consuming less discretionary foods, alcohol and takeaway food, having a higher DGI score, less likely to skip breakfast, eating more fruit and vegetable, spending more time in LTPA and less time watching TV.

The findings of 4.4 kg weight gain after cessation in quitters and 1.9 kg greater weight gain in quitters than continuing smokers are very similar to the 4.1 kg and 2.6 kg reported in our recent meta-analysis including 63,403 quitters and 388,432 continuing smokers from 35 prospective cohort studies [6]. Previous studies have reported that the magnitude of post-cessation weight gain is positively related to the heaviness of tobacco smoking partly because of varying impacts on metabolic rate [3,56], and this point was also supported by our data that showed taking into account baseline frequency of smoking increased the amount of weight gain after cessation by 38% (**Appendix 5 Table S1**). Time since quitting and number of quit attempts might influence the amount of weight gain after quitting given that most weight gain is reported to occur during the first few months of abstinence [5] and the positive relationship between heaviness of smoking and number of quit attempts [57]. However, we failed to detect a significant association in a subsample of quitters who were daily smokers at baseline, possibly due to the small sample size. Younger age has previously been associated with a higher risk of major weight gain after quitting [3,58], and a similar study in young people reported a greater weight gain of approximately 5 kg in quitters than continuing smokers [59]. Our finding of 1.9 kg excess weight gain is lower and may reflect our inclusion of more occasional smokers at baseline and shorter follow-up time [6].

In line with the notion that quitters may change their food preferences after cessation [23,24,27], our results confirmed some significant changes in dietary behaviours after quitting smoking; however, all these changes were towards healthier dietary behaviours. For example, both quitters and continuing smokers reported increased DGI and decreased

consumption of takeaway food as they aged, but the changes were greater in quitters than continuing smokers. These findings were consistent with national data from Australia [39] and the United States [60] which show that diet quality increases with age and adults' consumption of calories from fast food decreases with age, respectively. In addition, the difference in DGI score between continuing smokers and quitters at both baseline and follow-up (around 5 points, equivalent to one serving of fruit per day) might be clinically meaningful. It has been reported that an increment of one serving a day for fruit was associated with 6% reduction in the risk of all-cause mortality and 5% reduction in the risk of cardiovascular mortality [61]. Given the 5-year time interval from baseline to follow-up, and evidence suggesting that most post-cessation weight gain occurs in the first three months after quitting [5], it is possible that quitters gained weight shortly after cessation and then changed their dietary behaviours to control the post-cessation weight gain, such as consuming less discretionary foods and takeaway meals, less often skipping breakfast and having a higher DGI score. It is also possible that smokers quit smoking because they wanted to be healthier, so made other changes in behaviours simultaneously. Previous studies have found a clustering of lifestyle risk factors (smoking, excessive alcohol consumption, poor diet and physical inactivity) [62,63], and clustering of multiple risk behaviours increased with daily cigarette consumption [64].

We failed to find that dietary behaviours contributed to weight gain after quitting but the extent to which this is due to our inability to accurately measure energy intake, as noted by others [65], or due to the influence of non-dietary factors is uncertain. Indeed studies exploring energy intake specifically have had contradictory findings. Some studies using self-reported dietary intake indicate that smokers increase their energy intake shortly after quitting [25-27], while others find no change or a decrease in energy intake [66,67]. Evidence from a single clinical trial also found a very low calorie diet was not effective at preventing weight gain among quitters after 12 months follow-up [12]. More accurate and repeated objective measures of energy intake are needed if we are to properly understand the role of energy intake in weight gain after smoking cessation.

The only behaviour in the current study that became less healthy in quitters compared with continuing smokers was the increase in sitting time, but the difference in this change was

trivial. Therefore, it did not explain why smokers gained more weight after quitting. To the best of our knowledge, this is the first study exploring the effects of changes in sedentary behaviours on the relationship of weight gain and smoking cessation. In a cohort study of middle-aged women [20], which investigated whether change in exercise could modify weight gain after smoking cessation by comparing weight gain between continuing smokers who did not change their LTPA level and quitters categorized into groups according to change in LTPA (no change, increase by 8-16 metabolic equivalent of task (MET) hours per week, increase by > 16 MET hours per week), greater weight gain was observed among quitters with no change in LTPA, and the extent of weight gain was mitigated with an increase of LTPA. This result was further supported by data from participants of a one year RCT of smoking cessation treatment examining whether smokers' PA was associated with weight gain after a quit attempt using pedometer-measured total PA: it found that quitters who decreased their PA gained significantly greater weight than those who increased their PA or maintained a high level of activity [21]. A recent meta-analysis investigating the efficacy of a range of interventions to reduce weight gain among quitters concluded that there was insufficient evidence to support specific clinical recommendations [12].

As the behaviours we measured in our study did not seem to explain weight gain, questions arise as to its cause. The underlying mechanisms linking smoking cessation and weight gain are complex and still poorly understood. Apart from changing health behaviours, absence of nicotine could acutely increase appetite, decrease basal metabolic rate and metabolic efficiency [56]. Nevertheless, research on interventions with NRT to reduce post-cessation weight gain achieved little success. While NRT did appear to limit weight gain during treatment, the benefits were smaller after the treatment had stopped [12]. The evidence is insufficient to be sure whether the effects could persist in the long term. Although no strong clinical recommendations can be made to smokers who want to quit and prevent excess weight gain, it is important to acknowledge that weight gain after smoking cessation can be expected. This weight gain may in part reflect a return-to-normal weight whereby quitters end up weighing the same as they would have had they never smoked [58]. Further prospective studies with regular anthropometric measurements (e.g. weight, height, waist circumference and waist-hip ratio), health behaviours (e.g. diet, PA and fitness), energy expenditure and metabolic factors (e.g. basal metabolic rate), and smaller well controlled

clinical trials may help elucidate the complex mechanisms for post-cessation weight gain and therefore ways it may be prevented.

The strengths of the current study are its longitudinal design and its ability to examine changes in a range of dietary behaviours, alcohol consumption and PA behaviours accompanying smoking cessation. Some of these factors are reported for the first time in the literature, such as takeaway food consumption, DGI score, breakfast skipping, discretionary foods consumption and sedentary behaviours.

Some limitations should be acknowledged. First, self-reported smoking status may lead to misclassification of quitters and continuous smokers [68], and self-reported weight and height might result in underestimations of actual weight and BMI [69]; however, a correction factor was applied to reduce the error of self-reported weight and height, and the outcome of interest was difference in weight gain between quitters and continuing smokers during follow-up rather than the weight at each time point. Second, we did not collect serving size data in dietary questionnaires, and therefore, could not calculate the energy intake, which has been suggested as a main determinant of weight gain following cessation [27]; however, not all studies support this point [56] and the accuracy of energy intake from self-reported dietary recall is poor [65]. Third, there may be measurement error in dietary and PA behaviours as these data were collected by means of self-completed questionnaires though all measures are widely accepted in the literature. Reassuringly, the percentage of change in β coefficient was similar in magnitude when using pedometer-measured PA as with self-reported PA. Fourth, the sample size is small, limiting the ability to evaluate the effects of changes in dietary or PA behaviours among quitters separately. Fifth, we have only collected data 5-years apart so cannot distinguish whether the changes in diet and PA occurred before quitting, at the same time, or after. Sixth, a large amount of missing data for dietary and PA behaviours at follow-up was imputed; however, similar results were observed after excluding people with imputed data. Finally, this is a small Australian sample. Compared with data from a national survey of 24,000 people across Australia [70], our smokers were younger and less dependent on nicotine. In addition, some significant differences were evident between these study participants and the general population of similar age, and between those retained and lost to follow-up with respect to baseline socio-demographics.

These might limit the generalisability to the general population of smokers. However, as discussed above, our findings on the magnitude of post-cessation weight gain are very similar to a recent comprehensive meta-analysis of population-based prospective cohort studies [6], suggesting that these are not major sources of bias.

5.6 Conclusion

In summary, smoking cessation was associated with excess weight gain compared with those who continued to smoke. This weight gain was not explained by changes in dietary and PA behaviours. Future research is needed to elucidate the complex mechanisms underlying weight gain after smoking cessation and to develop effective strategies for its prevention.

5.7 Postscript

This chapter showed that quitting smoking was associated with 1.9 kg weight gain and it was not attenuated after adjustment for worsening dietary and PA behaviours. The association of another important health metric (HRQoL) with changing smoking status is presented in Chapter 6.

5.8 References

1. Flegal KM, Troiano RP, Pamuk ER, Kuczmarski RJ, Campbell SM. The influence of smoking cessation on the prevalence of overweight in the United States. *N Engl J Med.* 1995;333(18):1165-1170. doi:10.1056/nejm199511023331801.
2. Fernandez E, Chapman S. Quitting smoking and gaining weight: the odd couple. *BMJ.* 2012;345:e4544. doi:10.1136/bmj.e4544.
3. Williamson DF, Madans J, Anda RF, Kleinman JC, Giovino GA, Byers T. Smoking cessation and severity of weight gain in a national cohort. *N Engl J Med.* 1991;324(11):739-745. doi:10.1056/NEJM199103143241106.
4. Klesges RC, Meyers AW, Klesges LM, La Vasque ME. Smoking, body weight, and their effects on smoking behavior: a comprehensive review of the literature. *Psychol Bull.* 1989;106(2):204-230. http://www.ncbi.nlm.nih.gov/pubmed/2678202. Accessed November 10, 2015.
5. Aubin HJ, Farley A, Lycett D, Lahmek P, Aveyard P. Weight gain in smokers after quitting cigarettes: meta-analysis. *BMJ.* 2012;345:e4439. doi:10.1136/bmj.e4439.
6. Tian J, Venn A, Otahal P, Gall S. The association between quitting smoking and weight gain: a systemic review and meta-analysis of prospective cohort studies. *Obes Rev.* 2015. doi:10.1111/obr.12304.
7. Pomerleau CS, Zucker AN, Stewart AJ. Characterizing concerns about post-cessation weight gain: results from a national survey of women smokers. *Nicotine Tob Res.* 2001;3(1):51-60. doi:10.1080/14622200020032105.
8. Levine MD, Perkins KA, Marcus MD. The characteristics of women smokers concerned about postcessation weight gain. *Addict Behav.* 2001;26(5):749-756. doi:S0306-4603(00)00156-8.
9. Levine MD, Bush T, Magnusson B, Cheng Y, Chen X. Smoking-related weight concerns and obesity: differences among normal weight, overweight, and obese smokers using a telephone tobacco quitline. *Nicotine Tob Res.* 2013;15(6):1136-1140. doi:10.1093/ntr/nts226.
10. Jha P, Ramasundarahettige C, Landsman V, et al. 21st-century hazards of smoking and benefits of cessation in the United States. *N Engl J Med.* 2013;368(4):341-350. doi:10.1056/NEJMsa1211128.
11. Pirie K, Peto R, Reeves GK, Green J, Beral V. The 21st century hazards of smoking and benefits of stopping: a prospective study of one million women in the UK. *Lancet.* 2013;381(9861):133-141. doi:10.1016/S0140-6736(12)61720-6.
12. Farley AC, Hajek P, Lycett D, Aveyard P. Interventions for preventing weight gain after smoking cessation. *Cochrane Database Syst Rev.* 2012;1:CD006219. doi:10.1002/14651858.CD006219.pub3.
13. Spring B, Howe D, Berendsen M, et al. Behavioral intervention to promote smoking cessation and prevent weight gain: a systematic review and meta-analysis. *Addiction.* 2009;104(9):1472-1486. doi:10.1111/j.1360-0443.2009.02610.x.
14. Parsons AC, Shraim M, Inglis J, Aveyard P, Hajek P. Interventions for preventing weight gain after smoking cessation. *Cochrane Database Syst Rev.* 2009(1):CD006219. doi:10.1002/14651858.CD006219.pub2.
15. Yang M, Bhowmik D, Wang X, Abughosh S. Does combination pharmacological intervention for smoking cessation prevent post-cessation weight gain? A systemic review. *Addict Behav.* 2013;38(3):1865-1875. doi:10.1016/j.addbeh.2012.11.007.

16. Hill JO, Peters JC. Environmental contributions to the obesity epidemic. *Science.* 1998;280(5368):1371-1374. http://www.sciencemag.org/content/280/5368/1371.full.pdf. Accessed October 7, 2015.
17. Boulos R, Vikre EK, Oppenheimer S, Chang H, Kanarek RB. ObesiTV: how television is influencing the obesity epidemic. *Physiol Behav.* 2012;107(1):146-153. doi:10.1016/j.physbeh.2012.05.022.
18. Mozaffarian D, Hao T, Rimm EB, Willett WC, Hu FB. Changes in diet and lifestyle and long-term weight gain in women and men. *N Engl J Med.* 2011;364(25):2392-2404. doi:10.1056/NEJMoa1014296.
19. De Cocker KA, Van Uffelen JG, Brown WJ. Associations between sitting time and weight in young adult Australian women. *Prev Med.* 2010;51(5):361-367. doi:10.1016/j.ypmed.2010.07.009.
20. Kawachi I, Troisi RJ, Rotnitzky AG, Coakley EH, Colditz GA. Can physical activity minimize weight gain in women after smoking cessation? *Am J Public Health.* 1996;86(7):999-1004. http://www.ncbi.nlm.nih.gov/pubmed/8669525. Accessed March 13, 2014.
21. Gennuso KP, Thraen-Borowski KM, Schlam TR, et al. Smokers' physical activity and weight gain one year after a successful versus unsuccessful quit attempt. *Prev Med.* 2014;67C:189-192. doi:10.1016/j.ypmed.2014.07.040.
22. Le Strat Y, Rehm J, Le Foll B. How generalisable to community samples are clinical trial results for treatment of nicotine dependence: a comparison of common eligibility criteria with respondents of a large representative general population survey. *Tob Control.* 2011;20(5):338-343. doi:10.1136/tc.2010.038703.
23. Hall SM, McGee R, Tunstall C, Duffy J, Benowitz N. Changes in food intake and activity after quitting smoking. *J Consult Clin Psychol.* 1989;57(1):81-86. http://www.ncbi.nlm.nih.gov/pubmed/2925977. Accessed December 27, 2014.
24. Rodin J. Weight change following smoking cessation: the role of food intake and exercise. *Addict Behav.* 1987;12(4):303-317. http://www.ncbi.nlm.nih.gov/pubmed/3687515. Accessed June 20, 2015.
25. Thompson RL, Pyke SD, Scott EA, Thompson SG, Wood DA. Dietary change after smoking cessation: a prospective study. *Br J Nutr.* 1995;74(1):27-38. http://www.ncbi.nlm.nih.gov/pubmed/7547826. Accessed August 18, 2015.
26. Caan B, Coates A, Schaefer C, Finkler L, Sternfeld B, Corbett K. Women gain weight 1 year after smoking cessation while dietary intake temporarily increases. *J Am Diet Assoc.* 1996;96(11):1150-1155. doi:10.1016/S0002-8223(96)00296-9.
27. Stamford BA, Matter S, Fell RD, Papanek P. Effects of smoking cessation on weight gain, metabolic rate, caloric consumption, and blood lipids. *Am J Clin Nutr.* 1986;43(4):486-494. http://www.ncbi.nlm.nih.gov/pubmed/3962901. Accessed March 15, 2015.
28. Gilbert RM, Pope MA. Early effects of quitting smoking. *Psychopharmacology (Berl).* 1982;78(2):121-127. http://www.ncbi.nlm.nih.gov/pubmed/6817366. Accessed March 15, 2015.
29. Buijsse B, Feskens EJ, Schulze MB, et al. Fruit and vegetable intakes and subsequent changes in body weight in European populations: results from the project on Diet, Obesity, and Genes (DiOGenes). *Am J Clin Nutr.* 2009;90(1):202-209. doi:10.3945/ajcn.2008.27394.

30. Smith KJ, McNaughton SA, Gall SL, Blizzard L, Dwyer T, Venn AJ. Takeaway food consumption and its associations with diet quality and abdominal obesity: a cross-sectional study of young adults. *Int J Behav Nutr Phys Act.* 2009;6:29. doi:10.1186/1479-5868-6-29.
31. Nago ES, Lachat CK, Dossa RA, Kolsteren PW. Association of out-of-home eating with anthropometric changes: a systematic review of prospective studies. *Crit Rev Food Sci Nutr.* 2014;54(9):1103-1116. doi:10.1080/10408398.2011.627095.
32. Giovannini M, Agostoni C, Shamir R. Symposium overview: Do we all eat breakfast and is it important? *Crit Rev Food Sci Nutr.* 2010;50(2):97-99. doi:10.1080/10408390903467373.
33. Quatromoni PA, Pencina M, Cobain MR, Jacques PF, D'Agostino RB. Dietary quality predicts adult weight gain: findings from the Framingham Offspring Study. *Obesity (Silver Spring).* 2006;14(8):1383-1391. doi:10.1038/oby.2006.157.
34. Gall S, Jose K, Smith K, Dwyer T, Venn A. The childhood determinants of adult health study: a profile of a cohort study to examine the childhood influences on adult cardiovascular health. *Australasian Epidemiologist.* 2009;16(1):35-39. http://search.informit.com.au/documentSummary;dn=793534700501591;res=IELNZC. Accessed March 12, 2015.
35. Venn AJ, Thomson RJ, Schmidt MD, et al. Overweight and obesity from childhood to adulthood: a follow-up of participants in the 1985 Australian Schools Health and Fitness Survey. *Med J Aust.* 2007;186(9):458-460. http://www.ncbi.nlm.nih.gov/pubmed/17484707. Accessed June 6, 2014.
36. Smith KJ, Gall SL, McNaughton SA, Blizzard L, Dwyer T, Venn AJ. Skipping breakfast: longitudinal associations with cardiometabolic risk factors in the Childhood Determinants of Adult Health Study. *Am J Clin Nutr.* 2010;92(6):1316-1325. doi:10.3945/ajcn.2010.30101.
37. Berteus Forslund H, Lindroos AK, Sjostrom L, Lissner L. Meal patterns and obesity in Swedish women-a simple instrument describing usual meal types, frequency and temporal distribution. *Eur J Clin Nutr.* 2002;56(8):740-747. doi:10.1038/sj.ejcn.1601387.
38. Mishra G, Ball K, Arbuckle J, Crawford D. Dietary patterns of Australian adults and their association with socioeconomic status: results from the 1995 National Nutrition Survey. *Eur J Clin Nutr.* 2002;56(7):687-693. doi:10.1038/sj.ejcn.1601391.
39. McNaughton SA, Ball K, Crawford D, Mishra GD. An index of diet and eating patterns is a valid measure of diet quality in an Australian population. *J Nutr.* 2008;138(1):86-93. http://www.ncbi.nlm.nih.gov/pubmed/18156409. Accessed January 1, 2015.
40. Worsley A, Blasche R, Ball K, Crawford D. Income differences in food consumption in the 1995 Australian National Nutrition Survey. *Eur J Clin Nutr.* 2003;57(10):1198-1211. doi:10.1038/sj.ejcn.1601670.
41. Savige GS, Ball K, Worsley A, Crawford D. Food intake patterns among Australian adolescents. *Asia Pac J Clin Nutr.* 2007;16(4):738-747. http://www.ncbi.nlm.nih.gov/pubmed/18042537. Accessed January 1, 2015.
42. Australian Bureau of Statistics. *National Nutritional Survey Users' Guide 1995*. Canberra, ACT: Department of Health and Family Services; 1998. http://www.ausstats.abs.gov.au/ausstats/subscriber.nsf/0/CA25687100069892CA256889002102FD/$File/48010_1995.pdf. Accessed August 20, 2015.

43. Smith A, Kellet E, Schmerlaib Y. *The Australian Guide to Healthy Eating*. Canberra, ACT: Department of Health and Family Services; 1998. Accessed August 18, 2015.
44. Crawford D, Ball K, Mishra G, Salmon J, Timperio A. Which food-related behaviours are associated with healthier intakes of fruits and vegetables among women? *Public Health Nutr.* 2007;10(3):256-265. doi:10.1017/S1368980007246798.
45. Coyne T, Ibiebele TI, McNaughton S, et al. Evaluation of brief dietary questions to estimate vegetable and fruit consumption - using serum carotenoids and red-cell folate. *Public Health Nutr.* 2005;8(3):298-308. doi:S1368980005000418.
46. National Health and Medical Research Council. *Dietary Guidelines for Australian Adults*. Canberra, ACT: National Health and Medical Research Council; 2003. https://www.nhmrc.gov.au/_files_nhmrc/publications/attachments/n33.pdf. Accessed September 7, 2015.
47. McNaughton SA, Dunstan DW, Ball K, Shaw J, Crawford D. Dietary quality is associated with diabetes and cardio-metabolic risk factors. *J Nutr.* 2009;139(4):734-742. doi:10.3945/jn.108.096784.
48. Craig CL, Marshall AL, Sjostrom M, et al. International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc.* 2003;35(8):1381-1395. doi:10.1249/01.MSS.0000078924.61453.FB.
49. Cleland VJ, Schmidt MD, Dwyer T, Venn AJ. Television viewing and abdominal obesity in young adults: is the association mediated by food and beverage consumption during viewing time or reduced leisure-time physical activity? *Am J Clin Nutr.* 2008;87(5):1148-1155. http://www.ncbi.nlm.nih.gov/pubmed/18469233. Accessed January 1, 2014.
50. Tudor-Locke C, Williams JE, Reis JP, Pluto D. Utility of pedometers for assessing physical activity: convergent validity. *Sports Med.* 2002;32(12):795-808. http://www.ncbi.nlm.nih.gov/pubmed/12238942. Accessed January 15, 2015.
51. Azur MJ, Stuart EA, Frangakis C, Leaf PJ. Multiple imputation by chained equations: what is it and how does it work? *Int J Methods Psychiatr Res.* 2011;20(1):40-49. doi:10.1002/mpr.329.
52. Graham JW, Olchowski AE, Gilreath TD. How many imputations are really needed? Some practical clarifications of multiple imputation theory. *Prev Sci.* 2007;8(3):206-213. doi:10.1007/s11121-007-0070-9.
53. Australian Bureau of Statistics. *Census of population and housing table: social marital status by age by sex for time series. Catalog no. 20680.* Canberra, Australia: Australian Bureau of Statistics; 2006. https://www.kempsey.nsw.gov.au/library/census/pubs/2006-kempsey-shire-time-series/t05-social-marital-status-by-age-sex.pdf. Accessed March 8, 2016.
54. Australian Bureau of Statistics. *Year book Australia, 2012. Education and training.* Canberra, Australia: Australian Bureau of Statistics; 2012. http://www.abs.gov.au/ausstats/abs@.nsf/Lookup/by%20Subject/1301.0~2012~Main%20Features~Educational%20attainment~110. Accessed March 9, 2016.
55. Australian Bureau of Statistics. *National health survey: summary of results, 2004-05. Catalog no. 4364.0.* Canberra, Australia: Australian Bureau of Statistics; 2006. http://www.abs.gov.au/AUSSTATS/abs@.nsf/DetailsPage/4364.02004-05?OpenDocument. Accessed.

56. Chiolero A, Faeh D, Paccaud F, Cornuz J. Consequences of smoking for body weight, body fat distribution, and insulin resistance. *Am J Clin Nutr.* 2008;87(4):801-809. http://www.ncbi.nlm.nih.gov/pubmed/18400700. Accessed July 17, 2013.
57. John U, Meyer C, Hapke U, Rumpf HJ, Schumann A. Nicotine dependence, quit attempts, and quitting among smokers in a regional population sample from a country with a high prevalence of tobacco smoking. *Prev Med.* 2004;38(3):350-358. doi:10.1016/j.ypmed.2003.11.003.
58. Koster-Rasmussen R, Permin CA, Siersma V, et al. Back on track-Smoking cessation and weight changes over 9years in a community-based cohort study. *Prev Med.* 2015;81:320-325. doi:10.1016/j.ypmed.2015.09.025.
59. Fagerstrom KO, Kunze M, Schoberberger R, et al. Nicotine dependence versus smoking prevalence: comparisons among countries and categories of smokers. *Tob Control.* 1996;5(1):52-56.
60. Xie YJ, Ho SC, Su X, Liu ZM. Changes in Body Weight From Young Adulthood to Middle Age and Its Association With Blood Pressure and Hypertension: A Cross-Sectional Study in Hong Kong Chinese Women. *J Am Heart Assoc.* 2016;5(1). doi:10.1161/JAHA.115.002361.
61. Wang X, Ouyang Y, Liu J, et al. Fruit and vegetable consumption and mortality from all causes, cardiovascular disease, and cancer: systematic review and dose-response meta-analysis of prospective cohort studies. *BMJ.* 2014;349:g4490. doi:10.1136/bmj.g4490.
62. Schuit AJ, van Loon AJ, Tijhuis M, Ocke M. Clustering of lifestyle risk factors in a general adult population. *Prev Med.* 2002;35(3):219-224. http://www.ncbi.nlm.nih.gov/pubmed/12202063. Accessed March 2, 2015.
63. Poortinga W. The prevalence and clustering of four major lifestyle risk factors in an English adult population. *Prev Med.* 2007;44(2):124-128. doi:10.1016/j.ypmed.2006.10.006.
64. Chiolero A, Wietlisbach V, Ruffieux C, Paccaud F, Cornuz J. Clustering of risk behaviors with cigarette consumption: A population-based survey. *Prev Med.* 2006;42(5):348-353. doi:10.1016/j.ypmed.2006.01.011.
65. Dhurandhar NV, Schoeller D, Brown AW, et al. Energy balance measurement: when something is not better than nothing. *Int J Obes (Lond).* 2015;39(7):1109-1113. doi:10.1038/ijo.2014.199.
66. Allen SS, Hatsukami D, Christianson D, Brown S. Energy intake and energy expenditure during the menstrual cycle in short-term smoking cessation. *Addict Behav.* 2000;25(4):559-572. doi:S0306-4603(00)00074-5.
67. Allen AM, Kleppinger A, Lando H, Oncken C. Effect of nicotine patch on energy intake and weight gain in postmenopausal women during smoking cessation. *Eat Behav.* 2013;14(4):420-423. doi:10.1016/j.eatbeh.2013.07.007.
68. Connor Gorber S, Schofield-Hurwitz S, Hardt J, Levasseur G, Tremblay M. The accuracy of self-reported smoking: a systematic review of the relationship between self-reported and cotinine-assessed smoking status. *Nicotine Tob Res.* 2009;11(1):12-24. doi:10.1093/ntr/ntn010.
69. Danubio ME, Miranda G, Vinciguerra MG, Vecchi E, Rufo F. Comparison of self-reported and measured height and weight: implications for obesity research among young adults. *Econ Hum Biol.* 2008;6(1):181-190. doi:10.1016/j.ehb.2007.04.002.

70. Audrain-McGovern J, Benowitz NL. Cigarette smoking, nicotine, and body weight. *Clin Pharmacol Ther.* 2011;90(1):164-168. doi:10.1038/clpt.2011.105.

5.9 Appendix 5

Table S1. Effects of smoking characteristics at baseline on the magnitude of greater weight gain in quitters than continuing smokers

Smoking characteristics at baseline	Models	N	Post-cessation weight gain		Change in β*
			β	95% CI	
Frequency of smoking	Unadjusted	281	2.09	0.35, 3.83	
	Model 1†	281	1.93	0.18, 3.67	
	Model 1 + frequency of smoking	281	2.67	0.93, 4.40	38.3%
Number of cigarettes/day	Unadjusted	175	2.72	0.28, 5.16	
	Model 1†	175	2.44	0.03, 4.85	
	Model 1 + number of cigarettes/day	175	2.44	0.01, 4.88	0.2%
Smoking duration	Unadjusted	182	2.63	0.30, 4.95	
	Model 1†	182	2.33	0.02, 4.63	
	Model 1 + smoking duration	182	2.43	0.11, 4.75	4.5%
Pack-years	Unadjusted	174	2.50	0.06, 4.94	
	Model 1†	174	2.23	-0.17, 4.63	
	Model 1 + pack-years	174	2.25	-0.18, 4.68	0.8%

BMI: body mass index; CI: confidence interval.

* Relative to Model 1.

† Adjusted for age, sex, BMI, education and follow-up length.

Table S2. Mean±SD of post-cessation weight gain in quitters during follow-up, by time since quitting and number of quit attempts

Smoking characteristics	Post-cessation weight gain			P-value
	N	Mean	SD	
Time since quitting				0.895
≤ 2 years	26	6.1	7.2	
>2 years	34	5.8	7.9	
Number of quit attempts				0.691
≤ 2 times	36	4.6	8.1	
>2 times	28	3.8	7.0	

SD: standard deviation.

Table S3. Effects of changes in dietary and physical activity behaviours on the magnitude of BMI change after quitting smoking during follow-up, compared with continuing smoking

	Models	**β**	**95% CI**	**Change in β***
	Unadjusted	0.70	0.11, 1.30	
	Model 1†	0.68	0.08, 1.28	
Changing dietary behaviours	Model 1 + changing discretionary foods consumption	0.69	0.09, 1.29	1.02%
	Model 1 + changing fruit and vegetable consumption	0.68	0.08, 1.29	0.42%
	Model 1 + changing DGI score	0.70	0.10, 1.31	3.33%
	Model 1 + changing alcohol consumption	0.70	0.09, 1.31	2.79%
	Model 1 + change in eating takeaway food	0.70	0.09, 1.31	3.07%
	Model 1 + change in skipping breakfast	0.68	0.08, 1.28	-0.06%
	Model 1 + changing all dietary behaviours	0.73	0.11, 1.35	7.61%
Changing PA behaviours	Model 1 + changing total PA	0.70	0.10, 1.30	2.57%
	Model 1 + changing LTPA	0.78	0.18, 1.39	15.32%
	Model 1 + changing sitting time	0.68	0.08, 1.28	0.02%
	Model 1 + changing TV viewing time	0.68	0.08, 1.29	0.17%
	Model 1 + changing LTPA and sitting time	0.79	0.18, 1.39	15.61%
Changing dietary and PA behaviours	Model 1 + changing all dietary behaviours, LTPA and sitting time	0.81	0.19, 1.43	19.22%

* Relative to Model 1.

† Adjusted for age, sex, BMI, education and follow-up length.

BMI: body mass index; CI: confidence interval; DGI: diet guideline index; LTPA: leisure time physical activity; PA: physical activity; TV: television.

Sensitivity analyses were conducted by excluding participants with imputed data. **Table S4** and **S5** document the dietary and PA behaviours at baseline, follow-up and their changes during the follow-up period. **Table S6** presents the effects of changing dietary and PA behaviours on the magnitude of weight (kg) change after quitting smoking during follow-up, compared with continuing smoking (75 quitters and 107 continuing smokers).

The results were similar to those obtained when MI was performed.

For dietary behaviours in **Table S4**, at baseline, continuing smokers reported lower DGI score and higher daily consumption of alcohol than quitters. No other statistically significant difference was observed in baseline dietary behaviours. Similar differences were found at follow-up and quitters also consumed less discretionary foods and were more likely to have breakfast than continuing smokers. Compared with continuing smokers, quitters were less likely to consume takeaway food at least two times per week at follow-up but the difference did not reach statistical significance. There was no significant difference in changes in dietary behaviours between quitters and continuing smokers.

For PA behaviours in **Table S5**, at baseline, no significant difference was observed between quitters and continuing smokers. Quitters reported a higher level of LTPA and spent less time watching TV at follow-up. In addition, quitters spent more time sitting than continuing smokers although the difference did not reach statistical significance. There was no evidence of statistically significant difference in changes in PA behaviours between quitters and those who continued to smoke.

Table S6 describes the results for the linear regression analyses of smoking cessation on weight change in individuals with complete data. In the fully adjusted model (Model 1 + changing all dietary behaviours, LTPA and sitting time), smoking cessation was associated with 2.75kg greater weight gain than continuing smoking. Changing dietary and PA behaviours accompanying cessation could not account for this weight gain, with changes in β coefficients ranging from -1.44% to 16.75%.

Table S4. Dietary behaviours at baseline, follow-up and changes during follow-up, for continuing smokers and quitters, excluding participants with imputed data*

Dietary behaviours	Continuing smokers	Quitters	P-value
Servings of 'discretionary' foods /day			
Baseline	4.3±2.7	3.9±2.5	0.285
Follow-up	**4.3**±3.2	**3.4**±2.0	**0.028**
Change from baseline to follow-up†	-0.0±2.5	-0.5±1.8	0.141
Servings of fruit and vegetables/day			
Baseline	3.6±1.5	3.6±1.7	0.800
Follow-up	3.6±1.6	3.9±1.8	0.120
Change from baseline to follow-up†	-0.0±1.6	0.2±1.5	0.156
DGI score			
Baseline	**94.9±20.0**	**101.0±16.3**	**0.032**
Follow-up	**95.8±18.7**	**105.6±16.4**	**0.000**
Change from baseline to follow-up†	0.9±16.5	4.6±14.5	0.121
Alcohol consumption (grams/day)			
Baseline	**15.3±20.1**	**10.9±9.7**	**0.027**
Follow-up	**12.9±17.3**	**8.7±10.1**	**0.020**
Change from baseline to follow-up†	-2.4±22.0	-2.2±10.6	0.909
Consuming takeaway food (≥2/wk) (%)			
Baseline	31.3	29.9	0.837
Follow-up	29.3	16.9	0.055
Change from baseline to follow-up			
Neither baseline or follow-up	58.6	63.6	0.207
Baseline only	12.1	19.5	
Follow-up only	10.1	6.5	
Both baseline and follow-up	19.2	10.4	
Skipping breakfast (%)			
Baseline	35.7	37.7	0.791
Follow-up	**41.8**	**27.3**	**0.046**
Change from baseline to follow-up			
Neither baseline or follow-up	41.8	48.1	0.215
Baseline only	16.3	24.7	
Follow-up only	22.5	14.3	
Both baseline and follow-up	19.4	13.0	

Bold denotes statistically significant result.

* Mean±SD except for percentages; P-values determined by t test or person χ^2 test (where appropriate); sample size varied (range, 175-279) because of missing data.

† Calculated using follow-up values minus baseline ones.

DGI: diet guideline index; SD: standard deviation.

Table S5. Physical activity behaviours at baseline, follow-up and change during follow-up, for continuing smokers and quitters, excluding participants with imputed data*

Physical activity behaviours	Continuing smokers		Quitters		P-value
	Mean	SD	Mean	SD	
Total PA (minutes/week)					
Baseline	**872.7**	**564.7**	**694.7**	**435.2**	**0.029**
Follow-up	794.6	580.1	692.4	422.7	0.212
Change from baseline to follow-up†	-78.2	633.4	-2.3	544.6	0.421
Total LTPA (minutes/week)					
Baseline	129.4	176.5	130.3	148.9	0.971
Follow-up	124.6	165.7	177.6	174.9	0.051
Change from baseline to follow-up†	-4.8	172.1	47.3	196.5	0.075
Sitting time (minutes/day)					
Baseline	311.3	157.6	345.0	160.4	0.167
Follow-up	319.7	165.2	358.6	145.6	0.108
Change from baseline to follow-up†	8.5	170.2	13.5	179.8	0.850
TV viewing time (hours/day)					
Baseline	2.3	1.5	1.9	1.6	0.122
Follow-up	**2.3**	**1.6**	**1.8**	**1.1**	**0.034**
Change from baseline to follow-up†	0.0	1.7	-0.1	1.6	0.708

Bold denotes statistically significant result.

* P-values determined by t test; sample size varied (range, 161-173) because of missing data.

† Calculated using follow-up values minus baseline ones.

LTPA: leisure time physical activity; PA: physical activity; SD: standard deviation; TV: television.

Table S6. Effects of changing dietary and physical activity behaviours on the magnitude of weight (kg) change after quitting smoking during follow-up, compared with continuing smoking, excluding participants with imputed data

	Models	β	95% CI	Change in β*
	Unadjusted	2.14	-0.05, 4.33	
	Model 1†	2.09	-0.07, 4.26	
Changing dietary behaviours	Model 1 + changing discretionary foods consumption	2.19	0.02, 4.36	4.78%
	Model 1 + changing fruit and vegetable consumption	2.08	-0.10, 4.26	-0.48%
	Model 1 + changing DGI score	2.40	0.21, 4.59	14.83%
	Model 1 + changing alcohol consumption	2.18	0.07, 4.30	4.31%
	Model 1 + change in eating takeaway food	2.19	0.03, 4.35	4.78%
	Model 1 + change in skipping breakfast	2.44	0.24, 4.65	16.75%
	Model 1 + changing all dietary behaviours	2.64	0.46, 4.83	26.32%
Changing PA behaviours	Model 1 + changing total PA	2.06	-0.09, 4.20	-1.44%
	Model 1 + changing LTPA	2.29	0.16, 4.42	9.57%
	Model 1 + changing sitting time	2.16	-0.02, 4.33	3.35%
	Model 1 + changing TV viewing time	2.11	-0.07, 4.29	0.96%
	Model 1 + changing LTPA and sitting time	2.36	0.22, 4.50	12.92%
Changing dietary and PA behaviours	Model 1 + changing all dietary behaviours, LTPA and sitting time	2.75	0.62, 4.89	31.58%

* Relative to Model 1.

† Adjusted for age, sex, BMI, education and follow-up length.

BMI: body mass index; CI: confidence interval; DGI: diet guideline index; LTPA: leisure time physical activity; PA: physical activity; TV: television.

Table S7. Mean±SD of daily steps at baseline, follow-up and change during follow-up, for continuing smokers and quitters

Steps per day	Continuing smokers			Quitters			P-value
	n1	Mean	SD	n2	Mean	SD	
Baseline	128	9268	3471	106	9172	2998	0.823
Follow-up	52	8335	2635	58	8628	3133	0.599
Change over follow-up*	52	-865	3218	58	-204	2880	0.258

* Calculated using follow-up value minus baseline one.
SD: standard deviation.

Chapter 6

Smoking status and health-related quality of life: a longitudinal study in young adults

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Chapter 6. Smoking status and health-related quality of life: a longitudinal study in young adults

6.1 Preface

Chapter 4 and 5 quantified the weight gain after smoking cessation and further explored whether changing health behaviours could explain it. This chapter aims to research another health effect of changing smoking status in young adults: HRQoL.

6.2 Introduction

The physical health consequences of tobacco use have been studied extensively. More recently the effects on mental health and well-being have also attracted attention, with one line of research focusing on HRQoL. Cross-sectional studies have demonstrated impaired HRQoL in smokers compared with non-smokers [1-6]. Evidence from a few longitudinal studies has suggested that compared to never smokers, those who smoked at baseline had poorer physical HRQoL at follow-up [7,8] and those who continued to smoke from baseline to follow-up reported poorer HRQoL at follow-up [9]. Only one study had looked at the associations of change in smoking status on changes in HRQoL [10]; however, it only focused on the impact of cessation, changes in HRQoL among other groups were not examined. In addition, the participants of that study were registered female nurses with similar socioeconomic status, thus, the results may not be generalizable to men and other socioeconomic groups. Further, control for confounding may have been inadequate, with some potential confounders, such as alcohol consumption, psychiatric diagnosis and personality, which are strongly associated with smoking [11,12] and HRQoL [13,14], not considered.

A recent meta-analysis showed a significant improvement of mental HRQoL from baseline to follow-up in quitters compared with continuing smokers [15]. However, there was publication bias and moderate heterogeneity between studies [15]. Additionally, most included studies (seven in eight) were either secondary analyses of cessation interventions (n=1) or focused on people with chronic physical or psychiatric conditions (n=6).

We set out to overcome the aforementioned limitations and investigate the longitudinal relationship between change in smoking status and change in HRQoL among a population-based sample of Australian young adults with consideration of a wide range of potential

confounders. We hypothesised that those who quit smoking would experience an improvement in HRQoL, while in contrast, those who continued or resumed smoking would have a reduction in HRQoL.

6.3 Methods

6.3.1 Study population

Sampling procedures have been presented in the section of 2.3.1. **Figure 6-1** shows the recruitment and retention of participants in the current chapter.

6.3.2 Assessment of smoking status

Smoking status was defined via three questions. The first question asked all participants 'Over your lifetime, have you smoked at least 100 cigarettes, or a similar amount of tobacco?' Participants answering 'yes' were classified as ever smokers, and those answering 'no' were classified as never smokers. Ever smokers were classified into current or former smokers based on a second question 'How often do you now smoke cigarettes, cigars, pipes or any other tobacco products?' Participants who answered 'daily' or 'at least once a week' were classified as current smokers, and those who answered 'not at all' were further asked the third question 'In the past have you ever been a daily smoker?' Respondents answering 'yes' were classified as 'former smokers'. We excluded occasional smokers (less than 1 cigarette per week to the second question) and former smokers who had never been a daily smoker.

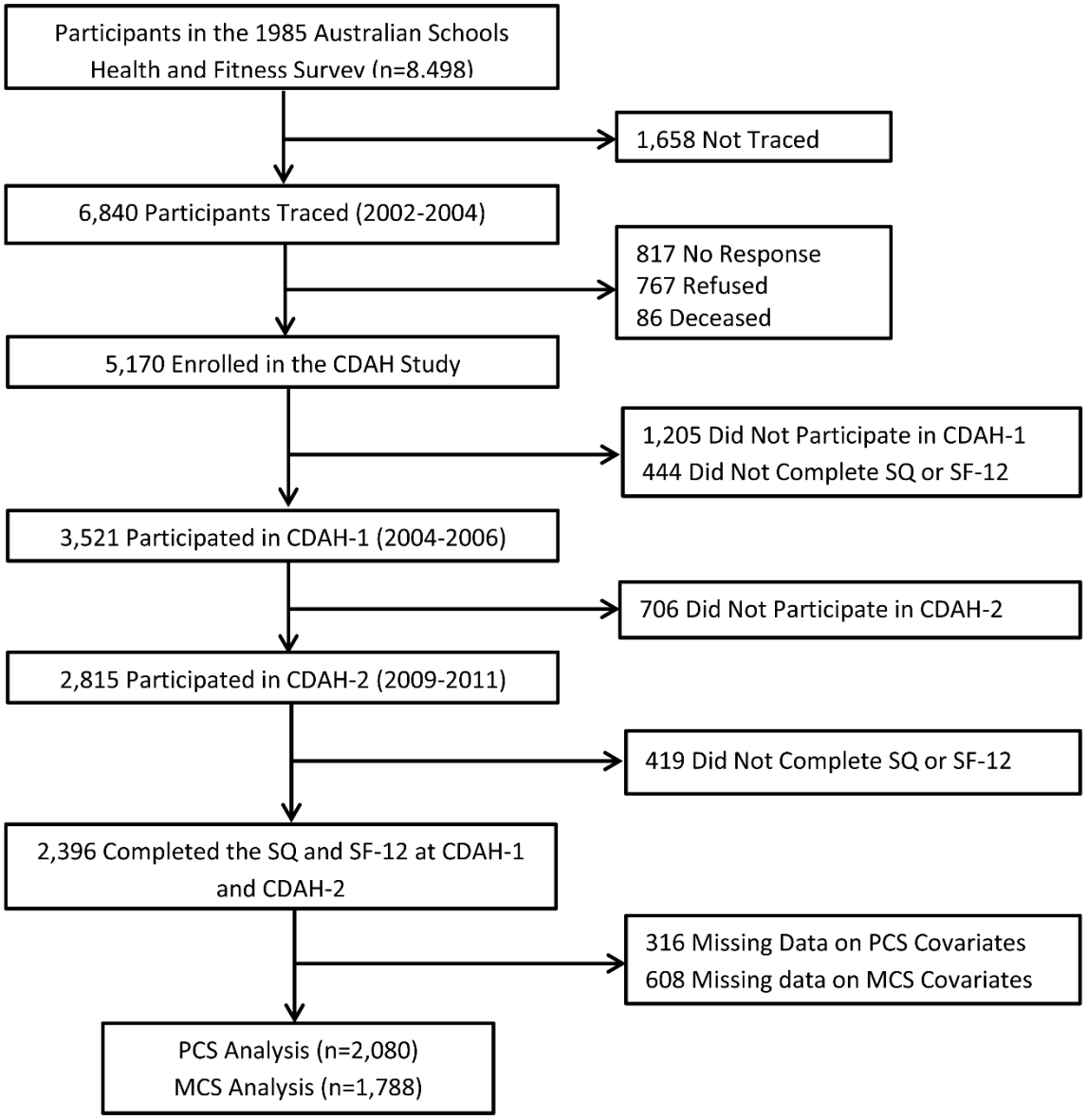

Figure 6-1 Flow chart of recruitment and retention of participants for Childhood Determinants of Adult Health Study, Australia, 1985-2011.

For the longitudinal analyses, the exposure variable was change in smoking status from baseline to follow-up, categorised as: stable never smokers (never smoker at both time points), stable former smokers (former smoker at both time points), continuing smokers (current smoker at both time points), quitters (current smoker at baseline and former smoker at follow-up) and resumed smokers (former smoker at baseline and current smoker at follow-up).

6.3.3 Assessment of HRQoL

HRQoL was measured by the SF-12 version 2 [17] at baseline and follow-up. SF-12 assesses 8 domains: physical functioning (2 items), role limitations due to physical problems (2 items), bodily pain (1 item), general health perceptions (1 item), vitality (1 item), social functioning (1 item), role limitations due to emotional problems (2 items), and mental health (2 items). These domains were summarized into two component scores: physical component summary (PCS) and MCS. Each domain was scored from 0 to 100. These scores were calculated based on US population normative values with a mean of 50 and a SD of 10. Higher scores denote better HRQoL. SF-12 has been shown to be a valid and reliable measure of health status in different age groups [17] and countries [18], including Australia [19] .

For the longitudinal analyses, we examined the absolute change of HRQoL as both continuous and categorical variables. The HRQoL change was calculated by subtracting PCS or MCS scores at baseline from those at follow-up. We used five points as the minimal clinical significant difference (MCSD) to define three categories of HRQoL change [20]: 'decreased' included persons who lost more than five points of HRQoL during follow-up; 'stable' included those for whom HRQoL stayed the same, decreased or increased within a five point range, and 'increased' included those who gained more than five points of HRQoL.

6.3.4 Covariates

Information on socio-demographic variables was self-reported at baseline, including age, sex, marital status (married or living as married versus other), education level (school only, vocational, university) and employment status (working versus not in the workforce). ARIA classifications (residing in major city versus other) were assigned to participants based on the census collection district of their residential address.

BMI was calculated from measured weight and height for most. Total PA per week was assessed using the IPAQ [21]. Most participants (73%) completed the long version, with the remainder completing the short version. Data from the short and long instruments were combined with participants categorized as low, moderate and high activity using the published scoring protocols (www.ipaq.ki.se/scoring.htm, accessed August 2010). The average number of daily steps was calculated for participants who reported wearing the pedometer for at least 8 hours on at least 4 days. Daily fruit and vegetable consumption were assessed using short questions [22]. The frequency of daily standard alcoholic drinks was estimated from self-reported usual frequency of consumption (range: 'never' to '6 or more times per day') of 10 types of alcoholic beverages listed in the FFQ multiplied by each beverage's average alcohol concentration. The NEO-Five Factor Inventory was used to assess the 'big five' personality traits: neuroticism, extraversion, openness, agreeableness, and conscientiousness [23]. A 15 item Index for Social Support assessed participants' perceptions and satisfaction with the social interaction available to them [24]. To control for poor health prior to beginning smoking, we used a question completed by participants in 1985 that asked 'Is your health usually?' with responses of 'very good', 'good', 'average', 'poor' and 'very poor'. For 97% of ever daily smokers the response to this question was given before they started daily smoking. For women, parity was also considered defined as the number of live births (one or more versus none).

Major depression, dysthymia, anxiety, alcohol and drug use disorders (dependence or abuse) within the previous 12 months were assessed with the self-administered Composite International Diagnostic Interview (CIDI-Auto, version 2.1) [25]. Diagnoses were only considered as potential confounders in the physical HRQoL analyses. Follow-up length between baseline and follow-up and baseline HRQoL were also considered as a potential confounder in the longitudinal analyses. We categorized the baseline HRQoL into five groups to control the ceiling and floor effects – there is no room to go higher or lower for participants who are at the top or bottom at baseline.

6.3.5 Statistical analysis

Student t-tests and chi-square tests were used to compare differences in means and proportions respectively. Linear regression was used to examine the cross-sectional

associations between smoking status and HRQoL, and longitudinal associations of baseline smoking status and change in smoking status with change in HRQoL from baseline to follow-up. Never smokers or stable never smokers were generally treated as the reference group, but we purposefully performed some comparisons between other smoking groups. The log multinomial model, which estimates RRs and 95% CIs for outcomes with multiple attributes [26], was used to assess the association of change in smoking status with clinically significant (>5 point) change in HRQoL with the stable category (change less than five points in either direction) as the excluded category. Whilst the categories of the response variable are ordered, the log multinomial model for nominal outcomes was used because none of the logit-link [27] or log-link [28] ordinal regression models could be fitted without substantial and statistically significant loss of model fit.

For all models, covariates were considered as potential confounders if they were causally related to the outcome, imbalanced between the exposure groups and caused a change of 10% or more in the effect estimate when included in a given regression model. Interactions between sex and smoking status or changes of smoking status on changes in HRQoL during follow-up were measured in all multivariable models. We did not separate men and women for analyses because there was no evidence from the literature that sex modified the association between smoking status and HRQoL.

The following sensitivity analyses were conducted. First, because only a subsample of participants completed the CIDI to get a DSM-IV psychiatric diagnosis, a sensitivity analysis was performed to examine the result of using current severe psychological distress determined from the MCS (scores ≤ 36) as the outcome instead [29]. This measure was available for a much larger sample. Second, we considered the effect of loss to follow-up using inverse probability weighting. The following factors were used to determine the weights: sex, marital status, residing in a major city, education level, BMI, IPAQ level, diagnosis of current severe psychological distress, physical HRQoL and smoking status for physical HRQoL analyses, and age, sex, marital status, education level, BMI, total alcohol drinks per day, neuroticism, extraversion, openness, agreeableness, mental HRQoL and smoking status for mental HRQoL analyses. Third, we examined the effect of using a different categorization for clinically significant difference for PCS and MCS scores. This was

defined as a half a SD of baseline HRQoL [20]. Fourth, to examine the potential influence of reverse causation, whereby those with poor health were more likely to quit smoking, we repeated our analyses by excluding people who reported quitting smoking due to health problems during follow-up. Finally, we conducted a sensitivity analysis to determine whether defining former smokers' smoking status according to length of abstinence changed the findings. Former smokers were those who had been a daily smoker and had stopped daily smoking more than one year ago.

A two-tailed *P* value less than 0.05 was considered statistically significant. All analyses were performed with STATA software, version 12.1 (Stata Corp, College Station, Texas 77845 USA).

6.4 Results

We restricted the analyses to participants with full information on the outcome and principal study factor and on potential confounders and modifiers, leading to different sample sizes for the analyses of physical (n=2,080) and mental (n=1,788) HRQoL (**Table 6-1**). The mean age at baseline was 32 years (range 26-37). Most participants were never smokers. The mean physical HRQoL score was 52.3 (SD 7.6), and the mean mental HRQoL score was 50.0 (SD 8.3).

Compared with those who did not participate in the follow-up, those who did were younger, more often female, had lower BMI, more often married or living as married, had a higher education level, were more often never-smokers, and had better physical HRQoL but poorer mental HRQoL at baseline (**Appendix 6-1**). There was no practically or statistically significant difference between these two groups in health status assessed in 1985.

6.4.1 Cross-sectional association between smoking and HRQoL at baseline

For physical HRQoL, persons who were current smokers had significantly lower mean scores than never (difference: -2.86, 95% CI: -3.69, -2.03) and former (difference: -2.36, 95% CI: -3.36, -1.37) smokers after adjustment for potential confounders (**Table 6-2**). The difference between the means for never and former smokers was less than one-half of a point, and not statistically significant.

Table 6-1 Baseline characteristics of participants

Characteristics	Physical HRQoL* (N=2,080)		Mental HRQoL* (N=1,788)	
	Mean / %	SD / n	Mean / %	SD / n
Age (years)	31.7	2.6	31.6	2.6
Sex (male)	41.6	866	40.5	724
Body mass index†	25.2	4.8	25.2	4.9
Married or living as married	70.0	1,455	29.4	525
Resides in a major city	72.7	1,512	74.3	1,324
Education				
Any university education	43.5	904	45.4	812
Vocational training	29.7	617	28.0	501
High school only	26.9	559	26.6	475
Working	83.3	1,727	84.5	1,505
Level of PA per week				
Low	11.3	235	10.3	173
Moderate	41.1	854	40.0	738
High	47.6	991	45.6	765
Fruit and vegetable consumption, serves/day	4.0	1.7	4.0	1.7
Standard alcoholic drinks, drinks/day	0.7	0.9	0.8	0.9
Diagnosis of severe psychological distress‡	8.0	167	8.7	156
Social support	45.4	3.6	45.3	3.7
Personality				
Neuroticism	19.7	3.8	19.7	3.9
Extraversion	26.9	3.6	26.9	3.6
Openness	24.4	3.1	24.5	3.1
Agreeableness	23.6	4.0	23.7	4.0
Conscientiousness	28.7	2.9	28.7	2.9
Parity (female only, having one or more live births)	57.1	664	52.5	555
Health status in 1985				
Very good	36.7	593	35.9	504
Good	44.5	719	44.6	626
Average/poor/very poor	18.8	303	19.5	274
Smoking status				
Never	60.5	1,259	61.0	1,091
Former	19.0	396	19.1	341
Current	20.4	425	19.9	356
Physical component summary	52.3	7.6	---	---
Mental component summary	---	---	50.0	8.3

BMI: body mass index; SD: standard deviation.

* Sample size varied (range, 1,615 to 1,681 for PCS and 1,404 to 1,782 for MCS) because of missing data.

† Weight (kg)/height $(m)^2$.

‡ Defined as MCS scores ≤ 36.

Table 6-2 Cross-sectional associations of smoking status and HRQoL at baseline

	Physical HRQoL				Mental HRQoL			
	No	Mean (SD)	Unadjusted Diff(95% CI)	Adjusted† Diff(95% CI)	No	Mean (SD)	Unadjusted Diff(95% CI)	Adjusted‡ Diff(95% CI)
Never smoker	1,259	52.83 (7.32)	Ref	Ref	1,091	50.73 (7.90)	Ref	Ref
Former smoker	396	52.77 (7.26)	-0.06(-0.92, 0.79)	-0.49(-1.32, 0.34)	341	49.41 (8.57)	**-1.32(-2.33, -0.31)****	-0.36(-1.31, 0.60)
Current smoker	425	50.35 (8.51)	**-2.48(-3.32, -1.65)*****	**-2.86(-3.69, -2.03)***[a]	356	48.42 (9.13)	**-2.31(-3.30, -1.31)*****	-0.91(-1.88, 0.07)

CI: confidence interval; Diff: difference in means; Ref: reference group.

** denotes *P*< or = 0.01; *** denotes *P*< or =0.001.

† Adjusted for age, sex, marital status, residing in major city, education level, BMI, IPAQ level, total alcohol drinks per day, and diagnosis of current severe psychological distress.

‡ Adjusted for age, sex, marital status, education level, BMI, total alcohol drinks per day, social support, and personality (neuroticism, agreeableness).

[a] Statistical difference compared with former smokers.

Both former and current smokers reported significantly worse mental HRQoL on average than never smokers in unadjusted analyses; however, the differences were greatly reduced and no longer statistically significant after adjustment for confounders (**Table 6-2**).

6.4.2 Longitudinal association between baseline smoking status and change in HRQoL

On average, PCS scores decreased over 5 years. Compared with never smokers, current smokers at baseline had a significantly greater reduction in physical HRQoL at follow-up. Former smokers had a smaller reduction (difference: -0.82, 95% CI: -1.64, 0.01, *P*=0.051) relative to never smokers.

In unadjusted analyses, mental HRQoL declined over time for never smokers on average, but improved among those who were former or current smokers at baseline. The differences were greatly reduced particularly for current smokers at baseline, and were no longer statistically significant after adjustment for confounders (**Table 6-3**).

6.4.3 Longitudinal association between change in smoking status and change in HRQoL

On average, those that continued to smoke and those that resumed smoking had larger reductions in PCS scores than stable never smokers (**Table 6-3**). Those that continued to smoke had larger reductions than those that quit on average (difference: -2.12, 95% CI: -3.51, -0.73), and those that resumed smoking had larger reductions than stable former smokers (difference: -2.08, 95% CI: -3.94, -0.21). There were much smaller differences between stable never smokers and quitters.

For changes in mental HRQoL, stable never smokers reported reductions in MCS scores, whereas all other groups showed improvements over follow-up. These differences were not statistically significant in either unadjusted or adjusted analyses (**Table 6-3**).

The associations between change in smoking status and clinically significant (>5 point) change in HRQoL over 5-years are presented in **Table 6-4**. In multivariable models, the risks of a clinically significant reduction in physical HRQoL for those who continued to smoke and those who resumed smoking were respectively 1.28 and 1.32 times that in stable never smokers. Resumed smokers had a similar elevation in risk relative to stable former smokers (RR: 1.39, 95% CI: 1.10, 1.75). The risk of a clinically significant improvement in physical HRQoL was 1.43 (95% CI: 1.03, 1.98) times higher for quitters than for continuing smokers.

Table 6-3 Associations of baseline smoking status and change in smoking status with change in HRQoL

	Change in physical HRQoL				Change in mental HRQoL			
	No	Mean(SD)	Unadjusted Diff(95% CI)	Adjusted† Diff(95% CI)	No	Mean(SD)	Unadjusted Diff(95% CI)	Adjusted‡ Diff(95% CI)
Baseline smoking status								
Never smoker	1,259	-2.18(8.62)	Ref	Ref	1,091	-0.93 (9.29)	Ref	Ref
Former smoker	396	-3.10(8.68)	-0.91(-1.92, 0.09)	-0.82(-1.64, 0.01)	341	0.41 (9.63)	**1.34(0.18, 2.51)***	0.78(-0.27, 1.83)
Current smoker	425	-2.70(9.68)	-0.52(-1.49, 0.46)	**-1.36(-2.20, -0.52)*****	356	0.39 (10.30)	**1.32(0.17, 2.46)***	0.08(-0.99, 1.15)
Change in smoking status								
Stable never	1,259	-2.18(8.62)	Ref	Ref	1,091	-0.93(9.29)	Ref	Ref
Stable former	329	-2.85(8.67)	-0.67(-1.74, 0.41)	-0.47(-1.35, 0.41)	288	0.27(9.60)	1.20(-0.04, 2.45)	0.67(-0.45, 1.78)
Resumed	67	-4.31(8.70)	-2.13(-4.31, 0.04)	**-2.55(-4.29, -0.80)**[a]	53	1.17(9.84)	2.10(-0.54, 4.74)	1.35(-0.98, 3.68)
Continuing	265	-3.38(9.48)	**-1.19(-2.37, -0.02)***[b]	**-2.17(-3.16, -1.19)***[b]	223	0.18(10.64)	1.11(-0.27, 2.49)	-0.31(-1.59, 0.96)
Quitter	160	-1.57(9.94)	0.61(-0.85, 2.07)	-0.05(-1.25, 1.15)	133	0.73(9.74)	1.66(-0.06, 3.39)	0.72(-0.83, 2.26)

CI: confidence interval; Diff: difference in means; HRQoL: health-related quality of life; Ref: reference group.

* denotes *P*< or =0.05; ** denotes *P*< or = 0.01; *** denotes *P*< or =0.001.

† Adjusted for age, sex, marital status, follow-up duration (days), baseline PCS (in analysis of change in smoking status), residing in major city, education, BMI, IPAQ level, total alcohol drinks per day, and diagnosis of current severe psychological distress.

‡ Adjusted for age, sex, marital status, follow-up duration (days), baseline MCS (in analysis of change in smoking status), education, BMI, total alcohol drinks per day, social support, and personality (neuroticism, agreeableness).

[a] Statistical difference compared with stable former smokers; [b] Statistical difference compared with quitters.

Table 6-4 Association between changes in smoking status and clinically meaningful changes (or 5 or more score points) in HRQoL

	N	Stable[†]		Decreased			Increased		
		n	%	n	%	RR(95% CI)[‡§]	n	%	RR(95% CI)[‡§]
Change in physical HRQoL									
Stable never	1,259	637	50.60	416	33.04	Ref	206	16.36	Ref
Stable former	329	156	47.42	125	37.99	0.95(0.83, 1.10)	48	14.59	0.92(0.72, 1.18)
Resumed	67	26	38.81	33	49.25	**1.32(1.07, 1.63)**[**a]	8	11.94	0.84(0.48, 1.47)
Continuing	265	114	43.02	105	39.62	**1.28(1.09, 1.50)**[**]	46	17.36	0.84(0.64, 1.10)[b]
Quitter	160	64	40.00	59	36.88	1.16(0.97, 1.40)	37	23.13	1.20(0.95, 1.53)
Change in mental HRQoL									
Stable never	1,091	518	47.48	324	29.70	Ref	249	22.82	Ref
Stable former	288	122	42.36	78	27.08	1.01(0.82, 1.25)	88	30.56	1.08(0.92, 1.27)
Resumed	53	24	45.28	10	18.87	0.66(0.37, 1.15)	19	35.85	1.29(0.93, 1.79)
Continuing	223	99	44.39	56	25.11	0.96(0.75, 1.24)	68	30.49	0.95(0.78, 1.15)
Quitter	133	62	46.62	37	27.82	1.02(0.76, 1.36)	34	25.56	1.00(0.78, 1.29)

CI: confidence interval; HRQoL: health-related quality of life; RR: relative risk; Ref: reference group.

[**] denotes *P*< or = 0.01

[†] Reference category

[‡] Physical HRQoL: adjusted for baseline age, sex, marital status, follow-up duration (days), categorical baseline PCS, residing in major city, education, BMI, IPAQ level, total alcohol drinks per day, and diagnosis of current severe psychological distress.

[§] Mental HRQoL: adjusted for baseline age, sex, marital status, follow-up duration (days), categorical baseline MCS, education, BMI, total alcohol drinks per day, social support and personality (neuroticism, agreeableness).

[a] Statistical difference compared with stable former smokers; [b] Statistical difference compared with quitters.

Change in smoking status was not significantly associated with elevation in risk of clinically significant change in mental HRQoL.

6.4.4 Sensitivity analyses

Results from the subsample of participants with DSM-IV psychiatric diagnoses measured using the CIDI instrument were consistent with the analyses that used current severe psychological distress from the MCS (scores ≤36) [29]. Sensitivity analyses conducted by re-analysing the data with inverse probability weighting produced similar patterns of results as the unweighted analyses (**Appendix 6-2**). The changes in the magnitude of statistically significant estimates were within 15% of those from unweighted analyses. Findings using half a SD of baseline HRQoL to define the MCSD were broadly similar to the original results (**Appendix 6-3**), but the clinically significant improvements of physical HRQoL for quitters relative to continuing smokers were no longer apparent. The results obtained after removing quitters who quit smoking due to health problems during follow-up were similar, with the changes in the magnitude of statistically significant estimates ranging from 0% to 6.0% (**Appendix 6-4**). Using length of abstinence to define the smoking status of former smokers made no appreciable difference in the interpretation of our results (**Appendix 6-5**).

6.5 Discussion

To the best of our knowledge, this is the first study examining the longitudinal relationship between change in smoking status and change in HRQoL. In this cohort of younger adults, we found that smoking was closely associated with poorer physical HRQoL. Continuing and resuming smoking was associated with a statistically significant reduction of physical HRQoL relative to quitting and maintaining cessation over 5 years. On average, these changes were not clinically significant; however, those that continued to smoke or those that resumed smoking had a higher risk of a clinically significant reduction of physical HRQoL than stable never smokers. The association between change in smoking status and mental HRQoL change was not clinically or statistically significant.

We found a strong association between baseline smoking status and physical HRQoL at follow-up with a greater reduction of PCS scores on average for current smokers than for never smokers. This finding is consistent with the few previous cohort studies of this association [7,8]. In a 26-year follow-up of a white male cohort, never smokers in midlife

reported better physical HRQoL than other smoker groups [8]. A similar association was reported in women [7]. In addition, there was a dose-response relationship between the number of cigarettes smoked per day at baseline and physical HRQoL at follow-up, with heavier smokers showing lower PCS scores [7,8]. However, not all studies support this finding. For example, in a sample of male veterans, smoking status was only negatively related with PCS cross-sectionally, with no longitudinal association reported [30].

The observed downtrend of physical HRQoL and uptrend of mental HRQoL over follow-up for continuing smokers and quitters was supported by the only comparable study in women [10]. An important finding was that, as hypothesised, change in smoking status was associated with change in physical HRQoL, with significant greater reductions in continuing smokers than in quitters. The impact of quitting smoking on changes in physical HRQoL has been reported in the Nurses' Health Study [10], but no comparison was performed between continuing smokers and quitters in that study. So far, there have been no reports of the relationship between resuming smoking and change in physical HRQoL. We showed that compared with those who maintained cessation, resumed smokers had a statistically and clinically significant reduction in PCS scores. This relationship was very robust in several sensitivity analyses.

In terms of how smoking may have impaired physical HRQoL, given the age of our sample, the most likely cause of reduced health is respiratory symptoms. Others have reported that smokers in their 20s have more wheezing, coughing and phlegm than non-smokers [31,32]. Therefore, our longitudinal finding of greater reduction of physical HRQoL in continuing or resumed smokers may reflect improvements in respiratory symptoms in quitters or stable former smokers. There may also be a cognitive explanation for the clinically significant improvement in physical HRQoL. Those who quit smoking or maintained cessation may have an altered concept of their health due to their perceived healthier lifestyle, rather than any objective improvement in health. This hypothesis is supported by qualitative research showing that younger people often reference their health status to health behaviours, whereas older individuals consider chronic conditions when reporting health status [33].

Consistent with a recent meta-analysis [15], relative to continuing smokers, those who quit smoking did not have a significant reduction of reported mental HRQoL. It is unexpected

that resumed smokers showed some improvement of mental HRQoL compared with those who maintained cessation, even though the association did not reach statistical significance. This could be partly explained by the neurobiological effects of nicotine on concentration, cognition, and pleasurable sensations [34]. Exploring in depth, we found that the baseline MCS scores were lower among resumed smokers (mean 48.43 (SD 9.67)) than among stable former smokers (mean 49.59 (SD 8.36), data not shown); therefore, there is more room to move up in the scale for resumed smokers than stable former smokers. This spectulation is supported by our finding that the category of baseline HRQoL was associated with the risk of having a clinically significant change of HRQoL with persons at the bottom of the scale at baseline having smaller risk of a clinically significant reduction, while having higher risk of a clinically significant improvement (data not shown). Also, this is in line with the self-medication hypothesis with those quitters who resume smoking having poorer mental health and possibly using cigarettes to regulate psychological distress [35]. The reasons why people quit or relapse smoking are many and are also likely to vary between individuals. Two widely recognised factors are level of nicotine dependence and psychological distress experienced after quitting, and it may be difficult for some smokers to overcome these issues and therefore achieve prolonged abstinence.

It is important to consider the public health implications of improved HRQoL following changes of smoking status. Previous campaigns have successfully informed people that smoking will more than likely shorten their life [36,37], but there is less awareness among smokers of the negative day to day health effects of smoking [38] and these findings may be used to highlight this association. This message may be particularly important among young adults as it contrasts with traditional anti-smoking messages that focus on preventing tobacco-related diseases, like cancer or CVDs, which may seem a distant reward for younger smokers and they tend to disregard such long-term health events [39].

Some limitations should be considered when interpreting these results. First, smoking status was self-reported at baseline and follow-up without biochemical verification, possibly leading to misclassification of smoking status. However, high levels of concordance have been reported between these two measures in other studies [40]. Second, those that completed follow-up were different to those lost to follow-up on some socio-demographics,

smoking status and HRQoL at baseline. Applying inverse probability weights to account for these differences did not appreciably change the results, suggesting that this was not a major source of bias. Third, we had missing data for some covariates, mostly psychiatric diagnoses, but the results were not appreciably changed in the sensitivity analysis. Fourth, there is potential for ceiling and floor effects in the analysis of a truncated response variable such as PCS or MCS scores, but the minimum PCS or MCS score was larger than 10 and the maximum was smaller than 70 at baseline and this made deterioration or improvement possible for all subjects. In addition, our results are adjusted for baseline HRQoL using binary covariates each corresponding to a discrete range of the PCS or MCS score to allow for differential patterns of response of change in score to the baseline value.

Our study also has some strengths. As mentioned previously, this is the first prospective study to comprise solely young adults. This is important as younger people are less likely than older people, who have most commonly been studied in relation to smoking and health outcomes, to suffer from health conditions that may independently alter assessments of HRQoL and make reverse causation a potential issue. Also, unlike other studies examining smoking status and HRQoL, a range of covariates have been considered in our study. Some of these were considered for the first time, such as fruit and vegetable consumption, measures of mental health (in physical HRQoL analysis), social support and personality. In our adjusted models, when we included measures of mental health in physical HRQoL analyses and included social support and personality in mental HRQoL analysis, the changes in effect estimate were 10% or more. Removing the confounding by these factors allows us to estimate the independent association between smoking and HRQoL.

6.6 Conclusion

To conclude, smoking by young adults was cross-sectionally associated with lower physical HRQoL and longitudinally associated with reductions in physical HRQoL. Quitters had an improvement in physical HRQoL relative to continuing smokers and no worsening of mental HRQoL, and people who resumed smoking had a greater reduction of physical HRQoL than those who maintained cessation. The expectation of short to medium-term gains in physical HRQoL as well as long-term health benefits may help motivate young adult smokers to quit.

6.7 Postscript

The findings presented in this chapter demonstrated short to medium-term gains in physical HRQoL and no deterioration in mental HRQoL among people who quit smoking relative to those who continued smoking and among people who stayed in cessation relative to those who relapsed to smoking. The next chapter will summarise all findings presented in research chapters, discuss their public health implications and suggest some future directions of research.

6.8 References

1. Toghianifar N, Najafian J, Pooya A, et al. Association of smoking status with quality of life in a cross-sectional population-based sample of Iranian adults: Isfahan Healthy Heart Program. *Asia Pac J Public Health.* 2012;24(5):786-794. doi:10.1177/1010539511403800.
2. Schmitz N, Kruse J, Kugler J. Disabilities, quality of life, and mental disorders associated with smoking and nicotine dependence. *Am J Psychiatry.* 2003;160(9):1670-1676.
3. Vogl M, Wenig CM, Leidl R, Pokhrel S. Smoking and health-related quality of life in English general population: implications for economic evaluations. *BMC Public Health.* 2012;12:203. doi:10.1186/1471-2458-12-203.
4. Wilson D, Parsons J, Wakefield M. The health-related quality-of-life of never smokers, ex-smokers, and light, moderate, and heavy smokers. *Prev Med.* 1999;29(3):139-144.
5. Dube SR, Thompson W, Homa DM, Zack MM. Smoking and health-related quality of life among U.S. Adolescents. *Nicotine Tob Res.* 2013;15(2):492-500. doi:10.1093/ntr/nts163.
6. Heikkinen H, Jallinoja P, Saarni SI, Patja K. The impact of smoking on health-related and overall quality of life: a general population survey in Finland. *Nicotine Tob Res.* 2008;10(7):1199-1207. doi:10.1080/14622200802163142.
7. Holahan CK, Holahan CJ, North RJ, Hayes RB, Powers DA, Ockene JK. Smoking status, physical health-related quality of life, and mortality in middle-aged and older women. *Nicotine Tob Res.* 2013;15(3):662-669. doi:10.1093/ntr/nts182.
8. Strandberg AY, Strandberg TE, Pitkala K, Salomaa VV, Tilvis RS, Miettinen TA. The effect of smoking in midlife on health-related quality of life in old age: a 26-year prospective study. *Arch Intern Med.* 2008;168(18):1968-1974. doi:10.1001/archinte.168.18.1968.
9. Guiterrez-Bedmar M, Segui-Gomez M, Gomez-Gracia E, Bes-Rastrollo M, Martinez-Gonzalez MA. Smoking status, changes in smoking status and health-related quality of life: findings from the SUN ("Seguimiento Universidad de Navarra") cohort. *Int J Environ Res Public Health.* 2009;6(1):310-320. doi:10.3390/ijerph6010310.
10. Sarna L, Bialous SA, Cooley ME, Jun HJ, Feskanich D. Impact of smoking and smoking cessation on health-related quality of life in women in the Nurses' Health Study. *Qual Life Res.* 2008;17(10):1217-1227. doi:10.1007/s11136-008-9404-8.
11. Lasser K, Boyd JW, Woolhandler S, Himmelstein DU, McCormick D, Bor DH. Smoking and mental illness: A population-based prevalence study. *JAMA.* 2000;284(20):2606-2610.
12. Munafo MR, Zetteler JI, Clark TG. Personality and smoking status: a meta-analysis. *Nicotine Tob Res.* 2007;9(3):405-413.
13. Goldney RD, Fisher LJ, Wilson DH, Cheok F. Major depression and its associated morbidity and quality of life in a random, representative Australian community sample. *Aust N Z J Psychiatry.* 2000;34(6):1022-1029.
14. van Straten A, Cuijpers P, van Zuuren FJ, Smits N, Donker M. Personality traits and health-related quality of life in patients with mood and anxiety disorders. *Qual Life Res.* 2007;16(1):1-8. doi:10.1007/s11136-006-9124-x.

15. Taylor G, McNeill A, Girling A, Farley A, Lindson-Hawley N, Aveyard P. Change in mental health after smoking cessation: systematic review and meta-analysis. *BMJ.* 2014;348:g1151. doi:10.1136/bmj.g1151.
16. Gall S, Jose K, Smith K, Dwyer T, Venn A. The childhood determinants of adult health study: a profile of a cohort study to examine the childhood influences on adult cardiovascular health. *Australasian Epidemiologist.* 2009;16(1):35-38.
17. Ware J, Jr., Kosinski M, Keller SD. A 12-Item Short-Form Health Survey: construction of scales and preliminary tests of reliability and validity. *Med Care.* 1996;34(3):220-233.
18. Gandek B, Ware JE, Aaronson NK, et al. Cross-validation of item selection and scoring for the SF-12 Health Survey in nine countries: results from the IQOLA Project. International Quality of Life Assessment. *J Clin Epidemiol.* 1998;51(11):1171-1178.
19. Sanderson K, Andrews G. The SF-12 in the Australian population: cross-validation of item selection. *Aust N Z J Public Health.* 2002;26(4):343-345.
20. Lim LL, Fisher JD. Use of the 12-item short-form (SF-12) Health Survey in an Australian heart and stroke population. *Qual Life Res.* 1999;8(1-2):1-8.
21. Craig CL, Marshall AL, Sjostrom M, et al. International physical activity questionnaire: 12-country reliability and validity. *Med Sci Sports Exerc.* 2003;35(8):1381-1395.
22. Smith KJ, McNaughton SA, Gall SL, Blizzard L, Dwyer T, Venn AJ. Takeaway food consumption and its associations with diet quality and abdominal obesity: a cross-sectional study of young adults. *Int J Behav Nutr Phys Act.* 2009;6:29. doi:10.1186/1479-5868-6-29.
23. Costa PTJ, McCrae RR. *Revised NEO Personality Inventory (NEO-PI-R) and NEO Five-Factor Inventory (NEO-FFI) professional manual.* Odessa, FL1992.
24. Henderson S, Duncan-Jones, R, McAuley, H. & Ritchie, K. The patient's primary group. *Br J Psychiatry.* 1978;132:74-86.
25. World Health Organization. *Composite International Diagnostic Interview (CIDI)*. Geneva: 1997. http://www.hcp.med.harvard.edu/wmhcidi/. Accessed 15 August 2014.
26. Blizzard L, Hosmer DW. The log multinomial regression model for nominal outcomes with more than two attributes. *Biom J.* 2007;49(6):889-902. doi:10.1002/bimj.200610377.
27. Hosmer Jr DW, Lemeshow S, Sturdivant RX. *Applied logistic regression.* Vol 398. 3rd ed. Canada: John Wiley & Sons; 2013.
28. Blizzard CL, Quinn SJ, Canary JD, Hosmer DW. Log-Link Regression Models for Ordinal Responses. *Open Journal of Statistics.* 2013;3(04):16-25.
29. Gill SC, Butterworth P, Rodgers B, Mackinnon A. Validity of the mental health component scale of the 12-item Short-Form Health Survey (MCS-12) as measure of common mental disorders in the general population. *Psychiatry Res.* 2007;152(1):63-71.
30. Borzecki AM, Lee A, Kalman D, Kazis LE. Do poor health behaviors affect health-related quality of life and healthcare utilization among veterans? The Veterans Health Study. *J Ambul Care Manage.* 2005;28(2):141-156.
31. Vianna EO, Gutierrez MR, Barbieri MA, Caldeira RD, Bettiol H, Da Silva AA. Respiratory effects of tobacco smoking among young adults. *Am J Med Sci.* 2008;336(1):44-49. doi:10.1097/MAJ.0b013e31815c3b47.

32. Urrutia I, Capelastegui A, Quintana JM, Muniozguren N, Basagana X, Sunyer J. Smoking habit, respiratory symptoms and lung function in young adults. *Eur J Public Health.* 2005;15(2):160-165.
33. Krause NM, Jay GM. What do global self-rated health items measure? *Med Care.* 1994;32(9):930-942.
34. Aubin HJ, Rollema H, Svensson TH, Winterer G. Smoking, quitting, and psychiatric disease: a review. *Neurosci Biobehav Rev.* 2012;36(1):271-284. doi:10.1016/j.neubiorev.2011.06.007.
35. Khantzian EJ. The self-medication hypothesis of substance use disorders: a reconsideration and recent applications. *Harv Rev Psychiatry.* 1997;4(5):231-244. doi:10.3109/10673229709030550.
36. Jha P, Ramasundarahettige C, Landsman V, et al. 21st-century hazards of smoking and benefits of cessation in the United States. *N Engl J Med.* 2013;368(4):341-350. doi:10.1056/NEJMsa1211128.
37. Pirie K, Peto R, Reeves GK, Green J, Beral V, Million Women Study C. The 21st century hazards of smoking and benefits of stopping: a prospective study of one million women in the UK. *Lancet.* 2013;381(9861):133-141. doi:10.1016/S0140-6736(12)61720-6.
38. Burns DM. Live fast, die young, leave a good-looking corpse. *Arch Intern Med.* 2008;168(18):1946-1947. doi:10.1001/archinte.168.18.1946.
39. Gough B, Fry G, Grogan S, Conner M. Why do young adult smokers continue to smoke despite the health risks? A focus group study. *Psychol Health.* 2009;24(2):203-220. doi:10.1080/08870440701670570.
40. Patrick DL, Cheadle A, Thompson DC, Diehr P, Koepsell T, Kinne S. The validity of self-reported smoking: a review and meta-analysis. *Am J Public Health.* 1994;84(7):1086-1093.

6.9 Appendix 6-1: Comparison of baseline characteristics of participants and non-participants

Table S1 Comparison of baseline characteristics of participants and non-participants*

	Physical HRQoL			Mental HRQoL		
	Participants (N=2,080)	**Non-participants (N=1,441)**	***P***	**Participants (N=1,788)**	**Non-participants (N=1,733)**	***P***
Age (years), Mean (SD)	31.7 (2.6)	31.9 (2.6)	0.106	31.6 (2.6)	32.0 (2.6)	<0.001
Men (%)	41.6	50.9	<0.001	40.6	50.6	<0.001
BMI†, Mean (SD)	25.2 (4.8)	26.0 (5.4)	<0.001	25.2 (4.9)	25.9 (5.2)	<0.001
Married/living as married (%)	70.0	66.6	0.037	70.6	66.5	0.008
Education (%)			<0.001			<0.001
Tertiary	43.5	30.0		45.4	30.3	
Vocational	29.7	34.6		28.0	35.5	
School only	26.9	35.4		26.6	34.3	
Smoking status (%)			<0.001			<0.001
Never	60.5	52.5		61.0	53.4	
Former	19.0	22.0		19.1	21.5	
Current	20.4	25.5		19.9	25.2	
Health status in 1985 (%)			0.463			0.783
Very good	36.7	36.1		35.9	37.1	
Good	44.5	43.2		44.6	43.4	
Average/poor/very poor	18.8	20.7		19.5	19.6	
PCS, Mean (SD)	52.3 (7.6)	50.2 (8.4)	<0.001	--	--	--
MCS, Mean (SD)	--	--	--	50.0 (8.3)	51.1 (10.0)	<0.001

BMI: body mass index; MCS: mental component summary; PCS: physical component summary; SD: standard deviation.

* Sample size varied (range, 1,108 to 1,437 for physical HRQoL analyses and 1,319 to 1,729 for mental HRQoL analyses) because of missing data.

† Weight (kg)/height $(m)^2$.

6.10 Appendix 6-2: Sensitivity analysis using inverse probability weighting technique

Sensitivity analyses were conducted by re-analysing the data using the technique of inverse probability weighting. Compared with the original models as showed in appendix 2, the associations remained significant at the same patters and the changes in the magnitude of significant associations were small, ranging from 0.0% to 14.7%.

Table S2 Cross-sectional associations of smoking status and HRQoL at baseline, applying inverse probability weighting

	Physical HRQoL			Mental HRQoL		
	N	Unadjusted	Adjusted[†]	N	Unadjusted	Adjusted[‡]
		Diff(95% CI)	Diff(95% CI)		Diff(95% CI)	Diff(95% CI)
Never smoker	1,259	Ref	Ref	1091	Ref	Ref
Former smoker	396	-0.13(-0.85, 0.60)	-0.55(-1.23, 0.13)	341	**-1.11(-2.10, -0.12)**[*]	-0.16(-1.14, 0.81)
Current smoker	425	**-2.16(-2.91, -1.40)**[***a]	**-2.44(-3.19, -1.68)**[***a]	356	**-2.19(-3.21, -1.17)**[***]	-0.89(-1.90, 0.12)

CI: confidence interval; Diff: difference in means; Ref: reference group.

[*] denotes *P*< or =0.05; [***] denotes *P*< or =0.001.

[†] Adjusted for age, sex, marital status, residing in major city, education, BMI, IPAQ level, total alcohol drinks per day, diagnosis of current severe psychological distress, and weight score.

[‡] Adjusted for age, sex, marital status, education, BMI, total alcohol drinks per day, social support, personality (neuroticism, agreeableness), and weight score.

[a] Statistical difference compared with former smokers.

Table S3 Associations of baseline smoking status and change in smoking status with change in HRQoL, applying inverse probability weighting

	Change in physical HRQoL			Change in mental HRQoL		
	Mean(SD)	Unadjusted Diff(95% CI)	Adjusted† Diff(95% CI)	Mean(SD)	Unadjusted Diff(95% CI)	Adjusted‡ Diff(95% CI)
Baseline smoking status						
Never smoker	-2.18(8.62)	Ref	Ref	-0.93 (9.29)	Ref	Ref
Former smoker	-3.10(8.68)	**-1.01(-1.98, -0.05)***	**-0.90(-1.74, -0.07)***	0.41 (9.63)	**1.18(0.03, 2.33)***	0.68(-0.32, 1.68)
Current smoker	-2.70(9.68)	-0.75(-1.76, 0.26)	**-1.40(-2.31, -0.48)****	0.39 (10.30)	**1.20(0.00, 2.39)***	0.00(-1.13, 1.13)
Change in smoking status						
Stable never	-2.18(8.62)	Ref	Ref	-0.93(9.29)	Ref	Ref
Stable former	-2.85(8.67)	-0.84(-1.88, 0.21)	-0.66(-1.55, 0.23)	0.27(9.60)	1.12(-0.10, 2.34)	0.58(-0.47, 1.64)
Resumed	-4.31(8.70)	-1.96(-3.92, 0.01)	**-2.21(-4.05, -0.37)***	1.17(9.84)	1.49(-1.29, 4.27)	1.22(-0.95, 3.38)
Continuing	-3.38(9.48)	**-1.34(-2.58, -0.11)***	**-2.17(-3.24, -1.09)***[a]	0.18(10.64)	0.66(-0.84, 2.16)	-0.62(-2.03, 0.78)
Quitter	-1.57(9.94)	0.17(-1.34, 1.68)	-0.21(-1.55, 1.12)	0.73(9.74)	**2.04(0.36, 3.72)***	0.95(-0.53, 2.44)

CI: confidence interval; Diff: difference in means; HRQoL: health-related quality of life; Ref: reference group.

* denotes *P*< or =0.05; ** denotes *P*< or = 0.01; *** denotes *P*< or =0.001.

† Adjusted for age, sex, marital status, follow-up duration (days), baseline PCS (in analysis of change in smoking status), residing in major city, education, BMI, IPAQ level, total alcohol drinks per day, diagnosis of current severe psychological distress, and weight score.

‡ Adjusted for age, sex, marital status, follow-up duration (days), baseline MCS (in analysis of change in smoking status), education, BMI, total alcohol drinks per day, social support, personality (neuroticism, agreeableness), and weight score.

[a] Statistical difference compared with quitters.

Table S4 Association between changes in smoking status and clinically meaningful changes (or 5 or more score points) in HRQoL, applying inverse probability weighting

	N	Stable[†]		Decreased			Increased		
		n	%	n	%	RR(95% CI)[‡§]	n	%	RR(95% CI)[‡§]
Change in physical HRQoL									
Stable never	1,259	637	50.6	416	33.04	Ref	206	16.36	Ref
Stable former	329	156	47.42	125	37.99	0.97(0.83, 1.14)	48	14.59	0.87(0.68, 1.13)
Resumed	67	26	38.81	33	49.25	**1.29(1.03, 1.62)**[*a]	8	11.94	0.76(0.41, 1.41)
Continuing	265	114	43.02	105	39.62	**1.36(1.14, 1.61)**[***]	46	17.36	0.81(0.61, 1.07)
Quitter	160	64	40	59	36.88	1.19(0.98, 1.44)	37	23.13	1.09(0.83, 1.44)
Change in mental HRQoL									
Stable never	1,091	518	47.48	324	29.70	Ref	249	22.82	Ref
Stable former	288	122	42.36	78	27.08	1.07(0.86, 1.34)	88	30.56	1.04(0.88, 1.25)
Resumed	53	24	45.28	10	18.87	0.68(0.37, 1.23)	19	35.85	1.24(0.86, 1.79)
Continuing	223	99	44.39	56	25.11	1.03(0.79, 1.33)	68	30.49	0.94(0.76, 1.16)
Quitter	133	62	46.62	37	27.82	0.95(0.70, 1.30)	34	25.56	1.08(0.84, 1.39)

CI: confidence interval; HRQoL: health-related quality of life; Ref: reference group; RR: relative risk.

[*] denotes *P*< or =0.05; [***] denotes *P*< or =0.001.

[†] Reference category

[‡] Physical HRQoL: adjusted for baseline age, sex, marital status, follow-up duration (days), categorical baseline PCS, residing in major city, education, BMI, IPAQ level, total alcohol drinks per day, diagnosis of current severe psychological distress, and weight score.

[§] Mental HRQoL: adjusted for baseline age, sex, marital status, follow-up duration (days), categorical baseline MCS, education, BMI, total alcohol drinks per day, social support, personality (neuroticism, agreeableness), and weight score.

[a] Statistical difference compared with stable former smokers.

6.11 Appendix 6-3: Sensitivity analyses by re-reanalysing data using 0.5 SD of baseline HRQoL values

Findings using half a SD of baseline HRQoL as the MCSD were broadly similar with the result with complete case analyses, with changes within 10.9% of the original significant associations. The clinically significant reduction of physical HRQoL for continuing smokers was at the borderline statistical significance level (RR: 1.14, 95% CI: 0.99, 1.32), and the clinically significant improvement of physical HRQoL for quitters disappeared relative to continuing smokers.

Table S5 Association between changes in smoking status and clinically meaningful changes in HRQoL, using half a SD of baseline HRQoL as the MCSD

	N	Stable[†]		Decreased			Increased		
		n	%	n	%	RR(95% CI)[‡§]	n	%	RR(95% CI)[‡§]
Change in physical HRQoL									
Stable never	1,259	503	39.95	480	38.13	Ref	276	21.92	Ref
Stable former	329	121	36.78	142	43.16	0.88(0.76, 1.02)	66	20.06	0.98(0.82, 1.17)
Resumed	67	17	25.37	39	58.21	**1.39(1.18, 1.64)**[***a]	11	16.42	0.82(0.53, 1.27)
Continuing	265	100	37.74	114	43.02	1.14(0.99, 1.32)	51	19.25	0.80(0.63, 1.01)
Quitter	160	58	36.25	62	38.75	1.02(0.85, 1.22)	40	25	1.04(0.83, 1.29)
Change in mental HRQoL									
Stable never	1,091	419	38.41	373	34.19	Ref	299	27.41	Ref
Stable former	288	108	37.50	85	29.51	0.94(0.78, 1.15)	95	32.99	1.07(0.92, 1.24)
Resumed	53	23	43.40	11	20.75	0.62(0.37, 1.05)	19	35.85	1.22(0.88, 1.69)
Continuing	223	94	42.15	60	26.91	0.89(0.71, 1.13)	69	30.94	0.89(0.73, 1.08)
Quitter	133	61	45.86	38	28.57	0.90(0.68, 1.19)	34	25.56	0.90(0.70, 1.16)

CI: confidence interval; Ref: reference group; RR: relative risk; MCSD: minimal clinical significant difference.

[***] denotes *P*< or = 0.001

[†] Reference category

[‡] Physical HRQoL: adjusted for baseline age, sex, marital status, follow-up duration (days), categorical baseline PCS, residing in major city, education, BMI, IPAQ level, total alcohol drinks per day, and diagnosis of current severe psychological distress.

[§] Mental HRQoL: adjusted for baseline age, sex, marital status, follow-up duration (days), categorical baseline MCS, education, BMI, total alcohol drinks per day, social support and personality (neuroticism, agreeableness).

[a] Statistical difference compared with stable former smokers.

6.12 Appendix 6-4: Sensitivity analyses by removing quitters who quit smoking owing to emerged health problems during follow-up

Table S6 Associations of baseline smoking status and change in smoking status with change in HRQoL, excluding quitters who quit smoking owing to emerged health problems during follow-up

	Change in physical HRQoL			Change in mental HRQoL		
	Mean(SD)	Unadjusted	Adjusted[†]	Mean(SD)	Unadjusted	Adjusted[‡]
		Diff(95% CI)	Diff(95% CI)		Diff(95% CI)	Diff(95% CI)
Baseline smoking status						
Never smoker	-2.18(8.62)	Ref	Ref	-0.93 (9.29)	Ref	Ref
Former smoker	-3.10(8.68)	-0.91(-1.91, 0.08)	**-0.83(-1.65, -0.01)**[*]	0.41 (9.63)	**1.34(0.18, 2.51)**[*]	0.79(-0.26, 1.83)
Current smoker	-2.73(9.64)	-0.55(-1.53, 0.43)	**-1.42(-2.26, -0.58)**[***]	0.49 (10.29)	**1.43(0.26, 2.59)**[*]	0.18(-0.90, 1.26)
Change in smoking status						
Stable never	-2.18(8.62)	Ref	Ref	-0.93(9.29)	Ref	Ref
Stable former	-2.85(8.67)	-0.67(-1.74, 0.41)	-0.48(-1.36, 0.40)	0.27(9.60)	1.20(-0.04, 2.45)	0.67(-0.44, 1.79)
Resumed	-4.31(8.70)	-2.13(-4.30, 0.04)	**-2.55(-4.29, -0.80)**[**a]	1.17(9.84)	2.10(-0.53, 4.74)	1.35(-0.98, 3.68)
Continuing	-3.38(9.48)	**-1.19(-2.37, -0.02)**[*b]	**-2.16(-3.15, -1.18)**[***b]	0.18(10.64)	1.11(-0.26, 2.49)	-0.32(-1.59, 0.95)
Quitter	-1.57(9.86)	0.61(-0.90, 2.11)	-0.12(-1.36, 1.11)	1.07(9.62)	**2.00(0.20, 3.81)**[*]	1.08(-0.53, 2.70)

CI: confidence interval; Diff: difference in means; Ref: reference group.

[*] denotes *P*< or =0.05; [**] denotes *P*< or = 0.01; [***] denotes *P*< or =0.001.

[†] Adjusted for age, sex, marital status, follow-up duration (days), baseline PCS (in analysis of change in smoking status), residing in major city, education, BMI, IPAQ level, total alcohol drinks per day, and diagnosis of current severe psychological distress.

[‡] Adjusted for age, sex, marital status, follow-up duration (days), baseline MCS (in analysis of change in smoking status), education, BMI, total alcohol drinks per day, social support, and personality (neuroticism, agreeableness).

[a] Statistical difference compared with stable former smokers; [b] Statistical difference compared with quitters.

Table S7 Association between changes in smoking status and clinically meaningful changes (or 5 or more score points) in HRQoL, excluding quitters who quit smoking owing to emerged health problems during follow-up

	N	Stable[†]		Decreased			Increased		
		n	%	n	%	RR(95% CI)[‡§]	n	%	RR(95% CI)[‡§]
Change in physical HRQoL									
Stable never	1,259	637	50.6	416	33.04	Ref	206	16.36	Ref
Stable former	329	156	47.42	125	37.99	0.95(0.82, 1.10)	48	14.59	0.92(0.73, 1.17)
Resumed	67	26	38.81	33	49.25	**1.30(1.05, 1.60)**[*a]	8	11.94	0.84(0.48, 1.48)
Continuing	265	114	43.02	105	39.62	**1.33(1.13, 1.56)**[***]	46	17.36	0.84(0.65, 1.09)
Quitter	148	58	39.19	56	37.84	**1.23(1.02, 1.48)**[*]	34	23.13	1.14(0.89, 1.46)
Change in mental HRQoL									
Stable never	1,091	518	47.48	324	29.7	Ref	249	22.82	Ref
Stable former	288	122	42.36	78	27.08	1.01(0.82, 1.25)	88	30.56	1.08(0.92, 1.27)
Resumed	53	24	45.28	10	18.87	0.65(0.37, 1.15)	19	35.85	1.30(0.93, 1.80)
Continuing	223	99	44.39	56	25.11	0.97(0.75, 1.25)	68	30.49	0.95(0.78, 1.16)
Quitter	120	55	45.83	33	27.5	1.00(0.73, 1.35)	32	26.67	1.04(0.82, 1.34)

CI: confidence interval; Ref: reference group; RR: relative risk;

[*] denotes *P*< or =0.05; [***] denotes *P*< or =0.001.

[†] Reference category

[‡] Physical HRQoL: adjusted for baseline age, sex, marital status, follow-up duration (days), categorical baseline PCS, residing in major city, education, BMI, IPAQ level, total alcohol drinks per day, and diagnosis of current severe psychological distress.

[§] Mental HRQoL: adjusted for baseline age, sex, marital status, follow-up duration (days), categorical baseline MCS, education, BMI, total alcohol drinks per day, social support and personality (neuroticism, agreeableness).

[a] Statistical difference compared with stable former smokers.

6.13 Appendix 6-5: Sensitivity analyses by re-defining former smokers' smoking status according to length of abstinence

Table S8 Cross-sectional associations of smoking status and HRQoL at baseline, re-defining former smokers' smoking status according to length of abstinence

	Physical Component Summary				Mental Component Summary			
	No	Mean (SD)	Unadjusted	Adjusted[†]	No	Mean (SD)	Unadjusted	Adjusted[‡]
			Diff(95% CI)	Diff(95% CI)			Diff(95% CI)	Diff(95% CI)
Never smoker	1,259	52.83 (7.32)	Ref	Ref	1,091	50.73 (7.90)	Ref	Ref
Former smoker[§]	315	52.84 (7.20)	0.01(-0.93, 0.94)	-0.39(-1.30, 0.52)	278	49.15 (8.69)	**-1.58(-2.68, -0.49)****	-0.48(-1.52, 0.56)
Current smoker	388	50.39 (8.51)	**-2.44(-3.30, -1.58)***[a]	**-2.86(-3.72, -2.00)***[a]	329	48.22 (9.26)	**-2.51(-3.53, -1.48)***	**-1.10(-2.11, -0.10)***

CI: confidence interval; Diff: difference in means; Ref: reference group.

* denotes *P*< or =0.05; ** denotes *P*< or = 0.01; *** denotes *P*< or =0.001.

[†] Adjusted for age, sex, marital status, residing in major city, education, BMI, IPAQ level, total alcohol drinks per day, and diagnosis of current severe psychological distress.

[‡] Adjusted for age, sex, marital status, education, BMI, total alcohol drinks per day, social support, and personality (neuroticism, agreeableness).

[§] Former smokers were those who had smoked at least 100 cigarettes, or a similar amount of tobacco over their lifetime, had been a daily smoker but had stopped daily smoking more than one year ago.

[a] Statistical difference compared with former smokers.

Table S9 Associations of baseline smoking status and change in smoking status with change in HRQoL, re-defining former smokers' smoking status according to length of abstinence

	Change in physical HRQoL				Change in mental HRQoL			
	No	Mean(SD)	Unadjusted	Adjusted[†]	No	Mean(SD)	Unadjusted	Adjusted[‡]
			Diff(95% CI)	Diff(95% CI)			Diff(95% CI)	Diff(95% CI)
Baseline smoking status								
Never smoker	1,259	-2.18(8.62)	Ref	Ref	1,091	-0.93 (9.29)	Ref	Ref
Former smoker[§]	315	-2.92(9.02)	-0.74(-1.83, 0.36)	-0.65(-1.56, 0.25)	278	0.37 (10.09)	**1.30(0.03, 2.57)**[*]	0.64(-0.51, 1.79)
Current smoker	388	-2.72(9.45)	-0.54(-1.55, 0.47)	**-1.37(-2.24, -0.50)**[**]	329	0.38 (10.45)	**1.31(0.12, 2.50)**[*]	-0.02(-1.13, 1.10)
Change in smoking status								
Stable never	1,259	-2.18(8.62)	Ref	Ref	1,091	-0.93(9.29)	Ref	Ref
Stable former	260	-2.52(9.00)	-0.33(-1.52, 0.85)	-0.22(-1.19, 0.75)	230	0.21(10.06)	1.14(-0.23, 2.52)	0.53(-0.70, 1.77)
Resumed	55	-4.81(8.96)	**-2.63(-5.02, -0.24)**[*]	**-2.70(-4.62, -0.78)**[**a]	48	1.13(10.29)	2.06(-0.74, 4.85)	1.13(-1.34, 3.60)
Continuing	265	-3.38(9.48)	**-1.19(-2.37, -0.02)**[*b]	**-2.20(-3.19, -1.21)**[***b]	223	0.18(10.64)	1.11(-0.28, 2.51)	-0.31(-1.59, 0.98)
Quitter	123	-1.32(9.27)	0.86(-0.78, 2.50)	0.37(-0.97, 1.72)	106	0.79(10.10)	1.73(-0.20, 3.65)	0.58(-1.13, 2.30)

CI: confidence interval; Diff: difference in means; HRQoL: health-related quality of life; Ref: reference group.

[*] denotes *P*< or =0.05; [**] denotes *P*< or = 0.01; [***] denotes *P*< or =0.001.

[†] Adjusted for age, sex, marital status, follow-up duration (days), baseline PCS (in analysis of change in smoking status), residing in major city, education, BMI, IPAQ level, total alcohol drinks per day, and diagnosis of current severe psychological distress.

[‡] Adjusted for age, sex, marital status, follow-up duration (days), baseline MCS (in analysis of change in smoking status), education, BMI, total alcohol drinks per day, social support, and personality (neuroticism, agreeableness).

[§] Former smokers were those who had smoked at least 100 cigarettes, or a similar amount of tobacco over their lifetime, had been a daily smoker but had stopped daily smoking more than one year ago.

[a] Statistical difference compared with stable former smokers; [b] Statistical difference compared with quitters.

Table S10 Association between changes in smoking status and clinically meaningful changes (or 5 or more score points) in HRQoL, re-defining former smokers' smoking status according to length of abstinence

	N	Stable[†]		Decreased			Increased		
		n	%	n	%	RR(95% CI)[‡§]	n	%	RR(95% CI)[‡§]
Change in physical HRQoL									
Stable never	1,259	637	50.6	416	33.04	Ref.	206	16.36	Ref.
Stable former	260	123	47.31	94	36.15	0.90(0.77, 1.06)	48	14.59	0.97(0.76, 1.24)
Resumed	55	19	34.55	29	52.73	**1.36(1.09, 1.69)**[**a]	8	11.94	0.93(0.53, 1.63)
Continuing	265	114	43.02	105	39.62	**1.31(1.12, 1.54)**[***]	46	17.36	0.84(0.64, 1.10)
Quitter	123	55	44.72	42	34.15	1.07(0.85, 1.34)	37	23.13	1.15(0.86, 1.54)
Change in mental HRQoL									
Stable never	1,091	518	47.48	324	29.70	Ref.	249	22.82	Ref.
Stable former	230	93	40.43	66	28.70	1.06(0.85, 1.33)	71	30.87	1.11(0.93, 1.33)
Resumed	48	20	41.67	10	20.83	0.75(0.43, 1.30)	18	37.50	**1.43(1.00, 2.03)**[*]
Continuing	223	99	44.39	56	25.11	0.96(0.75, 1.24)	68	30.49	0.97(0.79, 1.18)
Quitter	106	48	45.28	30	28.30	1.02(0.74, 1.40)	28	26.42	1.15(0.88, 1.49)

CI: confidence interval; Ref: reference group; RR: relative risk.

[*] denotes *P*< or =0.05; [**] denotes *P*< or = 0.01; [***] denotes *P*< or =0.001.

[†] Reference category

[‡] Physical HRQoL: adjusted for baseline age, sex, marital status, follow-up duration (days), categorical baseline PCS, residing in major city, education, BMI, IPAQ level, total alcohol drinks per day, and diagnosis of current severe psychological distress.

[§] Mental HRQoL: adjusted for baseline age, sex, marital status, follow-up duration (days), categorical baseline MCS, education, BMI, total alcohol drinks per day, social support and personality (neuroticism, agreeableness).

[a] Statistical difference compared with stable former smokers.

Chapter 7

Summary, implications, future directions and conclusions

Chapter 7 Summary, Implications, Future Directions and Conclusions

7.1 Aims of this thesis

Young adults have the highest prevalence of current smoking. Getting them to quit smoking will have the greatest benefits to their health and the society. However, quitting smoking, especially achieving prolonged abstinence, is a difficult journey for the majority of smokers. The ultimate goal of this thesis was to help young adults to quit smoking and prolong abstinence. Specifically, some factors that are either common in young adults (e.g. life-stage transitions) or known to be associated with lower cessation levels (e.g. post-cessation weight gain) were investigated.

As shown in **Figure 7-1**, the framework of this thesis encompassed two components: two analyses looking at predictors of smoking transitions and three analyses looking at health effects of smoking transitions. Awareness of predictors of smoking transitions can help to identify factors that predict successful cessation and identify people at risk of relapse. Strategies that work effectively in cessation are then matched to those who fail in quitting. Investigation of early effects of smoking transitions on health in young adults is also important in promoting cessation. This is partly because young smokers tend to disregard long-term health consequences of smoking [11]. Our findings can be used by general practitioners and other health professionals in communicating with young smokers at group and individual level, thereby increasing quit attempts and success rates.

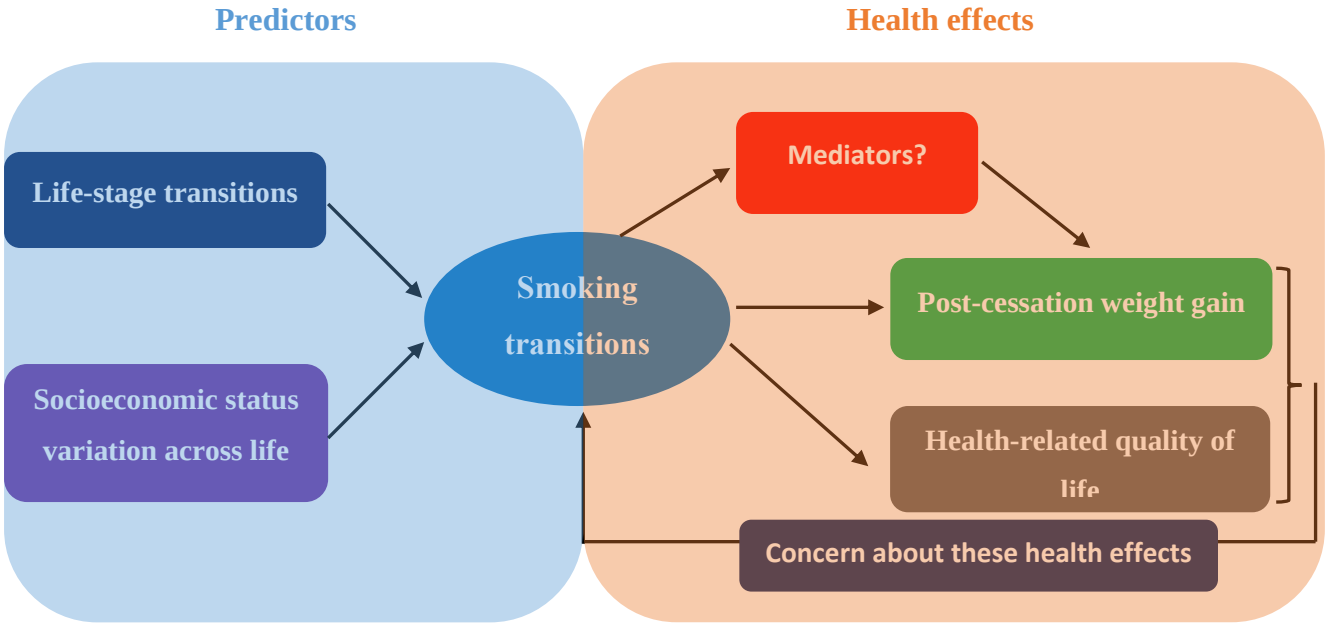

Figure 7-1 Thesis framework

The principal findings of this thesis, their public health implications and future research directions are summarised in the following sections.

7.2 Summary of results and public health implications

7.2.1 Partnering and parenting transitions and changes in smoking status

Investigations of the effects of partnering and parenting transitions on smoking continuity over five years in young adults were presented in **Chapter 2**. This was the first study examining the impacts of these factors among young men and women separately. It was found that transitions into relationships with a partner and entering parenthood were associated with beneficial changes in smoking behaviours in young adults, but they influenced men and women differently. The benefits of partnering on quitting smoking were greater for men than women, while transition into parenthood was of greater benefit to quitting smoking for women, especially for primiparous women.

As stated in the discussion section of **Chapter 2**, a partner's smoking status is a possible explanation of the greater benefits of being in a partnership seen in men than women. This hypothesis highlights the importance of encouraging the whole family, and indeed the wider peer group, to quit smoking. Several RCTs have examined the efficacy of family-based interventions on quit rates but produced mixed results. Some trials found family-based interventions worked effectively in promoting cessation [12,13], whereas others failed to

detect an increase in quit rates [14,15]. Park et al [16] recently updated a systematic review and meta-analysis to determine whether enhancing partner support helps smoking cessation when added as an adjunct to a smoking cessation program. Thirteen studies with more than 2000 participants were included. The definition of partners varied widely across the studies, including spouse, child, parent, friend, relative, co-worker, buddy and fellow cessation participants. They concluded that enhancing partner support for smokers in cessation programmes did not improve the abstinence rate at six months or longer post-treatment, nor did it increase the level of partner support. The conflicting findings do not necessarily mean family-based interventions are ineffective. A number of possible explanations have been proposed to account for the inconsistency, such as inadequate statistical power, short-term effects only, and adopted interventions ineffective in increasing partner support [16]. Given the importance of partner support in successful cessation [17,18] and the promise of family based interventions in other health behaviours [19,20] and addictions [21,22], additional research with larger samples is needed to further investigate the roles of family-based interventions on successful smoking cessation. Another possibility is a greater emphasis of the role of family and friends in supporting a person who is trying to quit in social marketing campaigns that aim at reducing smoking. However, most studies of the messages that are most effective in prompting quit attempts report that it is those with highly emotive or graphic health effects that have a greater effect rather than positive or instructional messages [23].

The apparent benefit on cessation of having a first child, compared to having following children, may suggest that focusing on women in their first pregnancy will be of particular benefit. It would appear that the women who remain smokers after their first child might be, so called “hardcore” smokers who are resistant to quitting [24]. To encourage more multiparous women to quit smoking, further research is needed to examine what factors motivate them to quit smoking in their first pregnancy, why the messages do not resonate in their following pregnancies and what are their triggers for relapsing. Pregnancy is already acknowledged as a ‘teachable moment’ where women may be more amenable to quitting smoking. In Australia, a national social marketing campaign about smoking cessation during pregnancy (National Smokefree Pregnancy Project) and a free quit smoking app for mums-

to-be (Quit for you – Quilt for two) have been launched to provide support to pregnant women and their partners on stopping smoking [25,26].

7.2.2 Life course SEP and smoking status in mid-adulthood

Investigations of which life course model best describes the association of SEP over early life and smoking status at mid-adulthood were presented in **Chapter 3**. The accumulation model and the sensitive period model were found to best describe the association between SEP and smoking in adulthood. Those exposed to low SEP for longer and those exposed during childhood and young adulthood reported higher risk of being a current smoker at mid-adulthood. The factors that might explain the increased risk of smoking associated with SEP across the life course were also examined. Parental smoking and rating that being a non-smoker was not important in childhood appeared to be influential. Together these two childhood factors accounted for 32% of the excess risk of smoking at mid-adulthood.

These results reiterate the importance of socio-economic inequalities in smoking. There is a great deal of effort directed towards reducing these inequalities including by increasing quit attempts and encouraging maintenance of abstinence in socio-disadvantaged groups. Given that differential uptake accounted for most of the SEP disparities in tobacco use rather than differential cessation rates, more work is needed to prevent the uptake of smoking among young adults, especially those from low SEP families [23].

An important contribution of the analyses presented in this chapter was the attempt to consider the root causes. People who smoked at mid-adulthood reported higher likelihood of starting life with smoking parents and having favourable attitudes about smoking than those who did not. These were found to account for some of the effect of low SEP across the life course on smoking. This finding demonstrates the crucial roles of parents in determining their children's future smoking status. Knowledge of the 'transmission' of smoking behaviours between generations may provide an incentive to prompt quitting smoking when people transit into parenthood. Possible ways through which parental smoking increases children's risk of being a smoker may include modelling of parental behaviour, greater perceived approval of smoking and increased susceptibility of smoking due to nicotine exposure in utero [27-29].

Population-wide approaches to promote educational achievement are important for reducing socioeconomic inequalities in a range of health behaviours and outcomes [30,31]. Attaining a greater level of education may discourage the uptake of smoking through improved health literacy. The associated higher incomes and membership of social groups with lower smoking prevalence also appears to have benefits for an individual's smoking behaviour [32]. It is evident that living in an area rated high in neighbourhood disorder increases the odds of being a smoker [33] and building a sense of community through participation in prosocial activities may help to reduce this risk [34].

There is also a role for comprehensive tobacco control programs, as these are believed to reduce disparities in tobacco use according to SEP [35]. According to the two latest systematic reviews, increasing tobacco taxes is the component with the greatest potential to reduce tobacco use associated with socioeconomic deprivation, including in youth [36,37]. Other interventions, such as legislation of smoke free policies, advertising bans and access controls seem unlikely to help narrow the gap between SEP groups without specific efforts to reach disadvantaged smokers [36,37].

Overall, the gross social inequalities in tobacco use present a challenge to the world and the gap between high and low SEP groups is estimated to widen with the trend to greater socioeconomic inequality [38], unless there is widespread cessation and effective smoking prevention strategies in low SEP groups. As emphasised by the WHO Commission on the Social Determinants of Health [39], looking at 'up-stream' causal factors related to smoking such as differential exposure to smoking cues is important. The current chapter found that exposure to parental smoking and having favourable attitudes toward smoking at childhood mediated the relationship between low SEP and high risk of smoking in adult life. As stated above, this finding highlights the lasting influences of parental smoking on their off-springs' smoking behaviours and points out the importance of discouraging the uptake of smoking and encouraging cessation at the age of entering parenthood.

7.2.3 Quitting smoking and gaining weight

Utilising a systematic review and meta-analysis method, the weight gained after smoking cessation and difference in weight gain between quitters and continuing smokers were quantified among 63,403 quitters and 388,432 continuing smokers from 35 population-

based prospective cohort studies (See **Chapter 4**). It was found that over approximately five years, quitters gained an average of 4.1 kg weight, which was 2.6 kg greater than the gain in continuing smokers. The amount of difference in weight gain was greater in women than men, and in studies conducted in North America than in Asia. Of note, quitting smoking specifically increased gain of abdominal fat, reflected by greater gain in waist circumference in quitters than continuing smokers.

The mechanisms linking smoking cessation and weight gain were further explored in 281 young Australian smokers by investigating the effects of dietary and PA behaviours. It was found that quitters tended to adopt healthier dietary and PA behaviours than continuing smokers, so these behaviours did not readily explain the post-cessation weight gain (See **Chapter 5**). So far, the reasons why people gain weight after quitting are not clear and numerous interventions have only a limited effect on mitigating post-cessation weight gain [40]. According to a recent meta-analysis of RCTs of first line smoking cessation drugs and interventions designed to limit weight gain after cessation, there was large variation in the magnitude of weight gain after cessation: 16-21% quitters lost weight, 35-38% gained less than 5 kg, 29-34% gained 5-10 kg, and 13-14% gained more than 10 kg [41]. The benefits of quitting smoking far outweigh continuing smoking regardless of weight gain [5-9]. Therefore, in practice, it is important to acknowledge the likelihood of weight gain and offer the optimum timing and interventions to prevent excess weight gain. Also, it is worth noting that although changes in diet and PA behaviours may not have explained post-cessation weight gain, it does not mean they did not have an effect in reducing weight gain – the weight gain may have been even greater without those positive behavioural changes among quitters. One potential way to better understand these associations could be through repeated measurements of anthropometry, weight-related health behaviours and metabolic factors perhaps using 'real time' devices to shed light on the complex mechanisms between smoking cessation and weight gain. This might assist with the design of strategies to prevent weight gain among quitters in the future.

7.2.4 Smoking status and HRQoL

Traditional tobacco control campaigns have focused on morbidity and mortality from tobacco-related diseases, such as cancer, respiratory diseases, stroke and CVDs. These

diseases take decades to develop and their prevention may seem a distant reward for young smokers. To persuade young adults to give up smoking, it could be important to increase the understanding of the detrimental effects of smoking that occur earlier. Assessment of HRQoL provides a way to achieve this aim, which is the exact purpose of **Chapter 6**. It was found that, for physical HRQoL change over five years follow-up, young people who quit smoking reported a statistically and clinically significant improvement compared with those who continued smoking, whereas former smokers who resumed smoking showed a statistically and clinically significant reduction compared with those who maintained cessation. No deterioration in mental HRQoL was observed after quitting smoking relative to continuing smoking. These findings underscore the immediate and negative health effects of smoking in the early lifespan and can be used to help discourage young adult smokers from initiating smoking and encourage them to quit and stay abstinent.

A better understanding of the extent to which young people are aware of the poor HRQoL caused by smoking and whether they care about these earlier health consequences will be important to understand. Perceptions of smoking-related risks and benefits play a key role in determining young people's smoking behaviours. Awareness of the harmful effects of smoking (i.e. physical risk and addiction risk) is associated with a decreased probability of initiating smoking [42,43]. Also, having negative beliefs about the health effects of smoking robustly predicted quitting smoking [17]. Nevertheless, only some youth have a realistic risk-benefit analysis of smoking [44]. Therefore, it is of importance to reinforce public education campaigns with messages about the earlier health effects of smoking. In addition, although there was a difference in predictive values between perceived short-term (i.e. smelling like an ashtray, getting a cough, have trouble breathing, getting colds) and long-term (i.e. getting lung cancer, having a heart attack and chronic trouble breathing) smoking-related risks, strengthening the short-term risks was suggested to work effectively in discouraging young people from starting smoking [43,45], and expectation of short-term health benefits is a suggested predictor of making a quit attempt [46-48].

Apart from deterioration in physical HRQoL, other possible immediate consequences of smoking in one's twenties or thirties include poorer lung function, reduced rate of lung

growth, poorer performance and endurance in physical fitness, more phlegm, faster resting heart rates, having early signs of CVDs and stroke [49].

This evidence can be used in consolidating future public education campaigns. Specifically, advertising in mass media is an effective approach at the population level, which allows messages about the health risks of smoking and the benefits of cessation to be repeatedly delivered to large audiences [50,51]. Exposure to such messages can directly influence people's view about smoking and their decision-making about quitting. It also prompts interpersonal (i.e. with partners, family and close friends) and public discussion about tobacco use, which might increase the likelihood of making a quit attempt and maintenance of abstinence. Furthermore, because large audiences are exposed to mass media, cessation may happen in smokers' social networks, including partners, family and close friends, who play important roles in influencing individual's smoking behaviours [18], including younger smokers [17]. This might further reinforce beneficial changes in smoking behaviours.

For many smokers who want to quit, doctors and other health professionals are the first point of contact to get advice and seek assistance on cessation strategies [52]. Compared with young people who did not smoke, those who did were three times more likely to see a doctor or health professional for varying reasons [53]. This is an opportunity to help young smokers quit by using doctors and health professionals in a face-to-face communication about the immediate harmful effects of smoking or using patient's own data on day to day deterioration in health. In Australia, three publications have been produced for assisting quitting in general practice (Smoking cessation guidelines for Australia general practice in 2004 [54], Smoking cessation pharmacotherapy: an update for health professionals in 2009 [55] and Supporting smoking cessation: a guide for health professionals in 2011 [56]). However, these clinical guidelines mainly focus on the cessation strategies and rarely contain the early detrimental health effects of smoking. Therefore, this is a potential vehicle for passing this chapter's findings to the hands of doctors and health professionals to better control tobacco use in young adults.

7.3 Future directions

This thesis has provided a valuable insight into some of the dynamic predictors and health effects of smoking transitions in young adults. These findings have important implications for current and future tobacco control programmes in this age group. Some suggestions for future research directions include:

- Understanding the factors underpinning the pronounced shifts in smoking following life-stage transitions
- Developing interventions to prevent uptake/promote quitting among young and young-middle aged adults related to partner and parenting transitions
- Exploration of the underlying mechanisms that link socioeconomic disadvantage over the life course with high risk of being a current smoker to inform strategies to address higher tobacco use in socioeconomically disadvantaged groups
- Investigation of the long-term effects of weight gain and other body composition changes after cessation on morbidity and mortality due to type 2 diabetes and CVDs, and whether this relationship differs between males and females, and between different smoking histories (pack-years, years since smoking cessation)
- Understanding why people gain weight after quitting smoking and most importantly, the ways it can be prevented
- Investigation of the efficacy of using day to day deterioration in HRQoL in promoting quitting and prolonged abstinence among young adults

7.4 Conclusions

This thesis examined the predictors and health effects of smoking transitions using a systematic review and meta-analysis and original data from a national cohort of young adults in Australia. Partnering and parenting transitions and SEP trajectories across the life course were found to predict smoking status or changes in smoking status. Compared with continuing smoking, quitting smoking led to greater weight gain, which was not explained by changing dietary and PA behaviours, and a significant improvement in physical HRQoL. These analyses have provided novel information on predictors of smoking cessation and the

associated health effects in young adults – a high priority group. The findings may help to promote smoking cessation and the maintenance of abstinence at the population and individual level.

7.5 References

1. G. B. D. Risk Factors Collaborators, Forouzanfar MH, Alexander L, et al. Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks in 188 countries, 1990-2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet.* 2015;386(10010):2287-2323. doi:10.1016/S0140-6736(15)00128-2.
2. Australian Institute of Health and Welfare. *National Drug Strategy Household Survey detailed report: 2013*. Canberra: Australian Institute of Health and Welfare; 2014. http://www.aihw.gov.au/publication-detail/?id=60129549469. Accessed 31 May 2016.
3. U.S. Department of Health and Human Services. *The health consequences of smoking-50 years of progress: a report of the surgeon general*. Atlanta, GA: 2014. http://www.ncbi.nlm.nih.gov/books/NBK179276/pdf/Bookshelf_NBK179276.pdf. Accessed 24 April 2016.
4. Robinson S, Harris H. Smoking and drinking among adults, 2009. *Newport: Office for National Statistics.* 2011.
5. Banks E, Joshy G, Weber MF, et al. Tobacco smoking and all-cause mortality in a large Australian cohort study: findings from a mature epidemic with current low smoking prevalence. *BMC Med.* 2015;13:38. doi:10.1186/s12916-015-0281-z.
6. Doll R, Peto R, Boreham J, Sutherland I. Mortality in relation to smoking: 50 years' observations on male British doctors. *BMJ.* 2004;328(7455):1519. doi:10.1136/bmj.38142.554479.AE.
7. Jha P, Ramasundarahettige C, Landsman V, et al. 21st-century hazards of smoking and benefits of cessation in the United States. *N Engl J Med.* 2013;368(4):341-350. doi:10.1056/NEJMsa1211128.
8. Pirie K, Peto R, Reeves GK, Green J, Beral V, Million Women Study C. The 21st century hazards of smoking and benefits of stopping: a prospective study of one million women in the UK. *Lancet.* 2013;381(9861):133-141. doi:10.1016/S0140-6736(12)61720-6.
9. Sakata R, McGale P, Grant EJ, Ozasa K, Peto R, Darby SC. Impact of smoking on mortality and life expectancy in Japanese smokers: a prospective cohort study. *BMJ.* 2012;345:e7093. doi:10.1136/bmj.e7093.
10. Centers for Disease Control Prevention. Quitting smoking among adults--United States, 2001-2010. *MMWR. Morbidity and mortality weekly report.* 2011;60(44):1513.
11. Gough B, Fry G, Grogan S, Conner M. Why do young adult smokers continue to smoke despite the health risks? A focus group study. *Psychol Health.* 2009;24(2):203-220. doi:10.1080/08870440701670570.
12. Huang FF, Jiao NN, Zhang LY, Lei Y, Zhang JP. Effects of a family-assisted smoking cessation intervention based on motivational interviewing among low-motivated smokers in China. *Patient Educ Couns.* 2015;98(8):984-990. doi:10.1016/j.pec.2015.02.017.
13. Tsoh JY, Burke NJ, Gildengorin G, et al. A Social Network Family-Focused Intervention to Promote Smoking Cessation in Chinese and Vietnamese American Male Smokers: A Feasibility Study. *Nicotine Tob Res.* 2015;17(8):1029-1038. doi:10.1093/ntr/ntv088.

14. Hemsing N, Greaves L, O'Leary R, Chan K, Okoli C. Partner support for smoking cessation during pregnancy: a systematic review. *Nicotine Tob Res.* 2012;14(7):767-776. doi:10.1093/ntr/ntr278.
15. Curry SJ, Hollis J, Bush T, et al. A randomized trial of a family-based smoking prevention intervention in managed care. *Prev Med.* 2003;37(6 Pt 1):617-626.
16. Park EW, Tudiver FG, Campbell T. Enhancing partner support to improve smoking cessation. *Cochrane Database Syst Rev.* 2012(7):CD002928. doi:10.1002/14651858.CD002928.pub3.
17. Cengelli S, O'Loughlin J, Lauzon B, Cornuz J. A systematic review of longitudinal population-based studies on the predictors of smoking cessation in adolescent and young adult smokers. *Tob Control.* 2012;21(3):355-362. doi:10.1136/tc.2011.044149.
18. Christakis NA, Fowler JH. The collective dynamics of smoking in a large social network. *N Engl J Med.* 2008;358(21):2249-2258. doi:10.1056/NEJMsa0706154.
19. Black DR, Gleser LJ, Kooyers KJ. A meta-analytic evaluation of couples weight-loss programs. *Health Psychol.* 1990;9(3):330-347.
20. Anonymous. Randomised controlled trial evaluating cardiovascular screening and intervention in general practice: principal results of British family heart study. Family Heart Study Group. *BMJ.* 1994;308(6924):313-320.
21. O'Farrell TJ, Clements K. Review of outcome research on marital and family therapy in treatment for alcoholism. *J Marital Fam Ther.* 2012;38(1):122-144. doi:10.1111/j.1752-0606.2011.00242.x.
22. Rowe CL. Family therapy for drug abuse: review and updates 2003-2010. *J Marital Fam Ther.* 2012;38(1):59-81. doi:10.1111/j.1752-0606.2011.00280.x.
23. Scollo MM, Winstanley MH. *Tobacco in Australia: Facts and issues*. Melbourne: Cancer Council Victoria; 2015. http://www.tobaccoinaustralia.org.au/. Accessed 11 April 2016.
24. Ip DT, Cohen JE, Bondy SJ, et al. Do components of current 'hardcore smoker' definitions predict quitting behaviour? *Addiction.* 2012;107(2):434-440. doi:10.1111/j.1360-0443.2011.03674.x.
25. Australian Department of Health and Aging. *Annual report 2007-2008*. Canberra: Department of Health and Aging; 2008. http://www.health.gov.au/internet/annrpt/publishing.nsf/Content/3C6696A0554501F1CA2575A5008138F0/$File/Full%20Report%20of%20the%202007-08%20Annual%20Report.pdf. Accessed 16 June 2016.
26. Australian Government Department of Health. Quit for you- Quit for two App. 2012.
27. Roberts KH, Munafo MR, Rodriguez D, et al. Longitudinal analysis of the effect of prenatal nicotine exposure on subsequent smoking behavior of offspring. *Nicotine Tob Res.* 2005;7(5):801-808. doi:10.1080/14622200500262840.
28. Al Mamun A, O'Callaghan FV, Alati R, et al. Does maternal smoking during pregnancy predict the smoking patterns of young adult offspring? A birth cohort study. *Tob Control.* 2006;15(6):452-457. doi:10.1136/tc.2006.016790.
29. Paul SL, Blizzard L, Patton GC, Dwyer T, Venn A. Parental smoking and smoking experimentation in childhood increase the risk of being a smoker 20 years later: the Childhood Determinants of Adult Health Study. *Addiction.* 2008;103(5):846-853. doi:10.1111/j.1360-0443.2008.02196.x.

30. Wilkinson RG, Marmot MG. *Social determinants of health: the solid facts.* World Health Organization; 2003.
31. Marmot M. The health gap: the challenge of an unequal world. *Lancet.* 2015;386(10011):2442-2444. doi:10.1016/S0140-6736(15)00150-6.
32. Graham H. Smoking, stigma and social class. *Journal of Social Policy.* 2012;41(01):83-99.
33. Miles R. Neighborhood disorder and smoking: findings of a European urban survey. *Soc Sci Med.* 2006;63(9):2464-2475. doi:10.1016/j.socscimed.2006.06.011.
34. Xue Y, Zimmerman MA, Caldwell CH. Neighborhood residence and cigarette smoking among urban youths: the protective role of prosocial activities. *Am J Public Health.* 2007;97(10):1865-1872. doi:10.2105/AJPH.2005.081307.
35. Garrett BE, Dube SR, Babb S, McAfee T. Addressing the Social Determinants of Health to Reduce Tobacco-Related Disparities. *Nicotine Tob Res.* 2015;17(8):892-897. doi:10.1093/ntr/ntu266.
36. Brown T, Platt S, Amos A. Equity impact of interventions and policies to reduce smoking in youth: systematic review. *Tob Control.* 2014;23(e2):e98-105. doi:10.1136/tobaccocontrol-2013-051451.
37. Hill S, Amos A, Clifford D, Platt S. Impact of tobacco control interventions on socioeconomic inequalities in smoking: review of the evidence. *Tob Control.* 2014;23(e2):e89-97. doi:10.1136/tobaccocontrol-2013-051110.
38. OECD. *Divided we stand: why inequality keeps rising*. 2011. https://www.oecd.org/els/soc/49170768.pdf. Accessed 10 January 2016.
39. Marmot M, Friel S, Bell R, Houweling TA, Taylor S, Commission on Social Determinants of H. Closing the gap in a generation: health equity through action on the social determinants of health. *Lancet.* 2008;372(9650):1661-1669. doi:10.1016/S0140-6736(08)61690-6.
40. Farley AC, Hajek P, Lycett D, Aveyard P. Interventions for preventing weight gain after smoking cessation. *Cochrane Database Syst Rev.* 2012;1:CD006219. doi:10.1002/14651858.CD006219.pub3.
41. Aubin HJ, Farley A, Lycett D, Lahmek P, Aveyard P. Weight gain in smokers after quitting cigarettes: meta-analysis. *BMJ.* 2012;345:e4439. doi:10.1136/bmj.e4439.
42. Aryal UR, Petzold M, Krettek A. Perceived risks and benefits of cigarette smoking among Nepalese adolescents: a population-based cross-sectional study. *BMC Public Health.* 2013;13:187. doi:10.1186/1471-2458-13-187.
43. Song AV, Morrell HE, Cornell JL, et al. Perceptions of smoking-related risks and benefits as predictors of adolescent smoking initiation. *Am J Public Health.* 2009;99(3):487-492. doi:10.2105/AJPH.2008.137679.
44. Arens CR, White TL, Massengill N. Attitudinal factors protective against youth smoking: an integrative review. *J Nurs Scholarsh.* 2014;46(3):167-175. doi:10.1111/jnu.12065.
45. Chang C. Psychological motives versus health concerns: predicting smoking attitudes and promoting antismoking attitudes. *Health Commun.* 2009;24(1):1-11. doi:10.1080/10410230802465241.
46. Li L, Feng G, Jiang Y, Yong HH, Borland R, Fong GT. Prospective predictors of quitting behaviours among adult smokers in six cities in China: findings from the International

Tobacco Control (ITC) China Survey. *Addiction.* 2011;106(7):1335-1345. doi:10.1111/j.1360-0443.2011.03444.x.

47. Li L, Borland R, Yong HH, et al. Predictors of smoking cessation among adult smokers in Malaysia and Thailand: findings from the International Tobacco Control Southeast Asia Survey. *Nicotine Tob Res.* 2010;12 Suppl:S34-44. doi:10.1093/ntr/ntq030.
48. Vangeli E, Stapleton J, Smit ES, Borland R, West R. Predictors of attempts to stop smoking and their success in adult general population samples: a systematic review. *Addiction.* 2011;106(12):2110-2121. doi:10.1111/j.1360-0443.2011.03565.x.
49. U.S. Department of Health and Human Services. *Preventing tobacco use among youth and young adults: a report of the surgeon general*. Atlanta, GA: U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Chronic Disease Prevention and Health Promotion, Office on Smoking and Health; 2012. http://www.surgeongeneral.gov/library/reports/preventing-youth-tobacco-use/exec-summary.pdf. Accessed 28 April 2016.
50. Durkin S, Brennan E, Wakefield M. Mass media campaigns to promote smoking cessation among adults: an integrative review. *Tob Control.* 2012;21(2):127-138. doi:10.1136/tobaccocontrol-2011-050345.
51. Bala MM, Strzeszynski L, Topor-Madry R, Cahill K. Mass media interventions for smoking cessation in adults. *Cochrane Database Syst Rev.* 2013(6):CD004704. doi:10.1002/14651858.CD004704.pub3.
52. McIntosh S, Ossip-Klein DJ, Hazel-Fernandez L, Spada J, McDonald PW, Klein JD. Recruitment of physician offices for an office-based adolescent smoking cessation study. *Nicotine Tob Res.* 2005;7(3):405-412. doi:10.1080/14622200500125567.
53. Arday DR, Giovino GA, Schulman J, Nelson DE, Mowery P, Samet JM. Cigarette smoking and self-reported health problems among U.S. high school seniors, 1982-1989. *Am J Health Promot.* 1995;10(2):111-116.
54. Zwar N, Richmond R, Borland R, Stillman S, Cunningham M, Litt J. *Smoking cessation guidelines for Australian general practice: practice handbook*. Canberra: Australian Government Department of Health and Ageing; 2004. http://www.health.gov.au/internet/main/publishing.nsf/content/21A287831207BB16CA257BF0001E0159/$File/smoking_flip.pdf. Accessed 8 June 2016.
55. Zwar N, Richmond R, Borland R, et al. *Smoking cessation pharmacotherapy: an update for health professionals (updated 2009)*. Melbourne: The Royal Australian College of General Practitioners; 2009. Accessed.
56. Zwar N, Richmond R, Borland R, et al. *Supporting smoking cessation: a guide for health professionals*. Melbourne: The Royal Australian College of General Practitioners; 2011. http://www.racgp.org.au/your-practice/guidelines/smoking-cessation/. Accessed 8 June 2016.

Appendix A

ASHFS questionnaire

AUSTRALIAN HEALTH & FITNESS SURVEY

1985

QUESTIONNAIRE

On the following pages you will find a number of questions about yourself, the exercise you do and your health.

We would like you to answer them all.

Please note that there are no right or wrong answers and that the answers you give are strictly private.

How to fill in your questionnaire:

1. I will read the questions with you and then you can answer each one yourself.
2. For most questions you will tick the box for the answer you want to give.

   For example:

   Do you watch television?

   Yes ..... ☑

   No ..... ☐

3. For other questions you will need to write the answer in the space provided. **Please print clearly.**
4. If you have any problems just ask me and I will help you with the answer.

Remember this is not a test, there are no right or wrong answers. What we want to know is what **you** do, how **you** think and how **you** feel.

COMMONWEALTH DEPARTMENT OF SPORT RECREATION & TOURISM

COMMONWEALTH DEPARTMENT OF HEALTH

NATIONAL HEART FOUNDATION OF AUSTRALIA

COMMONWEALTH SCHOOLS COMMISSION

2

1. We want to find out all the sports you have played regularly in the **last year.** Please include all sports you played with an organised team, group or club, or for your school, in the last year. Don't put in sports you have done as part of your P.E. classes but don't forget the sports you played during the last summer.
   Write the name of the sport in the first column and then fill in the other columns across the line with numbers. Do this for all the sports you have played in the last year.

Leave this column blank

SPORT Name of Sport	What time of year do you play this sport? Summer . 1 Winter . . . 2 Both 3	Who do you play this sport for? Club . . . . . 1 School . . . 2 Both . . . . . 3 Neither . . . 4	How good are you at it compared to others of your age? Better . . . . 1 Same . . . . 2 Worse . . . 3	Do you like playing this sport? Lots . . . . . 1 Some . . . . 2 Little. 3	Will you do it again? Yes . . . . . 1 No 2
e.g. GOLF	2	1	2	2	2
e.g. SWIMMING	1	3	1	1	1
.....	☐	☐	☐	☐	☐
.....	☐	☐	☐	☐	☐
.....	☐	☐	☐	☐	☐
.....	☐	☐	☐	☐	☐
.....	☐	☐	☐	☐	☐
.....	☐	☐	☐	☐	☐

3 (8)

9–15 ☐☐☐☐☐☐☐

16–22 ☐☐☐☐☐☐☐

23–29 ☐☐☐☐☐☐☐

30–36 ☐☐☐☐☐☐☐

37–43 ☐☐☐☐☐☐☐

44–50 ☐☐☐☐☐☐☐

2. In the **last week** what exercise have you had and what sports have you played. Begin with your travel to and from school and your Physical Education classes for the last week. Then we want to know about your school sport and any other sport or exercise you have done. Particularly anything you have done to improve your fitness.

Activity (describe fully)	How often (How many times last week?)	How long (How many hours and minutes were spent each session?) Hours : Mins	Effort (Did you huff and puff?) A lot . . . . . 1 Some . . . . . 2 Not much 3
Travel to school (Bicycle)	☐☐	☐ : ☐☐	☐
Travel to school (Walking)	☐☐	☐ : ☐☐	☐
School Physical Education	☐☐	☐ : ☐☐	☐
School sport: (Please print)			
.....	☐☐	☐☐ : ☐☐	☐
.....	☐☐	☐☐ : ☐☐	☐
Other activities: (Please print)			
.....	☐☐	☐☐ : ☐☐	☐
.....	☐☐	☐☐ : ☐☐	☐
.....	☐☐	☐☐ : ☐☐	☐
.....	☐☐	☐☐ : ☐☐	☐

51–56 ☐☐☐☐☐☐

57–62 ☐☐☐☐☐☐

63–68 ☐☐☐☐☐☐

4 (6)

9–17 ☐☐☐☐☐☐☐☐☐

18–26 ☐☐☐☐☐☐☐☐☐

27–35 ☐☐☐☐☐☐☐☐☐

36–44 ☐☐☐☐☐☐☐☐☐

45–53 ☐☐☐☐☐☐☐☐☐

54–62 ☐☐☐☐☐☐☐☐☐

Leave this column blank

3. Is your answer to the last question typical of your exercise pattern in the last **month?**

- I am usually more active ..... ☐ 1
- The same as usual ..... ☐ 2
- I am usually less active ..... ☐ 3

63

4. In most weeks do you get exercise or activity three or four times which makes you huff and puff and lasts at least 30 minutes each time?

- Yes ..... ☐ 1
- No ..... ☐ 2

64

5. What do you usually do at recess? *(Usually means 3 or more days a week.)*

- Sit and talk to friends ..... ☐ 1
- Walk around the school ..... ☐ 2
- Run around playing sports/games ..... ☐ 3
- Read/study for the next classes ..... ☐ 4
- Nothing much ..... ☐ 5
- Other (Please print) .....

65

6. What do you usually do at lunch time? *(Usually means 3 or more days a week.)*

- Sit and talk to friends ..... ☐ 1
- Walk around the school ..... ☐ 2
- Ride or walk home for lunch ..... ☐ 3
- Train for school sports teams ..... ☐ 4
- Play sport/games on the oval or in the school grounds ..... ☐ 5
- Study or do homework ..... ☐ 6
- Other (Please print) .....

66

7. Do you enjoy School Physical Education Classes?

- Very much ..... ☐ 1
- Quite a lot ..... ☐ 2
- Sometimes ..... ☐ 3
- Not much ..... ☐ 4
- Not at all ..... ☐ 5
- We don't have School Physical Education ..... ☐ 6
- I don't do Physical Education ..... ☐ 7

67

8. Do you enjoy School Sports?

- Very much ..... ☐ 1
- Quite a lot ..... ☐ 2
- Sometimes ..... ☐ 3
- Not much ..... ☐ 4
- Not at all ..... ☐ 5
- We don't have School Sport ..... ☐ 6
- I don't do Sport ..... ☐ 7

68

9. Do you enjoy Physical Activity? *(Vigorous playing or exercise that you do by choice)*

- Yes ..... ☐ 1 Why/Why not: .....
- No ..... ☐ 2 .....

69 70

Leave this column blank

10. How fit do you think you are compared to others of your age?

- Fitter than most ..... ☐ 1
- About average fitness ..... ☐ 2
- Not as fit as most ..... ☐ 3

71

11. Is your health usually?

- Very good ..... ☐ 1
- Good ..... ☐ 2
- Average ..... ☐ 3
- Poor ..... ☐ 4
- Very poor ..... ☐ 5

72

12. Do you enjoy school?

- Yes, all the time ..... ☐ 1
- Yes, most of the time ..... ☐ 2
- Sometimes Yes/sometimes No ..... ☐ 3
- Not very often ..... ☐ 4
- Never ..... ☐ 5

5 6

9

13. How good are you at school work compared to others of your age?

- Better than most ..... ☐ 1
- About the middle ..... ☐ 2
- Not as good as most ..... ☐ 3

10

14. What time did you go to bed and turn out the lights last night? *(For example 10:35)*

☐☐:☐☐ Hours Mins

11 14

15. What time did you wake up this morning? *(For example 06:15)*

☐☐:☐☐ Hours Mins

15 18

16. Here is a list that describes some of the ways people feel at different times. During the **past few weeks,** how often have you felt **(Tick one box on each line.)**

	Often	Sometimes	Never	
(a) On top of the world?	☐	☐	☐	19
(b) Very lonely or remote from other people?	☐	☐	☐	20
(c) Particularly excited or interested in something?	☐	☐	☐	21
(d) Depressed or unhappy?	☐	☐	☐	22
(e) Pleased about having accomplished something?	☐	☐	☐	23
(f) Bored?	☐	☐	☐	24
(g) Proud because someone complimented you on something you had done?	☐	☐	☐	25
(h) So restless you couldn't sit long in a chair?	☐	☐	☐	26
(i) That things were going your way?	☐	☐	☐	27
(j) Upset because someone criticised you?	☐	☐	☐	28

Leave this column blank

17. Have you ever smoked even part of a cigarette?

- No ☐ 1
- Yes, just a few puffs ☐ 2
- Yes, I have smoked fewer than 10 cigarettes in my life ☐ 3
- Yes, I have smoked more than 10 cigarettes in my life ☐ 4

28

18. How many cigarettes have you smoked in the last 7 days? ☐☐☐

30 32

19. How long have you been smoking regularly? *(Regularly means 1 or more times a week.)*

- I don't smoke ☐ 1
- Just started ☐ 2
- 1 month up to 6 months ☐ 3
- 7 months up to 1 year ☐ 4
- 1 year up to 2 years ☐ 5
- 2 years up to 4 years ☐ 6
- More than 4 years ☐ 7

33

20. How many people smoke at home? *(If you smoke — do not include yourself.)*

Number of persons ☐

34

21. Do you think you will be smoking this time next year?

- Yes ☐ 1
- No ☐ 2
- Don't know ☐ 3

35

22. Please say what you think about the following statements about smoking.

	Agree	Disagree	Don't know	
(a) It makes you catch cold easily	☐	☐	☐	36
(b) It slows your circulation and makes your fingers cold	☐	☐	☐	37
(c) It keeps you alert	☐	☐	☐	38
(d) It makes you unable to play sport well	☐	☐	☐	39
(e) It makes your breathing difficult	☐	☐	☐	40
(f) It can cause lung cancer	☐	☐	☐	41
(g) It calms your nerves	☐	☐	☐	42
(h) It can cause heart disease	☐	☐	☐	43
(i) It gives you bad breath	☐	☐	☐	44
(j) It keeps you slim	☐	☐	☐	45
(k) It is addictive (habit forming)	☐	☐	☐	46

Leave this column blank

23. How often do you usually drink alcohol?

- I don't drink alcohol ☐ 1
- Less than once a week ☐ 2
- On 1 or 2 days a week ☐ 3
- On 3 or 4 days a week ☐ 4
- On 5 or 6 days a week ☐ 5
- Every day ☐ 6

47

24. Do you usually eat something before starting school? *(Usually means 4 or more times a week.)*

- Yes ☐ 1
- No ☐ 2

48

25. In your opinion how important is it to you to —

	Very important	Of some importance	Of little importance	Not important	
(a) See a dentist once a year	☐	☐	☐	☐	49
(b) Have a doctor's checkup	☐	☐	☐	☐	50
(c) Know about your body and how it works	☐	☐	☐	☐	51
(d) Have a good night's sleep	☐	☐	☐	☐	52
(e) Eat a good diet	☐	☐	☐	☐	53
(f) Be a non-smoker	☐	☐	☐	☐	54
(g) Have a good body/figure	☐	☐	☐	☐	55
(h) Exercise regularly	☐	☐	☐	☐	56
(i) Not be fat	☐	☐	☐	☐	57
(j) Have friends	☐	☐	☐	☐	58
(k) Not be stressed and worried	☐	☐	☐	☐	59
(l) Not drink alcohol or only drink a little	☐	☐	☐	☐	60
(m) Know about fitness and how to stay fit	☐	☐	☐	☐	61

26. How well can you do the following?

	Never tried	Can't do	Can do about average	Can do very well	
(a) Play a musical instrument	☐	☐	☐	☐	62
(b) Ride a horse	☐	☐	☐	☐	63
(c) Play tennis	☐	☐	☐	☐	64
(d) Swim 100 metres non-stop	☐	☐	☐	☐	65
(e) Play a round of golf	☐	☐	☐	☐	66
(f) Ride on a surfboard/sailboard	☐	☐	☐	☐	67
(g) Ice or roller skate	☐	☐	☐	☐	68
(h) Play squash/badminton or racquetball	☐	☐	☐	☐	69
(i) Play video games	☐	☐	☐	☐	70
(j) Play billiards/pool or snooker	☐	☐	☐	☐	71
(k) Ride on a skateboard	☐	☐	☐	☐	72

27. In what suburb/town do you live? *(Shire if you live on a farm.)*

.....

28. When were you born? (eg 06 / May / 1970) day month year ☐☐ / ..... / 19☐☐ (day / month / year)

29. What sex are you?
- Male ..... ☐ 1
- Female ..... ☐ 2

30. In what country were you born? *(If you don't know the country put the city or town.)*

.....

31. In which Australian State or Territory were you born?
- Born outside Australia ..... ☐ 0
- A.C.T. ..... ☐ 1
- New South Wales ..... ☐ 2
- Victoria ..... ☐ 3
- Queensland ..... ☐ 4
- South Australia ..... ☐ 5
- Western Australia ..... ☐ 6
- Tasmania ..... ☐ 7
- Northern Territory ..... ☐ 8
- Don't know ..... ☐ 9

32. How many years since you first arrived in Australia? ☐☐ years

In the next question, when we ask about your brothers and sisters please include step-brothers and step-sisters.

33. How many older brothers do you have at home? ..... ☐
- How many older sisters do you have at home? ..... ☐
- How many younger brothers do you have at home? ..... ☐
- How many younger sisters do you have at home? ..... ☐

The next two questions are about where your natural parents (not your step-parents) were born. If you don't know the country, give the city or town, if you don't know write in "don't know".

34. In what country was your father born? .....

35. In what country was your mother born? .....

36. Do you speak a language, other than English, at home?
- No, English only .. ☐ 1
- Yes, we speak ..... ☐ 2

6 — Leave this column blank — 7

8 — Leave this column blank

The rest of the questions ask about your home and parents. By father we also mean stepfather or male guardian. For mother we also want you to include stepmother or female guardian.

37. During the school week, where do you live?
- At home with your mother and father ..... ☐ 1
- At home with your mother ..... ☐ 2
- At home with your father ..... ☐ 3
- In a boarding school/house ..... ☐ 4

38. Does your mother or father smoke at home?
- No ..... ☐ 1
- Yes, both smoke ..... ☐ 2
- Yes, mother ..... ☐ 3
- Yes, father ..... ☐ 4

39. Does your father exercise regularly (2 or more times a week)?
(For example does he — jog, play sport, do exercises, go to a gym, do aerobics.)
- Yes ..... ☐ 1
- No ..... ☐ 2
- Don't know ..... ☐ 3
- What activity does he do? .....

40. Does your mother exercise regularly (2 or more times a week)?
(For example does she — jog, play sport, do exercises, go to a gym, do aerobics.)
- Yes ..... ☐ 1
- No ..... ☐ 2
- Don't know ..... ☐ 3
- What activity does she do? .....

Appendix B

Assessment of smoking status

8784007497 17

7. Do you have any BIOLOGICAL sisters

O Yes

O No -->Skip to SECTION F

O Don't know -->Skip to SECTION F

IF YES

7a) Have any of your biological sisters been diagnosed with diabetes when under the age of 50?

O Yes O No O Don't Know

7b) If 'YES', please complete details below (space has been allowed for you to complete details for up to 3 sisters if necessary):

	Age at diagnosis (if known)	Did this result in her death?
1		O Yes O No
2		O Yes O No
3		O Yes O No

SECTION F: This section is about smoking tobacco

1. Over your lifetime, have you smoked at least 100 cigarettes, or a similar amount of tobacco?

O No --> SKIP TO SECTION G (Page 20)

O Yes

2. How often do you now smoke cigarettes, cigars, pipes or any other tobacco products?

O Daily

O At least once a week (but not daily) -->Skip to Question 7

O Less often than weekly -->Skip to Question 7

O Not at all -->Skip to Question 7

1497007491 18

3. When did you start smoking daily? Years of Age [ ][ ] OR Year [ ][ ][ ][ ]

4. What do you currently smoke?
(Please indicate types and enter how many you smoke)

4a)	O Manufactured cigarettes	[ ][ ][ ] Cigarettes per day
4b)	O Hand-rolled cigarettes	[ ][ ][ ] Grams per week*
4c)	O Cigars	[ ][ ][ ] Cigars per week
4d)	O Pipes full of tobacco	[ ][ ][ ] Grams per week*

*** A one and three quarter ounce pouch of tobacco equals 50 grams**

5. When you smoke manufactured cigarettes, which brand do you usually smoke?

I do not smoke manufactured cigarettes O

The brand I usually smoke is [ ]
(Please give as much detail as possible, eg Marlboro Lights)

6. Have there been any periods of time when you gave up daily smoking and then started smoking again?

No O -->Skip to SECTION G (Page 20)

Yes O

IF YES

6a) Were any of these periods greater than 3 months duration?

No O -->Skip to SECTION G (Page 20)

Yes O

IF YES 6b) What is the total amount of time that you stopped smoking for?
(Please add together all the periods of time when you stopped smoking)

Years [ ][ ] Months [ ][ ] Now skip to SECTION G (Page 20)

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7. In the past have you ever been a daily smoker?

No O -->Skip to SECTION G (Page 20)

Yes O

	Years of Age		Year
8. When did you start smoking daily?		OR	
9. When did you finally stop smoking daily?			

10. When you smoked daily, how much did you usually smoke?
(Please indicate types and enter the number smoked)

10a)	O Manufactured cigarettes	Cigarettes per day
10b)	O Hand-rolled cigarettes	Grams per week*
10c)	O Cigars	Cigars per week
10d)	O Pipes full of tobacco	Grams per week*

* A one and three quarter ounce pouch of tobacco equals 50 grams

11. When you smoked manufactured cigarettes, which brand did you usually smoke?

I did not smoke manufactured cigarettes O

The brand I usually smoked was

(Please give as much detail as possible, eg Marlboro Lights)

12. Prior to the time when you finally stopped daily smoking, were there any periods of time when you gave up daily smoking and then started smoking again?

No O -->Skip to SECTION G (Page 20)

Yes O

IF YES

12a) Were any of these periods greater than 3 months duration?

No O -->Skip to SECTION G (Page 20)

Yes O

IF YES 12b) What is the total amount of time that you stopped smoking for?
(Please add together all the periods of time when you stopped smoking)

Years Months

Appendix C

Assessment of HRQoL

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SECTION C: The following questions ask for your views about your health. This information will help keep track of how you feel and how well you are able to do your usual activities.

1. **In general would you say your health is:**

O Excellent O Very Good O Good O Fair O Poor

2. **The following questions are about activities you might do during a typical day. Does your health now limit you in these activities? If so, how much?**

	YES, limited a lot	YES, limited a little	NO, not limited at all
2a) Moderate activities, such as moving a table, pushing a vacuum cleaner, bowling or playing golf.	O	O	O
2b) Climbing several flights of stairs.	O	O	O

3. **During the past 4 weeks, how much of the time have you had any of the following problems with your work or other regular daily activities as a result of your physical health?**

	All of the time	Most of the time	Some of the time	A little of the time	None of the time
3a) Accomplished less than you would like	O	O	O	O	O
3b) Were limited in the kind of work or other activities.	O	O	O	O	O

4. **During the past 4 weeks, how much of the time have you had any of the following problems with your work or other regular daily activities as a result of any emotional problems (such as feeling depressed or anxious)**

	All of the time	Most of the time	Some of the time	A little of the time	None of the time
4a) Accomplished less than you would like	O	O	O	O	O
4b) Did work or other activities less carefully than usual	O	O	O	O	O

1849007492 9

5. During the past 4 weeks, how much did pain interfere with your normal work (including both work outside the home and housework)?

Not at all	A little bit	Moderately	Quite a bit	Extremely
O	O	O	O	O

6. These questions are about how you feel and how things have been with you during the past 4 weeks. For each question, please give the one answer that comes closest to the way you have been feeling.

How much of the time during the past 4 weeks:	All of the time	Much of the time	Some of the time	A little of the time	None of the time
6a) Have you felt calm and peaceful?	O	O	O	O	O
6b) Did you have a lot of energy?	O	O	O	O	O
6c) Have you felt downhearted and depressed?	O	O	O	O	O

7. During the past 4 weeks, how much of the time has your physical health or emotional problems interfered with your social activities (like visiting with friends, relatives etc.)?

All of the time	Most of the time	Some of the time	A little of the time	None of the time
O	O	O	O	O

Appendix D

The International Physical Activity Questionnaire

7394203480 ☐☐☐☐ 1

OFFICE USE ONLY

SCAN	VERIFY	CHECK

OFFICE USE ONLY

(Clinic staff attach bar code here)

SECTION A: CURRENT ACTIVITIES

The following questions will ask you about the time you spent being physically active in the last 7 days. Please think about the activities you do at work, as part of your house and yard work, to get from place to place, and in your spare time for recreation, exercise or sport.

Please answer each question even if you do not consider yourself to be an active person.

Think about all the **vigorous** and **moderate** activities that you have done **in the last 7 days**.

- **Vigorous** physical activities refer to activities that take **hard** physical effort and make you breathe much harder than normal.
- **Moderate** activities refer to activities that take moderate physical effort and make you breathe **somewhat** harder than normal

PART 1: WORK RELATED PHYSICAL ACTIVITY
The first section is about your work. This includes paid jobs, farming, volunteer work, course work, and any other unpaid work that you did outside your home.

Do not include unpaid work you might do around your home, like housework, yard work, general maintenance, and caring for your family. We ask about these in Part 3.

1. Do you currently have a job or do any unpaid work outside your home?

   No O --> **SKIP TO PART 2, TRANSPORTATION**

   Yes O

The next questions are about all the physical activity you did in the last 7 days as part of your paid or unpaid work. This does not include travelling to and from work.

2. During the **last 7 days**, on how many days did you do vigorous physical activities like heavy lifting, digging, heavy construction, or climbing up stairs **as part of your work**?
   Think about only those physical activities that you did for **at least 10 minutes** at a time.

   ☐ days per week

   O No vigorous job-related physical activity --> **SKIP TO Question 4**

3. How much time did you usually spend on one of those days doing vigorous physical activities as part of your work?

   ☐☐ hours ☐☐ minutes **Per day**

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4. Again, think about only those physical activities that you did for **at least 10 minutes** at a time. During **the last 7 days**, on how many days did you do **moderate** physical activities like carrying light loads as part of your work? *Please DO NOT include walking.*

☐ days per week

O No moderate job-related physical activity --> **SKIP TO Question 6**

5. How much time did you **usually** spend on **one** of those days doing **moderate** physical activities as part of your work?

☐☐ hours ☐☐ minutes **Per day**

6. During the last 7 days, on how many days did you walk for **at least 10 minutes** at a time as part of your work? Please do not count any walking you did to travel to, or from work.

☐ days per week

O No job-related walking--> **Skip to PART 2: TRANSPORTATION**

7. How much time did you usually spend on **one** of those days **walking** as part of your work?

☐☐ hours ☐☐ minutes **Per day**

PART 2: TRANSPORTATION PHYSICAL ACTIVITY
These questions are about how you travelled from place to place, including to places like work, stores, movies, and so on.

8. During the last 7 days, on how many days did you travel **in a motor vehicle** like a train, bus, car, or tram?

☐ days per week

O No motor transport --> **SKIP TO Question 10**

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9. How much time did you **usually** spend in a motor vehicle on **one** of those days.

☐☐ hours ☐☐ minutes **Per day**

Now think only about the <u>cycling</u> and <u>walking</u> you might have done to travel to and from work, to do errands, or to go from place to place.

10. During **the last 7 days**, on how many days did you **cycle** for **at least 10 minutes** at a time to go from place to place?

☐ days per week

O No cycling from place to place --> **SKIP TO Question 12**

11. How much time did you usually spend on **one** of those days **cycling** from place to place?

☐☐ hours ☐☐ minutes **Per day**

12. During the last 7 days, on how many days did you **walk** for at **least 10 minutes** at a time to go from place to place?

☐ days per week

O No walking from place to place --> **SKIP TO PART 3: HOUSEWORK, MAINTENANCE AND CARING FOR FAMILY**

13. How much time did you usually spend on **one** of those days **walking** from place to place?

☐☐ hours ☐☐ minutes **Per day**

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PART 3: HOUSEWORK, HOUSE MAINTENANCE, AND CARING FOR FAMILY
This section is about some of the physical activities you might have done in the last 7 days in and around your home, like housework, gardening, yard work, general maintenance work, and caring for your family

YARD WORK:

14. Think about only those physical activities that you did for **at least 10 minutes** at a time. During **the last 7 days**, on how many days did you do **vigorous** physical activities like heavy lifting, chopping wood, shovelling snow, or digging in the garden or yard?

☐ days per week

O No vigorous yard activity --> **SKIP TO Question 16**

15. How much time did you usually spend on **one** of those days doing **vigorous** physical activities **in the garden or yard**?

☐☐ hours ☐☐ minutes **Per day**

16. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do **moderate** activities like carrying light loads, sweeping, washing windows, and raking **in the garden or yard**?

☐ days per week

O No moderate yard activity --> **SKIP TO Question 18**

17. How much time did you usually spend on **one** of those days doing **moderate** physical activities **in the garden or yard**?

☐☐ hours ☐☐ minutes **Per day**

HOUSEWORK:

18. Once again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do **moderate** activities like carrying light loads, washing windows, scrubbing floors and sweeping **inside your home**?

☐ days per week

O No moderate activity at home --> **SKIP TO PART 4: RECREATION, SPORT, AND LEISURE-TIME PHYSICAL ACTIVITY**

19. How much time did you usually spend on **one** of those days doing **moderate** physical activities **inside your home**?

☐☐ hours ☐☐ minutes **Per day**

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PART 4: RECREATION, SPORT, AND LEISURE-TIME PHYSICAL ACTIVITY
This section is about all the physical activities that you did in the last 7 days solely for recreation, sport, exercise or leisure. Please do not include any activities you have already mentioned.

20. Not counting any walking you have already mentioned, during **the last 7 days**, on how many days did you **walk** for **at least 10 minutes** at a time in your leisure time?

☐ days per week

O No leisure walking --> **SKIP TO Question 22**

21. How much time did you usually spend on **one** of those days **walking** in your leisure time?

☐☐ hours ☐☐ minutes **Per day**

22. Think about only those physical activities that you did for **at least 10 minutes** at a time. During **the last 7 days**, on how many days did you do **vigorous** physical activities like: aerobics, running, fast bicycling, or fast swimming in your leisure time?

☐ days per week

O No vigorous activity in leisure time --> **SKIP TO Question 24**

23. How much time did you usually spend on **one** of those days doing vigorous physical activities in your leisure time?

☐☐ hours ☐☐ minutes **Per day**

24. Again, think about only those physical activities that you did for at least 10 minutes at a time. During the last 7 days, on how many days did you do **moderate** physical activities like: bicycling at a regular pace, swimming at a regular pace, and doubles tennis in your leisure time?

☐ days per week

O No moderate activity in leisure time --> **SKIP TO PART 5: TIME SPENT SITTING**

25. How much time did you usually spend on **one** of those days doing **moderate** physical activities in your leisure time?

☐☐ hours ☐☐ minutes **Per day**

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PART 5: TIME SPENT SITTING

These last questions are about the time you spend sitting while at work, at home, while doing course work and during leisure time. This may include time spent sitting at a desk, visiting friends, reading or sitting or lying down to watch television.

Do not include any time spent sitting in a motor vehicle that you have already told us about.

26. During the **last 7 days**, how much time did you usually spend **sitting** on a **weekday**?

☐☐ hours ☐☐ minutes **Per day**

27. During the last 7 days, how much time did you usually spend **sitting** on a **weekend** day?

☐☐ hours ☐☐ minutes **Per day**

We are also interested in finding out about your television viewing and computer use habits

28. Please estimate the total time **during the last week** that you spent watching television, videos or DVD's when it was the **main** activity that you were doing.
For example, you should not include time when the television was switched on and you were preparing a meal or ironing.

Total time Monday to Friday ☐☐ hours ☐☐ minutes

Total time Saturday and Sunday ☐☐ hours ☐☐ minutes

29. Please estimate how often **in a usual week** you would have each of the following while watching television

	Always (every day)	Usually (5-6 times/week)	Sometimes (3-4 times/week)	Rarely (1-2 times/week)	Never
A Meal	O	O	O	O	O
A Snack	O	O	O	O	O
A soft drink	O	O	O	O	O
An alcoholic drink	O	O	O	O	O

I do not watch television O

30. Please estimate the total time **during the last week** that you spent using a computer during the week and on weekends (this might be a personal computer at home or work, Playstation, X-box, Gameboy, etc).

Total time Monday to Friday ☐☐ hours ☐☐ minutes

Total time Saturday and Sunday ☐☐ hours ☐☐ minutes

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PART 6: STRENGTH TRAINING
We are interested in any strength/weight training that you may be involved in.

31. Are you currently involved in a muscle-strengthening program?

No O --> **SKIP TO Question 35**

Yes O

If YES, please specify the type(s) of muscle strengthening program you are involved in. *(Select all that apply.)*

O Free weights

O Weight training machines

O Other (please specify) ☐

32. How many days per week do you usually do these activities? ☐ days

33. What is the average duration of your workout? ☐☐ hours ☐☐ minutes

34. How long have you been involved in this routine? ☐☐ years ☐☐ months

35. Have you ever held a job for 12 months or more that regularly (more than once a week) involved vigorous activities (hard physical effort that made you breathe much harder than normal)?

No O -->**SKIP to SECTION B: PAST PHYSICAL ACTIVITIES**

Yes O

If '**YES**', please complete the table below by indicating:

- -the type of job you held
- -the year you started this job
- -how long you held the job for, in years and months
- -the average number of days per week you did vigorous activity in that job
- -the average amount of time you spent doing vigorous activity each day, in hours

(If you have held more than four jobs that involved vigorous activity, please list the four that you held the longest)

Occupation/Job type	Year started	Length of time in job Years	Length of time in job Month	Days per week	Hours per day