Population-wide Changes in Obesity:

Their Causes, Trends and Implications

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Summary

The prevalence of obesity is increasing worldwide. In Australia, for example, the prevalence of obesity doubled in two decades between 1980 and 2000, and continues to increase. Considering the substantial body of literature suggesting that obesity is the cause of much morbidity, premature mortality and an important component of increasing health care costs, it has become an issue high on the public agenda. But much remains unclear about what is commonly referred to as the 'obesity epidemic'.

This thesis examines trends in the whole population distribution of body weight and differences in trends based on two measures of excess body weight, using data from national population-based surveys from Australia and the United States, respectively. It also investigates the prevalence of obesity in Vietnam, a country with a historically low prevalence of obesity, using data from a population-based survey of northern Vietnam. Furthermore, this thesis examines individual-level predictors of weight gain, projects the likely future burden of obesity in Australia based on current incidence rates, and examines the impact of hypothetical obesity-reduction strategies, using data from Australian national population-based surveys, the National Death Index (NDI), and national mortality statistics.

The findings suggest that in high-income countries such as Australia and the United States, the increase in obesity is the result of an increase in body weight across the whole population distribution, with much greater increases at the higher end of the distribution. There also appears to be a greater increase in abdominal weight than overall body mass index (BMI) across the whole population distribution of body weight, suggesting that in each of the BMI categories the level of metabolic risk has increased. In Australia, a third of the population underwent substantial abdominal weight gain between 2000 and 2005. The main individual-level behavioural predictor of such a gain was poor diet quality; however a commentary article included in the thesis discusses how the focus on individual-level determinants should not overlook the context in which such behaviours are made.

Projecting recent measures of the incidence of overweight and obesity in Australia over the next two decades suggests that by 2025 approximately 34% of the population will be obese (compared to 21% in 2000). Of the normal-weight 25-29 year-olds in the year 2000, an estimated 36% will be overweight and 29% obese by age 60-64 years. Analysis of the effect

of a series of hypothetical interventions showed that population strategies that de-skew the distribution of body weight would be the most safe and effective.

In urban Hanoi, Vietnam, almost a third of adults were found to be overweight or obese in 2004, and one in ten were underweight, based on Asian-specific BMI cut-points.

It has long been suggested that obesity places a heavy health burden on individuals and an encumbrance on the health care system. By analysing trends in the whole population distribution of body weight, their causes, and the likely trajectory of the future burden of obesity, this thesis contributes to a better understanding of the increasing prevalence of obesity.

General declaration

In accordance with Monash University Doctorate Regulation 17/ Doctor of Philosophy and Master of Philosophy (MPhil) regulations the following declarations are made:

I hereby declare that this thesis contains no material which has been accepted for the award of any other degree or diploma at any university or equivalent institution and that, to the best of my knowledge and belief, this thesis contains no material previously published or written by another person, except where due reference is made in the text of the thesis.

This thesis includes five original papers published in peer reviewed journals and five unpublished publications. The core theme of the thesis is population-wide changes in obesity, their causes, trends and implications. The ideas, development and writing of all the papers in this thesis were the principal responsibility of myself, the candidate, working within the Department of Epidemiology and Preventive Medicine, under the supervision of Professor John McNeil, Dr Anna Peeters and Associate Professor Christopher Reid. The inclusion of co-authors reflects the fact that the work came from active collaboration between researchers and acknowledges input into team-based research.

In the case of the publications presented in Chapters 3 and 4, my contribution to the work involved the following:

Thesis chapter	**Publication title**	**Publication status**	**Nature and extent of candidate's contribution**
2.3	Methodological problem with comparing increases in different measures of body weight	Submitted	Design and drafting of manuscript (70%)
2.4.2	Predicting the effectiveness of prevention: A role for epidemiological modelling	Published	Conceptualisation and drafting of manuscript (75%)
3.1.1	Trends in BMI of urban Australian adults, 1980-2000	Published	Design, analysis and drafting of manuscript (70%)
3.1.2	Comparing trends in body mass index and waist circumference	Submitted	Design, analysis and drafting of manuscript (70%)
3.2.1	Underweight, overweight and obesity in adults	Published	Design, analysis and drafting

	of urban Hanoi, Vietnam		of manuscript (90%)
3.3	Predictors of increasing waist circumference in an Australian population	Submitted	Design, analysis and drafting of manuscript (80%)
3.4	Projected progression of the prevalence of obesity in Australia	Submitted	Design, analysis and drafting of manuscript (60%)
3.5	A new perspective on chronic disease prevention: why approaches to obesity prevention require more thought	Published (short version)	Design, analysis and drafting of manuscript (75%)
4.4.1	Public health campaigns and obesity – a critique of the evidence	Submitted	Conceptualisation and drafting of manuscript (80%)
4.4.2	Why education and choice won't solve the obesity problem	Published	Conceptualisation and drafting of manuscript (70%)

In the case of the co-authored publications presented in the Appendices which are related to the thesis but not forming part of the thesis, my contribution to the work involved the following:

App. 3	The duration of obesity and risk of diabetes	Submitted	Drafting of manuscript (5%)
App. 3	The number of years lived with obesity and the risk of mortality	Submitted	Drafting of manuscript (5%)
App. 3	Economic implications of obesity among people with atherothrombotic disease	Accepted	Conceptualisation and drafting of manuscript (10%)
App. 3	Setting population targets for successful obesity prevention	Submitted	Analysis and drafting of manuscript (20%)

I have renumbered sections of submitted or published papers in order to generate a consistent presentation within the thesis.

Signed: .....

Date: .....

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Research is a collaborative venture and I am appreciative of the expertise, support and kindness of my co-authors and many others with whom I have worked over my period of candidature. Many thanks to colleagues at the National Heart Institute in Hanoi, Vietnam and the Baker IDI Heart and Diabetes Institute here in Melbourne for providing access to their data and for their collaboration.

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I am also extremely appreciative to those friends, family and colleagues who read and commented on the thesis during its final days. Thank you to Dr Andrea Curtis, Dr Kelvin Walls, Rosanne Freak-Poli, Vanessa Stephens, Julie Novak, Anthony del Monaco and Alison Beauchamp. Thank you, too, for the sometimes ensuing debate!

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List of abbreviations, terms and definitions

AEA	–	Australasian Epidemiological Association
ABS	–	Australian Bureau of Statistics
AIHW	–	Australian Institute of Health and Welfare
AusAID	–	Australian Agency for International Development
AusDiab	–	Australian Diabetes, Obesity and Lifestyle Study
BMI	–	body-mass index
CAD	–	coronary artery disease
CD	–	census cluster district
cerVD	–	cerebrovascular disease
CVD	–	cardiovascular disease
COAG	–	Coalition of Australian Governments
DQI	–	dietary quality index
DRG	–	diagnosis related group
FFQ	–	food frequency questionnaire
GP	–	general practitioner
HCMC	–	Ho Chi Minh City
ICD	–	International Classification of Diseases
IEA	–	International Epidemiological Association
IHD	–	ischaemic heart disease
MBS	–	Medicare Benefit Schedule

MEC	–	mobile examination centre
NDI	–	National Death Index
NHANES	–	National Health and Nutrition Examination Studies
NHF	–	National Heart Foundation
NHMRC	–	National Health and Medical Research Council
NHS	–	National Health Survey
NNS	–	National Nutrition Survey
PAD	–	peripheral arterial disease
PBS	–	Pharmaceutical Benefits Scheme
PSC	–	Prospective Studies Collaboration
QALY	–	quality-adjusted life year
RFPS	–	National Heart Foundation Risk Factor Prevalence Study
RR	–	relative risk
SD	–	standard deviation
SE	–	standard error
SEIFA	–	socio-economic indexes for areas
VicHealth	–	Victorian Health Promotion Foundation
WC	–	waist circumference
WHO	–	World Health Organization
WHR	–	waist-to-hip ratio

List of publications, presentations and awards

Listed below are the candidate's first-author and co-authored publications, conference posters and presentations, and awards. All are relevant to the period of candidature.

Publications by the candidate relevant to the thesis

Walls HL, Peeters A, Reid C, Liew D, McNeil JJ. 2008. Predicting the effectiveness of prevention: A role for epidemiological modelling. *J Prim Prev*. 2008;29:295–305

Walls HL, Peeters A, Loff B, Crammond BR. 2009. Why education and choice won't solve the obesity problem. *Am J Public Health*. 2009;99:590-2

Walls HL, Peeters A, Pham TS, Nguyen NQ, Nguyen TTH, Do DL, Nguyen LV, Pham GK, Reid CM. Prevalence of underweight, overweight and obesity in urban Hanoi, Vietnam. *Asia Pac J Clin Nutr*. 2009; 18(2):234-9

Walls HL, Wolfe R, Haby MM, Magliano DJ, de Courten M, Reid CM, McNeil JJ, Shaw J, Peeters A. 2009. Trends in Body Mass Index in Urban Australians, 1980-2000. *Public Health Nutr*. 2009;22:1-8.

Walls HL, McNeil JJ, Peeters A. 2009. Population versus high-risk interventions for obesity. *Epidemiology*. 2009;20:929-930

Walls HL, Stevenson C, Abdullah A, Reid CM, McNeil JJ, Peeters A. 2009. Comparing trends in body mass index and waist circumference. *Submitted*.

Walls HL, Peeters A, Mannan H, Stevenson C. 2009. Methodological problem with comparing increases in different measures of body weight. *Submitted*.

Walls HL, McNeil JJ, Peeters A. 2009. Population interventions for obesity. *Epidemiology.* 2010. In Press.

Walls HL & Magliano DJ, Stevenson C, Backholer K, Mannan HR, Shaw J, Peeters A. 2009. Projected progression of the prevalence of obesity in Australia. Submitted.

Walls HL, Magliano DJ, Stevenson C, McNeil JJ, Ademi Z, Shaw J, Peeters A. Predictors of increasing waist circumference in an Australian population. Submitted.

Walls HL, McNeil JJ, Proietto J, Peeters A. Public health campaigns and obesity – a critique. Submitted.

Additional peer-reviewed publications

Ademi Z, **Walls HL**, Peeters A, Liew D, Hollingsworth B, Stevenson C, Deepak B, Steg G, Reid CM. Economic implications of obesity among people with atherothrombotic disease. *Int J Obes*. In press.

Abdullah A, Stoelwinder J, Shortreed S, Wolfe R, **Walls HL**, Stevenson C, de Courten M, Peeters A. The duration of obesity and risk of diabetes. Submitted.

Abdullah A, de Courten M, Stolewinder J, Stevenson C, **Walls HL**, Peeters A. The number of years lived with obesity and the risk of mortality. Submitted.

Backholer K, **Walls HL**, Magliano DJ, Peeters A. Setting population targets for measuring successful obesity prevention. Submitted.

Conference posters/presentations

Walls HL, McNeil JJ, Peeters A. 2009. A new perspective on chronic disease prevention: why approaches to obesity prevention require more thought. Conference: Australasian Epidemiological Association. Dunedin, New Zealand.

Walls HL, Wolfe R, de Courten M, Reid CM, Magliano DJ, McNeil JJ, Peeters A. 2008. Comparing trends in body mass index and waist circumference. Conference: 16th European Congress on Obesity. Geneva, Switzerland.

Walls HL, Wolfe R, Haby MM, Magliano DJ, de Courten M, Reid CM, McNeil JJ, Shaw J, Peeters A. 2007. Obesity Trends in Urban Australians, 1980-2000. Conference: AusDiab Consortium Meeting Annual Conference. Melbourne, Australia.

Walls HL, Magliano DJ, Reid C, McNeil J, Peeters A. 2007. Waist Circumference Trends in Australia, 1989-2000. Conference: 16th Annual Meeting of the Australian Society for the Study of Obesity. Canberra, Australia.

Walls HL, Haby M, Magliano DJ, Shaw J, Reid C, McNeil J, Peeters A. 2007. Trends in Body Mass Index in Australia, 1980-2000. Conference**:** Joint Scientific Meetings of the Australian Epidemiological Association and the International Epidemiological Association Western Pacific Region**.** Hobart, Australia.

Awards

Student Travel Bursary, Joint Scientific Meeting of the Australasian Epidemiological Association (AEA) and the International Epidemiological Association (IEA) Western Pacific Region. Hobart, Australia, 2007.

Top Ranked Scholar in Victoria, National Heart Foundation (NHF), 2008

Student Travel Bursary, Annual Scientific Meeting of the Australasian Epidemiological Association. Dunedin, New Zealand, 2009.

Best Student Oral Presentation, Annual Scientific Meeting of the Australasian Epidemiological Association (AEA). Dunedin, New Zealand, 2009.

Chapter 1: Introduction

The prevalence of obesity has increased worldwide over recent decades, in both low- and high-income countries (1, 2). In Australia, for example, the prevalence of obesity in adults more than doubled in the two decades between 1980 and 2000 (3), and in 2007/08, 26% of men and 24% of women were classified as obese (4). Comparable estimates (2007) for the US were 33% of men and 35% of women (5).

The development of obesity is caused by a chronic excess of energy intake over energy expenditure (6, 7). But the regulation of this 'energy equation' is the result of complex environmental and physiological influences, and their interactions (8).

A substantial body of literature documents the associations between obesity – particularly an abdominal or central pattern of fat distribution (9-14) – and a number of diseases and metabolic abnormalities (15-19). Thus, obesity appears to place a heavy health burden on individuals and is a significant encumbrance on health care systems. A better understanding of the trends in the population distribution of body weight, their causes, and the likely trajectory of the future burden of obesity will help inform health policy decision-making regarding the most appropriate obesity-reduction responses.

This introductory chapter provides a background on obesity – its definition and measurement, its burden of consequences, trends, determinants, and obesity-reduction strategies. The chapter concludes with an outline of the research undertaken as part of this thesis.

1.1 Definition and measurement of obesity

1.1.1 Measurement of body fat

The main stores of body fat (composed mainly of adipose in the form of triglyceride) are subcutaneous and intra-abdominal (19). Body fat also resides in muscle, particularly in

older people (19, 20). Total adiposity cannot be measured directly because of its diffuse and inaccessible nature, but there are several methods of measuring it indirectly.

The appropriateness of the measurement method used depends on the situation. Laboratory measures (such as body potassium, body density and total body water) are fairly accurate, but are also expensive, time-consuming, and thus impractical for use in large-scale population studies (21, 22). In epidemiological studies and clinical settings body fat and its distribution are estimated by anthropometric measures and associated indices (19, 23).

The anthropometric measure most commonly used in epidemiologic studies is body-mass index (BMI), also called the Quetelet index (19). BMI is calculated by dividing a person's weight in kilograms by the square of the height in metres (kg/m^2). It is a fast and inexpensive means of determining excess body weight and can be based on self-reported or clinically assessed height and weight. BMI is reliable in that it generally correlates well with laboratory-derived measures of adiposity, but does have the limitation of not distinguishing body fat mass from lean mass (19). Thus, individuals of low body-fat but unusually large muscle mass – trained athletes, for example – can be assigned BMIs in the 'obese' range. BMI may also be a less appropriate measure of body fat in older people, in whom there is a differential loss of lean mass (19). It does, however, remain a useful population-level indicator of the distribution of body weight (19).

Other common anthropometric measures of excess body weight are waist circumference (WC) and waist-to-hip ratio (WHR) (22). WC represents the circumference of the part of the abdomen between the rib cage and hips. There is currently no consensus regarding the optimal method for the measurement of WC (24). A systematic review identified eight protocols, the majority of which measured WC at either the umbilicus, the midpoint (midway between the distal border of the lowest rib and the superior border of the iliac crest) or the minimal waist (generally described as the smallest area of the waist) (24). Although variation in measurement can profoundly influence the absolute WC measure, no evidence was found to suggest that variation in WC measurement protocol had substantial influence on risk of morbidity or mortality (24).

The WHR is the ratio of the circumference of the waist to that of the hips. The WHR measurement was introduced on the assumption that it would predict fat distribution better than WC alone as hip circumference appears to be inversely related to morbidity.

WHR has been found to be the best anthropometric predictor of CVD risk in some studies (9, 25, 26), and was for some years a clinically-accepted method of identifying people with excess body weight. WC has been considered, however, a more practical measure of intra-abdominal fat mass and total body fat, due to the requirement for only one measurement and no subsequent calculations (9).

1.1.2 Body-mass index and waist circumference

1.1.2.1 Categorisation of body-mass index and waist circumference

World Health Organization (WHO) guidelines have defined body weight according to BMI, as follows (27, 28):

Table 1. Categories of body-mass index

BMI category	BMI – kg/m^2
Underweight	<18.5
Normal weight	18.5-24.9
Overweight	25.0-29.9
Obese	≥ 30.0
- Obese class I	30.0-34.9
- Obese class II	35.0-39.9
- Obese class III	≥ 40.0

Source: Adapted from World Health Organization (2000) (27).

WHO guidelines do not yet exist for WC (although consultation regarding these is currently taking place). Commonly-used categories for WC (in use in Australia and the United States, for example) are as follows (29):

Table 2. Categories of waist circumference

	WC category	WC – cm
Men	Low risk	<94.0
	Increased risk	94.0-101.9
	Substantially increased risk	≥ 102.0
Women	Low Risk	<80.0
	Increased risk	≥ 80.0-87.9
	Substantially increased risk	≥ 88.0

Note: The 'risk' to which the WC categories above refer is the morbidity/mortality associated with excess body weight.

Source: Adapted from Lean et al. (1995) (29).

Different cut-points are sometimes used for some ethnic groups, as discussed further in Chapter 1.1.2.3.

BMI and WC cut-points do not represent true risk-based thresholds (19); the cut-points to define their categories (for example, 'underweight', 'normal-weight', 'overweight' and 'obese') are somewhat arbitrary. This is due to the continuous and graded association between BMI/WC and health risks (19), at least after a certain point – due to the J-shaped relationship between body weight and mortality (as discussed further in Chapter 1.2.1) (30).

1.1.2.2 Relative merit

High BMI and WC have both been shown to be associated with adverse health outcomes (15-19). The specific conditions are described in Chapter 1.2. There is considerable controversy, however, regarding the contribution of abdominal visceral and subcutaneous fat to the development of disease (31-34), and thus the relative importance of BMI and WC as predictors of metabolic and cardiovascular disease.

BMI is a widely accepted index for assessing adiposity in adults and its associated health risks (35-37). However BMI has limitations, for example it does not distinguish between fat mass and lean tissue mass and may underestimate fatness in older adults who due to age-

related declines in muscle mass have a greater amount of body fat at a given BMI (16, 19). However at a population level, high and low BMI values are associated with an increased risk of morbidity and mortality (16, 30).

The use of WC (and WHR) is supported by a number of studies that have reported a stronger correlation with metabolic/cardiovascular risk for a central pattern of fat distribution (where excess body fat is distributed in the abdominal region) than a more peripheral fat distribution (where excess fat is distributed on areas such as the hips and thighs) (9-14). These increased risks may also apply to people with normal BMIs but large waist circumferences (38). However some studies suggest that WC is no more predictive than BMI of cardiovascular disease (CVD) events and mortality (39, 40).

The evidence to date is generally supportive of a stronger association between WC and CVD risk than with BMI, but the association between WC and other obesity-related diseases may be weaker than for BMI. Abbate et al. (2006) found BMI (more strongly related to weight load) was more strongly associated with radiographic knee osteoarthritis than WC and WHR (41). In population studies, there is also a greater need with BMI to ensure that groups such as smokers and the terminally ill are removed from the analysis else WC can appear the better measurement (42). Furthermore, WC is easier to ascertain than BMI, as BMI requires more equipment and a calculation. WC is associated with greater measurement error in the more severely obese (42). However incorrect BMI categorisation occurs for approximately 20% of adults, on the basis of measured height and weight, but only 2% are incorrectly classified (in the three WC categories) on the basis of self-measured WC (43). These and additional advantages and disadvantages of BMI and WC as measures of excess body weight are described in Table 3.

Table 3. Comparison of body-mass index and waist circumference

Body-mass index (BMI)	Waist circumference (WC)
Good predictor of total body fat and associated health risk at population level	Good predictor of total body fat and associated health risks at population level
Weak relationship with visceral fat	Best anthropometric measure of visceral fat
Routinely collected by general practitioners	Seldom collected by general practitiones
Used in many databases	Use in databases increasing rapidly
Less reliable in discriminating health risks when BMI <30 kg/m^2	Less reliable in discriminating health risks when BMI >40 kg/m^2
Confounded by differences in muscle mass	-
Requires shoes off	Requires upper clothing off
Sex differences ignored	Sex-specific cut-points
Conceptually complex; need calculation or chart for clinical use	Easily understood; easy home monitoring (no calculation needed)

In summary, BMI is a useful population-based indicator of overall adiposity and has some significant advantages for the assessment of excess body weight in individuals. However, WC has some important advantages over BMI, and may be more strongly associated in adults with cardiovascular risk.

1.1.2.3 Ethnicity considerations

Body fat composition varies considerably between ethnic groups (44, 45). Compared to their Caucasian counterparts, the average ratio of body fat to muscle is higher in people of Asian ethnicity and lower in Pacific people (46). Additionally, at the same height and weight as their Caucasian counterparts, people of Asian ethnicity appear to be at greater metabolic and cardiovascular risk (47). The most commonly used BMI and WC cut-points are based on research in predominantly US Caucasian populations, and thus different cut-points have been proposed for non-Caucasian populations. Based on the observed differences in the body fat to muscle ratio in different ethnic groups, lower cut-points for overweight and obesity have been proposed for Asian populations, and higher cut-points for Pacific populations (46).

A WHO expert consultation has addressed the debate about recommended BMI cut-points for determining overweight and obesity in Asian populations, and concluded that available data did not indicate clear BMI cut-points for all Asian populations (48). The cut-point for moderate risk is considered to vary between 22 and 25 kg/m^2 in different Asian populations; for high risk it varies between 26 and 31 kg/m^2. Due to the range of cut-points postulated for different Asian populations, the consultation made no attempt to redefine cut-points separately for these different groups (48). They agreed that the WHO BMI cut-off points should be retained as international classifications for all ethnic groups, and identified further potential public health action points along the continuum of BMI. The consultation also proposed methods by which countries could make decisions about the definitions of increased risk for their own populations (48).

While the use of ethnic-specific cut-points remains controversial, cut-points used should be guided by the objectives of the analysis (48).

1.2 Consequences of obesity

1.2.1 Health

1.2.1.1 Physical morbidity

Obesity has been associated with a range of adverse health conditions (15-19, 49). There is evidence of a causal relationship between obesity and many specific conditions[1], including:

- Cardiovascular: ischaemic heart disease, ischaemic stroke, congestive heart failure, ventricular hypertrophy and associated arrythmias and dizziness, cor pulmonale, varicose veins, pulmonary embolism (50-52);

[1] However it has not been clearly established to what extent the array of health problems associated with obesity are caused directly by obesity itself. There is further explanation of the complexity of the association between obesity and morbidity/mortality in Chapter 1.2.1.

- Endocrine: type 2 diabetes mellitus, polycystic ovarian syndrome (53), menstrual disorders, infertility (15, 54, 55);
- Gastrointestinal: gastroesophageal reflux disease, fatty liver disease, non-alcoholic steatohepatitis, colelithiasis (gallstones), hernia (56-58);
- Renal & genitourinary: urinary incontinence, glomerulopathy, hypogonadism (male), stillbirth (59-61);
- Integument[2]: stretch marks, acanthosis nigricans, lymphedema, cellulites, carbuncles, intertrigo (62);
- Musculoskeletal: hyperuricemia (predisposes to gout), immobility, osteoarthritis, lower back pain (63-65);
- Neurological: meralgia paresthetica, headache, carpal tunnel syndrome, dementia (66-69);
- Respiratory: dyspnea, obstructive sleep apnoea, Hypoventilation syndrome, Pickwickian syndrome, asthma (70);
- Psychological: depression, low self esteem, body dysmorphic disorder, social stigmatisation (71-73);
- Cancers esophagus, colorectal, breast, liver, gallbladder, pancreas, kidney (74).

The parallel increase in incidence of type 2 diabetes mellitus and obesity has been particularly striking (15, 75). The contribution of obesity to diabetes, the population attributable fraction, is high, approximately 89% in a recent (2005) Swiss study – the highest association of obesity with a health condition (76). Type 2 diabetes was formerly considered a disease of adults and referred to as adult-onset diabetes, however in high-income countries it is an increasingly common diagnosis in children (77, 78). Type 2

[2] Skin and appendages

diabetes is itself a risk factor for other complications – notably cardiovascular and renal disease, peripheral neuropathy, vision loss and pregnancy complications (79).

Table 4 presents the relative risks (RRs) for a selection of the conditions discussed above.

Table 4. Approximate relative risks of selected (as discussed in 2002 by the International Obesity TaskForce) conditions associated with obesity in high-income countries

Relative Risk*	Condition
Slightly increased risk (RR 1-2)	Cancer (breast, colon), reproductive hormone abnormalities, polycystic ovarian syndrome, impaired fertility, lower back pain, increased anaesthetic risk, foetal defects arising from maternal obesity
Moderately increased risk (RR 2-3)	Coronary heart disease, osteoarthritis, hyperuricaemia, gout
Greatly increased risk (RR>3)	Diabetes, gall bladder disease, hypertension**,** dyslipidaemia, insulin resistance, breathlessness, sleep apneoa

* RRs indicate the risk of the condition for an obese person compared to that of a non-obese person. For example, an obese person is 2-3 times more likely to suffer from coronary heart disease than a non-obese person.

Source: Adapted from Neilson et al., 2005 (76)

As previously discussed, the distribution of body fat appears to influence the risk of obesity-related disease. Abdominal obesity is more strongly correlated with insulin resistance, type 2 diabetes and cardiovascular disease than is peripheral distribution of body fat (9-14). The risk profile by which this occurs has been described as the 'metabolic syndrome', a cluster of diseases and risk factors centred on insulin resistance that greatly increase the risk of type 2 diabetes and cardiovascular disease (80). The metabolic syndrome is also associated with other disorders (for example, non-alcoholic steatohepatitis (81), obstructive sleep apnoea (82) and the polycystic ovarian syndrome (83)) and in Australia, affects between one and three in every ten adults, depending on the definition used (84).

Some estimates of the morbidity and mortality attributable to obesity have been undertaken despite complex methodological issues. The Australian burden of disease study (85) found that the burden of disease attributable to overweight and obesity was

40% of cardiovascular diseases and hypertension combined, 28% of diabetes and 14% of cancers. Globally, the estimated disease burden attributable to higher-than-optimal BMI is 21% of ischaemic heart disease (IHD), 23% of ischaemic stroke, 58% of type 2 diabetes, 12% of colon cancer and 8% of post-menopausal breast cancer (86).

1.2.1.2 Psychosocial morbidity

The effects of obesity on psychosocial morbidity are unclear (71, 87). A systematic review by Atlantis et al. (2008) found some support for a weak association between obesity and depression in prospective cohort studies (72). In the same review, cross-sectional studies from the US consistently showed associations between obesity and prevalence of depression for women but not men. The review reported that no association has been found in cross-sectional studies of populations of other countries.

There are a number of means by which obesity may increase the risk of depression and other psychological morbidity. These pathways include:

(1) dysregulation of the hypothalamic-pituitary-adrenocorticol system (72, 88);

(2) increased risk of co-morbidities of obesity, many of which are risk factors for depression (89);

(3) low levels of physical activity, often associated with obesity, affecting mental health (90).

Psychosocial factors among obese individuals may also be involved in the development of depression (72). The institutional and interpersonal mistreatment of obese people has been well documented (73, 91); discrimination that in itself predisposes to poor mental health (92, 93). Internalisation of negative weight-based stereotypes and poor self-image have been found to be associated with an increased risk of depression in obese individuals (72). In fact, psychosocial factors may increase the risk of depression regardless of weight status. A US cross-sectional study found that perception of overweight, rather than overweight or obesity themselves, significantly increased the odds of medium and high psychological distress for both men and women (72) (Atlantis 2007).

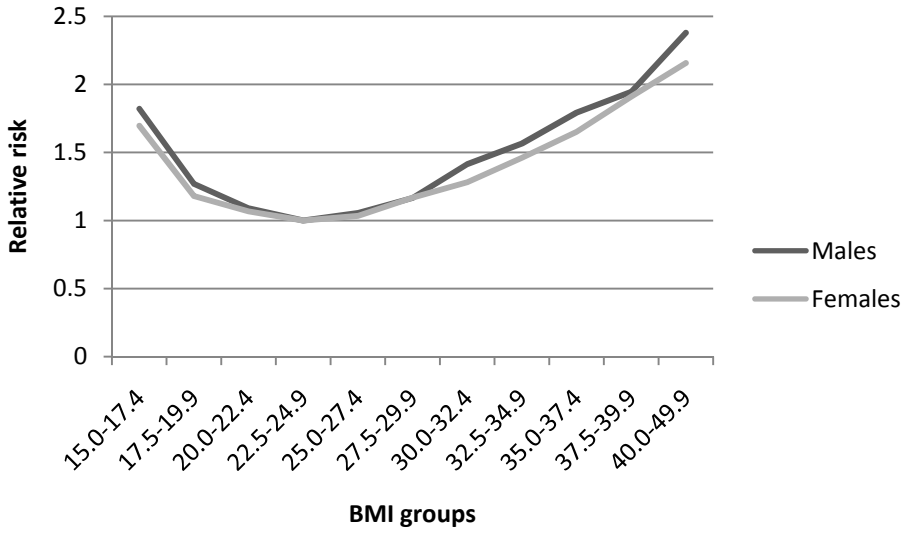
Studies have reported substantial population sub-group differences in such associations. For example, there is a generally stronger association between obesity and poor mental health in women than men (94). The social patterning of the stigmatisation of obesity – the targeting of such stigmatisation more at women than men, and the young rather than the old, for example – may explain the different associations between obesity and mental health by population sub-group. Differing socio-cultural norms by such factors as ethnicity, socio-economic status and gender also contribute to differences in such associations.

1.2.1 Mortality

The association between BMI and all-cause mortality was recently assessed by a collaborative meta-analysis of almost 900,000 participants from 57 prospective studies in multiple countries (30). In both sexes, mortality was lowest at a BMI of between 22.5 and 25.0 kg/m^2 – or upper normal-weight. Above this range, each 5 kg/m^2 of higher BMI was associated with an approximate 30% higher overall mortality. At a BMI of 30-35 kg/m^2, median survival was reduced by 2-4 years; at 40-45 kg/m^2 it was reduced by 8-10 years. The excess mortality below 22.5 kg/m^2 is not fully explained, but appears to be due mainly to respiratory causes. The increased mortality in people with a BMI below 22.5 kg/m^2 persisted after removing people who died in the first ten years of follow-up and after limiting the analysis to non-smokers (30, 95).

Figure 1 illustrates the J-shaped relationship between BMI and all-cause mortality, as assessed by the Prospective Studies Collaboration (PSC) (30).

Figure 1. Relationship between body mass index and all-cause mortality, as assessed by the Prospective Studies Collaboration (2009).



Source: Adapted from Prospective Studies Collaboration (2009) (30)

The recent study described above advances the debate regarding the effect of BMI levels on the risk of death (96, 97). However while severe obesity is generally associated with adverse health conditions and higher mortality, there is considerable evidence that survival from acute events such as myocardial infarction (98), heart failure (99) and dialysis for renal failure (100) is improved by mild to moderate obesity. This has been described as the 'obesity paradox'. Thus while obesity may increase the risk of developing ischaemic heart disease, by the time an acute event occurs, mild or moderate obesity may in fact be protective.

Explanations for the complexity of the associations found between obesity and morbidity/mortality may include:

- The arbitrary nature of weight categories. Health risks associated with obesity are graded and continuous, and assessment of risk within categories may be inaccurate depending on the distribution of the sample population within each category.
- 'Reverse causation', whereby some mortality in 'normal' or 'underweight' categories may be due to weight loss from terminal health conditions, creating the appearance of higher mortality among those with lower weights. The problem is

exacerbated by conditions that cause weight loss but remain undiagnosed for months or years (101).

- Confounding due to smoking. Assessments of the relationship between body weight and morbidity/mortality should be adjusted or stratified by smoking status (101)
- Improvements in public health and medical treatment of obesity-related conditions such as hypertension may be driving a reported weakening of the relationship between obesity and morbidity/mortality over time (102).
- Weight in old age, when most deaths occur, not reflecting earlier weight. For example, Stranberg et al. (2009) have reported that overweight and high-CVD risk in midlife with subsequent weight decrease predict the worst prognosis in later life (103).
- The possibility that body weight is not a significant risk factor for mortality. Despite the considerable body of evidence linking obesity and morbidity/mortality, a causal relationship has not been proven (104). Body weight may more simply represent a marker of lifestyle – in particular diet and exercise level – and genes. In addition to explaining the complexity of the associations found between obesity and morbidity/mortality, it would also explain differing associations between body weight and health outcomes between populations, defined by ethnicity, (sub)culture and time.
- Confounding by duration of obesity (105, 106).

1.2.3 Economic

Significant healthcare costs are associated with treating obesity and associated conditions (107), and many studies and governments' costings have linked obesity to higher annual medical expenditure (107-112).

Obesity reportedly accounts for 6-9% of total healthcare expenditure in most developed countries (113, 114). In the US, obesity in 2007 accounted for an estimated 6% of the healthcare budget, a figure exceeding US$100 billion (115). In Australia, an Access Economics Report has estimated the cost of obesity at A$58 billion. The total cost of obesity includes A$8.3 billion in financial costs (including lost productivity costs of A$3.6

billion, health care costs of A$2.0 billion and carer costs of A$1.9 billion) and A$49.9 billion in the value of lost wellbeing, which accounts for the years of healthy life lost through disability and/or premature death (116).

1.3 Prevalence and trends in obesity

1.3.1 International trends

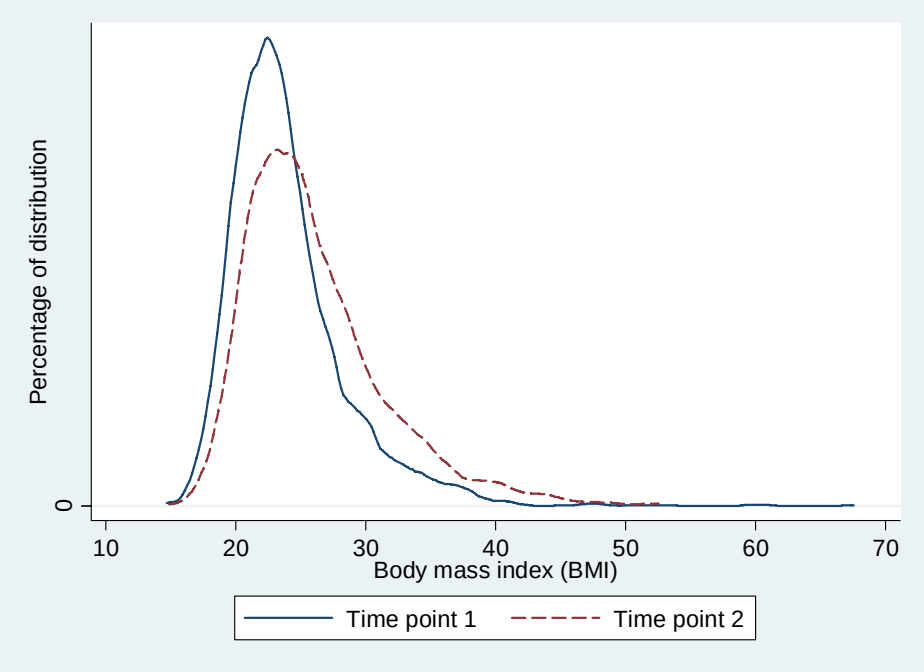
Over recent decades the prevalence of obesity has increased markedly in most high-income countries, and more recently in low-income countries although generally to a lesser extent (117-119). In England, obesity in adults tripled in 22 years from 6% of men and 8% of women in 1980 to 23% of men and 25% of women in 2002 (120). In China, the prevalence of overweight and obesity increased in all gender and age groups in all geographic areas between 1992 and 2002. The increase in overweight and obesity combined was from 15% to 22% based on WHO BMI cut-points; or from 20% to 30% using Chinese-ethnicity BMI cut-points (121). The prevalence of obesity in preschool children in urban areas of China increased from 2% in 1989 to 13% in 1997 (122). The prevalence of obesity in adults varies – from 2-3% in an increasingly small number of Asian countries to 40-70% in the Gulf States and >90% in some Pacific nations such as American Samoa in 2004 (86, 114, 123). In the US, an estimated 32% of the adult population (approximately 60 million people) were obese in 2004 and 66% were overweight or obese (124). However in most high and low-income countries alike – the prevalence of obesity in adults now exceeds 15%, which is the WHO threshold for defining the need for intervention in nutrition-related epidemics (114). Obesity can also co-exist with micronutrient malnutrition, particularly in low-income countries, where the diets of people living in poverty often contain adequate kilocalories to meet or exceed energy requirements but lack the dietary quality needed to promote optimal health (125).

1.3.2 Australian trends

In Australia, the age-standardised prevalence of obesity in adults more than doubled between 1980 and 2000, from 7% to 18% (using the same age and geographical restrictors) (3). In 2000 based on BMI, 21% of the population were considered obese, and a further 39% overweight. Based on WC, 26% were considered obese and 31% overweight. By either measure, approximately 60% of the adult Australian population were overweight or obese (3).

Changes over time in BMI distributions in Australia have not yet been examined. However studies from the United States and New Zealand suggest that the shift towards excess body weight is not uniform across different percentiles of body weight. Instead, examination of the changing BMI distribution suggests that most of the BMI increase is occurring at the higher percentiles of BMI; i.e., skewness is increasing (46, 126, 127). Figure 2 is a schematic illustration of increased *right-skew* in the distribution of BMI at a particular time point "Time point 2" compared with an earlier time point "Time point 1".

Figure 2. Illustration of an increase in right-skew over time in a distribution of body mass index



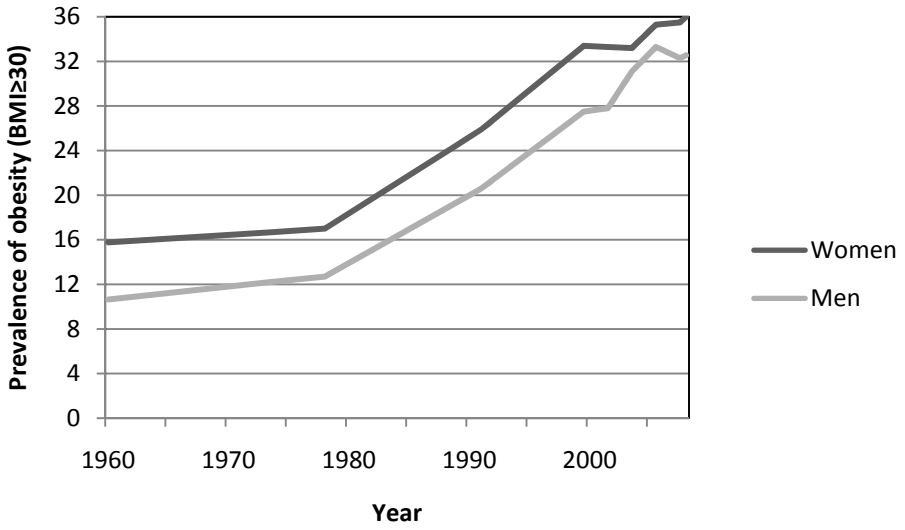
In the United States and New Zealand, it appears that much of the weight gain has involved already-obese people becoming even more obese. In New Zealand, the consequence has been an increase in the number of adults with extreme obesity, and a proportion of overweight people moving into the obese category and being replaced by a similar number of people from the normal weight category. Thus, between 1977 and 2003 the prevalence of obesity more than doubled, while the prevalence of overweight remained stable and the prevalence of normal-weight decreased.[3] This finding suggests a concept called 'differential susceptibility' – individuals exposed to an increasingly 'obesogenic' environment are differentially susceptible to weight gain (46).

The prevalence of obesity in New Zealand increased rapidly in the late 1980s, but slowed between 2002-03 and 2006-07 in both men and women (128).

Such a slowing is also evident in the United States (129) (see Figures 3 and 4). The prevalence of obesity was relatively stable in the United States between 1960 and 1980, but increased markedly in the 1980s and 1990s (based on data from the NHANES surveys and the National Health Examination Survey). Recent data suggest a period of relative stability. For women, the prevalence of obesity did not change statistically significantly in the decade to 2008. For men, there was a statistically significant increase over the same period, but no significant change between 2003-04 and 2007-08.

[3] The slight increase in height over this period (over the past 30 years, 1.3cm per decade in males and 0.8cm per decade in females) explains little of the BMI distributional shift.

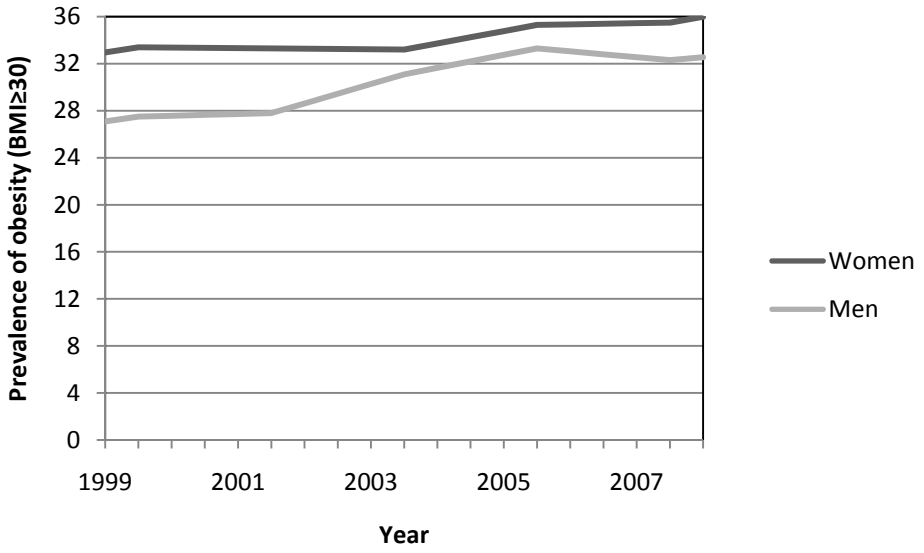
Figure 3. Approximation of trends in the age-adjusted prevalence of obesity (BMI≥30) in US men and women between 1960 and 2008



Note: The estimates prior to 1999-2000 are for ages 20-74 years, and from 1999-2000 are for ages 20+ years. Data were calculated for each half-year by extrapolating from NHANES surveys and the National Health Examination Survey conducted in 1960-62, 1971-74, 1976-80, 1988-94, 1999-2000, 2001-02, 2003-04, 2005-06 and 2007-08. Survey estimates were taken as relating to the mid-point of the survey period.

Source: Adapted from Flegal et al. (2002 and 2010) (129, 130).

Figure 4. Approximation of trends in the age-adjusted prevalence of obesity (BMI≥30) in US men and women between 1999 and 2008



Note: The estimates are for ages 20+ years. Data were calculated for each half-year by extrapolating from NHANES surveys conducted in 1999-2000, 2001-02, 2003-04, 2005-06 and 2007-08. Survey estimates were taken as relating to the mid-point of the survey period.

Source: Adapted from Flegal et al. (2010) (129, 130).

1.4 Determinants of obesity

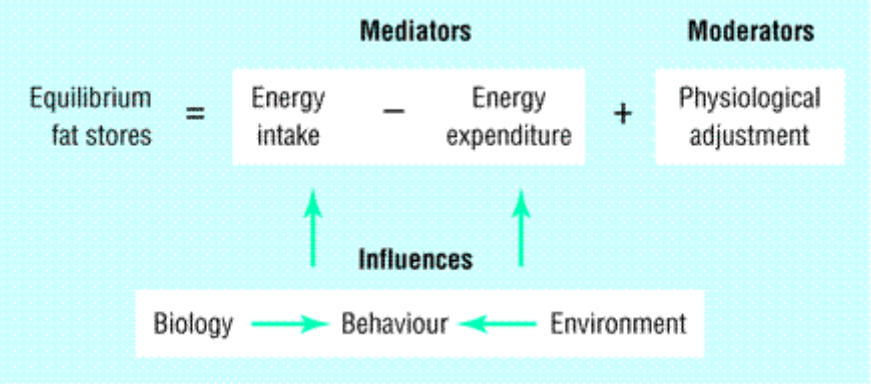
Excess body weight is caused by a chronic excess of energy intake (determined by food and beverage consumption) over energy expenditure (the most variable component of which is physical activity) (6, 7). But the regulation of this 'energy equation' is the result of complex social, cultural, environmental, genetic and hormonal influences, and their interactions (8). In short, body weight is a result of the interaction between the individual (his/her physiology) and the environment (131).

Some people are more genetically susceptible to weight gain than others. Rare genetic abnormalities that predispose to obesity have been identified (Prader-Willi syndrome and leptin receptor mutations (132, 133), for example) and there are also more common conditions associated with a modest increased risk of excess body weight, such as the polycystic ovarian syndrome (134). Furthermore, the baby-boom generation are approaching late middle-age, a period in the life course associated with a high prevalence

of obesity in high-income countries. However, the rapid increase in the prevalence of overweight and obesity during recent decades has occurred too quickly and is too widespread across age groups to be explained by genetic or demographic changes alone (28). Instead, it is likely to be the result of exposure to an 'obesogenic' environment – one that promotes sedentary lifestyles and the overconsumption of energy-dense foods and beverages (135, 136). Epigenetic factors may also be important in terms of environmentally-induced modulation of gene expression over individual lifetimes. For example, some studies are revealing environmental influences at critical stages of development, such as during foetal development and early childhood, that may predispose to obesity in adulthood (137-139).

Egger et al. (1997) have proposed an 'ecological model' (Figure 5) that outlines the three main influences on the body's body fat balance – biological, behavioural and environmental (135).

Figure 5. An ecological model for understanding body weight



Source: Egger et al. (1997) (135).

The environment can affect health and well-being in a number of ways: physiological, emotional, social, spiritual and intellectual. Environments are complex in nature and people occupy numerous environments concurrently and over time, and such environments can be broadly described as supportive (or not) of healthy eating patterns and physical activity levels (140). Modern environments, with readily-available tasty, attractively marketed convenience food high in refined sugar and saturated fat, and labour-saving technological innovation and time-pressured living that leads to reduced physical

activity (141), have sometimes been described as 'toxic' or 'obesogenic' (136). Thus our behaviour has changed in response to changes in the environments in which we live. It has been suggested that rather than people's behaviour, it is the environment that has become abnormal (142).

Egger et al. (1997) described the environment in terms of 'microenvironments' (eg. schools, workplaces, homes, neighbourhoods) and 'macroenvironments' (education and health system, government policy, societal attitudes and beliefs) (135). Macroenvironments directly influence microenvironments. These different types and levels of environment interact with each other and influence behaviour both directly and indirectly (140).

The complex individual/environment interaction suggests that the determinants of body weight (and their relative importance) are likely to differ between individuals, but also between different populations and population sub-groups. Demographic factors such as age, sex, ethnicity and socio-economic status are all important influences on physical activity levels and eating behaviours. Thus, those factors most predictive of weight gain in women are likely to be different from those in men due to the different environmental, social and physiological influences on their lives. The behaviours that contribute to energy expenditure among young children may be considerably different to those among older children or adults. Other studies have examined ethnic differences, relating these to cultural perceptions of body size and preferred body shape (143-145).

Thus, demographic differences in the prevalence of obesity, and in the relationship between obesity and socio-economic status should be unsurprising (79). In countries with indigenous populations, such as Australia, the indigenous people have a higher prevalence of obesity than non-indigenous people (146, 147) and in developed countries, socio-economic status is strongly inversely related to body weight and risk of obesity, especially among women (148-150). However there are within-country ethnic differences in this relationship (151).

Suggested drivers of the obesity epidemic include:

- Consumption of energy-dense food and beverages (152, 153);
- A high glycaemic diet (i.e., a diet that confers high postprandial blood sugar) (154, 155);
- Lack of activity (sedentary lifestyle) (156, 157);
- Lower relative cost of less healthy food (158, 159);

- Media and commercial advertising of unhealthy food (160-162);
- Fast food restaurants and 'eating out' (152, 163);
- Increased portion sizes (164);
- Weight cycling, caused by repeated attempts to lose weight by dieting (165);
- Eating disorders (e.g. binge eating) (166);
- The relatively low price of petrol (167);
- Television watching (and computer/video games) (168);
- Two-income households (169);
- Stress / Lack of relaxation (170, 171);
- Insufficient sleep (172, 173);
- Smoking cessation (174);
- Excess alcohol consumption (175);
- Changes in the built environment (176);
- Urban sprawl (and inaccessibility to public transport) (177, 178);
- Perception of unsafe environment (for example, leading to children not being allowed to walk to school) (179).

Environmental chemical contamination is another candidate as it is suggested that some chemicals, endocrine disruptors, may cause weight gain through their interference with the human endocrine system (180). However the evidence to support this is sparse, and further research is recommended.

In terms of the relationship between obesity, low socio-economic status, and the suggested drivers listed above, more disadvantaged environments are often characterised by factors such as poorer quality choices of foods and increased access to fast food outlets, less integrated public infrastructure and public transport in suburbs more distant from the central city. Such factors influence lifestyle choices. Furthermore, our behaviours are strongly influenced by the behaviours of those around us – behaviours that include participation in regular physical activity, levels of food and alcohol consumption and attitudes towards smoking. Thus in more disadvantaged areas, the behaviours of those around us may be less conducive to living healthily (150).

1.5 Responding to obesity

1.5.1 Societal response to obesity

There is considerable public debate regarding the contentious issue of the extent to which government should be involved in obesity prevention.

Civil libertarians and other advocates of 'personal responsibility' believe that authorities are unjustified in curtailing the autonomy of what they consider the self-determining individual, and resist regulatory attempts to intervene in citizen's private lives. They consider it an individual's 'right' to determine his or her own behaviour, rather than have the government impose regulation and control. Such advocates believe body weight to be an issue of personal responsibility (in the case of childhood obesity, the position is more divided but for many personal responsibility means parental responsibility), and resist regulatory or other intervention to create less obesogenic environments. They favour a focus on individual responsibility for lifestyle behaviours and any attendant consequences (181).

'Public interest' advocates argue that weight gain is a normal physiological response to what has become a pathological environment in which we live – an obesogenic environment with vast amounts of cheap, tasty, energy-dense food and sedentary leisure activities (135), even the most motivated individuals must sustain considerable effort to maintain healthy bodyweights, and often fail (182). These advocates believe the marketplace, as currently structured, has failed to deliver the best outcomes for people's health, and support regulations (on similar public health grounds as to the restrictions applied to tobacco products (183)), believing governmental intervention on the issue justifiable (184, 185). Such advocates take issue with those who place the rights of the individual over the rights of the population, particularly in regard to the more vulnerable and less assertive population sub-groups, such as children (182).

Historically, the 'personal responsibility' advocates have dominated in terms of the response to obesity, in that an individual-based approach has been adopted. Where a response has been enacted, the focus has been on social marketing and other 'health education' approaches (115). Such approaches endeavour to educate individuals on the

importance of good nutrition and physical activity, and to lose or maintain weight by generating a negative energy balance, achieved by decreasing energy intake through dietary means and/or increasing energy expenditure via an increase in physical activity. The implication of such an approach, however, is that a vastly growing proportion of the population are 'choosing' to gain weight (by eating too much and exercising too little). However, the existence of the strong societal pressure for lean bodies, demonstrated by the existence of the lucrative diet industry and health promotion campaigns reinforcing the risk of obesity, suggests that this is not the case (186-189). Individual choices are necessarily part of the total explanatory picture for adults, but the increasing prevalence of overweight and obesity suggests that the individual perspective is alone inadequate. The behaviours that influence body weight cannot be construed simply as those of individual choice when this choice is so greatly influenced by the social and physical environments in which we live (184).

Obesity is increasingly recognised as a manifestation of inappropriate societal structures shaping the way people live (115). It appears to be the end result of a large number of environmentally-influenced behaviours. Furthermore, it is a condition with considerable health, social and economic consequences (190, 191), its distribution within populations is inequitable, and there is a dearth of evidence suggesting substantive effectiveness of interventions, surgery aside (192-194). Thus, government intervention is considered, increasingly, an acceptable strategy for addressing the increasing societal burden of obesity.

Consequently, obesity now tops policy and programme agendas in many countries, with prevention of childhood obesity receiving particular attention (113). There is widespread agreement that action is urgently required, and that it should be comprehensive, sustained, and evidence based (113). The results of various population programmes encouraging weight loss and maintenance have been published, however the only substantially positive ones have been in children (195, 196), in whom behaviour can be controlled more easily. There is a lack of evidence of successful interventions in adults (197), and few modelling analyses have been undertaken of the relative effectiveness of the different approaches to obesity reduction. One exception is the ACE-Obesity (Accessing cost-effectiveness of obesity interventions in children and adolescents) study, which found a reduction in TV advertising of energy-dense foods and beverages to children to be the most cost-effective strategy assessed (198).

Considering the need to discriminate between potential obesity interventions, Swinburn et al. (2005) have suggested a method of categorising the 'promise' of an intervention (Table 5) (113).

Table 5. 'Promise' table for categorising potential interventions

Certainty of effectiveness*	Potential population impactβ		
	Low	**Moderate**	**High**
Quite high	Promising	Very promising	Most promising
Medium	Less promising	Promising	Very promising
Quite low	Least promising	Less promising	Promising

* The certainty of effectiveness is judged by the quality of the evidence, the strength of the programme logic, and the sensitivity and uncertainty parameters in the modelling of the population impact.

β Potential population impact takes into account efficacy (impact under ideal conditions), reach and uptake and it can be measured in a number of ways such as effectiveness, cost-effectiveness, or cost-utility.

Source: Swinburn et al. (2005) (113).

Interventions are commonly divided into two key types, as described below.

1.5.2 Obesity-reduction strategy types

Obesity-reduction strategies can be broadly categorised (199) as:

- 'High-risk' or 'individually-based' approaches – strategies that address bodyweight (weight loss or management) in already obese individuals and possibly also overweight, depending on the definition. Examples include:
    - Lifestyle or drug-based interventions for weight loss in obese individuals;
    - Surgical intervention to induce weight loss in severely obese individuals.
- 'Population' approaches – strategies that address bodyweight (weight loss or maintenance) in the whole population. Examples include:
    - Social marketing campaigns;

- The enactment of policy and environmental strategies to assist people adopt healthy behaviours.

'Targeted' approaches address bodyweight in high-risk demographic groups (sex, age and/or ethnic groups, for example) and can fit into either of the above approaches.

The high-risk approach seeks to protect those individuals at most risk of obesity-related conditions such as through the implementation of weight management programmes for at-risk individuals, by health practitioners. Each individual targeted has much to gain from the success of the intervention, and because of the possible gain for the individual, motivation is higher for those involved (provider and patient) (199).

However, there is contention regarding the utility of the high-risk approach (182). Most high-risk interventions for obesity (surgery aside) result in only small amounts of weight loss (often 3-5 kgs) (200), and the maintenance of weight lost is poor (201). Furthermore, while the high-risk approach is appropriate for people already obese, it does not affect the distribution of excess body weight in the whole community (199). It also relies on candidates for this approach contacting their general practitioner or other healthcare provider.

Population approaches address the cause of increasing body weight in the entire population (199) and can be considered a preventive measure. By preventing weight gain and its attendant health consequences, population approaches are a potentially cheaper method of addressing the obesity burden than treating those who reach high levels of excess body weight. In addition to the potential for greater cost-effectiveness, the changes that could be brought about through a population approach that results in a healthier lifestyle are also potentially larger and more sustainable than changes brought about by pharmacological or surgical interventions in just the high-risk individuals. The population-based approach also has the advantages of considering body weight from a continuous perspective, the perspective from which it relates to health outcomes, rather than focusing on arbitrarily-defined categories of weight, such as 'normal-weight', 'overweight' and 'obese' (202).

Reducing body weight by a small amount in the population, irrespective of whether obese or not, should have a greater impact on population health than targeting already-obese people. This is because most of the morbidity/mortality burden attributable to excess body

weight at a population level occurs not amongst the small number of obese or morbidly obese people, but amongst the much larger numbers of people with lesser degrees of overweight or obesity (203). Shifting the whole population distribution of weight should be more effective than high-risk strategies to reduce the obesity burden. This is the key advantage of the population approach to prevention, as first explained by Geoffrey Rose; leading to one of the fundamental axioms in disease prevention: 'A large number of people exposed to a small risk may generate many more cases than a small number exposed to high risk' (204).

The main disadvantages of the population approach are the potential for harm to those already in the lowest percentiles of body weight by lowering their weight even further (205), the difficulty of garnering support for an approach that, to quote the prevention paradox[4], 'brings large benefits to the community but offers little to each participating individual' (206), and the controversy regarding the health impact of overweight and mild obesity. There is also little evidence for the effectiveness of community-wide interventions targeting obesity (197, 207). Furthermore the poorest response to population approaches with an education focus, such as social marketing campaigns, is generally among lower socio-economic groups and those from minority or indigenous ethnic groups – generally those groups in whom the prevalence of obesity is highest.

[4] The 'prevention paradox', first described in 1981 by Geoffrey Rose, is a problem encountered by interventions to improve population health. Many interventions to improve population health confer relatively small benefits to any participating individual. Therefore for one person to benefit, many people have to change their behaviour, even though they receive no benefit from the change (206).

Table 6. Differences between high-risk and population approaches to obesity reduction

	High risk (or individual) approach	Population approach
Key measures	Body weight, BMI, WC	Prevalence of overweight and obesity, mean BMI, mean WC
Key aetiology questions	Why is this person obese (or gaining weight)?	Why does this population have a high (or rising) prevalence of obesity?
Main aetiological mechanisms	Genetic, metabolic, hormonal, behavioural	Environment, cultural, behavioural
Main management questions	Patient education, behavioural modification, drugs, surgery	Public education, improving food, physical activity environments, policy, planning
Volume of information on aetiology & management	Vast	Minimal
Driving forces for research & action	Immediate and powerful	Distant and weak
Potential for long term benefit to individuals	Modest	Modest
Potential for long-term benefit to populations	Modest	Significant
Sustainability	Poor	High
Cost	High	Potentially low

Source: Adapted from Swinburn et al. (2002) (208)

Few analyses have been conducted regarding the effects of the high-risk and population approaches on:

- The parameters of the body weight distribution, such as the mean, and prevalence of underweight, overweight and obesity;
- Obesity-related morbidity and mortality;
- Obesity-related healthcare costs.

Such research would make a substantial contribution to evidence regarding the relative utility of these approaches to reducing the obesity burden.

1.6 Thesis Outline

This thesis has been developed as an examination of population-wide trends in obesity, their causes and implications. Much is unclear about the population trends in body weight that are driving the increase in obesity, particularly in countries such as Australia, where surveillance of obesity has been limited to describing changes in the prevalence of overweight and obesity. Without understanding the detail of the increase in obesity prevalence we cannot accurately measure its burden, fully understand its drivers or ensure that obesity-reduction responses are targeted appropriately.

A broader goal of this thesis was to use the best available data, and data from a variety of sources, to provide a clearer understanding of population-wide trends in obesity. This broader goal included the characterisation of the prevalence of and trends in obesity in countries at both early and late stages in the obesity 'epidemic'.

Specifically, this thesis will describe the population trends in BMI in Australia, compare population trends in BMI and WC in the United States, and describe the prevalence of obesity in Hanoi, Vietnam. It will also examine the determinants of weight gain, the relative benefits of high-risk and population obesity-reduction strategies, and the future burden of obesity based on current incident probabilities. There is limited information available examining the determinants of a gain in WC, potentially with a stronger association to metabolic and cardiovascular risk than BMI. Thus, an analysis was undertaken to determine a set of behavioural risk factors with which to define an 'at risk' state for the onset of abdominal weight gain. The increases in the prevalence of obesity and trends in incidence driving this suggest an increasingly heavy burden of obesity over coming years. Thus, a projection of obesity in Australia based on current incidence probabilities of transitioning between normal-weight, overweight and obesity was undertaken. Furthermore, the relative utility of high-risk and population obesity-reduction strategies was examined in light of the findings from the population trends in body weight. Each of the primary topics of research addressed is listed and expanded upon below, and forms the basis for the research described in Chapter 3.

1.6.1 Trends in obesity

A deeper understanding of population-wide obesity trends is needed to develop effective strategies. Most information presented on trends in obesity has been focused on categories of BMI. Access to AusDiab and NHANES data provided the ability to look at these trends in greater detail.

1.6.1.1 Trends in body mass index

Overweight and obesity, commonly described according to BMI categories, are increasing worldwide (209). But much remains unknown about the nature of recent obesity trends. Specifically, it is unclear in Australia to what extent the current prevalence of obesity is a result of increases across the whole spectrum of BMI or limited mostly to the upper BMI ranges. It has been shown in the United States and New Zealand that increases in the prevalence of obesity have been accompanied by an increase in right-skew of the BMI distributions (46, 126, 127). However in Australia, the surveillance of obesity has been limited to describing changes in the prevalence of overweight and obesity. There has been no analysis of trends in the continuous distribution of BMI to date. This analysis examined BMI trends in urban Australian adults between 1980 and 2000, focusing on trends in the continuous distribution of BMI.

1.6.1.2 Differential trends in different measures of excess body weight

The nature of excess body weight may be changing over time to one of greater central adiposity. Our analyses in Australia suggested that WC has been increasing at a greater rate than BMI, and soon after beginning the analysis, Elobeid et al. (2007) reported that WC has been increasing at a greater rate than BMI in their US trends analyses (210). However it remained unclear if these differences are consistent across different population sub-groups and all body weights. WC measures the central distribution of excess body fat and appears to be more strongly correlated with metabolic and cardiovascular risk than BMI (9, 10). Thus, whether the nature of obesity is changing through a greater tendency to abdominal obesity, and in which population sub-groups, may have important implications

for the burden of obesity-related disease. This analysis examined the degree to which WC is increasing beyond that expected from changes in BMI alone, and examined the public health implications of the findings.

1.6.2 Obesity in a low-income country: the case of Vietnam

Although the population of Vietnam historically has a low overweight/obesity prevalence, this prevalence reportedly doubled in adults between 1992 and 2002 (211) (although the prevalences of overweight/obesity in this study were arguably low as they were based on BMI cut-points recommended by the WHO for Western populations rather than values appropriate for Asian populations). Analysis of 2004 data of urban adults in Ho Chi Minh City (HCMC), Vietnam's largest city with a population of approximately seven million, found that almost a third of adults were overweight or obese when using Asian-specific cut-points (212). Such an analysis is also of interest for Vietnam's capital, Hanoi, which has a population of approximately three million and is another large and dynamic city in a country that has undergone rapid economic and social change over the past fifteen years. Determining the prevalence of underweight, overweight and obesity in urban Hanoi and comparing these to values for HCMC will contribute to the evidence regarding body weight in Vietnam. It is also important, however, not only for Vietnam, but for the contribution it makes towards a deeper understanding of the global 'obesity epidemic'. This work determined the prevalence of BMI and WC categories in adults of urban Hanoi in 2004, using Asian and Vietnamese-specific cut-points, as well as commonly-used cut-points arguably more appropriate for Caucasians.

1.6.3 Predictors of increasing waist circumference

Few studies have examined predictors of increasing weight in adults, and we are not aware of any that have examined a range of behavioural or lifestyle factors potentially predictive of WC gain. However WC gain is of particular interest, considering findings presented earlier in this thesis (Chapter 1.6.1.2) and by others (210) that the nature of excess body weight may be changing over time to one of greater central adiposity, rather than a more peripheral distribution of body fat. For the most part, it is unknown whether factors associated with higher BMI and WC cross-sectionally are also driving increases in WC over

time. A set of behavioural factors with which to define an 'at risk' state for the onset of abdominal weight gain would be useful, as the development of effective prevention strategies requires better understanding of the causal role of lifestyle and health behaviours in regard to increasing WC over time. This analysis investigated relationships between lifestyle behaviours and an increase ≥5% over baseline WC to determine factors predictive of increasing WC over a five-year period in a cohort of Australian adults.

1.6.4 Predicting future obesity

Several projections of the prevalence of obesity have been conducted, both country-specific and globally. For example, projections for the United Kingdom suggest that by 2025, 47% of males and 36% of females will be obese (213). However the scientific literature only consists of studies predicting the future burden of obesity by extrapolating into the future past prevalences of obesity or distributions of body weight. This is potentially problematic, considering that the prevalence of obesity at any point in time is a result of decades of differing incidence rates of moving between normal-weight and overweight, and overweight and obese. Some of these rates reflect quite historic environments and lifestyles, and are markedly different to those of more recent times. A more accurate method would be to base future prevalence estimates on the most recent measures of incidence available. This analysis estimated the future burden of obesity in Australia using the most recent measures of incidence. The lifetime risk of obesity in a cohort of people aged 25-29 years in 2000 was also estimated.

1.6.5 Obesity-reduction strategies

In 1985 Geoffrey Rose demonstrated that preventive strategies enacting population-wide shifts in the distribution of risk factors would most affect the population burden of disease (204). Using plasma cholesterol and blood pressure as examples, he demonstrated that a small left-ward shift in the population distribution could decrease morbidity and mortality more than a large decrease in risk in the high-risk individuals. This approach has become a fundamental tenet of prevention. However obesity appears to provide a more complex situation. The increased risk conferred by cholesterol or blood pressure increases

progressively across the population distribution (at least compared to that conferred by body weight) (214, 215), but the risk conferred by body weight is J-shaped, affecting mainly the upper and lower ends of the weight distribution (30). Thus, a population approach that shifts the body mass index to lower levels may harm those at the lowest end of the distribution. Furthermore, the increasing prevalence of obesity in developed countries, and as we have shown in Australia is a result of an increased skewing of the distribution of body weight rather than a simple uniform shift (46, 126, 127). The unequal shifting of BMI across the population and the J-shaped relationship between body weight and morbidity/mortality suggest that high-risk strategies may be the most appropriate obesity-reduction response. This work analysed a range of theoretical responses to high-risk and population obesity-reduction strategies, and explored the response in terms of their effect on population mean BMI, percentage underweight, overweight and obese, and number of premature deaths prevented.

Chapter 2: Methods

The data sources used in this thesis will be introduced below, followed by an explanation of 'mean-difference' plots – a graphical method used in this thesis to present changes in population skew. The interpretation of mean-difference plots requires some explanation before their presentation in a later analysis (Chapter 3.1.1). This chapter also includes a description of the three types of epidemiological analysis subsequently used in this thesis (Chapter 3).

For each of the analyses presented in this thesis (Chapter 3), a methods section specific to that analysis is included as part of the published paper.

2.1 Data sources

The analyses in this thesis are based on data from nationally representative population-based surveys from Australia and the USA, a population-based survey from northern Vietnam, the Australian National Death Index (NDI), and national mortality data from the Australian Institute of Health and Welfare (AIHW). The surveys and data sources are described briefly below, and in most cases are described in more detail in the subsequent chapter (Chapter 3).

2.1.1 Australian data

The Australian data used are from five cross-sectional surveys: the 1980, 1983 and 1989 National Heart Foundation Risk Factor Prevalence Study (RFPS), the 1995 National Nutrition Survey (NNS) and the 1999-2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab).

National Heart Foundation Risk Factor Prevalence Study

The RFPS was a series of three national population-based surveys conducted by the National Heart Foundation (NHF) of Australia. The surveys, conducted in 1980, 1983 and

1989, were designed to obtain information on biomedical and behavioural risk factors in Australia and to monitor trends (216). Their target population was residents of Australian capital cities (Canberra and Darwin were not included in the 1980 and 1983 surveys (216)) listed on state electoral rolls and aged 25–64 years in the 1980 and 1983 surveys and 20–69 years in the 1989 survey. From initial target samples of 8375, 10 148 and 9328, the 1980, 1983 and 1989 studies involved 5603 (67 %), 7615 (75 %) and 6097 (65 %) respondents, respectively. Participants were administered a questionnaire, and underwent a physical examination and blood test (217).

National Nutrition Survey

The NNS was designed to collect information on nutrient intake, dietary habits and body measurements (216). It was a sub-sample of a larger population survey, the National Health Survey (NHS). The target population was residents of Australian States and Territories aged over 2 years. For the NHS, a nationwide sample of approximately 23 800 households was obtained. Of households selected to participate, 92% responded, with a total of 57 633 persons interviewed. Of those, 22 562 were selected to participate in the NNS. A total of 13 858 persons (61% of those selected) participated in the NNS (218). All participants were interviewed by trained nutritionists and also self-completed a Food Frequency Questionnaire to obtain detailed information of food and beverage consumption, and underwent a physical examination (219).

Australian Diabetes, Obesity and Lifestyle Study

AusDiab was a national population-based survey conducted in 1999/2000 by the International Diabetes Institute. The target population was residents of six states and the Northern Territory (216). It was designed to obtain estimates of the prevalence of diabetes and chronic health conditions, and a number of anthropometric measurements (216). Household questionnaires of the nationwide sample were completed in 67% of the households (n= 11 479) that could be contacted and contained at least one eligible person. A total of 20 347 eligible individuals were interviewed in these 11 479 households. The final survey sample (those attending the biomedical examination) included 11 247 adults (5049 men and 6198 women), representing 55% of those completing the household interview and 37% of those selected (220). The biomedical examination included an oral glucose

tolerance test, standard anthropometric measurements, blood pressure measurements and the administration of questionnaires (220).

National Death Index

The Australian National Death Index (NDI) is a database maintained by the Australian Institute of Health and Welfare (AIHW). It contains records of 2.3 million registered deaths that occurred between 1980 and 2002. The NDI has been found to have 94% sensitivity and 100% specificity for the identification of deaths (221).

In Australia, registration of death has been compulsory since the mid-1850s. Information about a death is recorded on a death certificate and includes the underlying cause of death (the disease or condition leading directly to death) and a list of other conditions contributing to that death. In most cases, this information is usually provided by a medical practitioner; however, it can also be provided by the government pathologist or the State Coroner in cases when the cause of death is unclear. Once completed, the death certificate and accompanying demographic and administrative information is registered with the Registrar of Births, Deaths and Marriages, and then passed to the NDI and the Australian Bureau of Statistics (ABS) for death registration and coding of cause of death, respectively. The ABS codes the cause of death according to the International Classification of Diseases, Version 9 (ICD-9) prior to 1997 and Version 10 (ICD-10) from 1997 (221).

2.1.2 American data

The National Health and Nutrition Examination Studies (NHANES) are nationally representative cross-sectional studies of the United States, conducted by the United States National Center for Health and Statistics. The studies have been designed to assess the health and nutritional status of adults and children in the United States. The surveys used in the analyses presented in Chapters 2.3 and 3.1.2 were conducted during the following time periods: 1988 to 1994 (NHANES III); 2001 to 2002; and 2005 to 2006. Participants in each survey were selected using a multi-stage, stratified cluster sampling design to be representative of the civilian, non-institutionalized United States population. Persons aged 60 and older, African-Americans and Mexican-Americans were oversampled. After being interviewed in their homes, participants were invited for a clinical examination at a mobile examination centre (MEC), where anthropometric data were collected. Only people with

data from the MEC examination were included in analyses. The household interview response rates and MEC response rates in the respective surveys were 86% and 78% in NHANES III (1988-94), 84% and 80% in 2001-02, and 80% and 77% in 2005-06. The sample design and weighting methodology across the three surveys were very similar and the studies included 31 311, 10 477 and 9950 individuals, respectively (222, 223). People who attended the MEC examination tended to be younger, and at least for the first survey, tended to be non-Hispanic Black or Mexican American and of lower socio-economic status (224).

2.1.3 Vietnamese data

The Vietnamese data used are from a population-based survey, the 2004 'Epidemiological survey on heart failure, its main causes and some of its risk factors in northern Vietnam' (225). The aim of this study was to describe the prevalence of heart failure and its risk factors in the northern provinces of Vietnam. Vietnam is divided into 58 provinces and five centrally-controlled municipalities; each province is further divided into districts. Sampling in this study included three randomly-selected districts in each of the four provinces of northern Vietnam: Hanoi (urban), Thai Binh (lowlands), Nghe An (coastal) and Thai Nguyen (midlands). The target population was persons aged 25 and older, from which the target sample was selected using a randomised cluster sampling method, at the individual level. The response rate in Hanoi was 73.9%, which included 1066 Hanoi residents and 3780 residents outside of Hanoi (the overall response rate for all regions combined was 84.1%). Participants were administered a questionnaire, physical examination and blood tests. Weight, height and WC were measured by trained personnel and measured in accordance with WHO guidelines (226).

2.2 Mean-difference plots

This section describes the use and interpretation of Tukey mean-difference plots in the illustration of body weight distributions changes over time, as used in Chapter 3.1.1.

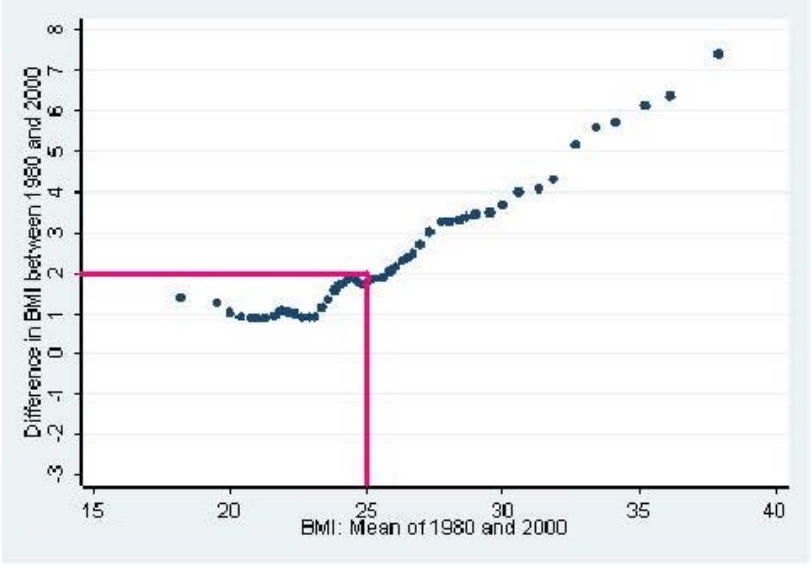
Tukey mean-difference plots – also known as Bland-Altman plots or difference plots – were developed by Bland and Altman for comparing measurement estimates in situations where

the ‘true’ value is unknown (227, 228). However they are also a useful tool for the illustration of changes in the distribution of body weight over time (46, 126). In later papers presented in this thesis (Chapters 3.1.1 and 3.5) changes in the distribution of BMI in Australia between surveys are illustrated using mean–difference plots (46, 126). A description of the plots is included here as their interpretation when presented in a later analysis may not be immediately clear.

In mean-difference plots of BMI, shifts in distributions of BMI are examined by comparing corresponding quantiles (for example, percentiles) from two distributions.

To construct a mean-difference plot of BMI, the difference between mean BMI in corresponding quantiles of different surveys are plotted on the y axis against the means of the same quantiles on the x-axis. For example (see Figure 6), if the mean BMI in the 50th percentile in 1980 was 24.0 kg/m^2 and the mean BMI in the 50th percentile in 1999/2000 was 26.0 kg/m^2, the plot would include a point for that percentile showing the combined mean (25.0) versus the difference (+2.0), demonstrating an increase of 2.0 kg/m2 for the 50th percentile. If the two distributions were the same, the quantiles would be equal and the differences would all be zero. The location of the plotted point on the y axis shows the direction and magnitude of the shift. The shift is assessed by judging differences from the horizontal line representing zero (126).

Figure 6. Example of a mean-difference plot



2.3 Comparing increases in different measures of body weight

A number of studies have compared changes over time in WC with BMI and suggested that the nature of excess body weight may be changing to one of greater abdominal obesity (210, 229-234). However a review of the literature comparing increases in BMI and WC, and the subsequent undertaking of such a comparison using Australian data revealed that some of the studies exploring this question have incorrectly based their conclusions on different proportional increases in the two measures of excess body weight. As we explain, using results presented in Chapter 3.1.2, this method is flawed due to the nature of the relationship between waist circumference and body mass index.

Walls HL, Peeters A, Mannan H, Stevenson C. Methodological problem with comparing increases in different measures of body weight. *Submitted*.

Declaration for Thesis Chapter 2.3

Walls HL, Peeters A, Mannan HR, Stevenson CE. Methodological problem with comparing increases in different measures of body weight. Submitted.

Declaration by candidate

In the case of Chapter 2.3, the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Design and drafting of manuscript.	70%

The following co-authors contributed to the work. Co-authors who are students at Monash University must also indicate the extent of their contribution in percentage terms:

Name	**Nature of contribution**	**Extent of contribution (%) (student co-authors only)**
A Peeters	Design and drafting of manuscript.	N/A
HR Mannan	Design and drafting of manuscript.	N/A
CE Stevenson	Design and drafting of manuscript.	N/A

Candidate signature	**Date**
[redacted]	8 February 2010

Declaration by co-authors

The undersigned hereby certify that:

(1) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors;
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, expect for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (2) granting bodies, (b) the editor or publisher of journals or other publication, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s):

Department of Epidemiology & Preventive Medicine, Monash University

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant].

Co-author signatures	**Date**
[redacted]	7/2/2010
[redacted]	8 February 2010
[redacted]	8 Feb 2010

Title:

Methodological problem with comparing increases in different measures of body weight

Running title:

Comparing increases in different measures of body weight

Authors:

Helen L Walls, Anna Peeters, Haider R Mannan, Christopher E Stevenson

Abstract:

A number of studies have compared proportional increases over time in waist circumference (WC) and body mass index (BMI). However this method is flawed. Here, we explain why comparisons of WC and BMI must take into account the relationship between them. Using data from two cross-sectional US surveys, we calculated the percentage change in the average BMI and the average WC between the two surveys and compared the results with a regression analysis of changes in WC relative to BMI. The crude percentage change in BMI (5.8%) was marginally greater than for WC(5.1%). But these percentages cannot be directly compared, as the relationship between the measures is described by a regression equation with an intercept term that does not equal zero. The coefficient of time from the regression equation will determine whether or not WC is on average larger for a given BMI at the second compared with the first time point.

Main text:

A number of studies have compared changes over time in waist circumference (WC), a measure of abdominal obesity, with body mass index (BMI) and suggested that the nature of excess body weight may be changing to one of greater abdominal obesity [1, 2, 3, 4, 5, 6, 7, 8]. Abdominal obesity appears to be more strongly correlated with metabolic and cardiovascular risk than BMI [9, 10], and thus whether the nature of the obesity epidemic is

changing to one of greater abdominal obesity may have important implications for the burden of obesity-related disease. However some studies exploring this question have based their conclusions on different proportional increases in the two measures of excess body weight [3, 4, 5, 7, 8]. This method is flawed, due to the nature of the relationship between WC and BMI.

We analysed data from two nationally representative cross-sectional surveys of the US population, the National Health and Nutrition Examination Studies (NHANES), conducted by the US National Center for Health and Statistics. The surveys were conducted during the time periods 1988 to 1994 (NHANES III), and 2005 to 2006. In each survey participants were selected using a multistage, stratified cluster sampling design to be representative of the civilian, non-institutionalised US population.

We analysed in two ways the change in WC and BMI between these two periods. First, we calculated the percentage change in the average BMI and the average WC between the two time points. Second, we fitted a regression equation with WC as the dependent (outcome) variable and BMI and time as predictor variables—along with a number of potential confounder variables [2].

In our analysis there was a marginally greater crude percentage change in BMI (5.8%) than WC (5.1%), but using regression analysis we demonstrated that increases in WC were in fact greater than expected based on increases in BMI [2]. Differences in the percentage change in BMI and WC do not indicate that one indicator has moved independently further than the other, due to the nature of the relationship between BMI and WC.

One way to understand this is to consider changes in temperature as measured in degrees Fahrenheit (°F) and degrees Celsius (°C). The relationship between these measures is described by:

$$°F = °C * 9/5 + 32$$

An increase in temperature from 25°C to 30°C corresponds to an increase from 77°F to 86°F, and the actual increase in temperature is of course identical, just measured on two different scales. But if the proportional increases in these measures are calculated without consideration of the relationship between them, there appears to be a much greater proportional increase in temperature when measured in degrees Celsius (20.0%) than in degrees Fahrenheit (11.7%). The size of the model intercept term relating the measures (in this case, 32) drives the misconception.

To explore this in a more theoretical manner using the relationship between WC and BMI, let's assume the relationship between WC and BMI does not change over time (and is not affected by confounders – assumed throughout these examples for ease of explanation). Such a relationship is based on this regression equation:

$$WC = \mu + \alpha * BMI \quad \text{.....} \quad (1)$$

In this equation, μ is the model intercept term and α is the coefficient of BMI. Thus, rearranging (1) to calculate the percentage change in WC gives us:

$$\frac{(WC_2 - WC_1)}{WC_1} = \frac{\alpha * (BMI_2 - BMI_1)}{\mu + \alpha * BMI_1} \quad \text{.....} \quad (2)$$

In this equation, WC_i, BMI_i and t_i are the values of WC, BMI and time at time i. If the model intercept term (μ) is zero, the BMI coefficients (α) cancel out and the percentage change in WC is equal to the percentage change in BMI. However, in general the model intercept term (μ) is not zero. Thus, whether WC increases more or less than BMI is dependent on the value of μ.

Let's explore a real-life scenario using estimates from our data [2]. The estimated mean values for BMI and WC were:

BMI_1 = 27.1 kg/m^2

BMI_2 = 28.7 kg/m^2

WC_1 = 93.3 cm

WC_2 = 98.1 cm

The BMI increase from 27.1 kg/m^2 to 28.7 kg/m^2 corresponds to a 5.8% increase and the WC increase from 93.3cm to 98.1cm corresponds to an increase of 5.1%. However we cannot directly compare these percentage increases and conclude that the change in BMI is greater than the increase in WC due to the fact that the relationship between the two measures is described by a regression equation with a model intercept term that does not equal zero. Thus, to explore whether BMI or WC has moved to a greater extent relative to the other we must consider regression equation (1) with the addition of a time variable:

$$WC = \mu + \alpha * BMI + \gamma * t \quad \text{.....} \quad (3)$$

Equation (3) implies that the coefficient of time (γ) will determine whether or not WC is on average larger for a given BMI at the second time point compared with the first time point.

The parameter estimates from our data were:

μ = 21.8

α = 2.2

γ = 0.85

Equation (3) implies that for a given BMI, the WC at the second time point will be on average 0.85 cm higher than the WC at the first time point. This clearly shows that for a given increase in BMI, the increase in WC is higher than expected, not lower, as predicted by the comparisons of proportional increases in BMI and WC. However this model is theoretical; the real-life application of this model will require adjustments needed for interactions between variables such as BMI and time, and consideration given to non-linear temporal effects.

The relationship between the percentage change in WC and the percentage change in BMI is not simple and differences in their percentage increases cannot be directly compared. Comparisons of increases in the two measures must account for the relationship between them as described by the regression equation.

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2.4 Types of epidemiological analysis

A range of epidemiological analysis types are used in this thesis: descriptive epidemiology, analytical epidemiology and epidemiological modelling. These types of analysis are explained below.

2.4.1 Descriptive and analytical epidemiology

Typically, epidemiology is divided into two components: descriptive epidemiology; and analytical epidemiology. Descriptive epidemiology analyses the pattern and frequency of health events in a population. It does this by examining the concepts of person, place, and time in relationship to health events. Analytical epidemiology uses data gathered by descriptive epidemiology to look for patterns suggesting causation, and thus explores the determinants – risk and protective factors – of health outcomes. Both descriptive and analytical epidemiology increase our understanding of the health of a population and provide information that can assist with reducing the incidence of undesirable events (235).

In Chapters 3.1 and 3.2, descriptive epidemiology is used to examine trends in body weight in Australia and the prevalence of underweight, overweight and obesity in Vietnam. In Chapter 3.3, analytical epidemiology is used to examine the individual determinants of a gain in WC.

2.4.2 Epidemiological modelling

As described in more detail in the subsequent paper, epidemiological modelling is a term encompassing a variety of techniques used to make population health inferences beyond the limits of available data sources. Other terms sometimes used to describe these techniques include ‘chronic disease modelling for health policy’ and ‘medical demography’. Epidemiological modelling involves the creation of theoretical populations or cohorts and their combination with particular data sources to analyse topics of interest, such as the effects of trends in risk factors or particular health outcomes, or the effects of preventive or therapeutic interventions.

In Chapter 3.5, epidemiological modelling is used to provide information on the future burden of obesity in Australia.

Walls HL, Peeters A, Reid CM, Liew D, McNeil JJ. Predicting the effectiveness of prevention: A role for epidemiological modelling. *J Prim Prev.* 2008;29:295-305.

Declaration for Thesis Chapter 2.4.2

Walls HL, Peeters A, Reid CM, Liew D, McNeil JJ. Predicting the effectiveness of prevention:

A role for epidemiological modelling. *J Prim Prev*. 2008;29:295-305.

Declaration by candidate

In the case of Chapter 2.4.2, the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Conceptualisation and drafting of manuscript	75%

The following co-authors contributed to the work. Co-authors who are students at Monash University must also indicate the extent of their contribution in percentage terms:

Name	**Nature of contribution**	**Extent of contribution (%) (student co-authors only)**
A Peeters	Conceptualisation and drafting of manuscript	N/A
CM Reid	Conceptualisation and drafting of manuscript	N/A
D Liew	Conceptualisation and drafting of manuscript	N/A
JJ McNeil	Conceptualisation and drafting of manuscript	N/A

Candidate signature	**Date**
[redacted]	8 February 2010

Declaration by co-authors

The undersigned hereby certify that:

(1) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors;
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, expect for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (2) granting bodies, (b) the editor or publisher of journals or other publication, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s):
Department of Epidemiology & Preventive Medicine, Monash University
Department of Medicine, The University of Melbourne

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant].

Co-author signatures	Date
[redacted]	5/2/10
[redacted]	7/2/2010
[redacted]	10 February 2010

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant].

Co-author signatures	Date
[redacted]	9/2/10

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ORIGINAL PAPER

Predicting the Effectiveness of Prevention: A Role for Epidemiological Modeling

Helen L. Walls · Anna Peeters · Christopher M. Reid · Danny Liew · John J. McNeil

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Abstract It is well known that the current combination of aging populations and advances in health technology is resulting in burgeoning health costs in developed countries. Prevention is a potentially important way of containing health costs. In an environment of intense cost pressures, coupled with developments in disease prevention and health promotion, it is increasingly important for decision-makers to have a systematic, coordinated approach to the targeting and prioritization of preventive strategies. However, such a systematic approach is made difficult by the fact that preventive strategies need to be compared over the long term, in a variety of populations, and in real life settings not found in most trials. Information from epidemiological models can provide the required evidence base. In this review, we outline the role of epidemiological modeling in this context and detail its application using examples. *Editors' Strategic Implications*: Policymakers and researchers will benefit from this description of the utility of epidemiological modeling as a means of generating translational evidence that helps to prioritize data-based prevention approaches and bridge the gap between clinical research and public health practice.

Keywords Epidemiological modelling · Health policy · Prevention · Prioritisation

Aging populations and advances in health technology have resulted in burgeoning health costs in developed countries. Prevention is a potentially important way of containing health costs and already comprises a large portion of health expenditure

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in Australia. An example of increasing prevention focus is evident in the rising cost of the Australian Pharmaceutical Benefits Scheme (PBS) (Stocks et al. 2005). Between 2002 and 2005, PBS expenditure, currently over A$5.3 billion annually and 15% of total Commonwealth health expenditure, increased by more than 60%, representing growth ahead of other major health expenditure categories. The prescription of preventive medications played a major role in this. For example, in the year to June 2005, the cost of subsidizing lipid-lowering agents (A$918 million) exceeded that of any other drug group and the cost increase (an increase of A$83 million) also exceeded that of any other drug group.

Approaches to disease prevention are often divided between those targeting high-risk individuals, such as drug-therapy to induce weight loss in persons with obesity, and those affecting population-wide shifts in a particular outcome, such as measures to increase physical activity and improve nutrition in the entire population. In an environment of intense cost pressures, coupled with developments in disease prevention and health promotion, it will be increasingly important for decision-makers to have a systematic, coordinated approach to targeting and prioritizing prevention strategies. The approach will need to address the optimal mix of funding for prevention and curative interventions, and questions such as: to what extent public funding of preventive initiatives should be capped; whether subsidies should be limited to individuals at more immediate risk of developing disease; whether age limits should be set for preventive drug use; and whether targets should be included in treatment guidelines. Therefore, evidence-based justifications for funding decisions will be required.

A likely response to increasing cost pressures faced by decision-makers, as is already occurring in Australia (Doran and Henry 2006), is to shift more of the costs of preventive medications to consumers. This poses problems, however, as patients are currently provided with little intuitively useful information about the results they may expect from their treatment; they remain largely ignorant about how a treatment may affect their length of life or their likelihood of surviving free of disease to a particular age, and are generally unaware of the relative benefits of drug and lifestyle-based preventive strategies in terms of these outcomes. They are in a position to decide, for example, whether a combination of lifestyle change and low-dose aspirin might achieve similar health benefits to a statin but at a substantially lower cost.

Epidemiological Modeling

Definition

These challenges may be greatly assisted by information from epidemiological modeling (Liew et al. 2002). Epidemiological modeling is one term encompassing a variety of techniques used to make population health inferences beyond the limits of available data sources. Other terms sometimes used to describe these techniques include ‘chronic disease modeling for health policy’ and ‘medical demography’. Epidemiological modeling involves the creation of theoretical populations or cohorts and their combination with particular data sources to analyze the effects of

trends in risk factors or particular health outcomes, as well as of preventive and therapeutic interventions. Such effects are not readily estimated from available data sources. Commonly, it combines evidence from clinical trials and epidemiological studies with that from population-wide disease and demographic data. By doing this, epidemiological modeling generates *translational* evidence that bridges the gap between clinical trials and clinical and public health practice. There are a number of different epidemiological modeling methods used, including life tables, decision trees and Markov models. Estimates derived from such methods include absolute benefit, number needed to treat, life expectancy, lifetime risk, and disability-adjusted life years lost. Epidemiological modeling renders explicit the costs and benefits involved in different strategies or treatments and enables comparisons between different approaches not readily compared in real life; for example, it enables comparisons between drug and lifestyle strategies for reducing rates of heart disease or diabetes. More detailed information on economic evaluations of prevention strategies can be found in Haddix et al. (2003). Epidemiological modeling facilitates decision-making informed by the best available evidence (Liew et al. 2002), making explicit information about the long-term benefits, risks and costs of interventions.

Real-world Advantages

The potential effect of any intervention must be assessed within the context of long-term administration, costs, competing mortality and morbidity risks, demographic change and disease trends. Despite recent moves to make clinical trials more relevant to clinical practice (Pike et al. 2003), evidence from clinical trials generally provides limited information regarding the 'real-life' utility of interventions and is insufficient to critically inform clinical and public health practice (Simon et al. 1995). Epidemiological modeling predicts long-term outcomes, which is important as the costs and benefits of preventive interventions often occur years into the future, well beyond the period able to be studied by most clinical trials.

Additionally, epidemiological modeling has the ability to take into account a wide range of patients and outcomes, varying compliance over time, and the costs and cost-effectiveness of interventions. It has the 'real world' advantage of being able to consider outcomes in patients with multiple health problems and possibly receiving treatments apart from those tested in trials (Liew et al. 2002; Simes 2002), and can allow for the simulation of interventions targeted at people in various risk categories, thus identifying sub-groups for which effectiveness and cost-effectiveness are optimal. Comparative modeling can also be undertaken of an intervention delivered to the whole population versus targeted to a specific high-risk group. It also has the ability to bring attention to the need for the collection of critical missing data and can capture the uncertainty that surrounds the impact of a preventive intervention through use of stochastic models (McEwan et al. 2006), in which inputs are entered as uncertainty ranges rather than as single values. The impact of assumptions made in the model can also be assessed, and those assumptions with the greatest bearing on the uncertainty can be identified (sensitivity analyses). The

benefits of epidemiological modeling are particularly obvious in *primary* preventive strategies, which are delivered over the long-term (often life-long) to large numbers of often healthy people. In this situation, the costs and potential side effects of interventions take on added significance.

Measurement of Outcomes

Outcomes from different interventions can be measured in a number of ways. *Absolute* benefit, assessed by epidemiological modeling, is most relevant to decision-making, as it takes into account the underlying risk of disease. The greater the underlying risk of disease, the greater the absolute benefit (Malenka et al. 1993; Simes 2002). Reporting efficacy in terms of *relative* risk reduction, as is typical in clinical trails, is advantageous in that it provides information on the strength of a treatment's effect, allows comparison between treatment strategies, and makes it easier to use the information as relative risk reductions tend to be similar across subpopulations. However, it is often erroneous to base treatment decisions on relative benefits, which evidence suggests remain constant across population subgroups, when potential absolute benefits vary according to the level of absolute risk (i.e., the actual rate or number of events). For example, the proportional benefit of cholesterol-lowering on subsequent coronary heart disease incidence is similar for patients with and without pre-existing heart disease, but absolute benefits of treatment in terms of morbidity and mortality vary widely; treatment is much more cost-effective when delivered as secondary prevention to those with pre-existing heart disease than as primary prevention to those without it.

Presentation and Interpretation of Outcomes

Information from epidemiological modeling can be summarized by intuitive diagrams and outputs such as survival curves, life expectancy at different levels of risk and number of deaths prevented due to treatment. Interventions can be ranked, as cost-effectiveness analyses allow various interventions to be compared in terms of differences in costs and differences in effects (both beneficial and adverse) (Marshall 2003). Such prioritization allows for efficient and equitable allocation of limited healthcare resources (Brandeau et al. 2005). In those many cases when long-term costs and benefits are of interest, epidemiological modeling is critical.

Limitations

The assumptions made in epidemiological modeling are also, however, its greatest limitation. The information derived is only as good as the input data used. Costs and uncertainty, for example, can be difficult to estimate. Costs can be particularly difficult to estimate in the inter-agency funding environment of many prevention programs. Also difficult to estimate are quality-adjusted life years (QALYs), a measurement of both the quality and quantity of life lived, which often are used as a means of quantifying benefit received in prevention strategies. Due to the

multiplicative nature of error, small errors in input data can produce much larger absolute errors in outcome estimates. For these reasons, the input data used and assumptions made need to be clearly described and uncertainty analyses of key input data need to be performed. The need for explicit description of assumptions made was also a key recommendation of a publication evaluating the strengths and limitations of policy models for coronary heart disease (Unal et al. 2006).

Applications of Epidemiological Modeling

Information from epidemiological modeling is useful to a wide range of health decision-makers, and in particular, informs public health, health policy, and clinical practice.

Public Health and Health Policy

For decision-makers interested in burden-of-disease predictions, or selecting between alternative strategies under resource constraints, information from epidemiological modeling can be very useful.

Epidemiological modeling is critical for predicting current total and potential future health burden (Murray and Lopez 1997). Table 1 shows a World Health Organization (WHO) prediction of the rank order in 2030 of the 15 leading causes

Table 1 Change in rank order of deaths for the 15 leading causes, world, 2002–2030

Rank	2002		2030	
	Disease or injury (ranked 1–15)	% total deaths	Disease or injury (ranked 1–15)	% total deaths
1	Ischaemic heart disease	12.6	Ischaemic heart disease	13.1
2	Cerebrovascular disease	9.7	Cerebrovascular disease	10.3
3	Lower respiratory infections	6.9	HIV/AIDS	8.7
4	HIV/AIDS	4.8	Chronic obstructive pulmonary disease	7.9
5	Chronic obstructive pulmonary disease	4.8	Lower respiratory infections	3.5
6	Perinatal conditions	4.3	Diabetes mellitus	3.1
7	Diarrhoeal diseases	3.3	Trachea, bronchus, lung cancers	3.0
8	Tuberculosis	2.7	Road traffic accidents	2.8
9	Trachea, bronchus, lung cancers	2.2	Tuberculosis	2.4
10	Road traffic accidents	2.1	Perinatal conditions	2.1
11	Diabetes mellitus	1.7	Stomach cancer	1.8
12	Malaria	1.6	Hypertensive heart disease	1.8
13	Hypertensive heart disease	1.6	Self-inflicted injuries	1.5
14	Self-inflicted injuries	1.5	Nephritis and nephrosis	1.3
15	Stomach cancer	1.5	Liver cancer	1.3

Note: Adapted from Mathers and Loncar (2005)

of death globally (Mathers and Loncar 2005). It is an example of the use of epidemiological modeling for making health burden predictions. Epidemiological modeling also enables the estimation of economic consequences of public health interventions, for example smoking cessation (Barendregt et al. 1997).

Questions that are not amenable to immediate direct observation can also be answered by epidemiological modeling. Examples include whether improving risk factor profiles in the population would lead to more or less disability (the compression of disability debate; Fries 1980; Mamun et al. 2004), or whether improvement in treatments for cardiovascular disease (CVD) was responsible for the cessation of the stroke mortality rate decline (Peeters et al. 2003) and the US coronary disease mortality decline (Ford et al. 2007).

The allocation of resources is more readily justified with the addition of cost-effectiveness evidence from epidemiological modeling; ensuring that products/proposals are supported by evidence of cost-effectiveness assures purchasers that they are receiving value for money. For example, the cost-effectiveness of drug and lifestyle-based preventive strategies within groups defined by absolute disease risk can be determined by epidemiological modeling. This allows for a more systematic targeting of resources and apportioning of funds. Table 2 is a league table comparing the cost-utility of a range of interventions to prevent and treat diabetes. The data show that diabetes is a costly disease, and interventions to prevent and treat diabetes very widely in terms of cost per health outcome gained (Zhang et al. 2004).

Information from epidemiological modeling can also help decision-makers select between community and high-risk approaches to prevention, select sub-populations for whom treatment is most appropriate, and answer questions regarding the consequences of varying compliance, and the lag-time between drug initiation and

Table 2 Cost-utility of interventions to prevent and treat type 2 diabetes from the perspective of the single payer

Intervention	Study population	Cost per QALY ($)
Primary prevention		
Lifestyle intervention	Impaired glucose tolerance	34,600
Metformin therapy	Impaired glucose tolerance	109,600
Tertiary prevention		
Screening for retinopathy	Type 2 diabetes	3,400
Nephropathy prevention		
Treating all patients with angiotensin-converting enzyme inhibitors	Type 2 diabetes	8,800
Foot care program	Type 1 or type 2 diabetes with various risks of amputation	Cost saving to 7,100
Intensive glucose control	Newly diagnosed type 2 diabetes	35,300
Intensive blood pressure control	Newly diagnosed type 2 diabetes	−2,400
Cholesterol control	Newly diagnosed type 2 diabetes	63,200
Smoking cessation	Newly diagnosed type 2 diabetes	12,500

Note: Adapted from Zhang et al. (2004). QALY = Quality of Life Year

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effect (Liew et al. 2002; Nelson et al. 2005). Such information may, for example, help clarify the most appropriate age at which treatment should commence or conclude.

Intuitive outputs presented in a policy-relevant format, such as survival curves and associated measures such as life expectancy and disability-adjusted life years, may also be useful for decision-makers. They enable the potential population impacts of an intervention known to alter a type of risk, such as cardiovascular risk, to be easily assessed. For example, Fig. 1 describes the probability of being alive and the probability of being alive *and* free of CVD for male smokers and non-smokers at different ages (Mamun et al. 2004).

Clinical Practice

Information from epidemiological modeling assists clinicians in targeting individuals for whom treatment is safest and most cost-effective. The intuitive diagrams and outputs of epidemiological modeling also allow consumers to make better-informed choices in regard to their preventive treatments, and assist health professionals to describe to patients the likely outcomes of preventive treatments.

Alternative strategies, such as whether to follow a drug-based intervention or make lifestyle changes, can be portrayed in such a way to allow consumers greater participation in decisions affecting the length and quality of their lives. Such information can help consumers understand the likelihood of being disease-free in later years or the implications of such things as obesity and cigarette smoking on disease-free life-expectancy and on the community burden of disease, and the likely impact of particular interventions on their health. Empirical evidence on the association between different methods of risk communication and patients' preference and behavior is sparse. It is becoming increasingly recognized, however,

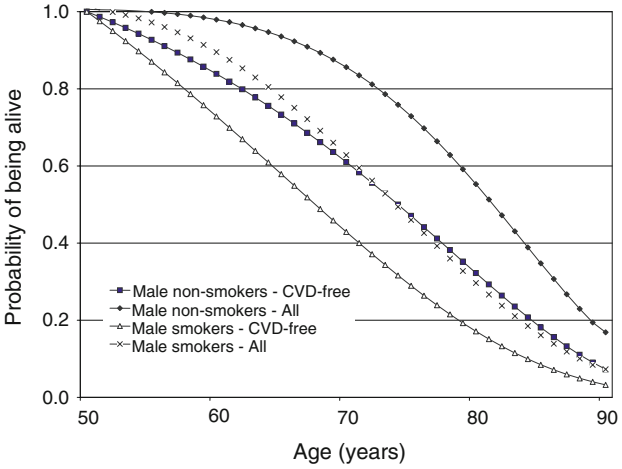


Fig. 1 Cardiovascular disease (CVD)-free and total survival curves for males. *Note*: Adapted from Mamun et al. 2004

that such risk communication should use measures of absolute risk (rather than relative risk) and concepts such as number needed to treat, lifetime risk and survival curves, and that these should be presented within a meaningful context (Beir 2001; Edwards et al. 2002). Such information is generally not available directly from clinical trails and requires the combination of trial data with the relevant context data for particular population groups.

Benefits of preventive therapies are typically presented in terms of proportional risk reductions, but despite the convenience, it is misleading to make treatment decisions based on such data. Treatment should be based on individuals' absolute risks and other competing risks, which will vary from patient to patient and be highly dependent on treatment duration. With their assessment of absolute risk, epidemiological models assist clinicians with selecting the most effective and tolerable interventions for specific patients while taking into account cost considerations, an area increasingly accepted as a responsibility of clinical decision-making (Jackson 2001).

Figure 2 illustrates the differential effects of delivering a hypothetical intervention to the two extreme deciles of underlying coronary risk (McNeil et al. 2001). Successful risk reduction in any individual or group of individuals is predicted to lead to a lowering of the risk throughout life, and the area between the pre- and post-intervention disease-free survival curves equates to the predicted (disability-adjusted) years of life saved. This also forms the basis of a simple approach to cost-effectiveness analysis. The disease-free survival curves show the effect of a hypothetical intervention delivered to a 55-year-old individual at the top and bottom deciles of absolute cardiovascular risk compared to baseline. Probability of survival to a particular age 'free of disease', in this case CVD, can be derived. The model also allows projection of 'survival free of cardiovascular disease' and can simulate the anticipated effect of drug and non-drug interventions. It can assist patients with

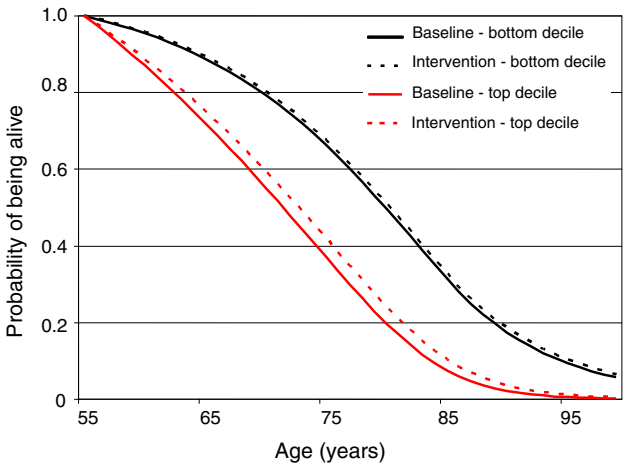


Fig. 2 Disease-free survival curves, comparing hypothetical intervention to baseline, for the top and bottom deciles of absolute risk *Note*: Adapted from the CVD prevention model (McNeil et al. 2001)

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deciding whether treatment is appropriate, considering the sub-group to which they belong and their level of absolute risk (McNeil et al. 2001).

Other Applications

Health insurers may use information from epidemiological modeling to determine which health interventions to cover. The health returns of interventions covered by employers in benefit packages can also be compared.

To assist in curtailing increasing pharmaceutical costs, pharmaceutical purchasing criteria could be adapted to take evidence from epidemiological modeling into account. Treatments of unproven cost-effectiveness could be categorized differently from those of proven cost-effectiveness, perhaps a new "conditional listing," (Johnson and Lassere 2003). For example, statin therapy has been shown to be a cost-effective intervention for populations at high risk of CVD (>4%/year), but not cost-effective for populations at low risk (<1%/year). For populations with moderate risk (1–4%/year) the available evidence is contradictory (Franco et al. 2005). From such findings, statins could be placed in the "conditional listing" for treating those at moderate risk of CVD, at least until further evaluations have been conducted. Lim et al. (Lim et al. 2001) have suggested that while current Australian PBS criteria target patients at risk of coronary heart disease, they could be improved in terms of identifying people *most* at risk.

In Australia, there is increasing pressure for the PBS to evaluate the cost-effectiveness of highly specialized and 'orphan' drugs (that address rare diseases) (Reid et al. 2006). One method of balancing the uncertainty of longer-term cost-effectiveness with public demand for access to effective but expensive drugs is the establishment of drug registries, which function as risk-sharing schemes between Government and drug sponsors, in order to collect data necessary for epidemiological modeling to ascertain the cost-effectiveness of specialized drugs and orphan drugs. One such example is the Australian Bosentan Patient Registry (Reid et al. 2006).

Conclusion

Despite future developments in disease prevention and health promotion, financial pressures and questions around issues such as whom to treat and when to commence or conclude therapy will remain. As long as community resources are limited, objective and equitable ways of selecting preventive treatments will be needed. Epidemiological modeling will increasingly be required to extrapolate the results of clinical trials and epidemiological studies to a broader context, one more relevant to 'real life'. Furthermore, epidemiological modeling will remain important for predicting current total and potential health burden.

Epidemiological models can assist policy-makers in making better-informed choices in terms of which preventive options to fund, improve the ability of clinicians to provide accurate preventive advice, and increase the ability of consumers to make decisions about preventive strategies that relate to themselves and their communities.

Decision-making must take into account more than information from epidemiological modeling alone—decisions must be informed by societal values and preferences as much as by evidence of benefit and value-for-money. However, epidemiological modeling is a powerful means of deriving quantitative evidence for health decision-making.

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Chapter 3: Results

The results of the analyses undertaken in this thesis are presented below, preceded by a brief explanation of the particular analysis. In cases where the final published version of the analysis was a shortened version of the full analysis, the full analysis is presented in this chapter and the published version is included in the Appendices (Appendix 1). Correspondence received from these publications is also included in the Appendices (Appendix 2).

3.1 Trends in body weight

3.1.1 Trends in body mass index

Overweight and obesity are increasing worldwide (209). But much remains unknown about the nature of the recent obesity trends, particularly in countries such as Australia, where the surveillance of obesity has been limited to describing changes in the prevalence of overweight and obesity. Yet the data to enable further analysis are available.

Here we examine BMI trends in urban Australian adults between 1980 and 2000, focusing on trends in the continuous distribution of BMI.

Walls HL, Wolfe R, Haby MM, Magliano DJ, de Courten M, Reid CM, McNeil JJ, Shaw J, Peeters A. Trends in BMI of urban Australian adults, 1980-2000. *Public Health Nutrition*. 2009;22:1-8.

Declaration for Thesis Chapter 3.1.1

Walls HL, Wolfe R, Haby MM, Magliano DJ, de Courten M, Reid CM, McNeil JJ, Shaw J, Peeters A. Trends in BMI of urban Australian adults, 1980-2000.*Public Health Nutr*. 2009;22:1-8.

Declaration by candidate

In the case of Chapter 3.1.1, the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Design, analysis and drafting of manuscript	70%

The following co-authors contributed to the work. Co-authors who are students at Monash University must also indicate the extent of their contribution in percentage terms:

Name	**Nature of contribution**	**Extent of contribution (%) (student co-authors only)**
R Wolfe	Statistical advice and drafting of manuscript	N/A
MM Haby	Design and drafting of manuscript	N/A
DJ Magliano	Design and drafting of manuscript	N/A
M de Courten	Design and drafting of manuscript	N/A
CM Reid	Drafting of manuscript	N/A
JJ McNeil	Drafting of manuscript	N/A
J Shaw	Drafting of manuscript	N/A
A Peeters	Design, analysis and drafting of manuscript	N/A

Candidate signature	**Date**
[redacted]	8 February 2010

Declaration by co-authors

The undersigned hereby certify that:

(1) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors;
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least part of the publication in their field of expertise;

(3) they take public responsibility for their part of the publication, expect for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (2) granting bodies, (b) the editor or publisher of journals or other publication, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s):
Department of Epidemiology & Preventive Medicine, Monash University
School of Population Health, The University of Melbourne
Baker IDI Heart and Diabetes Institute

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant].

Co-author signatures	**Date**
[redacted]	11 February 2010
[redacted]	10/2/2010
[redacted]	10 February 2010
[redacted]	5/2/10
[redacted]	10/2/10
[redacted]	8/2/10
[redacted]	7/2/2010

(5) potential conflicts of interest have been disclosed to (a) granting bodies, (b) the editor or publisher of journals or other publication, and (c) the head of the responsible academic unit; and

(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

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[redacted]	9/2/2010

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Trends in BMI of urban Australian adults, 1980–2000

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Abstract

Objective: To analyse changes in the distribution of BMI in Australia between 1980 and 2000.
Design: Data were from the 1980, 1983 and 1989 National Heart Foundation Risk Factor Prevalence Study, the 1995 National Nutrition Survey and the 1999/2000 Australian Diabetes, Obesity and Lifestyle Study. Survey participants were aged 25–64 years and resident in Australian capital cities. BMI was calculated as weight divided by height squared (kg/m^2), where weight and height were measured using standard procedures.
Results: Mean BMI was higher in 2000 than 1980 in all sex and age groups. The age-adjusted increase was 1·4 kg/m^2 in men and 2·1 kg/m^2 in women. The BMI distribution shifted rightwards for all sex and age groups and became increasingly right-skewed. The change between 1980 and 2000 ranged from a decrease of 0·04 kg/m^2 at the lower end of the distribution for men aged 25–34 years to an increase of 7·4 kg/m^2 at the higher end for women aged 55–64 years. While the prevalence of obesity (BMI ≥ 30 kg/m^2) doubled, the prevalence of obesity class III (BMI ≥ 40 kg/m^2) increased fourfold.
Conclusions: BMI in urban Australian adults has increased and its distribution has become increasingly right-skewed. This has resulted in a large increase in the prevalence of obesity, particularly the more severe levels of obesity. It will be important to monitor changes in the different classes of obesity and the extent to which obesity interventions both shift the BMI distribution leftwards and decrease the skew of the distribution.

Keywords
Obesity
Body mass index
Trends
Australia

Overweight and obesity are increasing worldwide[1]. In Australia the prevalence of obesity has doubled in two decades and increases are projected to continue[2]. However, much remains unclear about the population BMI trends that are driving the increase. Specifically, it is unclear to what extent the current prevalence of obesity is a result of increases across the whole spectrum of BMI or limited mostly to the upper BMI ranges. It is possible that the BMI distribution in today's population has a larger mean, more variability, increased skewness or any combination of these aspects in comparison with historical age-matched sections of the population.

It has been shown in the USA and New Zealand that increases in the prevalence of obesity have been accompanied by an increase in right skew of the BMI distributions[3–5]. In Australia, the surveillance of obesity has been limited to describing changes in the prevalence of overweight and obesity. There has been no analysis of trends in the continuous distribution of BMI. Without understanding the detail of the increase in obesity prevalence we cannot accurately measure its burden, fully understand its drivers or ensure that obesity prevention and treatment campaigns are targeted appropriately. Here, we examine BMI trends in urban Australian adults between 1980 and 2000, focusing on trends in the continuous distribution of BMI.

Methods

The present study compares data from five cross-sectional surveys: the 1980, 1983 and 1989 National Heart Foundation Risk Factor Prevalence Study (RFPS), the 1995 National Nutrition Survey (NNS) and the 1999/2000 Australian Diabetes, Obesity and Lifestyle Study (AusDiab).

Data sources

National Heart Foundation Risk Factor Prevalence Study
The RFPS comprised three national population-based surveys of the prevalence of risk factors for heart disease conducted in 1980, 1983 and 1989. Their target population

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was residents of Australian capital cities listed on state electoral rolls and aged 25–64 and 20–69 years. From initial target samples of 8375, 10 148 and 9328, the 1980, 1983 and 1989 studies involved 5603 (67 %), 7615 (75 %) and 6097 (65 %) respondents, respectively. Participants were administered a questionnaire, and underwent a physical examination and blood test[6]. For the current analysis participants with missing values for measured height or weight, those aged <25 and >65 years and women recorded as pregnant at the time of survey were excluded, leaving 5550, 7562 and 7667 participants from the 1980, 1983 and 1989 surveys for analysis, respectively. For each survey, there were no further missing values for country of birth or smoking status.

National Nutrition Survey

The NNS was a sub-sample of a larger population survey, the National Health Survey (NHS). The target population was residents of Australian States and Territories and aged over 2 years. For the NHS, a nationwide sample of approximately 23 800 households was obtained. Of households selected to participate, 92 % responded, with a total of 57 633 persons interviewed. Of those, 22 562 were selected to participate in the NNS. A total of 13 858 persons (61 % of those selected) participated in the NNS[7]. Participants with missing values for measured height or weight, aged <25 and >65 years, not living in capital cities and women recorded as pregnant at the time of survey were excluded, leaving 4783 participants for analysis. There were no further missing values for country of birth or smoking status.

Australian Diabetes, Obesity and Lifestyle Study

AusDiab was a national population-based survey conducted in 1999/2000, the methods for which are described in detail elsewhere[8]. Household questionnaires of the nationwide sample were completed in 67 % of the households (*n* 11 479) that could be contacted and contained at least one eligible person. A total of 20 347 eligible individuals were interviewed in these 11 479 households. The final survey sample (those attending the biomedical examination) included 11 247 adults (5049 men and 6198 women), representing 55 % of those completing the household interview and 37 % of those selected. Participants with missing values for measured height or weight, who were pregnant at the time of survey, not living in capital cities or aged <25 and >65 years were excluded, leaving 5529 participants for analysis. While there were no further missing values for the separate analysis by country of birth, missing smoking responses were excluded for the analysis by smoking status, leaving 5463 participants for that analysis.

BMI

BMI was calculated as weight divided by height squared (kg/m^2), where weight and height were measured using standard procedures. Standard cut-offs were applied to the BMI scores to classify people into underweight (<18·5 kg/m^2), normal weight (18·5–24·9 kg/m^2), overweight (25·0–29·9 kg/m^2) and obese (≥30·0 kg/m^2), and to classify obese people into obese class I (30·0–34·9 kg/m^2), obese class II (35·0–39·9 kg/m^2) and obese class III (≥40·0 kg/m^2)[9,10].

Analysis

Study-specific sample weights were used to represent the age, sex and state distribution of the Australian population at the time of each survey.

Analyses of BMI were stratified by sex and age group (age categorised in 10-year groups between ages 25 and 64 years) and conducted using the Intercooled Stata statistical software package version 9·0 (StataCorp, College Station, TX, USA).

The prevalence of each BMI category and mean BMI were calculated in each survey. Linear regression was used to analyse the changes in BMI adjusted for age and sex. Changes in the distribution of BMI between surveys are illustrated by Tukey mean–difference plots[3,5]. In these plots, the difference between mean BMI in corresponding quantiles of different surveys is plotted against the total mean of the combined quantiles.

Analyses were also conducted according to 'country of birth' (with strata defined as Australian-born and overseas-born) and stratified according to smoking status (with strata defined as 'never smoker' and 'past or current smoker'). The definitions of country of birth and smoking status were consistent across surveys.

Results

Across the five surveys, the number of participants in each sex and age group ranged from 379 to 1237. The distributions of age group, sex and proportion Australian-born were similar in each survey (with the exception of age group in AusDiab). The response fractions differed – they were higher in the RFPS, lower in the NNS and substantially lower in AusDiab – and the percentage of never smokers was higher in AusDiab than in the previous four surveys (Table 1).

Mean BMI was higher in 2000 than 1980 in all sex and age groups. The age-adjusted increase was 1·4 kg/m^2 in men and 2·1 kg/m^2 in women. The mean increase was significantly greater for women than men, but the changes in mean BMI by age group were not significantly different. Figure 1 shows that the crude increase over the 20 years for the different age and sex groups ranged from 1·0 to 2·6 kg/m^2. Three distinct phases were apparent during the 20-year period. A modest increase in BMI was observed between 1980 and 1989 (approximately 0·04 kg/m^2 per year for men, 0·10 kg/m^2 for women), followed by a more rapid increase between 1989 and 1995 (approximately 0·19 kg/m^2 per year for men, 0·21 kg/m^2 per year for women), and from 1995 to 2000 a

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Table 1 Demographic information from the five surveys among urban Australian adults aged 25–64 years, 1980–2000

	1980 RFPS (*n* 5550)		1983 RFPS (*n* 7562)		1989 RFPS (*n* 7667)		1995 NNS (*n* 4783)		2000 AusDiab (*n* 5529)	
	n	%	*n*	%	*n*	%	*n*	%	*n*	%
Males	2765	50	3738	49	3802	50	2302	48	2513	45
Females	2785	50	3824	51	3865	50	2481	52	3016	55
	%		%		%		%		%	
Response fraction to original survey data collection	67		75		65		61		37	
Age group distribution										
25–34 years	27		27		26		30		15	
35–44 years	24		28		32		28		27	
45–54 years	26		23		23		24		34	
55–64 years	23		23		20		18		23	
Australian-born	72		70		71		67		68	
Never smokers	46		47		49		48		56	

RFPS, Risk Factor Prevalence Study; NNS, National Nutrition Survey; AusDiab, Australian Diabetes, Obesity and Lifestyle Study.

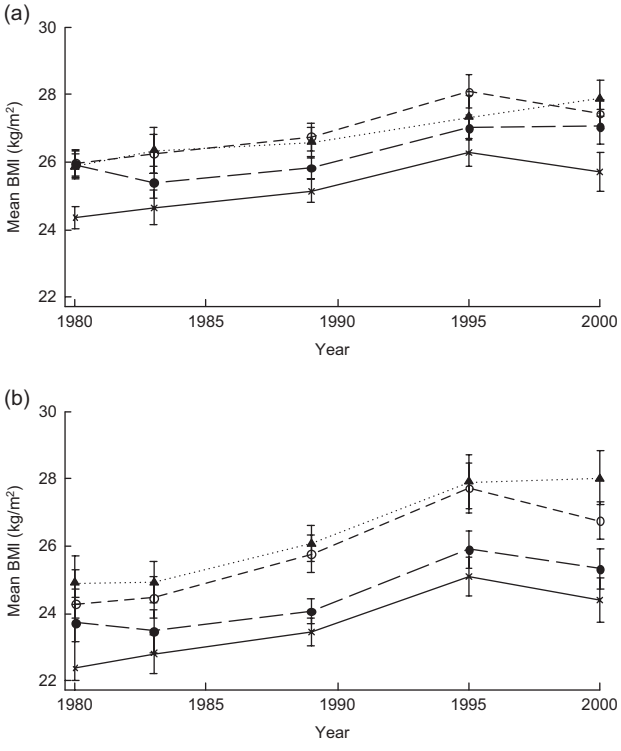


Fig. 1 Mean BMI between 1980 and 2000 of urban Australian males (a) and females (b) aged 25–64 years by age group (——×——, 25–34 years; ——●——, 35–44 years; ––○––, 45–54 years; · · · · ▲ · · · ·, 55–64 years), with 95 % confidence intervals represented by vertical bars

decrease (approximately 0·05 kg/m^2 per year for men, 0·04 kg/m^2 per year for women). These trends were similar across most male and female age groups.

We assessed changes in the shape of the distribution for the 20-year period (1980–2000) and individually for the three distinct time periods (1980 to 1989, 1989 to 1995 and 1995 to 2000). Figure 2 illustrates the changing shape of the BMI distribution between 1980 and 2000, and 1980 and 1995. In general, there is a rightwards shift in the whole distribution, and the tail is also stretching further out (an increase in skew). The right-skewing of the distribution is more pronounced for women than men. The greatest increases in skew were generally in the period with the most rapid increases in mean BMI (1989–95). In the last period (1995–2000), there was a leftwards shift of the total population BMI distribution.

Figure 3 compares the 1980 BMI distributions with those of 2000. The shape of the line formed by the points demonstrates the nature of the shift. It shows that for all age and sex groups, there was upward shift of the entire BMI distribution and increased skewness. The absolute magnitude of the differences varied widely, from a decrease of 0·04 kg/m^2 at the lower end of the distribution for males aged 25–34 years to an increase of 7·4 kg/m^2 at the higher end of the distribution for females aged 55–64 years. In general, the magnitude of increase for the higher end of the distribution was greater for women than men, suggesting a greater increase in skew for women. Skew also increased when the data were analysed according to relative rather than absolute change between 1980 and 2000.

There was some variation in the degree of increase by sex and age groups. For women aged 35–44 and 55–64 years and men aged 35–44 years, the increase was substantially higher at the highest end of the distribution with relatively less variation in the increase elsewhere. There was little variation in the increase by BMI for men aged 45–54 and 55–64 years.

Table 2 illustrates the consequence of these changes for the prevalence of underweight, normal weight, overweight and obesity. The prevalence of obesity increased by 6·6 and 10·5 percentage points in men and women between 1980 and 2000, leading to a prevalence of 17·2 % in men and 19·3 % in women. The prevalence of overweight was higher in men than women in all time periods. The prevalence of obesity was higher in men in 1980, but by 2000 was higher in most age groups of women. Between 1995 and 2000 the prevalence of obesity decreased for all age groups except those aged 55–64 years.

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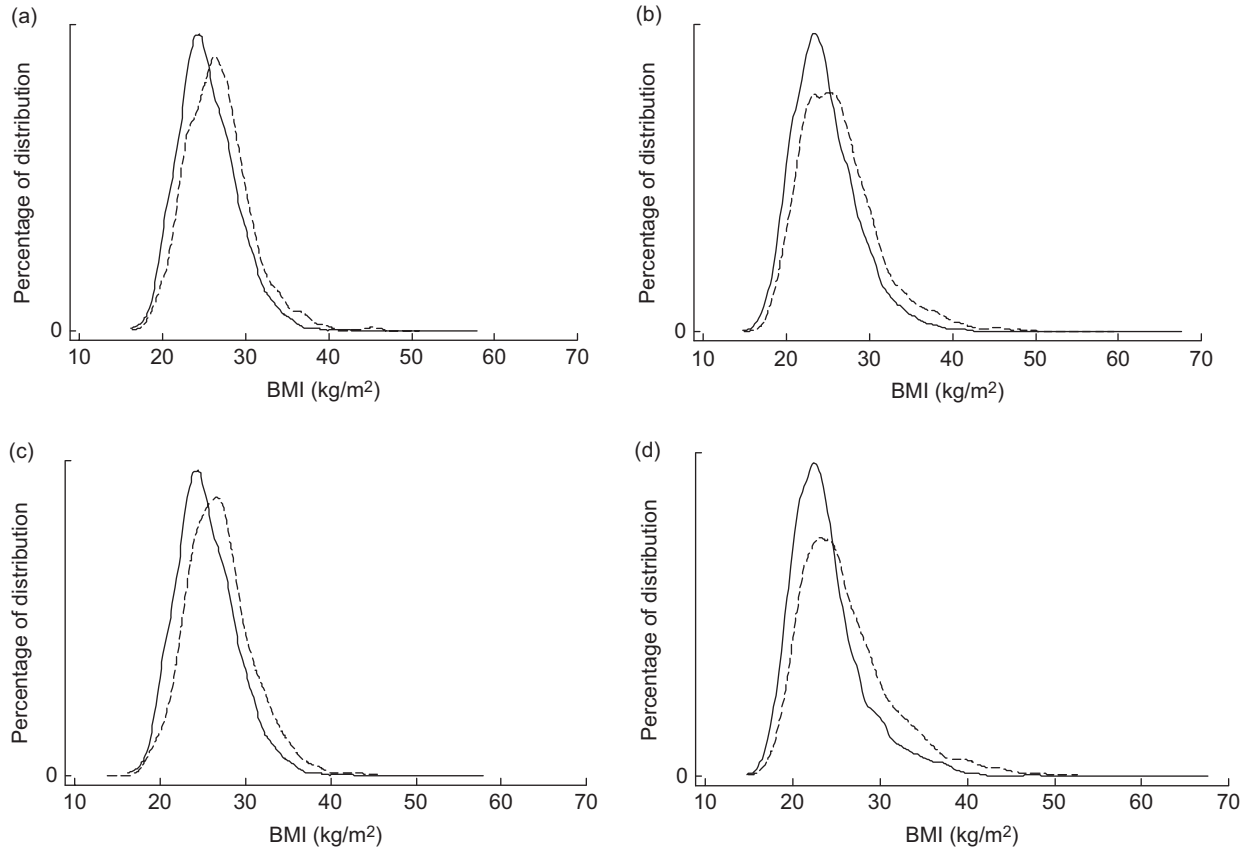


Fig. 2 Distribution of BMI in 1980 (——), 1995 and 2000 (– – –) in urban Australian adults aged 25–64 years by sex: (a) 1980 and 2000, males; (b) 1980 and 2000, females; (c) 1980 and 1995, males; (d) 1980 and 1995, females

Table 3 illustrates the consequences of the increase in skew for the different categories of obesity. As a percentage of those with a BMI $\geq 30\,kg/m^2$, obese class I decreased from 90·1 % to 81·4 % in men and from 71·3 % to 60·4 % in women. The most rapidly increasing obesity class was class II for men and class III for women.

Analyses of height and weight indicated that these BMI trends were driven largely by trends in weight. Mean height increased between 1980 and 2000 (1·8 cm for men and 1·4 cm for women) but this did not account for the changes observed in the BMI distribution; based on the observed increases in height across the distribution, less right skewness in BMI is expected. In contrast, mean weight increased markedly (by 5·7 kg in men and 6·4 kg in women) and there was an increase in the right skew of the weight distribution. Similar trends in skew and mean BMI between 1980 and 2000 to our whole-population results were found for Australian-born participants and people who had never smoked (data not shown).

Discussion

Between 1980 and 2000 the mean BMI of urban Australian adults increased. The age-adjusted increase was $1·4\,kg/m^2$ in men and $2·1\,kg/m^2$ in women. The increase in BMI was not uniform. It was greater at the higher end of the distribution than the lower end, ranging from a decrease of $0·04\,kg/m^2$ to an increase of $7·4\,kg/m^2$. Thus, the BMI distribution became increasingly skewed. The prevalence of obesity approximately doubled. As a percentage of total obesity, obesity class I decreased while obesity class III more than doubled.

There were substantial differences in the degree and rate of increase by age, sex and across time. The most rapid increase was in the early 1990s. After this time the rate of increase appears to have slowed in some groups. The only continued increase was in men and women aged 55–64 years.

The major potential limitation to our analysis is the comparability of the surveys from which the results are derived. The three RFPS surveys were similarly conducted and had relatively high response fractions, but the NNS and AusDiab studies were methodologically different and had lower response fractions (61 % and 37 %, respectively). These low response fractions could lead to either an under- or an over-representation of BMI levels. However an under-representation is most likely, as non-response has been linked to having a sedentary lifestyle and lower socio-economic status (also associated with

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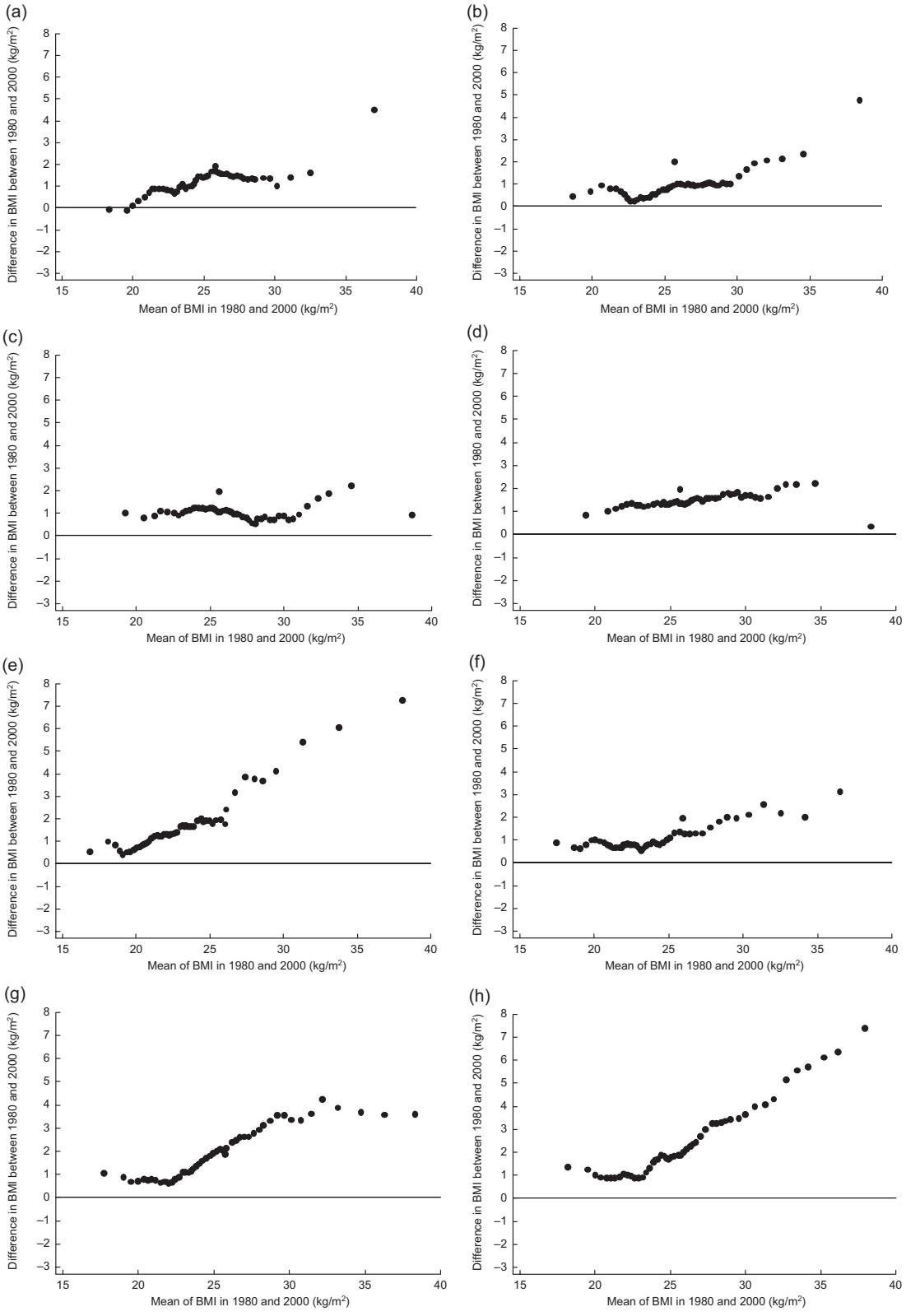


Fig. 3 Tukey mean–difference plots of BMI change between 1980 and 2000 in urban Australian adults aged 25–64 years by sex and age group: (a) males, 25–34 years; (b) males, 35–44 years; (c) males, 45–54 years; (d) males, 55–64 years; (e) females, 25–34 years; (f) females, 35–44 years; (g) females, 45–54 years; (h) females, 55–64 years. Data are presented as fifty data points representing the BMI distribution split into fifty centiles

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Table 2 Percentage of urban Australian adults aged 25–64 years in each BMI category in 1980 and 2000

		Males				Females			
		1980		2000		1980		2000	
	Age (years)	%	95 % CI	%	95 % CI	%	95 % CI	%	95 % CI
Underweight (<18·5 kg/m^2)	25–34	0·5	0·2, 1·3	0·6	0·2, 2·5	6·1	4·2, 8·8	2·8	1·3, 6·1
	35–44	1·0	0·2, 3·7	0·3	0·0, 1·5	3·7	2·4, 5·8	1·7	0·7, 4·0
	45–54	0·6	0·2, 1·5	0·0	0·0, 0·0	2·8	1·7, 4·4	0·5	0·2, 1·1
	55–64	1·0	0·4, 2·5	0·1	0·0, 0·6	2·1	1·1, 3·8	0·6	0·2, 2·0
	Total	0·7	0·4, 1·3	0·3	0·0, 0·7	4·0	3·1, 5·0	1·5	0·9, 2·4
	n (total)	19		9		111		45	
Normal weight (18·5–24·9 kg/m^2)	25–34	62·0	57·4, 66·3	46·1	38·7, 53·6	79·1	75·0, 82·6	63·8	56·2, 70·7
	35–44	44·4	39·5, 49·5	35·1	29·8, 40·7	67·4	62·2, 72·2	61·1	56·3, 65·6
	45–54	43·0	38·5, 47·7	27·6	23·6, 31·9	58·5	54·0, 62·8	44·2	40·0, 48·4
	55–64	39·2	34·9, 43·7	24·6	20·2, 29·6	53·1	48·5, 57·6	36·0	31·1, 41·3
	Total	49·1	46·6, 51·5	34·0	31·3, 37·0	66·4	64·1, 68·6	52·5	49·8, 55·2
	n (total)	1308		763		1796		1429	
Overweight (25·0–29·9 kg/m^2)	25–34	31·0	27·0, 35·4	42·3	35·1, 50·0	11·1	8·4, 14·4	21·6	15·6, 29·1
	35–44	44·8	39·8, 50·0	49·6	44·0, 55·2	19·4	15·5, 24·0	22·5	18·8, 26·7
	45–54	42·0	37·2, 46·4	53·2	48·6, 57·8	27·5	23·7, 31·6	31·1	27·4, 35·0
	55–64	45·4	40·9, 50·0	48·9	43·5, 54·3	31·5	27·5, 35·9	34·3	29·4, 39·6
	Total	39·6	37·3, 42·0	48·5	45·6, 51·5	21·0	19·0, 22·9	26·8	24·4, 29·3
	n (total)	1149		1216		626		908	
Obese (≥30·0 kg/m^2)	25–34	6·5	4·6, 9·3	11·0	7·7, 15·6	3·7	2·4, 5·6	11·9	8·0, 17·4
	35–44	9·8	7·2, 13·1	15·1	11·7, 19·2	9·5	6·5, 13·6	14·7	11·8, 18·2
	45–54	14·7	11·5, 18·5	19·2	16·0, 22·9	11·3	8·7, 14·6	24·3	20·9, 28·0
	55–64	14·4	11·4, 18·1	26·4	22·0, 31·3	13·4	10·5, 16·8	29·1	24·5, 34·1
	Total	10·6	9·3, 12·2	17·2	15·3, 19·3	8·8	7·5, 10·3	19·3	17·3, 21·4
	n (total)	289		525		252		634	

Table 3 Percentage of urban Australian adults aged 25–64 years in each obesity category (based on BMI) in 1980 and 2000

		Males				Females			
		1980		2000		1980		2000	
		%	95 % CI	%	95 % CI	%	95 % CI	%	95 % CI
Obese class I (30·0–34·9 kg/m^2)	% of all BMI categories	9·6	8·3, 11·1	14·0	12·3, 15·9	6·3	5·2, 7·5	11·7	10·1, 13·4
	% of obese (≥30·0 kg/m^2)	90·1	84·3, 93·9	81·4	76·3, 85·7	71·3	62·9, 78·5	60·4	54·8, 65·8
	n	260		417		182		392	
Obese class II (35·0–39·9 kg/m^2)	% of all BMI categories	0·8	0·5, 1·5	2·7	2·0, 3·7	2·0	1·4, 2·3	5·2	4·2, 6·5
	% of obese (≥30·0 kg/m^2)	7·9	4·5, 13·5	15·9	11·9, 20·9	23·3	16·6, 31·7	27·1	22·3, 32·5
	n	24		88		57		155	
Obese class III (≥40·0 kg/m^2)	% of all BMI categories	0·2	0·0, 0·5	0·5	0·3, 0·8	0·5	0·2, 0·9	2·4	1·8, 3·2
	% of obese (≥30·0 kg/m^2)	2·0	0·8, 5·0	2·7	1·4, 4·9	5·3	2·7, 10·3	12·5	9·5, 16·3
	n	5		20		13		87	

higher body weight, in developed countries)[11,12]. Bias could also have been introduced from the different times at which data were collected: for example, the NNS data were collected in all regions between February 1995 and March 1996; whereas the AusDiab data were collected over a 21-month period, with 2 months in each region. Comparable future population BMI studies will be important.

To help overcome potential incomparability between surveys due to both differences in sampling and true changes in population demographics, we performed analyses stratified by key demographic variables. We found that differences in population demographics and the prevalence and intensity of smoking are unlikely explanations of the trends, as we observed similar trends in different age and sex groups, in the Australian-born subgroup (the best available proxy measure for ethnicity) and in populations of never smokers, and for these subgroups between 1980 and 1995 as for 1980 to 2000.

The slowing between 1995 and 2000 in the previously increasing BMI trend may be driven by methodological issues. However, our main conclusions – increasing mean BMI, increasing right skew and large increases in the more

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severe categories of obesity – also hold true for other time period comparisons (1980 to 1989 and 1980 to 1995).

Although self-reported body weight trends continued increasing to 2005[13], the recent slowing in the rate of increase in mean BMI has also been shown for some population groups in New Zealand and the USA[5,14,15] and in a slowing of the annual rate of overweight/obesity increase based on measured BMI in Australian children[16]. The link between the rate of BMI increase and the degree of increase in distributional skew is also supported by New Zealand findings[5].

The data are sourced from five cross-sectional surveys (they do not reflect a cohort of individuals) and describe changing population 'snapshots' of the BMI distribution. Longitudinal analysis of participants in the AusDiab study (1999/2000 to 2005) showed that gain in weight, BMI and waist circumference have been greatest in individuals of normal weight and in younger people from 2000 to 2005[17].

There was some variation in the degree of increase by sex and age groups. The increase was substantially higher at the highest end of the distribution, with relatively less variation in the increase elsewhere, for women aged 35–44 and 55–64 years and men aged 35–44 years. This suggests that not all population subgroups have responded in the same way to the drivers of increased body weight. There was little variation in the increase for men aged 45–54 and 55–64 years.

The increase in right skew has implications for the 'obese' BMI category. The overall prevalence of obesity has increased, but the type of obesity becoming increasingly prevalent is that at the extreme end of the distribution. Similar findings have been shown in the USA[18]. These findings suggest that analyses focusing on the prevalence of total obesity are likely to underestimate the true obesity burden and highlight the importance of monitoring trends across the whole population distribution of BMI.

Challenges remain to understand the drivers of these patterns and to determine the most appropriate response. The major drivers of the BMI distributional shift are likely to be changing dietary and physical activity patterns due to the increasingly obesogenic environment in which we live[5]. Our findings suggest that this environment is not acting equally across the population and that environmental measures to shift BMI distributions leftwards may be insufficient on their own to substantially reduce the burden of obesity[3,19]. Approaches to reducing the burden of obesity need to shift both the BMI distribution leftwards and target the higher BMI subgroups. In addition, it will be important to analyse potential social inequalities in these trends.

In conclusion, the mean BMI of urban Australian adults increased between 1980 and 2000 for all sex and age groups. The increase was fastest during the early 1990s, and thereafter stagnated or declined. Over the 20 years, there were much greater BMI increases at the higher end of the distribution. This resulted in an increasingly skewed distribution, and has resulted in a large increase in the prevalence of obesity. However, it is the prevalence of the more severe levels of obesity that has increased the most. It will be important to monitor the true increase in the burden of obesity by looking at changes in the different classes of obesity and the extent to which obesity interventions shift the BMI distribution leftwards and decrease the skew of the distribution.

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3.1.2 Differential trends in different measures of excess body weight

After conducting the analysis of trends in BMI in Australia, preliminary comparisons of Australian data suggested that between 1989 and 1999/2000 WC had increased further than expected based on increases in BMI. However the methods of measuring WC in the two surveys were different, limiting the value of comparing the data. Thus, we decided to explore this issue with US data, for which there are comparable measurements of WC over time.

While we were conducting the analysis Elobeid et al. (2007) published a report similar to the one we were preparing and also using NHANES data, suggesting that WC had increased at a greater rate than BMI in their US trends analyses (210). However it was unclear if these differences were consistent across different population sub-groups and the range of body weight. Considering that abdominal obesity is more closely linked to adverse cardiovascular outcomes than BMI it is important to know the prevailing sub-group trends in WC as well as BMI.

Here, we determine whether BMI and WC are increasing proportionately amongst population sub-groups, and examine the public health implications of the findings.

Walls HL, Stevenson CE, Abdullah A, Reid CM, McNeil JJ. Peeters A. Comparing trends in body mass index and waist circumference. 2009.

Declaration for Thesis Chapter 3.1.2

Walls HL, Stevenson CE, Abdullah A, Reid CM, McNeil JJ. Peeters A. Comparing trends in body mass index and waist circumference. Submitted.

Declaration by candidate

In the case of Chapter 3.1.2, the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Design, analysis and drafting of manuscript	70%

The following co-authors contributed to the work. Co-authors who are students at Monash University must also indicate the extent of their contribution in percentage terms:

Name	**Nature of contribution**	**Extent of contribution (%) (student co-authors only)**
CE Stevenson	Statistical advice, analysis and drafting of manuscript	N/A
A Abdullah	Drafting of manuscript	5%
CM Reid	Drafting of manuscript	N/A
JJ McNeil	Drafting of manuscript	N/A
A Peeters	Design, analysis and drafting of manuscript	N/A

Candidate signature	**Date**
[redacted]	8 February 2010

Declaration by co-authors

The undersigned hereby certify that:

(1) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors;
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, expect for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (2) granting bodies, (b) the editor or publisher of journals or other publication, and (c) the head of the responsible academic unit; and

(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s):
Department of Epidemiology & Preventive Medicine, Monash University

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant].

Co-author signatures	**Date**
[redacted]	8 Feb 2010
[redacted]	5 February 2010
[redacted]	5/2/10
[redacted]	10/2/10
[redacted]	7/2/2010

Title

Comparing trends in body mass index and waist circumference

Authors

Helen L Walls, Christopher E Stevenson, Asnawi Abdullah, Christopher M Reid, John J McNeil, Anna Peeters

Abstract

Objective

To determine whether body mass index (BMI) and abdominal girth are increasing proportionately amongst different population sub-groups and the range of bodyweight. In addition, we examine the public health implications of these findings.

Design

Three cross-sectional population biomedical surveys of 31311, 10477, and 9950 US individuals.

Subjects

Survey participants were aged ≥20 years, sampled from the civilian, non-institutionalised US population and participated in National Health and Nutrition Examination Studies (NHANES) in 1988-94 (NHANES III), 2001-02, and 2005-06.

Measurements

Measured height, weight and waist circumference (WC) data from each survey. BMI was calculated as weight divided by height (kg/m^2).

Results

Between 1988-94 and 2005-06, BMI increased by an average of 1.8 kg/m^2 and WC by 4.7cm (adjusted for sex, age, race-ethnicity and education). The increase in WC was more than could be attributed simply to increases in BMI. This independent increase in WC (of on average, 0.9cm) was consistent across the different weight groups, sexes, education levels and race-ethnicity groups. It was present in the younger (<50 years) age groups but not in the older age groups (>50 years). Overall, in each BMI category, the prevalence of having a low-risk WC decreased and the prevalence of an increased-risk or substantially-increased-risk WC increased.

Conclusion

Amongst US adults participating in these NHANES studies, the increase in abdominal girth was proportionately greater than the increase in BMI. This difference was largely confined to the younger individuals. These results suggest that the adverse health consequences associated with obesity may be increasingly underestimated by trends in BMI alone. Since abdominal girth is closely linked to adverse cardiovascular outcomes, it is important to know the prevailing trends in both of these parameters.

Key words

Body mass index, waist circumference, trends, risk

Introduction

The nature of excess body weight may be changing over time, to one of greater central adiposity rather than a more peripheral distribution of body fat. Recently, Elobeid et al. reported that WC has been increasing at a greater rate than BMI in their US trends analyses (1). Our analyses in Australia have also suggested similar differences in the rate of change of the BMI and WC distributions, and in their degree of skew (2, 3). However it is unclear if these differences are consistent across different population

sub-groups and the range of body weight. Since abdominal obesity is more closely linked to adverse cardiovascular outcomes than BMI it is important to know the prevailing trends in both of these parameters.

WC measures the central distribution of excess body fat – fat distributed in the abdominal region rather than on the hips and thighs – and is more strongly correlated with metabolic and cardiovascular risk than is BMI (4, 5). Thus, whether the nature of the obesity epidemic is, indeed, changing – to one of greater abdominal obesity, for example – may have important implications for the burden of obesity-related disease. Comparing recent trends in BMI and WC will help elucidate whether the nature of excess body weight is changing over time, and provide insight into whether BMI is capturing risk as well as generally accepted.

The aim of this paper is to analyse the degree to which abdominal overweight is increasing beyond what would be expected from changes in BMI alone. In particular, we have studied these differential changes within different population sub-groups determined by age, sex, education, race-ethnicity and body weight, and suggest possible public health implications of the findings. We analysed changes in WC and BMI between 1988-94 and 2005-06 using the comparable, nationally representative cross-sectional NHANES surveys in the US.

Methods

Samples

This study examined data from three nationally representative cross-sectional surveys of the US population, the National Health and Nutrition Examination Studies (NHANES), conducted by the US National Center for Health and Statistics. The surveys were conducted during the following time periods: 1988 to 1994 (NHANES III), 2001 to 2002,

and 2005 to 2006. Participants in each survey were selected using a multistage, stratified cluster sampling design to be representative of the civilian, non-institutionalized US population. Persons aged 60 and older, African Americans and Mexican Americans were oversampled. After being interviewed in their homes, participants were invited for a clinical examination at a mobile examination centre (MEC), during which time anthropometric data were collected. Only people with data from the MEC examination were included in analyses. The household interview response rates and MEC response rates in the respective surveys were 86% and 78% in NHANES III (1988-94), 84% and 80% in 2001-02, and 80% and 77% in 2005-06. The sample design and weighting methodology across the three surveys was very similar and the studies included 31311, 10477, and 9950 individuals, respectively. For this analysis, we excluded participants with missing height, weight, waist circumference and education data, aged <20, and women recorded as pregnant at the time of survey, leaving 15349, 4343 and 4176 records for analysis.

Measurement procedures

Body measurements were performed using the Centers for Disease Control and Prevention standardized methods and equipment. Participants were measured by two health technicians (an examiner and a recorder). The examiner and recorder were trained using the Centers for Disease Control and Prevention standard procedures for obtaining measurements. The recorder assisted the examiner with positioning of the participants and the reading process to minimize the errors (6).

WC was measured using a steel measuring tape to the nearest 0.1cm at the high point of the iliac crest at minimal respiration when the participant was in a standing position. The examiner stood behind the participant, palpated the hip area for the right iliac crest, marked a horizontal line at the high point of the iliac crest, and crossed the line to indicate the midaxillary line of the body. The examiner then stood on the participant's right side and placed the measuring tape around the trunk in a horizontal

flat surface at the level marked on the right side of the trunk. The recorder observed the participant to ensure that the tape was parallel to the floor and that the tape was snug but did not compress the skin (6).

Weight was measured on a Toledo self-zeroing weight scale (Toledo Scale, Inc, Columbus, OH), and height was measured with a stadiometer to the nearest millimeter (6).

We applied the following cut-offs to the WC measurements to classify people into 'low risk' (<94cm for men; <80cm for women), 'increased risk' (≥94-<102cm for men; ≥80-<88cm for women), and 'substantially increased risk' (≥102cm for men, ≥88cm for women) (7). BMI was calculated as weight divided by height (kg/m^2). We applied standard cut-offs to the BMI scores to classify people into underweight (<18.5 kg/m^2), normal weight (18.5-24.99 kg/m^2), overweight (25-29.99 kg/m^2) and obese (≥ 30 kg/m^2) (8).

Other variables

In addition to BMI and WC, other variables included in this paper are sex, age group (categorized as age 20-29, 30-39, 40-49, 50-59, 60-69 and 70+), education (completed high school and did not complete high school), and race-ethnicity (non-Hispanic White, non-Hispanic Black, Mexican American, Other race[1] (incl. mixed race) and Other Hispanic). We also looked at the following two time periods: 1988-94 to 2001-03 and 2001-03 to 2005-06.

[1] The 'Other race' category is not strictly comparable between NHANES II and the other two surveys. For NHANES III those who indicated more than one race (multiracial) and then selected a main race as black (non-Hispanic) or white (non-Hispanic) were recorded into those respective categories. This recoding was not carried out in the data of the subsequent surveys. However the number recoded was small and the effect on our analyses negligible.

Analysis

We calculated the mean WC and BMI in each survey. Analyses were stratified by sex, age group (age was categorised in 10-year groups from age 20), and race-ethnicity (race-ethnicity was categorised as 'Non-Hispanic White', 'Non-Hispanic Black, "Mexican-American, 'Other race [including mixed race]', 'Other Hispanic') and analysed in SAS (SAS version 9.1.3. SAS Institute Inc). Linear regression was used to analyse the changes in the level of WC for a given level of BMI over time. Logistic regression was used to calculate the odds of being in particular WC categories in the two time periods, for particular BMI categories. The analysis of the survey data took into account the complex, multistage, cluster sampling design.

Results

Across the three surveys, the number of participants in each sex and race group ranged from 47 to 3376. Table 1 illustrates the participants' demographic characteristics. The distributions of mean age and sex were similar in each of the surveys, as were the response rates. The distribution of race varied between surveys. Overall, the proportion of Non-Hispanic Whites increased and the proportion of Non-Hispanic Blacks and Mexican Americans decreased. The proportion of people who had completed high school also increased across the three surveys.

Between 1988-94 and 2005-06, increases in both BMI and WC were observed. Mean BMI increased from 27.1 kg/m^2 to 28.7 kg/m^2, and mean WC increased from 93.3 cm to 98.1 cm.

After adjusting for sex, age, race-ethnicity and education, BMI increased between 1988-94 and 2005-06 by 1.8 kg/m^2 and WC by 4.7 cm (Table 2). After additional

adjustment for BMI, WC was found to have increased on average by 0.9 cm. In contrast, after additional adjusting for WC, there was no significant change in BMI.

There were independent increases in WC in all three BMI categories (Table 3). As a result, on average in 2005-06 individuals in the normal-weight, overweight and obese groups had waists 0.6 cm, 0.8 cm and 1.0cm larger than individuals in the normal-weight, overweight and obese groups in 1988-94, respectively. There were similar independent increases of WC in males and females, people with high and low education levels, and in the three most common race-ethnicity groups. We tested interactions between changes over time and BMI categories, age groups, sex, education level and race-ethnicity. Interactions with age group and time period were found to be statistically significant. There were independent increases in WC in those aged 20-29, 30-39 and 40-49 but not those aged 50-59, 60-69 and 70+. The consequence of this was that on average in 2005-06 individuals aged 20-29, 30-39 and 40-49 had waists 1.8 cm, 1.2 cm and 0.7 cm larger than their counterparts in 1988-94. There was a significant independent increase in WC in the first time period (1988-94 to 2001-03) but not in the second (2001-03 to 2005-06), possibly due to the shorter second time period. However after taking into account the length of these time periods, there was a suggestion of a greater annual change in the later time period (Table 3).

The consequence of these changes was that within each of the commonly used categories of normal-weight, overweight and obesity the level of metabolic risk increased for males and females aged <50 years but not for those aged ≥50 years (Figure 1). In 1988-94 of those aged <50 years in the normal-weight category, 3.0% of men and 29.5% of women had an increased-risk or substantially-increased-risk WC, but in 2005-06 this had increased to 5.9% and 43.0% respectively. Similarly, while the proportion of males and females aged <50 years with BMI<30 (non-obese) in 1988-94 with a substantially-increased-risk WC was 5.1% and 25.2%, by 2005-06 it had

increased to 11.5% and 33.4%, respectively. The proportion of males and females aged <50 years with BMI>30 (obese) *without* a substantially-increased-risk WC decreased from 11.6% and 1.9% in 1988-94 to 6.9% and 0.6% in 2005, respectively.

After adjusting for age, race-ethnicity and education, the implications of these changes in prevalence were that for overweight women there was a 2-fold increase between 1988-94 and 2005-06 in the odds of being in the increased-risk or substantially-increased-risk WC categories. There were also greater odds of being in the increased-risk or substantially-increased-risk WC categories for normal-weight and overweight men and normal-weight women; however these results were not statistically significant (although borderline for normal-weight women).

Discussion

Between 1988-94 and 2005-06 there were increases in both the BMI and WC of American adults. BMI increased by 1.8 kg/m^2 and WC by 4.7cm. But there was also an independent increase in WC over and above that for BMI, of on average 0.9 cm (adjusting for sex, age, race-ethnicity and education). The independent increase in WC occurred for each of the BMI categories, in both sexes, education levels and in the three most common race-ethnicity groups. However the independent increases occurred in younger people (aged <50) but not in older people (aged >50). There was a suggestion of a greater increase in the more recent time period (2001-03 to 2005-06), but this was not statistically significant.

The consequence of these differential changes in weight and WC was that within each of the normal-weight, overweight and obese BMI categories the level of metabolic risk may have increased for males and females aged <50 years. Overall, in each BMI category, the prevalence of having a low-risk WC decreased and the prevalence of an

increased-risk or substantially-increased-risk WC increased. These changes affected the normal-weight category as well as the overweight and obese BMI categories.

Our findings are consistent with a number of studies that have suggested an independent increase in WC over and above that for BMI (2, 9, 10, 11, 12, 13). In a study of English adults (aged 18 to 64) between 1993/4 and 2002/3, Wardle et al. (2008) reported "dramatic increases" in central adiposity across the range of body weights. They found little change in the BMI of slim adults between 1993/4 and 2002/3 but a 2cm increase in the WC of even the slimmest women (10^{th} percentile) (12). In studies of US and German adults, Li et al. (2007) and Liese et al. (2001) found that relative increases in the prevalence of overall obesity as measured by BMI was less than the relative increases in abdominal obesity as measured by WC (9, 13). In Finnish adults, Lahti-Koski et al. (2000; 2007) also found greater relative increases in mean WC than mean BMI (10, 11). Li et al. (2007) found that the difference was particularly pronounced among men and women aged 20 to 29 years and among those of normal-weight BMI, and that for the total population, the rate of increase was greater in the later time period (1999/00 to 2003/04) than the earlier time period (1988/94 – 1999/00) (13). However differences in the percentage change in BMI and WC in fact contribute little to informing whether one indicator has moved independently further than the other, due to the nature of the relationship between BMI and WC. In our analysis, whilst there was a marginally greater percentage change in BMI (5.9%) than WC (5.1%), we demonstrated that increases in WC were greater than expected based on increases in BMI. Elobeid et al. (2007) conducted the only study to specifically look at increases in WC independent of increases in BMI. The strengths of our study are that we looked at the increase in WC independent of increases in BMI over a range of demographic groups, and that we quantified these absolute changes in WC.

One of the limitations of the current study is the short time period between the two more recent surveys, rendering it difficult to make clear inferences about changes over

time. There were no differences in sampling design between the surveys and the survey response rates were similar, reducing the impact of bias from these sources. But the results are based on data are sourced from three cross-sectional surveys (they do not reflect a cohort of individuals) and describe changing population 'snapshots' of the BMI and WC distribution. That the mean WC values are close to the cut-off values for abdominal obesity could explain why a small increase in WC could lead to a large increase in the prevalence of increased risk WC (13). However this appears not to be the case in this study as we have used linear regression to explore the continuous changes in WC over and above BMI.

It has been postulated that population-level changes such as a changing population ethnic-makeup or other demographic factors could explain the changes in body fat distribution, however our analyses suggest that that is not the case as we have obtained similar results even when stratifying by demographic factors such as race-ethnicity. Declining physical activity levels may be an important contributor to this phenomenon [14]. Whilst the evidence base is sparse, other suggested changes include sleep deprivation, stress (suggested to cause abdominal fat deposition as a result of increased cortisol levels), endocrine disruptors and certain pharmaceuticals (14).

Of particular interest is that the changes are occurring across race, education classes, in both sexes, and across the range of body weights, including in people considered 'normal weight'. The nature of the changes suggests that using solely BMI to monitor the impact of increasing weight status in the population may lead to an underestimation of the associated health burden. That the independent increase in WC has been shown in people aged <50 years but not older age groups suggests that the changes in our environment have impacted more on younger adults, or that they are the result of more recent environmental changes.

In conclusion, there were increases in both BMI and WC in American adults between 1988-94 and 2005-06, but WC increased over and above the increase in BMI. This independent increase in WC occurred across the three BMI categories, in both sexes, education levels and in the three most common race-ethnicity groups. To date these independent changes have been restricted to younger people. The consequence of these changes is that in each of the BMI categories the level of metabolic risk has increased. This has important implications for the measurement of risk associated with obesity.

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Figure legends

Table 1. Demographic information from the three surveys

Table 2. Changes in WC and BMI between 1988-94 and 2005-06 using linear regression analysis

2A. Changes in WC

2A. Changes in BMI

Table 3. Changes in WC independent of BMI between 1988-94 and 2005-06 (unless otherwise specified) using linear regression analysis

Table 4. Odds[1] of being in WC categories of ≥94cm for men or ≥88cm for women in 2005-06 compared to 1988-94 for particular BMI categories

Figure 1. Changes in proportion of WC categories within BMI categories between 1988-94 and 2005-06 for males and females aged <50 years and ≥50 years

1A. Males aged <50 years

1B. Females aged <50 years

1C. Males aged ≥50 years

1D. Females aged ≥50 years

Table 1. Demographic information from the three surveys

	1988-94 NHANES III	2001-02	2005-06
No. included in this study (N)	15 349	4343	4176
Response rate[1]	78%	80%	77%
Age (yrs) – Mean, (SD)	47.9 (0.5)	49.3 (0.5)	49.2 (0.8)
Sex – % male, (SD)	47.9 (0.9)	50.3 (0.5)	51.3 (0.7)
Education – % completed high school, (SD)	59.8 (1.0)	70.3 (1.1)	73.0 (1.6)
Race-ethnicity			
% Non-Hispanic White, (SD)	41.6 (1.6)	52.5 (3.0)	50.3 (3.9)
% Non-Hispanic Black, (SD)	27.6 (1.3)	19.8 (3.3)	23.1 (3.2)
% Mexican-American, (SD)	26.8 (1.0)	21.0 (2.5)	19.5 (1.7)
% other race[2]**, (SD)**	1.5 (0.2)	2.6 (0.4)	3.9 (0.4)
% other Hispanic, (SD)	2.5 (0.5)	4.1 (1.1)	3.1 (0.7)

[1]The proportion of the selected sample that attended a clinical examination at a mobile examination centre (MEC).

[2] Includes multi-racial.

Table 2. Changes in WC and BMI between 1988-94 and 2005-06 using linear regression analysis

2A. Changes in WC

Model	Change in WC (cm)
Adjusting for age, sex, race-ethnicity & education	4.73 (95% CI 3.85 – 5.61)
As above plus adjustment for BMI	0.86 (95% CI 0.53 – 1.19)

2A. Changes in BMI

Model	Change in BMI (kg/m^2)
Adjusting for age, sex, race-ethnicity & education	1.75 (95% CI 1.43 – 2.09)
As above plus adjustment for WC	-0.02 (95% CI -0.15 - 0.11)

Table 3. Changes in WC independent of BMI between 1988-94 and 2005-06 (unless otherwise specified) using linear regression analysis

		Change in WC (cm) (95% CI)
BMI categories	**Normal weight population (BMI 18.5-25 kg/m^2)**	0.62 (0.25 – 0.98) *
	Overweight population (BMI 25-30 kg/m^2)	0.75 (0.29 – 1.16) *
	Obese population (BMI≥30 kg/m^2)	0.95 (0.43 – 1.48) *
Age group β	**20-29**	1.84 (1.38 – 2.30) *
	30-39	1.24 (0.74 – 1.73) *
	40-49	0.72 (0.15 – 1.28) *
	50-59	-0.02 (-0.75 – 0.70)
	60-69	-0.25 (-0.23 – 0.73)
	70+	0.67 (-0.12 – 1.47)
Sex	**Males**	0.79 (0.51 – 1.07) *
	Females	0.72 (0.13 – 1.30) *
Education	**Did not complete high school**	0.68 (0.18 – 1.17) *
	Completed high school	0.92 (0.60 – 1.25) *
Race-ethnicity	**Non-Hispanic White**	0.89 (0.33 – 1.45) *
	Non-Hispanic Black	0.50 (0.13 – 0.87) *
	Mexican American	0.95 (0.52 – 1.39) *
	Other race (incl. mixed race)	0.93 (-0.60 – 2.46)
	Other Hispanic	0.88 (-0.07 – 1.82)
Time periods β	**1988-94 – 2001-03**	0.58 (0.25 – 0.91) *
	Annual change	0.05 (0.02 – 0.08) *
	2001-03 – 2005-06	0.30 (-0.09 – 0.70)
	Annual change	0.09 (-0.03 – 0.2)

Adjustment for age, sex, race-ethnicity, education and BMI where relevant.

β Indicates that the test for interaction with change of time suggested significance for this category.

* Indicates a statistically significant increase in WC independent of BMI.

Table 4. Odds[1] of being in WC categories of ≥94cm for men or ≥88cm for women in 2005-06 compared to 1988-94 for particular BMI categories

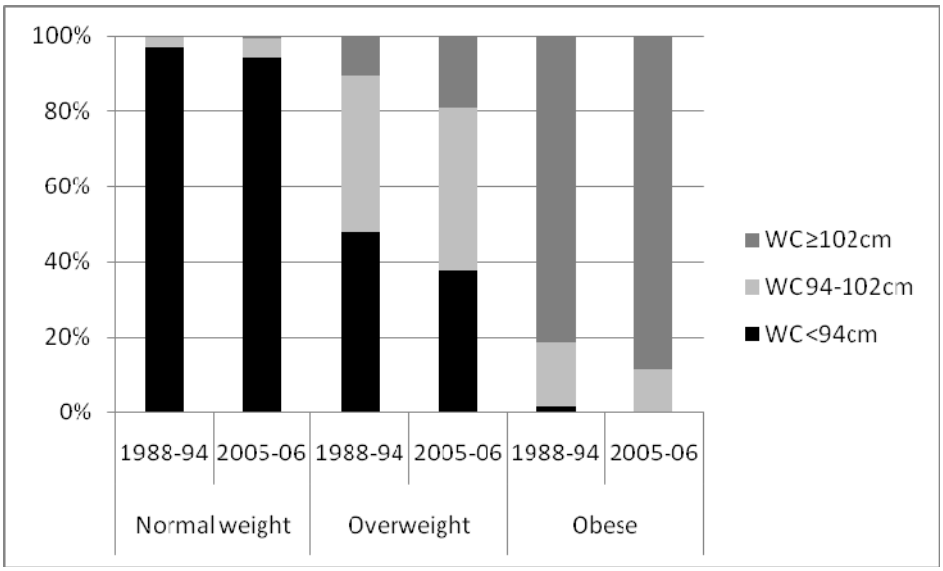
BMI category	Males	Females
Normal-weight	1.10 (0.82, 1.46)	1.27 (1.00, 1.63)
Overweight	1.18 (0.94, 1.49)	*2.00 (1.16, 3.45)

[1] Adjusted for age, race-ethnicity and education

* Indicates a statistically significant result.

Figure 1. Changes in proportion of WC categories within BMI categories between 1988-94 and 2005-06 for males and females aged <50 and ≥50 years

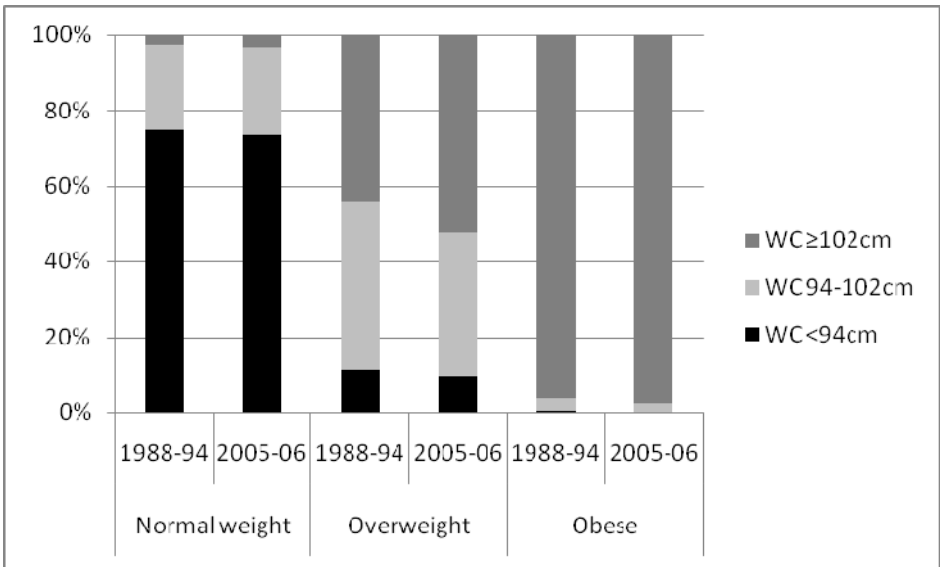
1A. Males aged <50 years



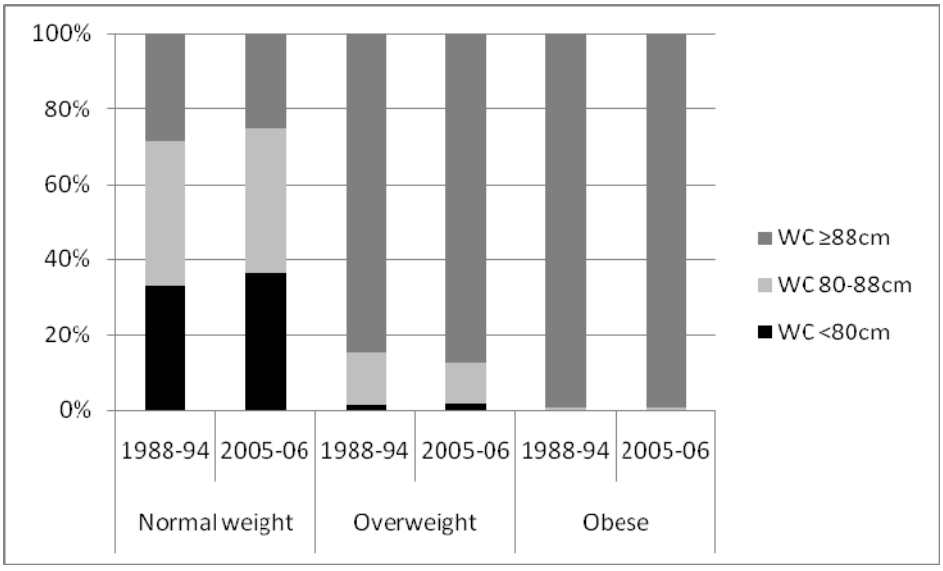
1B. Females aged <50 years



1C. Males aged ≥50 years



1D. Females aged ≥50 years



3.2 Obesity in a low-income country – the case of Vietnam

The prevalence of obesity has increased in almost all countries worldwide, including the poorest (1). In low-income countries, the rise in obesity has been linked to what has been called the 'nutrition transition' (closely related to the demographic and epidemiologic transitions (236)), which has led to decreased physical activity and the increased consumption of energy-dense food (236-238). While obesity was historically considered a symbol of wealth in many of these countries, there appears to be an emerging shift to greater overweight and obesity among the poorest in these countries (238, 239). The paradoxical coexistence of obesity and malnutrition has been widely documented – the diets of people living in poverty often contain adequate kilocalories to meet or exceed energy requirements but lack the dietary quality needed to promote optimal health (125, 240). Weight gain in low-income countries has also been more characteristic of urban rather than rural areas (238).

Vietnam (officially the Socialist Republic of Vietnam) is a South-East Asian country of more than 87 million people (241). Historically, Vietnam has been an agricultural civilisation based on wet rice cultivation. The Vietnam War (1959-1975) destroyed much of Vietnam's economy, and since taking power, the Government has created a planned economy – from 1986 incorporating elements of a market economy as part of an economic reform package called 'doi moi' (212). Since the 1990s Vietnam has achieved high annual growth in GDP, and is one of the world's fastest growing economies (242). However it remains relatively poor, with a per capita income in 2008 of about US$1024 (243).

Analysis of the body weight data of a low-income country such as Vietnam is important for the country in question, and also important in terms of the deeper understanding of the global 'obesity epidemic' to which it contributes.

3.2.1 Body weight in urban Hanoi

The prevalence of overweight and obesity in Vietnam has historically been very low, but this is changing rapidly. Nationally, the prevalence of overweight and obesity in adults more than doubled between 1992 and 2002 and the prevalence of underweight declined

significantly (211). A cross-sectional survey in 2004 of urban adults in Ho Chi Minh City (HCMC), Vietnam's largest city with a population of approximately seven million, found that almost a third of adults were overweight or obese based on Asian-specific BMI cut-points (212). Such an analysis is also of interest for Hanoi, Vietnam's capital with a population of approximately three million and, like HCMC, a large and dynamic city in a country that has undergone rapid economic and social change over recent years. These changes are likely to have contributed to a decrease in infectious diseases and malnutrition and an increase in risk factors for chronic disease, such as obesity (212).

Here, we determine the prevalence of BMI and WC categories in adults of urban Hanoi in 2004, using Asian- and Vietnamese-specific cut-points as well as commonly-used cut-points arguably more appropriate for Caucasians.

Walls HL, Peeters A. Pham TS, Nguyen NQ, Nguyen TTH, Do DL, Nguyen LV, Pham GK, Reid CM. Prevalence of underweight, overweight and obesity in urban Hanoi, Vietnam. Asia Pac J Clin Nutr. 2009;18:234-9

Declaration for Thesis Chapter 3.2.1

Walls HL, Peeters A. Pham TS, Nguyen NQ, Nguyen TTH, Do DL, Nguyen LV, Pham GK, Reid CM. Prevalence of underweight, overweight and obesity in urban Hanoi, Vietnam. *Asia Pac J Clin Nutr*. 2009; 18:234-9

Declaration by candidate

In the case of Chapter 3.2.1, the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Design, analysis and drafting of manuscript	90%

The following co-authors contributed to the work. Co-authors who are students at Monash University must also indicate the extent of their contribution in percentage terms:

Name	**Nature of contribution**	**Extent of contribution (%) (student co-authors only)**
A Peeters	Design and drafting of manuscript	N/A
TS Pham	Design and execution of survey and verification of manuscript	N/A
NQ Nguyen	Design and execution of survey and verification of manuscript	N/A
TTH Nguyen	Design and execution of survey and drafting of manuscript	N/A
DL Do	Design of survey and verification of manuscript	N/A
LV Nguyen	Advised survey design and verified manuscript	N/A
GK Pham	Advised survey design and verified manuscript	N/A
CM Reid	Design of survey and drafting of manuscript	N/A

Candidate signature	**Date**
[redacted]	8 February 2010

Declaration by co-authors

The undersigned hereby certify that:

(1) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors;

(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, expect for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (2) granting bodies, (b) the editor or publisher of journals or other publication, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s):
Department of Epidemiology & Preventive Medicine, Monash University
Vietnam National Heart Institute, Bachmai Hospital

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant].

Co-author signatures	**Date**
[redacted]	7/2/2010
[redacted]	5/2/10

(1) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors;
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, expect for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (2) granting bodies, (b) the editor or publisher of journals or other publication, and (c) the head of the responsible academic unit; and
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[redacted]	9/2/2010
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[redacted]	9/2/2010
[redacted]	9/2/2010
[redacted]	9/2/2010

Short Communication

Prevalence of underweight, overweight and obesity in urban Hanoi, Vietnam

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This study aims to assess the prevalence of underweight, overweight and obesity among adults in urban Hanoi, Vietnam; and compare these results to previous estimates among adults in urban Ho Chi Minh City. Survey participants were residents in urban Hanoi, Vietnam and aged between 25-74 years. Data from a cross-sectional biomedical survey conducted in 2004 were collected; which included a questionnaire, physical examination and blood tests. The age-standardised prevalence of overweight and obesity in 2004, using Asian-specific body mass index cut-offs, were 28.6% and 2.1%, respectively. The prevalence of overweight/obesity (combined) was similar in males (29.7%) and females (31.5%), and generally increased with age. The prevalence of overweight/obesity was considerably lower if the standard cut-off values of the World Health Organization were used. The age-standardised prevalence of underweight was 13.3%; and that of 'increased risk'/'substantially increased risk' waist circumference (combined) was 27.9% in males and 25.7% in females, respectively. Almost one in three adults in urban Hanoi were overweight or obese in 2004 and more than one in ten were underweight (based on Asian-specific cut-off values). This prevalence of overweight/obesity is similar to that for adults in urban Ho Chi Minh City, but the prevalence of underweight is lower. While low body weight remains a concern, overweight and obesity are an increasing problem for urban Vietnamese adults.

Key Words: underweight, overweight, obesity, BMI, Vietnam

INTRODUCTION

Overweight and obesity are increasing in countries worldwide, developing and developed alike.[1] Analysis of body weight trends in Vietnam, a populous country with a historically low overweight/obesity prevalence, found that the prevalence of overweight and obesity in adults has more than doubled between 1992 and 2002 and the prevalence of underweight declined significantly.[2] The prevalences of overweight/obesity in this study were arguably low as they were based on body mass index (BMI) cut-off values recommended by the World Health Organization (WHO) for Western populations rather than values appropriate for Asian populations. However a cross-sectional survey conducted in 2004 of urban adults in Ho Chi Minh City (HCMC), Vietnam's largest city with a population of approximately seven million, found that almost a third of adults were overweight or obese when using Asian-specific cut-off values.[3] Analysis of such prevalences in Hanoi, Vietnam's capital with a population of approximately three million, has not yet been undertaken. But such an analysis is of interest as Hanoi, like HCMC, is a large and dynamic city in a country that has undergone rapid economic and social change over the past fifteen years. The introduction of social and economic policy reforms and the lifting of the embargo placed on Vietnam by the US government catalysed these changes; changes that are likely to have contributed to a decrease in infectious diseases and malnutrition and an increase in risk factors for chronic disease, such as obesity.[3]

Determining the prevalence of underweight, overweight and obesity in urban Hanoi and comparing these prevalences to those for HCMC will contribute to the evidence regarding body weight in Vietnam. Here, we determine the prevalence of BMI and waist circumference (WC) categories in adults of urban Hanoi in 2004, using Asian and Vietnamese-specific cut-offs, as well as commonly-used cut-off values arguably more appropriate for Caucasians.

MATERIALS AND METHODS

This study analyses data from a population-based survey, the *2004 Epidemiological survey on heart failure, its main causes and some of its risk factors in northern Vietnam*.[4] The aim of this study was to describe the prevalence of heart failure and its risk factors in the northern provinces of Vietnam. The target population was persons

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aged 25 and older living in northern Vietnam, from which the target sample was selected using a randomised cluster sampling method, at the individual level. Vietnam is divided into 58 provinces and five centrally-controlled municipalities; each province is further divided into districts. Sampling in this study included three randomly-selected districts in each of the four provinces of northern Vietnam: Hanoi (urban), Thai Binh (lowlands), Nghe An (coastal) and Thai Nguyen (midlands). The response rate in Hanoi was 73.9%, 1066 Hanoi residents and 3780 outside of Hanoi (the overall response rate for all regions combined was 84.1%). Participants were administered a questionnaire, physical examination and blood tests. Weight, height and WC were measured by trained personnel and measured in accordance to WHO guidelines, such that height was measured without shoes, weight with only light clothes, and waist circumference was measured at the part of the trunk midway between the lower costal margin (bottom of lower rib) and the iliac crest (top of the pelvic bone) with the person standing. The measurement was taken at the end of a normal expiration.[5]

Definitions of underweight, overweight and obesity

Two sets of BMI and WC cut-off values were used in this analysis, values proposed to be appropriate for Vietnamese and Asian populations, as well as widely-used values which are often considered more appropriate for Caucasians. This second set of values will be referred to as the "comparator" cut-off values.

The "comparator" BMI cut-off values used in this analysis to classify people into underweight, normal weight, overweight and obesity are WHO standard cut-offs, as follows: underweight (<18.5 kg/m^2), normal weight (18.5-24.99 kg/m^2), overweight (25-29.99 kg/m^2) and obese (≥ 30 kg/m^2).[6] The Asian-specific cut-offs are as follows: underweight (<18.5 kg/m^2), normal weight (18.5-22.99 kg/m^2), overweight (23-27.49 kg/m^2) and obese (≥ 27.5 kg/m^2).[6] These Asian-specific cut-off values are consistent with what the WHO has suggested as overweight and obesity 'action trigger points for public health action' for Asian populations.[7] The cut-off values for underweight and normal weight are the same as in the "comparator" definition but the cut-off values for determining overweight and obesity are lower.

Cut-off values for the categorisation of WC are less defined than those for BMI. We have chosen common-used "comparator" values described by Lean et al. (1995), which classify people into 'low risk' (<94 cm for men; <80 cm for women), 'increased risk' (≥94-<102 cm for men; ≥80-<88 cm for women), and 'substantially increased risk' (≥102 cm for men, ≥88 cm for women).[8] As for Asian-specific WC cut-offs, we have chosen to use those proposed by Cuong et al. (2007) as optimal for people of Vietnamese ethnicity.[3] This definition also allows easy comparison with prevalence estimates from HCMC. The Vietnamese-specific WC cut-offs classify people into 'low risk' (<79 cm for males; <77 cm for females), 'increased risk' (79-<86 cm for males, 77-<86 cm for females), and 'substantially increased risk' (≥86 cm for males and females).[3]

Analysis

Analysis of BMI and WC were stratified by sex and age groups (age categorised in 10-year groups between ages 25 and 74 years). The statistical software, Stata (Intercooled Stata, Version 9.0, StataCorp) was used for the analysis. To account for different age structures in the comparison population from HCMC, total-population prevalence estimates were age-standardised based on data from the 1999 census (*Population & Housing Census 1999*), using the direct standardisation method. Cuong et al. (2007) used the same standard population in their HCMC study of body weight.[3] The prevalence of the BMI and WC categories were calculated for each survey, using both Asian and Vietnamese-specific BMI and WC cut-off values as well as higher "comparator" values, as previously described.

RESULTS

The number of participants in each sex and age group ranged from 52 to 168. The mean BMI was 22.0 kg/m^2 and the mean WC was 73.8 cm. Males accounted for a smaller proportion (37.0%) than females (Table 1).

Tables 2 and 3 illustrate the prevalence of the BMI and WC categories in 2004. Overall the age-standardised prevalence of underweight, overweight and obesity using Asian-specific BMI cut-off values were: for males 14.5%, 27.5% and 2.2%; and for females 12.6%, 29.4% and 2.1%. However the prevalence of overweight and obesity using "comparator" cut-off values were: for males 13.7% and 0.3%, and for females 12.2% and 0.5% (the prevalence of underweight was the same). The prevalence of underweight ranged from 2.0% for females aged 55-64 to 23.1% in males aged 65-74, and the prevalence of overweight/obesity (combined) using Asian-specific cut-off

Table 1. Demographic information from the 2004 survey population.

Characteristics of the 2004 survey population	
Number (n)	978
Response rate	73.9%
Mean age (years), (SD)	
25-34	29.8 (2.9)
35-44	40.2 (2.9)
45-54	48.9 (2.9)
55-64	59.6 (2.9)
65-74	69.8 (2.9)
Percentage of sample represented by age group (%) (% in 1999 Census data)	
Males	
25-34	15.7 (38.3)
35-44	21.8 (30.0)
45-54	31.3 (14.8)
55-64	17.1 (9.4)
65-74	14.3 (7.5)
Females	
25-34	18.9 (35.1)
35-44	25.0 (29.1)
45-54	27.3 (15.6)
55-64	16.3 (11.1)
65-74	12.5 (9.1)
Mean BMI (SD)	22.0 (2.9)
Range of BMI (lowest, highest)	14.1, 32.9
Mean WC (SD)	73.8 (8.0)
Range of WC (lowest, highest)	52.0, 102.5101
% male	37.0%

Table 2. Age-specific prevalence (%) of BMI categories in 2004 (with 95% confidence intervals) for males and females

	Age	Asian-specific BMI cut-offs		'Comparator' BMI cut-offs	
	(years)	Males	Females	Males	Females
Underweight	25-34	21.1 (12.3 – 33.6)	22.4 (15.7 – 30.9)	21.1 (12.3 – 33.6)	22.4 (15.7 – 30.9)
	35-44	8.9 (4.3 – 17.5)	5.8 (3.1 – 10.9)	8.9 (4.3 – 17.5)	5.8 (3.6 – 10.9)
	45-54	10.6 (6.1 – 17.8)	4.8 (2.4 – 9.3)	10.6 (6.1 – 17.8)	4.7 (2.4 – 9.3)
	55-64	6.5 (2.4 – 16.0)	2.0 (0.5 – 7.8)	6.5 (2.4 – 16.0)	2.0 (0.5 – 7.7)
	65-74	23.1 (13.6 – 36.5)	20.9 (13.1 – 31.3)	23.1 (13.6 – 36.5)	20.8 (13.1 – 31.3)
	Total†	14.5 (10.0 – 19.1)	12.6 (9.5 – 15.7)	14.5 (10.0 – 19.1)	12.6 (9.5 – 15.7)
Normal weight	25-34	54.4 (41.4 – 66.8)	59.5 (50.3 – 68.0)	64.9 (51.7 – 76.2)	73.3 (64.5 – 80.6)
	35-44	64.6 (53.4 – 74.3)	62.3 (54.4 – 69.7)	79.8 (69.4 – 87.2)	81.8 (74.9 – 87.2)
	45-54	49.6 (40.4 – 58.7)	45.2 (37.9 – 52.8)	72.3 (63.6 – 80.0)	69.6 (62.3 – 76.1)
	55-64	50.0 (37.8 – 62.3)	49.0 (39.3 – 58.7)	77.4 (65.3 – 86.2)	75.0 (65.6 – 82.5)
	65-74	50.0 (36.7 – 63.3)	45.5 (34.7 – 56.7)	61.5 (47.7 – 73.7)	64.9 (53.7 – 74.8)
	Total†	55.8 (49.7 – 61.9)	55.9 (51.5 – 60.3)	71.5 (65.9 – 77.1)	74.7 (71.0 – 78.6)
Overweight	25-34	22.8 (13.7 – 35.5)	18.1 (12.1 – 26.2)	14.0 (7.2 – 25.7)	4.3 (1.8 – 10.0)
	35-44	24.1 (15.9 – 34.7)	31.2 (24.3 – 38.9)	11.4 (6.0 – 20.5)	12.3 (8.0 – 18.6)
	45-54	38.1 (29.6 – 47.4)	43.5 (36.1 – 51.1)	15.0 (9.5 – 22.9)	25.0 (19.0 – 32.1)
	55-64	38.7 (27.4 – 51.4)	42.0 (32.7 – 51.9)	16.1 (8.9 – 27.5)	20.0 (13.3 – 29.0)
	65-74	26.9 (16.6 – 40.5)	31.2 (32.7 – 51.9)	15.4 (7.9 – 27.9)	13.0 (7.1 – 22.5)
	Total†	27.5 (22.2 – 32.8)	29.4 (25.6 – 33.1)	13.7 (9.5 – 18.0)	12.2 (9.7 – 14.7)
Obese	25-34	1.8 (0.2 – 11.5)	0.0	0.0	0.0
	35-44	2.5 (0.6 – 9.6)	0.7 (0.1, 4.5)	0.0	0.0
	45-54	1.8 (0.4 – 6.8)	6.6 (3.7 – 11.5)	1.8 (0.4 – 6.8)	0.6 (0.1 – 4.1)
	55-64	4.8 (1.6 – 14.0)	7.0 (3.4 – 14.0)	0.0	3.0 (1.0 – 8.9)
	65-74	0.0	2.6 (0.7 – 9.8)	0.0	1.3 (0.2 – 8.7)
	Total†	2.2 (0.4 – 3.9)	2.1 (1.2 – 3.0)	0.3 (-0.1 – 0.6)	0.5 (0.1 – 0.9)

Abbreviations: BMI, body mass index; WHO, World Health Organization
† Age-standardisation (direct method) based on Vietnamese National Census April 1999

Table 3. Age-specific prevalence (%) of WC categories in 2004 (with 95% confidence intervals) for males and females

	Age	Vietnamese-specific WC cut-offs		'Comparator' WC cut-offs	
	(years)	Males	Females	Males	Females
Low risk	25-34	82.5 (70.3 – 90.3)	88.8 (81.6 – 93.4)	100.0	97.4 (92.3 – 99.2)
	35-44	73.4 (62.6 – 82.0)	79.2 (72.1 – 84.9)	98.7 (91.5 – 99.8)	94.2 (89.1 – 96.9)
	45-54	61.1 (51.8 – 69.6)	55.4 (47.8 – 62.7)	98.2 (93.2 – 99.6)	73.8 (66.6 – 79.9)
	55-64	53.2 (40.8 – 65.3)	53.0 (43.2 – 62.6)	100.0	70.0 (60.3 – 78.2)
	65-74	65.4 (51.6 – 77.0)	54.6 (43.4 – 65.3)	100.0	66.2 (55.0 – 75.9)
	Total†	72.1 (67.0 – 77.2)	74.3 (71.0 – 77.7)	99.4 (98.5 – 100.0)	87.4 (85.2 – 89.7)
Increased risk	25-34	8.8 (3.7 – 19.5)	11.2 (6.6 – 18.4)	0.0	2.6 (0.8 – 7.7)
	35-44	22.8 (14.8 – 33.4)	20.8 (15.1 – 27.9)	1.3 (0.2 – 8.5)	5.8 (3.1 – 10.9)
	45-54	28.3 (20.8 – 37.3)	36.9 (29.9 – 44.5)	0.9 (0.1 – 6.1)	20.2 (14.8 – 27.0)
	55-64	32.4 (21.8 – 44.9)	35.0 (26.3 – 44.8)	0.0	24.0 (16.6 – 33.4)
	65-74	17.3 (9.2 – 30.1)	35.1 (25.3 – 46.3)	0.0	31.2 (21.8 – 42.3)
	Total†	19.0 (14.7 – 23.3)	22.4 (19.1 – 25.7)	0.5 (-0.3 – 1.3)	10.8 (8.7 – 13.0)
Substantially increased risk	25-34	8.8 (3.7 – 19.5)	0.0	0.0	0.0
	35-44	3.8 (1.2 – 11.2)	0.0	0.0	0.0
	45-54	10.6 (6.1 – 17.8)	7.7 (4.5 – 12.9)	0.9 (0.1 – 6.1)	6.0 (3.2 – 10.7)
	55-64	14.5 (7.7 – 25.7)	12.0 (6.9 – 20.0)	0.0	6.0 (2.7 – 12.7)
	65-74	17.3 (9.2 – 30.1)	10.4 (5.3 – 19.5)	0.0	2.6 (0.7 – 9.8)
	Total†	8.9 (5.5 – 12.2)	3.3 (2.2 – 4.3)	0.1 (-0.1 – 0.4)	1.7 (1.0 – 2.5)

Abbreviations: WC, waist circumference; WHO, World Health Organization
† Age-standardisation (direct method) based on Vietnamese National Census April 1999

values ranged from 18.1% for females aged 25-34 to 50.1% in females aged 45-54.

Overall the age-standardised prevalence of low-risk, increased-risk and substantially-increased risk WC using Vietnamese-specific WC cut-off values were for males 72.1%, 19.0% and 8.9%; and for females 74.3%, 22.4% and 3.3%. However the prevalence of these categories using 'comparator' cut-off values were for males 99.4%,

0.5% and 0.1%; and for females 87.4%, 10.8% and 1.7%. Using Vietnamese-specific cut-off values, the prevalence of increased-risk WC ranged from 8.8% in males aged 25-34 to 36.9% in females aged 45-54, and substantially-increased-risk WC from 0.0% in females aged 25-34 and 35-44 to 17.3% in males aged 65-74.

Generally, the prevalences of overweight and obesity increased with age (decreasing after age 55-64) and were slightly higher in females than males. The prevalence of underweight was highest in the oldest and youngest age groups (age 25-24 and 65-74), and was slightly higher in males than females.

DISCUSSION

These results show that both underweight and overweight are prevalent in urban Hanoi adults. Overweight (including obesity) was found in 29.7% of males and 31.5% of females; and increased-risk or substantially-increased-risk WC in 27.9% of males and 25.7% of females respectively. Underweight was found in 13.3% of the population. However the use of Asian and Vietnamese-specific cut-offs describes a much greater prevalence of excess body weight than the "comparator" or commonly-used cut-off values that has been considered to be more appropriate for Western populations. Using these higher cut-off values, the prevalence of underweight remains the same, but the prevalence of overweight/obesity and increased-risk or substantially-increased-risk WC were considerably lower (for overweight/obesity, 14.0% of males and 12.7% of females; for increased-risk or substantially-increased-risk WC, 0.6% of males and 12.5% of females).

The prevalences, based on Asian and Vietnamese-specific cut-off values, are not dissimilar to those found in HCMC and are substantially higher than those reported by Tuan et al. (2008) based on a national (rural *and* urban) population and not using Asian or Vietnamese-specific ethnicity cut-off values.[2] However in HCMC adults the prevalence of underweight was substantially higher (20.4%), the prevalence of overweight slightly lower (26.2%) and the prevalence of obesity higher (6.4%). The prevalence of 'increased risk' WC was slightly lower (18.7%) and the prevalence of 'substantially increased risk' WC similar (10.0%).

These Hanoi findings are further support the notion of a 'nutrition transition' occurring in Vietnam. Cuong et al. (2007) have suggested that Vietnam is at an early stage in the nutrition transition compared with that of many other Asian countries, as the problem of overweight in Vietnam is currently less severe.[3] However the finding of a doubling in the prevalence of overweight and obesity in Vietnam between 1992 and 2002 by Tuan et al. (2008) suggests a fairly rapid transition.[2] A cross-country study of the nutrition transition has found that changes are occurring rapidly, and at increasingly early stages of a countries' economic and social development.[9] Moreover, Popkin (2002) suggested that the rate of change with regard to obesity in low- and middle-income countries is much greater than that observed in high-income countries.[10] Hop et al. (1997) also found a positive secular trend in the height and weight of Vietnamese children.[11]

We found that the age-standardised prevalence of overweight/obesity (combined) was similar in men and women. This supports the HCMC findings and those discussed by Cuong et al. (2007), and is consistent with the results of the majority of studies worldwide.[3] However this was not the case when we applied the traditional BMI cut-offs, emphasising the influence of the chosen cut-off values on results.

Cuong et al (2007) found that the prevalence of overweight and obesity (based on BMI) more than doubled between the youngest and oldest age groups in the study (ages 20-29 and 50-60),[3] which is similar to our finding of a doubling between age groups 25-34 and 55-64. However we found that the prevalence was lower in the older age group (65-74). We also found that the prevalence of underweight was, for both men and women, substantially higher in those aged 25-34 and 65-74 than those of other age groups (those aged 35-44, 45-54 and 55-64); a pattern much weaker in the HCMC findings. The explanation for these differences is unclear, but may be explained by cultural and environmental differences between the two cities.

This study is based on a population health survey with a reasonably high response rate (73.9%) and measured height, weight and waist circumference. The findings are similar to those for adults in urban areas of HCMC. However, these overweight/obesity estimates are likely to be larger, and the underweight estimates lower, than those occurring in rural Vietnam, a pattern shown by Tuan et al. (2008) and observed in a number of low-income countries.[2,12] Due to a relatively small sample size and wide confidence intervals, the patterns by age and sex are generally not statistically significant. The Vietnamese-specific WC cut-offs used are those proposed by a single study.[3] Further research into body weight in rural areas is needed, as is research into the most appropriate BMI and WC cut-off values for people of Asian ethnicity. Analysis of social variations in body weight, such as by smoking status and socio-economic status is also recommended. Our findings suggest that the opportunity currently exists to intervene at what appears to be an early stage of the nutrition transition, and thus understanding the context of these changes and why different age and sex groups may be affected differently is important.

In conclusion, while low body weight remains a concern, overweight/obesity is an increasing problem in urban Vietnamese adults. The speed of such changes may be quite different from, and studies suggest more rapid than, those observed in high-income countries. As the nutrition transition unfolds in Vietnam, regular and comprehensive monitoring of trends with regard to body weight in the population is essential, as is the development of appropriate programmes and policy responses.

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AUTHOR DISCLOSURES

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Short Communication

Prevalence of underweight, overweight and obesity in urban Hanoi, Vietnam

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越南河內市的成人體重過輕、體重過重及肥胖的盛行率

此研究目的為評估在越南河內市區的成人體重過輕、體重過重及肥胖的盛行率，並將這個結果與之前在胡志明市針對成人所做的評估作比較。調查對象為居住在越南河內市區，且年齡在 25-74 歲之間的成人。資料是來自於 2004 年收集的一個橫斷性生物醫學調查，其包含一份問卷、身體檢查及血液檢驗。使用亞洲身體質量指數的切點，2004 年年齡標準化體重過重及肥胖盛行率分別是 28.6%及 2.1%。體重過重/肥胖(合併)的盛行率男性(29.7%)與女性(31.5%)相似，且普遍隨著年齡增加。假如採用世界衛生組織的標準切點，則體重過重/肥胖的盛行率則大幅降低。年齡標準化的體重過輕盛行率為 13.3%。腰圍在'危險性增加'/'大大增加危險性'(合併)的男性及女性分別為 27.9%及 25.7%。2004 年在河內市 3 位成年人中將近有 1 位是體重過重或肥胖，並且 10 位中超過 1 位是體重過輕(依據亞洲的切點)。這個體重過重/肥胖的盛行率與胡志明市的成年人相似，但體重過輕的盛行率則較低。當體重過低仍然是被關注的議題時，越南城市的成人體重過重與肥胖是一個逐漸增加的問題。

關鍵字：體重過輕、體重過重、肥胖、身體質量指數、越南

3.3 Predictors of increasing waist circumference

In earlier chapters, it has been shown that the increase in the prevalence of obesity is driven primarily by a skewing of the population distribution of body weight over time. Understanding the causal factors driving this increase in right-skew would inform the development of more effective obesity-reduction strategies. Drivers of WC are of particular interest, considering that the nature of excess body weight may be changing over time to one of greater central adiposity, as presented in Chapter 1.6.1.2 and supported by a study by Elobeid et al (2007) (210). It is unknown whether the factors driving increases in BMI over time and cross-sectionally associated with WC are also driving increases in WC over time. A set of behavioural factors with which to define an 'at risk' state for the development of abdominal weight gain would be useful, as the development of effective prevention strategies requires a better understanding of the causal role of lifestyle and health behaviours with regards to increasing WC over time.

Here, we identify predictors of increasing WC over a five-year period in a contemporary population of Australian adults.

Walls HL, Magliano DJ, McNeil JJ, Ademi Z, Stevenson C , Shaw J, Peeters A. Predictors of increasing waist circumference in an Australian population. *Submitted*.

Declaration for Thesis Chapter 3.3

Walls HL, Magliano DJ, McNeil JJ, Ademi Z, Stevenson CE, Shaw J, Peeters A. Predictors of increasing waist circumference in an Australian population. Submitted.

Declaration by candidate

In the case of Chapter 3.3.1, the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Design, analysis and drafting of manuscript	80%

The following co-authors contributed to the work. Co-authors who are students at Monash University must also indicate the extent of their contribution in percentage terms:

Name	**Nature of contribution**	**Extent of contribution (%) (student co-authors only)**
DJ Magliano	Design, analysis and drafting of manuscript	N/A
JJ McNeil	Design and drafting of manuscript	N/A
Z Ademi	Analysis and drafting of manuscript	5%
CE Stevenson	Statistical advice and drafting of manuscript	N/A
J Shaw	Drafting of manuscript	N/A
A Peeters	Design, analysis and drafting of manuscript	N/A

Candidate signature	**Date**
[redacted]	8 February 2010

Declaration by co-authors

The undersigned hereby certify that:

(1) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors;
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, expect for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (2) granting bodies, (b) the editor or publisher of journals or other publication, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s):
Department of Epidemiology & Preventive Medicine, Monash University
Baker IDI Heart and Diabetes Institute

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant].

Co-author signatures	Date
[redacted]	10/2/2010
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[redacted]	8 Feb 2010
[redacted]	8/2/10
[redacted]	7/2/2010

Baker IDI Heart and Diabetes Institute

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant].

Co-author signatures	Date
[redacted] PHD CANDIDATE	12.02.2010

Title: Predictors of increasing waist circumference in an Australian population

Running title: Predictors of increasing waist circumference

Author list:

Helen L Walls, Dianna J Magliano, John J McNeil, Zanfina Ademi, Christopher Stevenson, Jonathan Shaw, Anna Peeters

Abstract

Objective:

To identify predictors of increasing waist circumference (WC) over a 5-year period in a contemporary population of Australian adults.

Design:

Baseline and follow-up data from a national adult cohort participating in the Australian Diabetes, Obesity and Lifestyle Study (AusDiab).

Subjects:

2521 men and 2726 women aged ≥25 at baseline who participated in AusDiab and provided anthropometric measurements at baseline (1999/2000) and follow-up (2005).

Results:

An increase ≥5% of baseline WC occurred in 27% of men and 38% of women over the 5-year period. In the multivariate analysis of the total population, there was a higher risk of ≥5% gain in baseline WC in women, younger people, people with a lower baseline WC, people who never married compared to married/de facto, current smokers compared to never-smokers, people with a poorer diet quality, and people with a low energy intake. However there was no significant association with many expected predictors of waist gain such as physical activity. There were some associations between other lifestyle factors and change of WC by sex, age, level of education and across WC categories, but the associations differed across these groups.

Conclusions:

Gain of ≥5% of baseline WC occurred in a significant proportion of men and women over the 5-year period. Of the behavioural factors, poor diet quality was the key predictor of an increase ≥5% of baseline WC in this cohort. The findings highlight the need to understand better the causal role of lifestyle in regard to increasing WC over time.

Introduction

Recent reports have demonstrated faster increases in WC than BMI [1, 2], suggesting that the nature of excess body weight may be changing over time to one of greater central adiposity, rather than a more peripheral distribution of body fat [1, 2]. This has significant implications, considering that WC measures the central or abdominal distribution of excess body fat, which appears to be strongly correlated with metabolic and cardiovascular risk [3, 4]. However little is known about the drivers of increases in WC.

Few studies have examined predictors of increasing weight in adults, and we are not aware of any that have examined a range of behavioural of lifestyle factors potentially predictive of abdominal weight gain. Table 1 summarises those studies exploring predictors of change in BMI, weight and/or WC. The only study we were able to source that explored

behavioural predictors of change in WC examined a limited selection of predictors in women [5]. Previous cross-sectional analyses have found lifestyle factors such as higher television viewing time and lower levels of physical activity to be associated with higher waist circumference (WC) [6-9], and studies analysing predictors of gain in BMI/weight have found associations with factors such as higher television viewing time and lower levels of physical activity [5, 10-14]. Sternfeld et al. (2004) found that low levels of total physical activity were associated with increasing WC in their longitudinal analysis of American women. But for the most part, it is unknown whether such behavioural factors are also driving increases in WC over time. A set of behavioural factors with which to define an 'at risk' state for the onset of abdominal weight gain would be useful, as the development of effective prevention strategies requires a better understanding of the causal role of lifestyle and health behaviours in regard to increasing WC over time.

The aim of this study was to identify factors predictive of increasing WC – defined as a WC measurement at follow-up ≥5% of baseline WC – over a 5-year period in a cohort of Australian adults. Specifically, the study aimed to analyse relationships between lifestyle behaviours including levels of physical activity, television viewing time, smoking, alcohol consumption, energy intake, diet quality and portion size and change in WC.

Methods

Participants

The Australian Diabetes, Obesity and Lifestyle Study (AusDiab) is a cross-sectional, national, population-based survey conducted during 1999-2000 of 11247 Australian adults (aged ≥25 years). From the 17129 eligible households, 20347 adults completed a household interview and 11247 (55.3%) had a biomedical examination after an overnight fast, giving an overall response rate of 37% [15]. The majority (87.9%) of participants were born either in Australia or the United Kingdom; 96% spoke English at home; 0.8% were Aboriginal or Torres Strait Islander [16]. In 2004-2005, all participants (n=11247) were invited to a follow-up examination. Of the 10788 participants eligible for follow-up testing in 2004-05, 6537 (61%) presented for the biomedical examination and/or blood tests [16].

At baseline and follow-up, questionnaires were administered, anthropometric measurements were taken and a fasting blood sample was collected. The AusDiab study methodology has been described in more detail previously [15, 16]. In this study, the sample population was defined as participants who attended at both baseline and follow-up and had measures of WC at both examinations (n=5247).

All survey participants provided informed consent. The study was approved by the ethics committees of the Baker IDI Heart and Diabetes Institute and Monash University.

Measures

Changes in waist circumference between baseline and follow-up were categorised into three categories. A gain in WC was defined as a follow-up WC measurement increase of ≥5% of baseline WC. No change in WC was defined as a follow-up WC measurement of within 5% of baseline WC, and a loss of WC was defined as a follow-up WC measurement decrease of >5% of baseline WC. The following cut-offs to the WC measurements were also applied to classify people into 'low risk' (<94cm for men; <80cm for women), 'increased risk' (≥94-<102cm for men; ≥80-<88cm for women), and 'substantially increased risk' (≥102cm for men; ≥88cm for women) [17]. Ethnic-specific cut-points for WC were not used in this analysis as the proportion of Asian participants at baseline was low (3.5%) and not substantially different at follow-up.

Demographic attributes, smoking habits and educational attainment were assessed using an interviewer-administered questionnaire [16].

Physical activity

Physical activity was measured via an interviewer-administered Active Australia questionnaire, which considered participation in predominantly leisure-time physical activities (including walking for transport) during the previous week [18]. Total physical activity time was calculated as the sum of the time spent walking (if continuous and for ≥10 minutes) or performing moderate-intensity activity, plus double the time spent in vigorous-intensity physical activity [19].

Television viewing time

Self-reported television viewing time was calculated as the total time spent watching television or videos in the previous week, and is considered a reliable and valid estimate of television viewing time among adults [20].

Dietary quality

Dietary intake was assessed using a self-administered validated food frequency questionnaire (FFQ) [21], which included 74 items (with 10 frequency options), with additional questions on food habits, portion size and consumption of alcoholic beverages. Nutrient intakes from the FFQ were used to derive the Diet Quality Index [22, 23] and summarised to reflect 10 dietary characteristics – total fat, saturated fat, dietary cholesterol, fruit, vegetables, grains, calcium, iron, dietary diversity and dietary moderation. Scores from each of the ten components were summed for a highest possible score of 100 points, which represents a best-scenario dietary quality [24].

Portion size

Average daily serving sizes of potatoes, vegetables, steak and casserole/other meat were calculated as a single 'portion size factor' (PSF), which identified whether on average, a median size serve was consumed (PSF=1), more than the median (PSF>1) or below the median (PSF<1) [25].

SEIFA – disadvantage

Socio-economic status was measured using an Index of Disadvantage code from the Socio-economic Indexes for Areas (SEIFA). The index is derived from attributes such as low income, low educational attainment, high unemployment and jobs in low skilled occupations. The index is constructed so that high values reflect high scoio-economic status (relative advantage) and low values reflect low socio-economic status (relative disadvantage) [26, 27].

Statistical analyses

Logistic regression was used to predict the likelihood of a gain in WC compared to maintaining baseline WC. In Table 3, the results are presented for each predictor (1) unadjusted; (2) adjusted for sex and age; and (3) adjusted for sex and age group plus key

'fixed' demographics, all demographics, or all demographics plus all lifestyle predictors, depending on the specific predictor. Thus, those variables entered into the two multivariate logistic regression models in Table 3 were: (Model 1) sex and age group; (Model 2) sex and age group plus for variables marked "a" – country of birth and Aboriginal and Torres Strait Islander status; variables marked "b" – everything listed under "a" plus education, occupation, marital status, whether living in an Australian capital city; variables marked "c" everything listed under "a" and "b" plus baseline WC, physical activity, television viewing, smoking status, diet quality, alcohol, energy intake and portion size. The variables entered into the multivariate logistic regression in subsequent tables and figures were those variables described by "c" above, namely country of birth, Aboriginal and Torres Strait Islander status, education, occupation, marital status, whether living in an Australian capital city, physical activity, television viewing, smoking status, diet quality, alcohol, energy intake and portion size. The findings from the fully-adjusted multivariate analyses are discussed in the results.

Analyses were conducted in Stata (Intercooled Stata, Version 9.0, StataCorp) and took into account the complex, two-stage, cluster sampling design of the AusDiab study.

Results

As shown in Table 2 (univariate results), people who underwent WC gain (≥5% gain in baseline WC) had a lower mean baseline WC than the people who maintained baseline WC (within 5% of baseline WC) and people who underwent WC loss (≥5% loss in baseline WC). A higher proportion of females than males underwent WC gain and a higher proportion of people aged 25-54 years than aged 55+ years. White-collar workers were more likely to undergo WC gain than other occupational groups. Retirees and blue-collar workers were the least likely to undergo WC gain.

People who underwent WC gain viewed less television per week, had a lower energy intake, and consumed smaller average portion sizes than people who maintained baseline WC or underwent a loss of baseline WC. People who underwent WC gain were more likely to be current smokers than never-smokers or ex-smokers.

Only people who underwent WC gain and WC maintenance have been included in the analyses to follow, as it is the comparison between these two groups in which we are interested. People who underwent WC loss are likely to be a heterogeneous group who have lost weight for a variety of reasons, including illness.

In the multivariate analysis of the total population (Table 3), people who underwent WC gain were more likely to be female, have a lower baseline WC, to be aged 25-34 years than aged >35 years, and have been never married than married/de facto. Of the behavioural variables, people who underwent WC gain were more likely to be current smokers than never-smokers, to have a poorer diet quality, and to have a low energy intake. There was also a suggestion of a protective effect of a lower level of television viewing. We explored whether the predictive effect of current smoking was driven by people quitting smoking over the 5-year period. For the fully-adjusted multivariate analysis, compared to the never-smokers the odds of WC gain for current smokers who continued smoking over the 5-year period and current smokers who quit over the 5-year period were 1.01(0.80-1.28) and 2.05(1.34-3.14). To ensure that the lack of association between other variables such as physical activity and WC gain and was not due to the definition of change in WC we undertook analyses using three different definitions of change in WC, and the results were similar.

To explore whether effect modification influenced the findings we analysed the potential behavioural predictors of WC gain by sex and age. Figure 1 illustrates that in the multivariate analysis of males, there were no significant predictors of a gain in WC, although there was a suggestion of a protective effect of diet quality. In the multivariate analysis of females, those who underwent WC gain were more likely to be current smokers than never-smokers, and there was a suggestion of a protective effect of diet quality.

Figure 2 illustrates that in the multivariate analysis of people aged 25-54 years, people who underwent WC gain were more likely to have a poorer diet quality. Comparing people aged 25-34 years and 35-44 years (data not shown), in the younger age group people who underwent WC gain were more likely to have a poorer diet quality, and more likely to have a low energy intake, whereas in the older age group people who underwent WC gain were more likely to watch 2-4 hours/day of television compared with <2 hours/day. In people aged 55+ years (Figure 2), people who underwent WC gain were more likely to engage in higher levels of physical activity. In people aged 65+ years (data not shown), people who underwent WC gain were more likely to engage in higher levels of physical activity.

We have stratified by WC category at baseline to explore whether the findings are affected by the choice of comparator group – between people who underwent ≥5% gain in WC with those who have maintained their baseline WC, but who may have also gained WC recently (but prior to the survey period) (Table 4).

Table 4 illustrates that in the multivariate analysis of people with a low-risk WC at baseline people who underwent WC gain were more likely to watch 2-4 hours/day of television than <2 hours/day. In people with an increased-risk WC at baseline, people in the highest quartile of energy intake were less likely to undergo WC gain. In people with a substantially-increased-risk WC at baseline, people who underwent WC gain were more likely to watch <2 hours/day of television than 4+ hours/day, be current smokers than never-smokers and to have a poorer diet quality.

Overall, there were few significant associations with behavioural predictors. The lack of associations in the low-risk group suggests that the choice of comparator groupings is appropriate, as people who were defined as having maintained their baseline WC are less likely in the low-risk group to have undergone a recent gain in WC. The relative lack of right-skew in the distribution of gain in WC in people who did not undergo a change in baseline WC (data not shown) also supports this.

To test the sensitivity of our results to the definition of WC gain used, we conducted a series of analyses for the primary (total population) analysis using different definitions of gain in WC – a 5% gain in WC versus the total population (including people with WC gain, WC maintenance and WC loss); a ≥2% gain in WC versus change of between -2% and +2%; and differences between quartiles of gain in WC (to explore absolute changes in WC). The results did not differ greatly from those presented here.

Discussion

An increase ≥5% of baseline WC occurred in over a quarter of men and over a third of women between 2000 and 2005. Women, younger people, people who had never married, and people with a lower baseline WC were more likely to gain ≥5% of baseline WC. The key behavioural predictor of WC gain was poor diet quality.

Overall, the patterns were very similar by age and sex, with the exceptions of poor diet quality as a clear predictor of WC gain in people aged 25-54 but not for people aged 55+ years, and a high level of physical activity as a predictor of WC gain in the older age group but not in the younger. This finding of a high level of physical activity as a predictor of WC gain in older people may be a consequences of illness being associated with WC loss and lower levels of physical activity.

Previous studies exploring behavioural predictors of an increase in BMI and weight have found a range of predictive factors, including increased television viewing time, consumption of energy-dense foods, consumption of meat and low levels of physical activity (see Table 1). The only study of which we are aware exploring predictors of an increase in WC found physical activity and current smoking to be predictors of WC gain over a three-year period in US women aged 42-52 years [5]. While we also found current smoking was predictive of WC gain in our analysis, this was shown to be driven by people quitting over the study period. Current smoking in itself was not a predictor. Low physical activity levels did not feature as a predictor of WC gain in our analysis, as they did in many of the longitudinal studies. Cross-sectional studies have reported fairly consistent associations between behaviours considered to be either risk or protective factors for BMI/WC gain – such as television viewing, physical activity, diet quality, fruit and vegetable consumption and portion size [20, 28-30]. We found significant associations with diet quality – which also encapsulates fruit and vegetable consumption – and the suggestion of a greater likelihood of WC gain associated with greater portion size and some categories (generally not the highest) of increased television viewing. Regroupings of categories such as TV viewing did not substantially affect these results. With the exception of the group aged 55+ years, for whom high levels of physical activity were associated with WC gain, we found no association between physical activity and WC gain. This may be due to the more sophisticated measurement of physical activity in the study by Sternfeld et al. (2004) (which measured physical activity in various domains, including sports/exercise, household/caregiving and daily routine [5]. The general lack of predictors in our study may be due to the period of follow-up (five years), the study design, the definition of WC gain, or the predictors being "set" at an earlier stage in the life course.

Poor dietary quality in this context refers to a poor diet quality relative to national recommendations for adults. The index on which the measure is based has been found to appropriately explain variation in a wide range of food and nutrients thought to contribute

to health, and is considered a practical means of evaluating the overall quality of diet in adults. No one component of the DQI contributes disproportionately more than any other and thus it is difficult to explain the dietary components or patterns explaining the observed trends [22]. The relationship between a low energy intake and gain in WC is surprising, particularly considering the inclusion of physical activity levels as a confounder in the multivariate analysis.

There were some limitations to the study. The follow-up period of 5 years may be inappropriate for the identification of many predictors of WC gain. Five years may be too long, in that behaviours may have changed substantially over that length of time, limiting the relevance of the baseline behaviours. Similar findings of comparable (or shorter) periods revealed other predictors. The definitions of WC gain (increase ≥5% of baseline WC) and WC maintenance (change within 5% of baseline WC) may affect the results. However our results were not substantially different when we explored other groupings, such as a 5% gain in WC versus the rest of the population (including people with WC gain, WC maintenance and WC loss), and ≥2% increase and change from 2% of baseline WC (data not shown). Furthermore, we analysed differences by absolute change in WC, and again the results did not differ substantially. We chose not to adjust for baseline WC in the multivariate analyses as such adjustment would likely result in spurious associations (although the analysis with this adjustment was generally not very different, data not shown) [31].

We have also presented the results by WC groupings, to explore whether the two comparator groups are too similar – the people who maintained baseline WC having recently undergone WC gain themselves. If the results are affected by the maintainers having also gained WC recently, the difference between the two groups should be most obvious in the lowest WC category – the low-risk WC category. However the lack of associations persisted, even in the low-risk WC group, for whom a recent WC gain in people who maintained baseline WC is unlikely. This suggests that the findings are not affected by the comparator group.

The potential selection bias in AusDiab is an important limitation to our study, due to the healthy volunteer bias, and the 37% and 61% response rates to the first and second surveys, respectively. Loss to follow-up may mean that the proportion of people who underwent WC gain is not wholly reflective of population incidence rates [16]. In this case, an underestimation of some of the odds of WC gain may be likely as non-response has

been linked to having a sedentary lifestyle and lower socioeconomic status (also associated with a higher body weight in developed countries) [32-34]. There may also be greater self-report bias for levels of some behavioural factors in people of higher body weights; which may explain, for example, our finding of a greater odds of WC gain amongst people with a substantially-increased-risk WC in people who viewed <2 hours/day of television than in people who viewed 4+ hours/day.

The difference in the significant predictors in this analysis and those identified in cross-sectional analyses may be explained by life course epidemiology, which considers health and disease in adults to be influenced independently, cumulatively and interactively by various biological and social factors throughout life. Thus an outcome in adulthood such as weight gain would be attributable to biological and social exposures operating during gestation, childhood, adolescence, young adulthood and later adult life [35], for which a factor such as adult TV viewing might be a marker. Thus in this example, adult TV viewing and leisure time PA might be cross-sectional markers of an accumulated poor lifestyle rather than a causative factor in adult life.

The suggestion that many factors found associated with high body weight in cross-sectional analyses are markers of accumulated poor lifestyles, but not in themselves strong predictors of future WC gain has important implications. Rather than a narrow focus on "reducing TV viewing" or "reducing portion size", for example, a broader focus on "healthy lifestyles" may be more important – and one that begins at gestation/childhood and last life-long.

In conclusion, in an Australian cohort of men and women aged 25 years and over, a significant proportion experienced a gain in WC of ≥5% of baseline WC over a 5-year period. Poorer diet quality was found to be the key behavioural predictor of WC gain in this population. Many of the factors associated with higher WC in cross-sectional analyses were not predictive of a gain in WC. These findings highlight the need to understand better the causative role of lifestyle and health behaviours in regard to increasing WC over time.

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Figure legends

Table 1. Summary of literature exploring predictors of change in BMI, weight or waist circumference in adults

Table 2. Proportion of participants in each category of WC change, by socio-demographic characteristics and behaviours (at baseline except where indicated)

Table 3. Univariate and multivariate associations of potential predictors of WC gain in total population

Table 4. Multivariate odds ratios (ORs) of potential predictors of WC gain in people of low-risk, increased-risk and substantially-increased-risk waist circumference

Figure 1. Multivariate odds ratios (95% CIs) of potential predictors of WC gain for males and females

Figure 2. Multivariate odds ratios (95% CIs) of potential predictors of WC gain in people aged 25-54 years and 55+ years

Table 1. Summary of literature exploring predictors of change in BMI, weight or WC in adults

Study	Year	Country	Age and sex of population	Measured variables	Key predictors of outcome	Follow-up
French et al. [10]	1994	USA	Age years; males and females (n=3552)	Weight	**Weight loss:** increased exercise **Weight gain:** history of participation in formal weight loss programme, dieting to lose weight at baseline, increased consumption of fries, dairy products, sweets and meat	2 years
HPFS* [11]	1998	USA	40-75 years of age; males (n=19 478)	Weight	**Weight loss:** vigorous activity, being on a diet **Weight gain:** television viewing, eating between meals, quitting smoking and history of voluntary weight loss	2,4,6,8 years
FTC * [36]	1998	Finland	18-54 years of age, twin pairs; males and female (n=5867)	Weight	**Weight gain:** high level of stress at baseline **Weight gain for women:** low levels of life satisfaction and high score for neuroticism	6 years
POP* [37]	2000	USA	20- 45 years of age; males and females (n=1044)	Weight	**Weight gain:** dietary fat intake, low levels of physical activity **Weight loss:** high levels of physical activity, decreased food intake	3 years
CHNS* [12]	2001	7 provinces in China	20-45 years of age; males and females (n=2488)	BMI	**Weight gain:** light work related levels of physical activity	8 years
WHA * [13]	2002	Australia	18-23 age of years; females (n=8726)	BMI	**Weight maintenance:** managerial/professional occupations, never married, currently studying, not mothers, less time sitting and over consumption of takeaway food	4 years

EPIC* [14]	2002	Germany	24-2 age of years for males; and 19-70 for females (n=17 369)	Weight	**Weight gain:** women – consumption of high energy, high fat food (fats sauces, and meat); men – energy-dense foods (sweets) **Weigh loss:** women – cereals	2 years
IRAS* [38]	2003	USA	40-69 age of year; males and females (n=1194)	BMI, WC	**Weight loss:** diabetic state, high fasting insulin concentrations	5 years
SWAN* [5]	2004	USA	42-52 age of year ; females (n=3064)	Weight, WC	**WC gain:** baseline age, follow-up time, low levels of physical activity, race, perceived health, health status, smoking **Weight gain:** follow-up time, low levels of physical activity, race, perceive health, health status and smoking	3 years
Janghorbani et al. [39]	2008	Iran	Mean age 51.3; males and females with type II DM (n=12 650)	BMI	**Weight loss:** female, lower education, longer duration of DM, never smokers, insulin treatment regimen and higher HbA1c at baseline **Weight gain:** class II and III obesity at baseline	9.1 years

***Health Professionals Follow-up Study, * Finnish Twin Cohort, *Malmo Diet and Cancer prospective cohort Study, *China Health and Nutrition survey, *Australian Longitudinal Study on Women's Health, * European Prospective Investigation into Cancer and Nutrition (EPIC)-Potsdam cohort, *Insulin Resistance Atherosclerosis Study, * Study of Women's Health across the Nation**

Table 2. Proportion of participants in each category of WC change, by socio-demographic characteristics and behaviour (at baseline except where indicated)

Item	n	WC gain n=1748 (33.31%)	WC maintenance n=2821(53.76%)	WC loss n=678(12.92%)
WC(cm) (mean, SE)	5247	86.95(0.48)	92.71(0.40)	94.50 (0.52)
Demographic characteristics				
Sex (%, SE)				
Males	2521	27.49 (1.64)	60.42 (1.28)	12.10 (0.88)
Females	2726	38.70 (1.62)	47.62 (1.24)	13.68 (0.96)
Age groups (%, SE)				
25-34 years	392	38.27 (2.32)	45.66 (2.30)	16.07 (1.80)
35-44 years	1229	35.88 (1.94)	53.30 (1.51)	10.82 (1.00)
45-54 years	1630	36.87 (1.80)	51.29 (1.37)	11.84 (1.12)
55-64 years	1117	29.36 (2.21)	56.76 (1.71)	13.88 (1.37)
65-74 years	654	26.61 (2.04)	59.94 (2.26)	13.46 (1.10)
75+ years	225	24.00 (3.28)	55.56 (3.00)	20.44 (3.21)
Country of birth (%, SE)				
Australia/NZ	4065	34.12 (1.53)	53.28 (1.11)	12.60 (0.76)
UK/Northern Ireland	591	30.29 (2.08)	53.64 (2.14)	16.07 (1.66)
Other	591	30.80 (2.70)	57.19 (2.62)	12.01 (1.32)
Aboriginal and Torres Strait Islander status (%, SE)				
Yes	40	25.00 (-)	70.00 (-)	5.00 (-)
No	5207	33.38 (1.47)	53.64 (1.03)	12.98 (0.74)
Conpletion of high school education (%, SE)				
Yes	2539	33.99 (1.56)	53.13 (1.12)	12.88 (0.95)
No	2708	32.68 (1.69)	54.36 (1.36)	12.96 (0.83)
Occupation (%, SE)				
Professional	1654	34.22 (2.05)	54.05 (1.63)	11.72 (0.96)

White collar	846	40.90 (2.11)	48.46 (1.70)	10.64 (1.57)
Blue collar	717	27.75 (1.77)	58.30 (1.56)	13.95 (1.29)
Home duties	359	36.21 (2.71)	49.30 (2.79)	14.48 (2.05)
Unemployed, students	58	32.76 (7.11)	51.72 (5.90)	15.52 (4.61)
Retirees	587	28.62 (2.22)	58.09 (1.93)	13.29 (1.55)
Pensioners	1026	31.19 (1.86)	53.70 (1.71)	15.11 (0.99)
SEIFA – disadvantage (%, SE)				
<1000	1757	32.56 (-)	54.52 (-)	12.92 (-)
≥1000	3490	33.70 (1.66)	53.38 (1.13)	12.92 (0.85)
Marital status (%, SE)				
Married / de facto	4313	32.76 (1.53)	54.49 (1.04)	12.75 (0.89)
Widowed / separated / divorced	682	35.78 (1.95)	50.88 (2.07)	13.34 (1.16)
Never married	252	36.11 (3.13)	49.21 (2.72)	14.68 (2.78)
Living in an Australian capital city (%, SE)				
Yes	3299	32.74 (1.36)	52.86 (0.98)	14.40 (0.88)
No	1948	34.29 (1.77)	55.29 (1.08)	10.42 (1.14)
Behavioural characteristics				
Physical activity time (hours/wk) (mean, SE)	5247	4.79(0.20)	4.75(0.16)	4.43(0.24)
TV viewing time (mins/wk) (mean, SE)	5247	709.71(19.63)	762.46(16.34)	817.28 (23.96)
TV viewing time (%, SE)				
<2 hrs/day	341	34.31 (3.13)	52.79 (2.79)	12.90 (2.22)
2-4 hrs/day	450	41.56 (2.58)	49.56 (2.48)	8.89 (1.20)
4+ hrs/day	4446	32.41 (1.44)	54.26 (1.06)	13.33 (0.76)
Smoking status (%, SE)				
Never smoker	3029	33.51 (1.57)	54.18 (1.09)	12.31 (0.83)
Ex-smoker	1605	31.40 (1.84)	54.58 (1.61)	14.02 (1.02)
Current smoker	613	37.36 (2.11)	49.59 (2.23)	13.05 (1.38)
Diet quality (DQI/100)	5247	66.44(0.44)	66.90(0.40)	67.71 (0.52)

(mean, SE)				
Alcohol consumption (%, SE)				
Current non-drinkers	732	30.46 (2.23)	54.10 (1.84)	15.44 (1.65)
Light drinkers	3181	34.80 (1.48)	52.85 (1.08)	12.35 (0.73)
Moderate to heavy drinkers	1334	31.33 (1.91)	55.77 (1.46)	12.89 (1.09)
Fruit consumption (%, SE)				
Don't eat fruit	100	39.00 (4.98)	47.00 (4.50)	14.00 (3.64)
<2 / day	1105	32.94 (1.90)	53.67 (1.84)	13.39 (1.19)
3+ / day	4042	33.28 (1.50)	53.96 (1.11)	12.77 (0.80)
Vegetable consumption (%, SE)				
<2 / day	62	32.26 (6.04)	58.06 (6.11)	9.68 (3.60)
3-4 / day	4439	33.30 (1.49)	54.18 (1.16)	12.53 (0.82)
5+ / day	746	33.51 (2.22)	50.94 (2.02)	15.55 (1.30)
Energy intake (kj/day/1000) (mean, SE)	5247	7962.83(73.21)	8396.50 (64.34)	8376.00(143.77)
Portion size (mean, SE)	5247	1.16(0.01)	1.20(0.01)	1.19 (0.02)

Table 3. Univariate and multivariate associations of potential predictors of WC gain in total population

Item	Univariate odds ratio (OR)	Multivariate[1] odds ratio (OR)	Multivariate[2] odds ratio (OR)
WC[c]	0.97(0.96-0.97) *	0.97(0.97-0.98) *	0.97(0.97-0.98) *
Demographic characteristics			
Sex[a]			
Males	REF	REF	REF
Females	1.79(1.57-2.04)*	1.79(1.57-2.04)*	1.79(1.57-2.03)*
Age groups[a]			
25-34 years	REF	REF	REF
35-44 years	0.80(0.63-1.02)	0.75(0.59-0.96)*	0.75(0.59-0.95)*
45-54 years	0.86(0.68-1.09)	0.82(0.65-1.04)	0.82(0.65-1.04)
55-64 years	0.62(0.49-0.77)*	0.59(0.47-0.73)*	0.59(0.47-0.73)*
65-74 years	0.53(0.42-0.66)*	0.51(0.41-0.64)*	0.51(0.41-0.64)*
75+ years	0.52(0.33-0.80)*	0.50(0.32-0.78)*	0.50(0.32-0.78)*
Country of birth group[a]			
Australia/NZ	REF	REF	REF
UK/Northern Ireland	0.88(0.72-1.08)	0.95(0.77-1.18)	0.95(0.77-1.17)
Other	0.84(0.66-1.07)	0.87(0.68-1.12)	0.86(0.67-1.11)
Aborginal and Torres Strait Islander status[a]			
Yes	REF	REF	REF
No	1.74(0.69-4.37)	1.94(0.77-4.91)	1.97(0.78-5.01)
Completion of high school education[b]			
Yes	REF	REF	REF
No	0.94(0.82-1.08)	0.95(0.82-1.10)	0.94(0.80-1.11)
Occupation [b]			
Professional	REF	REF	REF

White collar	1.33(1.09-1.63)	1.09(0.87-1.36)	1.11(0.89-1.38)
Blue collar	0.75(0.63-0.89) *	0.82(0.68-1.00)*	0.84(0.69-1.03)
Home duties	1.16(0.89-1.52)	0.86(0.64-1.15)	0.90(0.67-1.20)
Unemployed, students	1.00(0.53-1.88)	0.97(0.51-1.83)	0.97(0.52-1.81)
Retirees	0.78(0.63-0.96) *	1.03(0.76-1.38)	1.02(0.76-1.37)
Pensioners	0.92(0.73-1.15)	1.09(0.82-1.46)	1.09(0.83-1.43)
SEIFA – disadvantage			
<1000	REF	REF	REF
≥1000	1.06(0.86-1.30)	1.06(0.86-1.31)	1.07(0.85-1.34)
Marital status [b]			
Married / de facto	REF	REF	REF
Widowed/separated/divorced	1.17(0.98-1.39)	1.16(0.98-1.38)	1.15(0.96-1.38)
Never married	1.22(0.99-1.50)	1.30(1.04-1.63)*	1.30(1.04-1.61)*
Living in an Australian capital city [b]			
Yes	REF	REF	REF
No	1.00(0.80-1.26)	1.00(0.79-1.26)	0.99 (0.78-1.26)
Behavioural characteristics			
Physical activity time[c] **(mins/week)**	1.00(0.99-1.01)	1.01(1.00-1.02)	1.01(1.00-1.02)
TV viewing time[c]			
<2 hrs/day	REF	REF	REF
2-4 hrs/day	1.29(0.97-1.72)	1.35(0.99-1.84)	1.34(0.99-1.82)
4+ hrs/day	0.92(0.70-1.20)	1.05(0.79-1.38)	1.03(0.78-1.36)
Smoking status[c]			
Never smoker	REF	REF	REF
Ex-smoker	0.93(0.82-1.06)	1.05(0.93-1.19)	1.04(0.91-1.18)
Current smoker	1.22(0.99-1.49)	1.28(1.03-1.59)*	1.25(1.00-1.55)*
Diet quality[c] **(DQI/100) (mean, SE)**	0.74(0.45-1.23)	0.59(0.34-1.03)	0.57(0.33-1.00)*
Alcohol consumption[c]			
Current non-drinkers	REF	REF	REF

Light drinkers	1.17(0.98-1.39)	1.14(0.95-1.36)	1.12(0.94-1.33)
Moderate-heavy drinkers	1.00(0.84-1.18)	1.12(0.93-1.35)	1.03(0.86-1.24)
Fruit consumption[c]			
Don't eat fruit	REF	REF	REF
<2 / day	0.74(0.48-1.14)	0.70(0.45-1.10)	0.75(0.47-1.19)
3+ / day	0.74(0.49-1.13)	0.68(0.44-1.06)	0.78(0.49-1.24)
Vegetable consumption[c]			
<2 / day	REF	REF	REF
3-4 / day	1.11(0.62-1.96)	1.17(0.65-2.09)	1.27(0.69-2.33)
5+ / day	1.18(0.64-2.17)	1.15(0.63-2.12)	1.25(0.67-2.36)
Energy intake[c] (Kj/day/1000)	0.96(0.94-0.97)*	0.98(0.96-1.00)*	0.97(0.95-1.00)
<6476	REF	REF	REF
6476-7688	0.95(0.79-1.14)	1.00(0.83-1.21)	1.00(0.82-1.21)
7688-9676	0.84(0.71-0.98)*	0.94(0.79-1.12)	0.95(0.78-1.16)
9676+	0.70(0.610.79)*	0.83(0.70-0.97)*	0.79(0.65-0.96)*
Portion size[c]	0.78(0.70-0.87)*	0.94(0.80-1.11)	1.17(0.95-1.43)
(Standard portion factor=1)			

[1] Multivariate analysis adjusted for sex and age group.

[2] Multivariate analysis adjusted for: variables marked "a" – sex, age group, country of birth and Aborginal and Torres Strait Islander status; variables marked "b" – everything listed under "a" plus education, occupation, marital status, whether living in an Australian capital city; variables marked "c" – everything listed under "a" and "b" plus physical activity, television viewing, smoking status, diet quality, alcohol, energy intake and portion size.

*Indicates a statistically significant result.

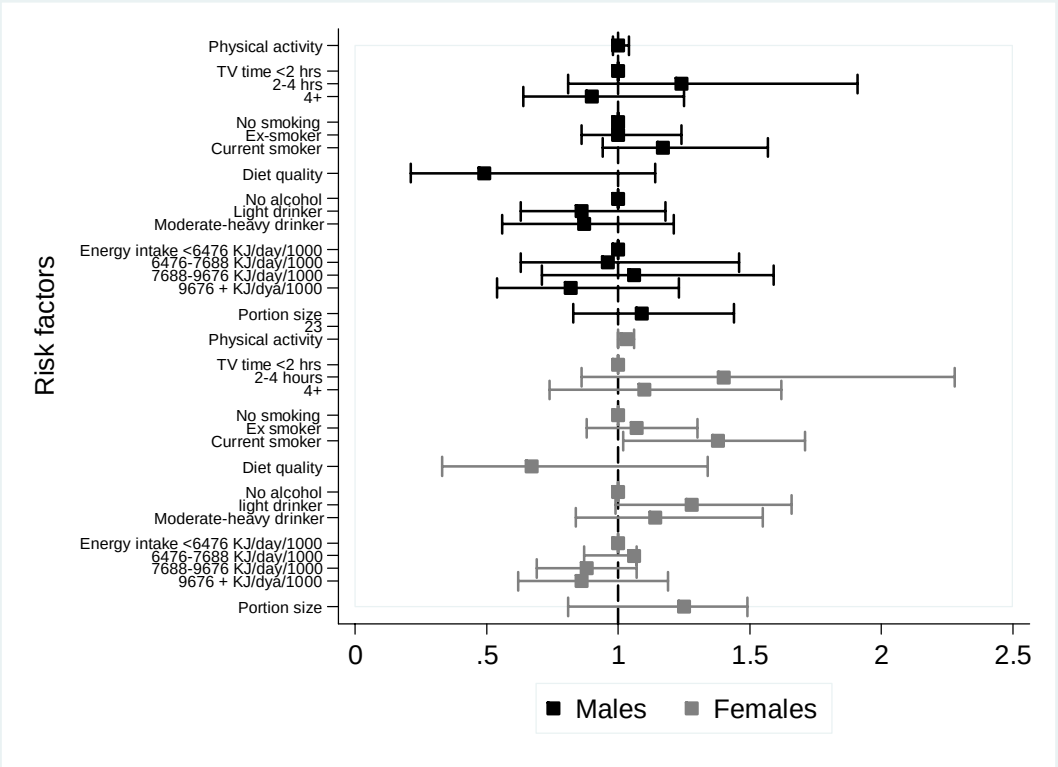
Table 4. Multivariate[1] odds ratios (ORs) of potential predictors of WC gain in people of low-risk, increased-risk and substantially-increased-risk WC

Variables	Multivariate[1] odds ratio (OR)		
	Low-risk WC	Increased-risk WC	Substantially-increased-risk WC
Physical activity time (mins/week)	1.00(0.99-1.02)	1.02(0.98-1.05)	1.00(0.97-1.02)
TV viewing time			
<2 hrs/day	REF	REF	REF
2-4 hrs/day	1.87(1.17-2.98)*	1.20(0.68-2.11)	0.68(0.36-1.31)
4+ hrs/day	1.24(0.81-1.91)	1.47(0.92-2.34)	0.61(0.38-0.99)*
Smoking status			
Never smoker	REF	REF	REF
Ex-smoker	1.05(0.85-1.32)	1.21(0.93-1.57)	1.13(0.85-1.52)
Current smoker	0.99(0.73-1.35)	1.48(0.91-2.41)	1.63(1.07-2.48)*
Diet quality (DQI/100) (mean, SE)	0.60(0.27-1.34)	1.12(0.38-3.25)	0.19(0.06-0.63)*
Alcohol			
Current non-drinkers	REF	REF	REF
Light drinkers	1.08(0.82-1.43)	1.43(0.92-2.24)	0.84(0.59-1.19)
Moderate-heavy drinkers	0.97(0.68-1.39)	1.59(0.99-2.54)	0.78(0.54-1.14)
Energy intake (kj/day/1000)	0.97(0.93-1.01)	0.96(0.90-1.01)	0.99(0.94-1.05)
<6476	REF	REF	REF
6476-7688	1.15(0.85-1.55)	0.84(0.56-1.25)	1.01(0.70-1.45)
7688-9676	1.02(0.76-1.38)	0.90(0.61-1.32)	0.89(0.62-1.29)
9676+	0.95(0.75-1.20)	0.59(0.38-0.90)*	0.81(0.52-1.27)
Portion size	1.23(0.90-1.69)	1.42(0.92-2.19)	0.97(0.66-1.43)
(standard portion factor =1)			

[1] Multivariate analysis adjusted for sex, age group, country of birth, Aborginal and Torres Strait Islander status, education, occupation, marital status, whether living in an Australian capital city, physical activity, television viewing, smoking status, diet quality, alcohol, energy intake and portion size.

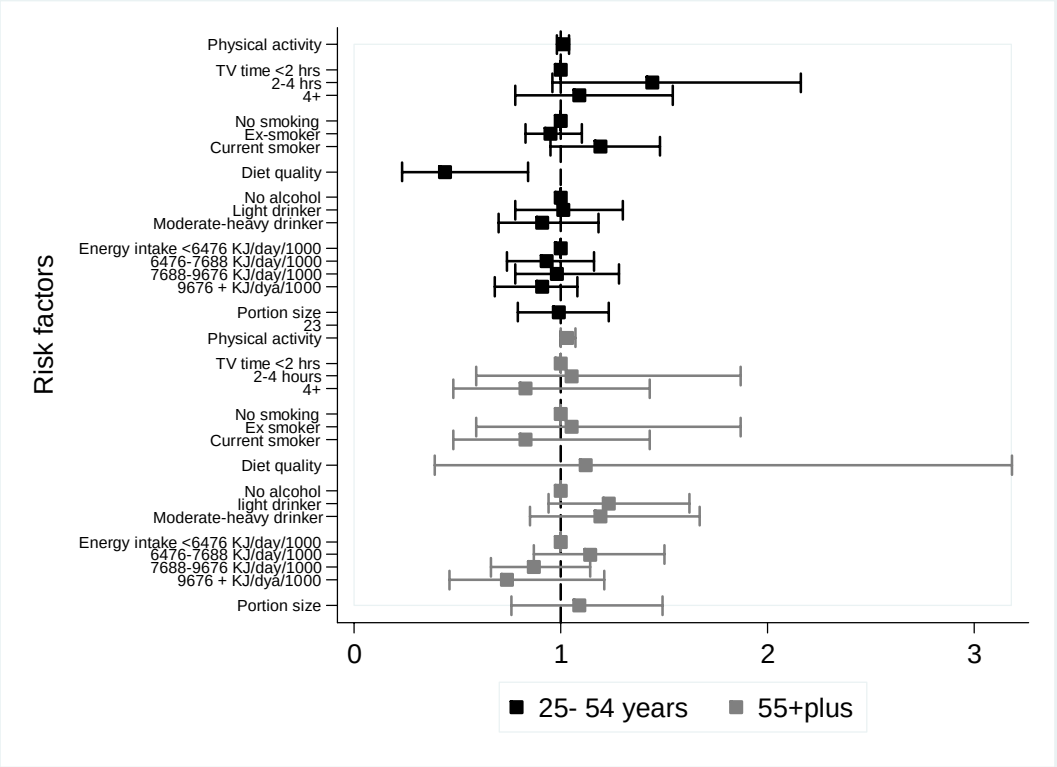
*Indicates a statistically significant result.

Figure 1. Multivariate[1] odds ratios (95% CIs) of potential predictors of WC gain for males and females



[1] Multivariate analysis adjusted for sex, age group, country of birth, Aborginal and Torres Strait Islander status, education, occupation, marital status, whether living in an Australian capital city, physical activity, television viewing, smoking status, diet quality, alcohol, energy intake and portion size.

Figure 2. Multivariate[1] odds ratios (95% CIs) of potential predictors of WC gain in people aged 25-54 years and 55+ years



[1] Multivariate analysis adjusted for sex, age group, country of birth, Aborginal and Torres Strait Islander status, education, occupation, marital status, whether living in an Australian capital city, physical activity, television viewing, smoking status, diet quality, alcohol, energy intake and portion size.

3.4 Predicting future obesity

The prevalence of obesity among Australian adults has progressively increased over recent decades, doubling between 1980 and 2000 (3, 244). Similar increases are evident worldwide (28), driven by an increase in incidence trends. Several projections of the future burden of obesity have been conducted, both country-specific and global projections. But these have been based on extrapolations of past prevalence or body weight distributions into the future. A more accurate method, as explained in the paper to follow, is to base future prevalence estimates on the most recent measures of incidence available.

Here, we estimate the future prevalence of obesity in Australia using the most recent measures of incidence of overweight and obesity.

Walls HL & Magliano DJ, Stevenson C, Backholer K, Mannan HR, Shaw J, Peeters A, Projected progression of the prevalence of obesity in Australia. *Submitted*.

Declaration for Thesis Chapter 3.4

Walls HL & Magliano DJ, Stevenson CE, Backholer K, Mannan HR, Shaw J, Peeters A.

Projected progression of the prevalence of obesity in Australia. Submitted.

Declaration by candidate

In the case of Chapter 3.4.1, the nature and extent of my contribution to the work was the following:

Nature of contribution	Extent of contribution (%)
Design, analysis and drafting of manuscript	60%

The following co-authors contributed to the work. Co-authors who are students at Monash University must also indicate the extent of their contribution in percentage terms:

Name	Nature of contribution	Extent of contribution (%) (student co-authors only)
DJ Magliano	Design, analysis and drafting of manuscript	N/A
CE Stevenson	Design, statistical advice and drafting of manuscript	N/A
K Backholer	Design, analysis and drafting of manuscript	N/A
HR Mannan	Design, statistical advice and drafting of manuscript	N/A
J Shaw	Drafting of manuscript	N/A
A Peeters	Design, analysis and drafting of manuscript	N/A

Candidate signature	Date
[redacted]	8 February 2010

Declaration by co-authors

The undersigned hereby certify that:

(1) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors;
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, expect for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;

(5) potential conflicts of interest have been disclosed to (2) granting bodies, (b) the editor or publisher of journals or other publication, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s):
Department of Epidemiology & Preventive Medicine, Monash University
Baker IDI Heart and Diabetes Institute

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant].

Co-author signatures	**Date**
[redacted]	10/2/2010
[redacted]	8 Feb 2010
[redacted]	8/2/10
[redacted]	8 February 2010
[redacted]	8/2/10
[redacted]	7/2/2010

Title: Projected progression of the prevalence of obesity in Australia

Running title: Projected prevalence of obesity in Australia

Study design: Multi-state life tables derived from a national, population-based longitudinal study

Author list:

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Abstract

Objective

This analysis estimates the future prevalence of obesity in Australia using measures of overweight and obesity incidence.

Design

Multi-state life tables derived from a national, population-based longitudinal study.

Setting

Participants in the Australian Diabetes, Obesity and Lifestyle (AusDiab) study (baseline 2000, follow-up 2005).

Participants

Australian adults aged ≥25 years in 2000.

Main outcome measures

Projected prevalence of normal-weight, overweight and obesity.

Results

Between 2000 and 2025 the adult prevalence of normal-weight will decrease from 40.6% to 28.1% and the prevalence of obesity will increase from 20.5% to 33.9% in Australia. By the time those people aged 25-29 in 2000 reach 60-64 years, 22.1% will be normal-weight and 42.4% will be obese. On average, normal-weight females aged 25-29 years in 2000 will live another 56.2 years: 26.6 years as normal-weight, 15.6 years as overweight and 14.0 years as obese. Normal-weight males aged 25-29 years in 2000 will live another 51.5 years: 21.6 years as normal-weight, 21.1 years as overweight and 8.8 years as obese.

Conclusion

Assuming that the rates of weight gain observed in the first five years of this decade are maintained, our findings suggest a 70% increase in the prevalence of obesity amongst Australian adults over coming decades, and a high probability for contemporary normal-weight young adults of becoming overweight or obese. Therefore, even maintaining current levels of obesity will likely require considerable intervention.

"What this study adds" box

Section 1 Projections of the future prevalence of obesity are essential for health policy decision-making. However, no such projections have been conducted using measures of overweight and obesity incidence – the most accurate method available.

Section 2 Our findings suggest a 70% increase in the prevalence of obesity amongst Australian adults over coming decades; and a high probability of future obesity amongst even contemporary normal-weight young adults. Effective intervention to reduce current levels of weight gain is essential if we are to avoid the predicted burden of obesity.

Introduction

The prevalence of obesity amongst Australian adults has progressively increased over recent decades, doubling between 1980 and 2000 (1, 2). Similar increases are evident worldwide (3), primarily driven by an increase in incidence. Indeed, a recent US study reported a 2-3 fold increase in incidence of overweight and obesity between 1950 and 2000 (4). Such increases are problematic considering the association between obesity and chronic disease, and the attendant burden on the health care system. Understanding the impact that current trends will have on the future prevalence of obesity is essential for determining both the nature and size of public-health interventions required to reduce or simply to maintain current levels of obesity.

Several projections of the future prevalence of obesity have been conducted, both country-specific and global (5-12). For example, projections for England suggest that by 2025, 47% of males and 36% of females will be obese, an increase of 96% and 50%, respectively, over 2004 levels (9). However these studies predict the future burden of obesity by extrapolating into the future past prevalence of obesity or distributions of body weight. This can be problematic, considering that the prevalence of obesity at any point in time is a result of decades of differing rates of moving between normal-weight and overweight, and overweight and obese. Some of these rates reflect historic environments and lifestyles, and are markedly different to those of more recent times. A more accurate method would be to base future prevalence estimates on the most recent measures available of transitions between these states.

Using the most recent measures of overweight and obesity incidence, from the Australian Diabetes, Obesity and Lifestyle (AusDiab) study, we aimed to estimate the future burden of obesity in Australia. In addition, we estimated the lifetime risk of overweight and obesity for a cohort of people aged 25-29 years in 2000.

Methods

Study population

AusDiab is a national, population-based survey, with baseline measurements of 11247 Australian adults aged ≥25 years conducted during 1999-2000. From the 17129 eligible households, 20347 adults completed a household interview and 11247 (55%) had a biomedical examination after an overnight fast, giving an overall response rate of 37% (13). The majority (88%) of participants were born in either Australia or the United Kingdom, 94% spoke English at home, and 0.8% were Aboriginal or Torres Strait Islander (13). In 2004-2005, all participants (*n*=11247) were invited to a follow-up examination. Those who refused further contact (*n* =128), were deceased (n=310), had moved overseas or into a nursing facility classified as high-care, or had developed a terminal illness (*n*=21) were considered ineligible. Of the 10788 participants eligible for follow-up testing in 2004-05, 6400 (59%) presented for the biomedical examination and/or blood tests (14). The AusDiab study methodology has been reported in more detail previously (13, 14). For the current analysis, participants with missing values for measured height or weight were excluded, leaving 11067 participants at baseline and 6350 at follow-up for analysis.

The study was approved by the ethics committees of the International Diabetes Institute and Monash University, and approval for matching the AusDiab cohort to the National Death Index (NDI) was granted by the Australian Institute of Health and Welfare. All survey participants provided informed consent.

Measurements

At baseline and follow-up, questionnaires were administered, anthropometric measurements were taken and a fasting blood sample was collected. Vital status in AusDiab was assessed annually by linking the AusDiab (*n*=11247) cohort to the Australian NDI. The NDI has a 93.7% sensitivity and 100% specificity for the identification of deaths (15). People unable to be matched to the NDI were assumed to be alive. This study included all deaths up until 1 June 2005 occurring among participants whose height and weight measurements were recorded and whose vital status could be determined at

follow-up. BMI cut-off values classified people into normal weight (<24.9 kg/m^2), overweight (25.0-29.9 kg/m^2) and obese (≥30.0 kg/m^2) (3, 16).

Transition probabilities

Transition probabilities between states were derived for the total population, and separately for males and females, and in five-year age-groups between 25-29 and 85+ years. Incidence was derived from participants whose height and weight measurements were recorded at baseline and either had these measurements repeated at follow-up (*n*=6285) or were recorded as having died within the 5-year time period (*n*=348).

As illustrated in Figure 1, transitions were allowed only between adjacent states. Small numbers of people moved from normal weight to obese (1 male and 7 females) and obese to normal-weight (1 male and 5 females) over the five-year period. However because of the small numbers of such transitions, only transitions to adjacent states were allowed. The people who moved from normal-weight to obese, for example, were considered to have moved from normal-weight to overweight. Back transitions from obese to overweight and overweight to normal-weight were also allowed. Thus the transition probabilities include: (1) normal-weight to normal-weight; (2) normal-weight to overweight; (3) normal-weight to dead; (4) overweight to normal-weight; (5) overweight to overweight; (6) overweight to obese; (7) overweight to dead; (8) obese to overweight; (9) obese to obese; (10) obese to dead. People missing data at the follow-up survey (2005) but not recorded as dead were assumed to have the same probability of transitioning between states as people who were not missing at follow-up.

The transition probability of becoming overweight from normal-weight was calculated – for the total population and separately for males and females – as the number of new cases of overweight divided by the normal-weight population at baseline. The transition probability of becoming obese from overweight was calculated as the number of new cases of obesity divided by the overweight population at baseline. Age-specific mortality probabilities for the Australian population (17) were apportioned across the three BMI categories to reflect the distribution of mortality by BMI category in AusDiab. Due to small numbers in the 85+ years age group, the mortality rate used was taken from national mortality statistics, and assumed not to differ between BMI categories.

We applied the Lowess smoother to all transition probabilities between ages 25 and 85 years to reduce the amount of 'noise' in transition probabilities (18) and influential outliers were removed. A sensitivity analysis was conducted to assess the impact of the smoothing of probabilities and removal of influential outliers.

Life table models

Abridged 5-year multi-state life-tables were constructed with three alive states (normal-weight, overweight and obese). These models were based on the transition probabilities outlined above. In the construction of multi-state life tables the transition probabilities followed the Markov assumption that implies that the probability of moving from one state to another depends only on the current state and is independent of all preceding states. An exponential probability of mortality was assumed within each 5-year age group. Starting prevalences for the 25-29-year age group were weighted to reflect the Australian population, and based on AusDiab.

Stationary life tables

Two sets of static multi-state life-tables were created. The first simulate the 5-yearly progression of a cohort of 25-29-year-olds with the 2000 prevalence of normal-weight, overweight, and obesity. The second simulate the 5-yearly progression of a cohort of normal-weight 25-29-year-olds in the year 2000. Both sets of life tables were exposed to the incidence and mortality observed between 2000 and 2005 throughout their future lifetime and followed until death or age 85+ years.

Dynamic life-tables

The dynamic multi-state life-tables were created to analyse the future population impact of the current transition rates between the year 2000 and 2025. The dynamic models are a series of linked multi-state life-tables, one for each 5 years between 2000 and 2025. In these linked life-tables, the distribution of people between the different states at a given age (a) and time (t) depends on the distribution at age (a-1) and time (t-1) and on the transition probabilities between states between (t-1) and (t). The baseline multi-state life-table population, representing the age structure in Australia in the year 2000 was created by applying the age-specific population numbers for the year 2000, and the age-specific prevalence of normal-weight, overweight and obesity observed in AusDiab, to the

stationary life-table structure described above. The mortality rate was assumed first, to remain constant and second, to decrease by 2.2% per year, reflecting the average decrease in all-cause mortality rates in Australia between 2000 and 2004 (17). Each 5 years a new cohort of 25-29-year-olds entered the model, based on population projections between 2008 and 2025 from the Australian Bureau of Statistics (14), and assuming the prevalence of normal-weight, overweight and obesity at age 25-29 years changed to the same extent as that of those aged 30-34 years. From these dynamic models, age-specific and total prevalence of normal-weight, overweight and obesity were derived for each 5 year-period between 2000 and 2025.

Analyses were performed for the total population, and for men and women, using sex-specific transition probabilities. The life-table models were constructed in Microsoft Excel 2007.

Results

Of the 11067 participants included at baseline (2000) with height and weight measurements, 6285 returned for follow-up (2005) (Table 1). There was no difference between the mean age at baseline of the population at baseline and follow-up. The unweighted prevalence at baseline of normal-weight, overweight and obesity was 37.6%, 40.1% and 22.3%, respectively. Over the 5-year period, there were 345 deaths recorded – 137 in those of normal-weight, 140 in those of overweight and 68 in those with obesity.

We analysed the body weight trajectory of a cohort of normal-weight 25-29-year-olds from year 2000 (Table 2A) and found that by the time this cohort reaches age 60-64 years, 27.5% will be normal-weight, 35.6% will be overweight, 29.3% will be obese and 7.6% will be dead. Of those alive at age 60-64 years, an estimated 29.7% will be normal-weight, 38.6% will be overweight and 31.7% obese.

By the time the cohort of people aged 25-29 years in 2000 with the 2000 prevalence of normal-weight, overweight and obese reach age 60-64 years, 20.2% will be normal-weight, 32.5% overweight, 38.7% obese and 8.6% dead. Of those alive at age 60-64 years, an estimated 22.1% will be normal-weight, 35.5% will be overweight and 42.4% will be obese.

By the time the cohort of people aged 25-29 years in 2000 with the 2000 prevalence of normal-weight, overweight and obese reach age 60-64 years, at some time in their lives 59.6% of females and 51.8% of males will have been obese (Table 3). Over their entire lifetime (until age 85+ years), an estimated 66.3% of females and 59.4% of males will have been obese.

We examined the average number of years and the percentage of their remaining lifespan that females and males will live in the normal-weight, overweight and obese states. On average, females of the 2000 distribution of BMI categories aged 25-29 years will live another 56.0 years; 19.2 years normal-weight, 15.6 years overweight and 21.2 years obese. Males aged 25-29 years will live another 51.0 years on average; 13.2 years normal-weight, 21.4 years overweight and 16.4 years obese. Figure 2 illustrates that as a proportion of their remaining lifespan, females aged 25-29 years will live on average 34.3% normal-weight, 27.8% overweight and 37.9% obese, whilst males aged 25-29 years will live on average 25.8% normal-weight, 42.0% overweight and 32.2% obese.

Between 2000 and 2025 the prevalence of normal-weight in Australian adults is predicted to decrease from 40.6% (48.3% of females and 32.8% of males) to 27.9% (31.4% of females and 24.8% of males). The prevalence of obesity is predicted to increase from 20.5% (22.0% of females and 19.1% of males) to 34.0% (40.3% of females and 34.6% of males) (Figure 3). For the age groups 25-44 years, 45-64 years and 65+ years, the prevalence of normal-weight will decrease between 2000 and 2025 from 48.6%, 31.8% and 36.7% to 30.2%, 25.9% and 27.7% respectively; while the prevalence of obesity in these age groups will increase from 16.7%, 26.2% and 19.6% to 29.2%, 37.3% and 36.9%, respectively. Adjusting predictions for the historic 2.2% (approximately) annual decline in mortality rates had little effect on the results.

Discussion

Of the normal-weight 25-29-year-olds in the year 2000, by age 60-64 years an estimated 29.3% will be obese. As a proportion of their remaining lifespan, females and males aged 25-29 years in 2000 will live 37.9% and 32.2% of it with obesity, respectively. Overall,

between 2000 and 2025 the prevalence of normal-weight will decrease from 40.6% to 28.1% and the prevalence of obesity will increase from 20.5% to 33.9%.

By the time the 25-29-year olds of 2000 reach age 60-64 years, over a third will be obese, and at some time in their lives over half will have had a BMI≥30 kg/m^2. The difference between these estimates is due to the approximate 10% probability at each age group of transitioning into the overweight category from obese.

Our projections are somewhat lower than previously published projections (5, 9, 10, 12). For example, projections for England – currently with a similar prevalence of overweight and obesity to Australia – suggest that by 2025 47% of males and 36% of females will be obese (9). Stewart et al. (2009) have reported that by 2020 approximately 45% of Americans will be obese. The starting prevalence of obesity is higher for the US. However, the difference in projections between ours and other studies may be due to the methods of projection used. Foresight, for example, obtained their projections for the United Kingdom by implementing non-linear regression analysis of historical cross-sectional data of the BMI distribution of the population, whereas we project the burden of obesity based on the national transition probabilities observed between 2000 and 2005.

This is the first time such a method has been used to obtain obesity projections. A recent paper demonstrated that use of a similar incidence-based model could predict future trends by validating against historical follow-up data in the United States (19). Recent evidence suggests that the rate of increase in BMI has slowed in recent years in Australia (2) and some other countries (20-24), presenting a serious limitation to methods of projection based on past prevalence. If incidence rates had, indeed, slowed between 2000 and 2005 relative to earlier years, this would directly drive differences between our results, which use recent incidence data, and other results, which are influenced by historical rates of rise in the BMI distribution.

Recent Australian data (2007/08) on the prevalence of obesity suggests that since 2000 there has been a continued progression towards an increasingly greater prevalence of obesity (25), and our projected prevalence estimates for 2005 and 2010 are fairly similar to these observed prevalences.

Important limitations to our study are that BMI transition and mortality probabilities were derived from different sources, and also that the study is subject to the potential selection bias of the AusDiab study – due to the exclusion of individuals not living in private residential homes (eg. prisoners, hospitalised patients and people residing in nursing homes), the potential healthy volunteer bias, and the 37% and 61% response rates to the first and second surveys, respectively. Loss to follow-up may mean that the incidence rates are not wholly reflective of population incidence rates (14). However in this case, an underestimation of BMI levels is most likely, as people absent at follow-up were less likely to have completed tertiary education (14). Lower educational attainment is a proxy marker of low socio-economic status, associated with higher body weight in developed countries (26-28). The starting or "input" prevalences of normal-weight, overweight and obesity for 25-29 years olds, to which age-specific incidence transition probabilities are later applied, has considerable influence over the projected prevalence results. An increase in prevalence of obesity in this group is likely, considering the upwards transitioning of all other age groups, but the age-specific transition probabilities for this first age group are unknown. Thus, for the dynamic life tables we chose to increase these starting prevalences in the years 2010 to 2025 to the same extent as the increase in the age group above (30-34 years).

As discussed, the key advantage of our study is that the future prevalence estimates are based on recent measures of incidence. Other strengths include the measured height and weight informing our BMI calculations, and the high quality of the mortality follow-up data.

Our projections of the obesity burden will be realised over coming decades if nothing occurs to affect 2000-2005 transition rates over time. But as the trend over recent decades has been towards increasingly obesogenic environments and a higher prevalence of obesity, our projections could easily be surpassed. A study by Parikh et al. (2007) reported that in the US the incidence rates of overweight and obesity increased two-fold and three-fold respectively, between the 1950s and 1990s (4). Our findings suggest that effective intervention to reduce current levels of weight gain is essential if we are to avoid the predicted burden of obesity.

In conclusion, our findings suggest a 70% increase in the prevalence of obesity amongst Australian adults over coming decades; and a high probability of future obesity amongst

even contemporary normal-weight young adults. Avoiding such levels of obesity will likely require considerable intervention.

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Figure 1. Pictorial model of the age-specific transitions

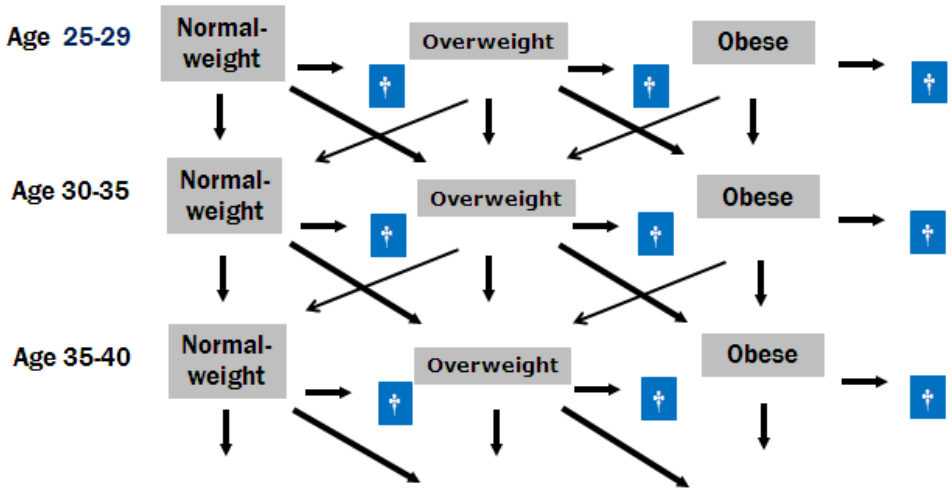


Table 1. Study population characteristics at baseline (1999/2000) and 5-year follow-up (2005)

Characteristic	Females	Males	Total
Population at baseline			
***n* at baseline**	6072	4995	11067
Mean age at baseline, years (SD)	51.4 (14.4)	51.9 (14.3)	51.6 (14.4)
Number with normal weight at baseline (%)	2667 (43.9%)	1492 (29.9%)	4159 (37.6%)
Number with overweight at baseline (%)	1974 (32.5%)	2462 (49.3%)	4436 (40.1%)
Number with obesity at baseline (%)	1431 (23.6%)	1041 (20.8%)	2472 (22.3%)
Number of deaths in those with normal weight (%)	58 (2.2%)	79 (5.3%)	137 (3.3%)
Number of deaths in those with overweight (%)	46 (2.3%)	94 (3.8%)	140 (3.2%)
Number of deaths in those with obesity (%)	32 (2.2%)	36 (3.5%)	68 (2.8%)
Population at follow-up			
***n* at follow-up**[a]	3425	2860	6285
Mean age at baseline, years (SD)	51.4(14.4)	51.9(14.3)	51.6(14.4)
Number with normal weight at baseline (%)	1542 (44.4%)	849 (29.7%)	2396 (37.8%)
Number with overweight at baseline (%)	1146 (33.1%)	1418 (49.6%)	2560 (40.7%)
Number with obesity at baseline (%)	763 (22.3%)	593 (20.7%)	1356 (21.6%)
Number of transitions of:			
• **normal-weight to normal-weight**	1187	644	1831
• **normal-weight to overweight**	326	204	530
• **normal-weight to obese**	7	1	8
• **overweight to normal-weight**	113	81	194
• **overweight to overweight**	781	1136	1917
• **overweight to obese**	248	201	449
• **obese to normal-weight**	5	1	6
• **obese to overweight**	70	50	120
• **obese to obese**	688	542	1230
Five-year probability of transition normal-weight to overweight[b]	21.4%	24.0%	22.4%
Five-year probability of transition overweight to obese[b]	21.7%	14.2%	17.5%

Values in parentheses are 95% CIs

The figures presented have not been weighted to account for the age and sex distribution of the Australian adult population. Thus, the figures do not correspond to those of other reports regarding, for example, the prevalence of normal-weight, overweight and obesity in Australia. They are also not the figures used for comparison with the projections of normal-weight, overweight and obesity in 2025 (which have been weighted, and are compared to weighted 2000 prevalences).

[a] Participants who could not be allocated a BMI value or whose vital status could not be confirmed at follow-up were excluded.

[b] Participants who died during the 5-year period were not included in the denominator. The probability of transition includes only those alive at follow-up.

Table 2A. Age-specific prevalence of normal-weight, overweight and obesity in a cohort of normal-weight 25-29-year-olds using current incidence and mortality rates held constant for the life of the cohort

Age (years)	Alive (%)			Dead (%)	Of those alive (%)		
	Normal-weight	Overweight	Obese		Normal-weight	Overweight	Obese
25-29	100.0	0.0	0.0	0.0	100.0	0.0	0.0
30-34	77.1	22.9	0.0	0.0	77.1	22.9	0.0
35-39	61.5	33.7	4.6	0.3	61.7	33.8	4.6
40-44	50.3	38.2	10.7	0.8	50.7	38.5	10.8
45-49	42.0	39.4	17.1	1.6	42.6	40.0	17.4
50-54	35.8	38.8	22.7	2.7	36.8	39.9	23.3
55-59	31.1	37.4	26.9	4.6	32.6	39.2	28.1
60-64	27.5	35.6	29.3	7.6	29.7	38.6	31.7
65-69	24.6	33.2	29.8	12.4	28.1	37.9	34.0
70-74	22.0	29.9	28.4	19.6	27.4	37.2	35.4
75-79	19.1	25.2	24.4	31.4	27.8	36.7	35.5
80-84	14.9	19.2	17.1	48.8	29.1	37.5	33.3
85+	9.5	12.8	8.2	69.6	31.3	41.9	26.8

Data refer to the percentage of a cohort of normal-weight 25-29-year-olds.

Table 2B. Age-specific prevalence of normal-weight, overweight and obesity in a cohort of 25-29-year-olds with initial prevalence of normal-weight, overweight and obesity as observed in the year 2000, and using current incidence and mortality rates held constant for the life of the cohort

Age (years)	Alive (%)			Dead (%)	Of those alive (%)		
	Normal-weight	Overweight	Obese		Normal-weight	Overweight	Obese
25-29	53.9	31.4	14.7	0.0	53.9	31.4	14.7
30-34	44.9	37.2	17.6	0.4	45.0	37.3	17.6
35-39	37.8	39.1	22.2	0.9	38.1	39.4	22.4
40-44	32.3	38.8	27.4	1.5	32.8	39.4	27.9
45-49	27.9	37.3	32.4	2.4	28.6	38.3	33.2
50-54	24.7	35.6	36.2	3.6	25.6	36.9	37.5
55-59	22.2	34.0	38.3	5.5	23.5	36.0	40.5
60-64	20.2	32.5	38.7	8.6	22.1	35.5	42.4
65-69	18.6	30.6	37.5	13.2	21.5	35.3	43.2
70-74	17.3	28.0	34.6	20.2	21.6	35.1	43.3
75-79	15.5	23.8	29.0	31.7	22.7	34.9	42.4
80-84	12.7	18.0	19.9	49.4	25.0	35.6	39.4
85+	8.6	11.4	9.4	70.7	29.2	38.8	32.1

Data refer to the percentage of a cohort of 25-29-year-olds initially with 2000 prevalence of overweight and obesity

Table 3. Lifetime probability of ever being obese, in a cohort of 25-29-year-olds with initial prevalence of normal-weight, overweight and obesity, as observed in the year 2000, and using current incidence and mortality rates held constant for the life of the cohort

Age	Cumulative incidence of obesity		
(years)	Females	Males	Total
25-29	13.6	15.6	14.7
30-34	23.5	21.9	21.1
35-39	32.8	27.5	27.9
40-44	40.5	33.1	34.6
45-49	46.9	38.5	40.9
50-54	52.1	43.5	46.5
55-59	56.1	48.0	51.3
60-64	59.6	51.8	55.2
65-69	62.3	54.8	58.1
70-74	64.3	57.0	60.2
75-79	65.5	58.4	61.5
80-84	66.2	59.1	62.1
85+	66.3	59.4	62.3

Data refer to the percentage of a cohort of 25-29-year-olds initially with initial prevalence of overweight and obesity, as applied in the year 2000.

Figure 2. Percentage of remaining years of life lived in normal-weight, overweight and obese categories for people age 25-29 years with initial prevalence of normal-weight, overweight and obesity as observed in the year 2000.

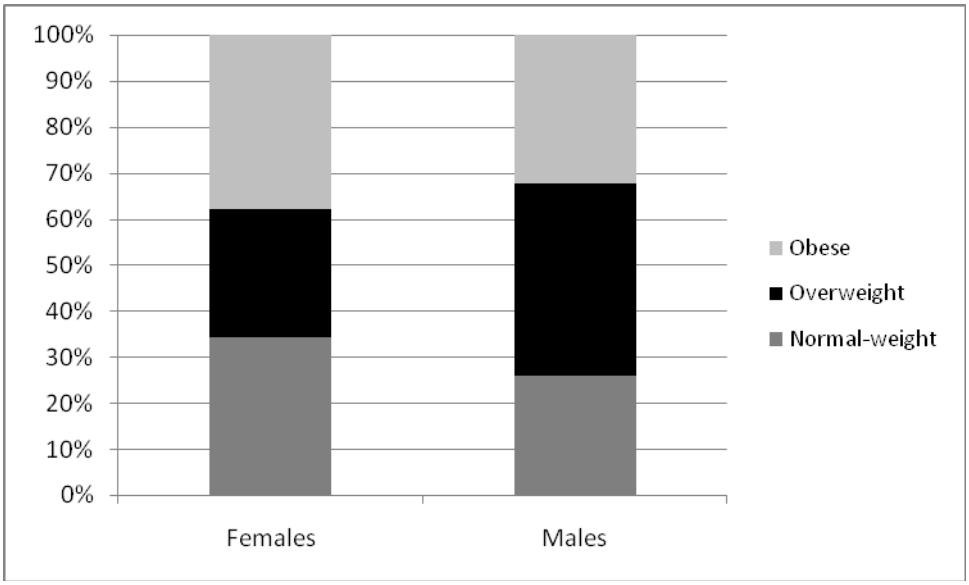


Figure 3. Projected prevalence of normal-weight, overweight and obesity for the total population from 2000 to 2025 using current incidence and mortality rates held constant for the life of the cohort

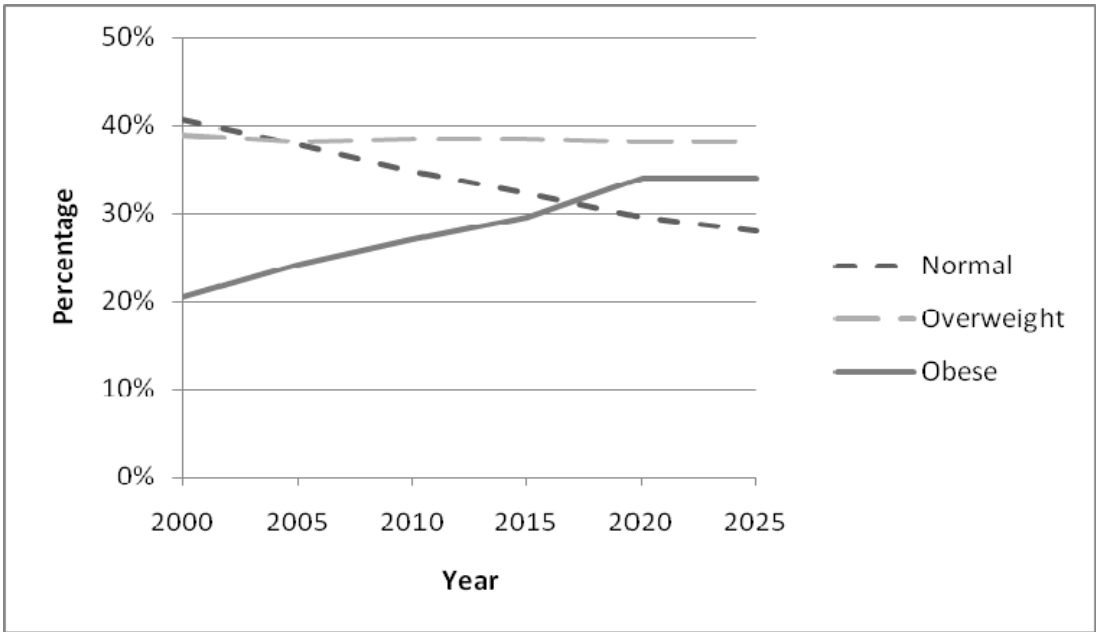


Table 4. Projected prevalence of normal-weight, overweight and obesity from 2000 to 2025 using current incidence and mortality rates held constant for the life of the cohort

A. Total

BMI category	Age group (years)	Prevalence (%)					
		2000	2005	2010	2015	2020	2025
Normal-weight	**25-44**	48.6	44.7	40.6	36.7	33.2	29.8
	45-64	31.8	31.7	30.7	29.3	27.3	25.9
	65+	36.7	32.5	29.7	28.5	27.1	27.7
Overweight	**25-44**	34.7	35.6	37.7	39.3	40.4	41.0
	45-64	42.0	39.7	38.4	37.7	37.2	36.8
	65+	43.7	41.1	39.3	37.9	34.9	35.4
Obesity	**25-44**	16.7	19.7	21.7	24.0	26.4	29.2
	45-64	26.2	28.6	30.9	33.0	35.6	37.3
	65+	19.6	26.4	31.0	33.7	37.9	36.9

B. Females

BMI category	Age group (years)	Prevalence (%)					
		2000	2005	2010	2015	2020	2025
Normal-weight	**25-44**	59.8	53.8	48.4	41.9	36.5	30.9
	45-64	37.3	39.0	36.6	35.3	32.5	30.0
	65+	39.9	37.0	35.7	33.8	33.5	33.7
Overweight	**25-44**	23.9	25.7	27.2	27.3	27.1	26.5
	45-64	33.4	32.1	31.1	30.9	30.4	29.8
	65+	37.1	32.1	30.5	29.7	29.3	28.9
Obesity	**25-44**	16.3	20.5	21.4	30.8	36.4	42.7
	45-64	29.3	28.9	32.3	33.8	37.1	40.2
	65+	22.9	30.8	33.8	36.6	37.3	37.4

C. Males

BMI category	Age group (years)	Prevalence (%)					
		2000	2005	2010	2015	2020	2025
Normal-weight	**25-44**	37.5	37.8	37.4	36.2	33.8	31.2
	45-64	27.4	24.8	24.8	22.9	22.2	21.8
	65+	31.5	27.1	23.0	20.2	20.1	19.5
Overweight	**25-44**	45.1	41.7	39.7	38.8	38.0	36.9
	45-64	50.0	47.4	45.7	43.6	42.1	41.2
	65+	52.0	51.9	50.1	49.7	48.0	47.0
Obesity	**25-44**	17.4	20.5	22.9	25.0	28.2	31.9
	45-64	22.6	27.8	29.5	33.5	35.8	36.9
	65+	16.6	21.0	26.9	30.1	31.9	33.5

3.5 Obesity-reduction strategies

A detailed understanding of the benefits and risks associated with changes to the distribution of body weight is necessary for choosing the most appropriate obesity-reduction responses, and ultimately, to reverse the potential harm associated with the current body weight trajectory. To date, however there has been little evaluation of the effect of obesity-reduction strategies, and particularly in terms of their effect on the whole-population distribution of body weight.

The paper was accepted for publication as a 'Letter to the Editor' (titled: 'Population versus high-risk interventions for obesity', and included in Appendix A). To follow is the full-length version of the paper, in which we contrast population and high-risk strategies for reducing the burden of obesity in terms of their impact on characteristics of the BMI distribution and the number of premature deaths prevented per annum in Australian adults.

Walls HL, McNeil JJ, Peeters A. A new perspective on chronic disease prevention: why approaches to obesity prevention require more thought. *Epidemiology*.

Note: A letter received in response to this article, and our response (titled: 'Population interventions for obesity') to this letter are included in Appendix B.

Declaration for Thesis Chapter 3.5

Walls HL, McNeil JJ, Peeters A. A new perspective on chronic disease prevention: why approaches to obesity prevention require more thought.

Declaration by candidate

In the case of Chapter 3.4.1, the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Design, analysis and drafting of manuscript	75%

The following co-authors contributed to the work. Co-authors who are students at Monash University must also indicate the extent of their contribution in percentage terms:

Name	**Nature of contribution**	**Extent of contribution (%) (student co-authors only)**
JJ McNeil	Design and drafting of manuscript	N/A
A Peeters	Design and drafting of manuscript	N/A

Candidate signature	**Date**
[redacted]	8 February 2010

Declaration by co-authors

The undersigned hereby certify that:

(1) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors;
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, expect for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (2) granting bodies, (b) the editor or publisher of journals or other publication, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s):

Department of Epidemiology & Preventive Medicine, Monash University

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant].

Co-author signatures	**Date**
[redacted]	10/2/10
[redacted]	7/2/2010

Type of manuscript

Commentary

Manuscript title

A new perspective on chronic disease prevention: why approaches to obesity prevention require more thought

Authors

Helen L Walls, John J McNeil, Anna Peeters

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Abstract

In 1985 Geoffrey Rose demonstrated that the most effective preventive strategies would achieve population-wide shifts in the distribution of risk factors. But obesity provides a more complex situation. The unequal shifting of body mass index (BMI) across the population and the J-shaped relationship between body weight and morbidity/mortality might suggest that uniform population-wide shifts in BMI will not adequately reverse current trends, and have the potential to cause harm at the lower end of the BMI spectrum. We illustrate this by modelling theoretical obesity-reduction strategies, using the 1980 and 2000 Australian BMI distributions as comparators, and calculating the number of premature deaths prevented per annum associated with each scenario. We demonstrate that the ideal approach would be a population-wide intervention directed towards de-skewing the distribution of body weight – reducing the body weight of people at the higher end of the distribution the most; and leaving largely unchanged the body weight of people at the lower end. A detailed understanding of the benefits and risks associated with changes to the distribution of body weight is necessary for choosing the most appropriate obesity-reduction responses, and ultimately, to reverse the potential harm associated with the current body weight trajectory.

Main text

In 1985 Geoffrey Rose demonstrated that those preventive strategies most likely to affect the population burden of disease would achieve population-wide shifts in the distribution of risk factors.[1] The large number of individuals making up the bulk of the distribution and exposed to a small risk is responsible for more morbidity and mortality than those at the extreme right of the distribution despite their lower individual risk.[1 2] Using plasma cholesterol and blood pressure as examples, Rose demonstrated that a small leftward shift in the population distribution could in theory decrease morbidity/mortality more than a large decrease in risk confined to the 'high risk' individuals. This approach has become a fundamental tenet of prevention.

In the case of obesity the situation is more complex as a result of key differences between the nature of the distributions of and risks conferred by cholesterol/blood pressure and those conferred by body weight [Table 1]. Whilst the increased risk conferred by cholesterol or blood pressure increases progressively across the population distribution the increased risk conferred by increasing body weight is J-shaped, and affects mainly the upper end of the weight distribution. In addition, the increasing prevalence of obesity in Australia and other developed countries is a result of an increased skewing of the distribution of body weight rather than a simple uniform shift as with cholesterol/blood pressure. Analysis of body mass index (BMI) trends in the US, Australia and New Zealand show that the greatest changes have been in the upper half of the distribution with the BMI's of the lower half having altered very little.[3 4 5]

In Australia, we have analysed in detail the trends in BMI between 1980 and 2000 in urban adults aged between 25 and 64 and have found large increases in the skew of the distribution over this time [Figures 1 and 2]. Consequently, in Australia the BMI of the bottom 2% of the population's BMI distribution was 0.8 BMI kg/m^2 greater in 2000 than in 1980, whereas the BMI of the top 2% of the population's BMI distribution was 4.3 BMI units greater. Figure 2 indicates that the association between the difference in BMI between 1980 and 2000 and the actual level of BMI for a given percentile of BMI is linear and the increasing susceptibility runs across the entire BMI distribution.

As mentioned by Rose and discussed in detail by Adams et al. (2005), a successful population approach to prevention works because everyone benefits a little (achieving a

combined large effect), even people in the lowest part of the distribution.[2 6] However because of the nature of the relationship between body weight and morbidity/mortality, a population approach achieving a uniform shift in body weight across the population may result in harm to those at the lowest end of the distribution.

The unequal shifting of BMI across the population and the J-shaped relationship between body weight and morbidity/mortality suggest that a high-risk strategy may be an appropriate obesity-reduction response. Figure 3 illustrates that the number of deaths associated with BMI is fairly consistent across BMI categories for BMIs above 27 kg/m^2. However the skewed BMI distribution (likely to reflect an interaction between genes and the environment) also suggests that a population approach would be appropriate if it de-skewed the body weight distribution of the population. The goal of a population-wide intervention might appropriately be directed towards restoring an environment (in the broadest sense of the word) that will de-skew the distribution of body weight – reducing the body weight of people at the higher end of the distribution the most; and leaving largely unchanged the body weight of people at the lower end of the distribution.

We analysed a selection of theoretical responses to obesity-reduction strategies, using descriptors of the 1980 and 2000 Australian BMI distribution as comparators, and also the number of premature deaths prevented per annum in Australians aged 25-64 associated with each scenario (Table 2). For this exercise we have used estimates of the association between BMI categories and mortality as described in a recent study (2009) of 900,000 adults in 57 prospective studies.[7]

Scenario 1 is a population approach that reduces all levels of the population distribution by 1.5 BMI units (as was observed in Cuba during its economic crisis of 1989-2000[8]). Scenario 2 is a high-risk approach that reduces the weight of the obese individuals (BMI ≥30kg/m^2) by 5kg (the order of which may be expected by current lifestyle and dietary interventions[9 10 11]). Scenario 3 is a more targeted high-risk approach that reduces the weight of all the morbidly obese individuals (BMI ≥40kg/m^2) by 50kg (a figure achievable by surgical intervention[12 13]). Scenario 4 is a hypothetical population approach that acts unequally across the range of body weight such that it de-skews the distribution, achieving the 1980 distribution of BMI.

In terms of BMI indicators, the comparison illustrates that a closer approximation to the mean and percentage of overweight/obesity and obesity of the earlier (1980) distribution is

achieved by the population-based strategies (Scenario 1) than the high-risk strategies (Scenarios 2 and 3). However there are large increases in the prevalence of underweight with this strategy. (This is an important problem with population-wide scenarios enacting equal reversal of weight gain amongst the different risk groups). Interestingly, it would take a reduction in the whole distribution by 2 BMI units – greater than that observed during Cuba's economic crisis – to return the distribution to its mean of 1980. One of the high-risk strategies (Scenario 2) achieves a good outcome for the prevalence of obesity. Scenario 3 does little for the percentage overweight/obesity *or* obesity, but is arguably very beneficial for those individuals targeted. Scenario 4, by definition, achieves an approximation of the 1980 BMI distribution.

Based on the recently reported association between BMI categories and mortality, Scenario 4 is clearly the most effective of the strategies at preventing premature deaths in Australians aged 25-64 (48501 per annum). This population strategy prevents almost a third as many deaths (18318 per annum). Of the high-risk approaches, Scenario 2 is relatively effective at preventing deaths (22602 per annum). Change in risk is clearly an important consideration, potentially altering conclusions quite markedly.

As the scenarios demonstrate, high-risk approaches can be relatively effective strategies, particularly until we are certain we are not creating harm at the lower end of the distribution by using population strategies. But there are other reasons for not dismissing the population approach. As Rose discussed, the extremes of a distribution are determined by what is happening in the middle. Generally, a population approach is the ultimate answer to a health problem that has a strong environmental determinant.[2] Furthermore, weight loss resulting from interventions targeting the high-risk groups is generally not maintained (with weight loss through bariatric surgery being an exception). Population approaches have the potential to be most effective in the long-term, and the scenarios demonstrate that they are also useful in the short-term. But the potential for an increase in the risk of underweight should be investigated in more detail, because of its potential implications for the public health response to obesity. An effective population strategy will lower body weight in the population, but do so unequally across the different levels of body weight such that the underweight remain largely untouched and skew decreases, otherwise the higher end of the distribution will not be effectively addressed. Such a strategy could restore the 1980 distribution of body weight (or that of another earlier time). Research to understand the effect of any population strategy across the entire

distribution is crucial. It is also likely that a combination of high-risk and population approaches will be required.

The most appropriate obesity-reduction response is less clear than the response in some other areas of prevention. With obesity, it is not a simple choice of high-risk versus population-based approaches. Our analyses combined with the observations of Geoffrey Rose illustrate three critical points: (1) we need to monitor population changes across the whole distribution of body weight; (2) we need to further research causes of the inequality of the shifts; and (3) we need to ensure obesity-reduction strategies are evaluated with consideration of these factors. A detailed understanding of the benefits and risks associated with changes to the distribution of body weight is necessary to successfully reverse the potential harm associated with the current body weight trajectory.

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Figure legends

Table 1. Comparison of characteristics of cholesterol-reduction and obesity-reduction strategies

Table 2. Population impact of theoretical obesity-reduction strategies

Figure 1. Distribution of BMI in 2000 in urban adult Australians aged 25-64 years from capital cities

Figure 2. Mean-difference plot of BMI change between 1980 and 2000 in urban adult Australians aged 25-64 years from capital cities

Figure 3. Excess number of deaths per annum due to body weight in Australian men aged 25-64 years, based on different estimates of mortality associated with body weight

Table 1. Comparison of characteristics of cholesterol-reduction and obesity-reduction strategies

	Cholesterol-reduction	Obesity-reduction
Current distribution of risk factor	Approximately normal (limited skewing)	Increasingly skewed
Exposure/Disease relationship	Progressively increases	J-shaped

Table 2. Population impact of theoretical obesity-reduction strategies

	Mean BMI	% underweight (BMI≥18.5)	% overweight + obese (BMI≥25)	% obese (BMI≥30)	No. of deaths in Australians aged 25-64 prevented per annum*
Australia in 1980	24.8	2.3	41.7	9.7	
Australia in 2000	**26.7**	**1.0**	**59.4**	**21.0**	
Australia in 2000, modified by the following intervention types:					
Scenario 1: 1.5 BMI unit (leftwards) uniform shift in pop.	25.2	4.5	45.5	14.6	18318
Scenario 2: 5kg weight loss in obese (BMI ≥30kg/m^2)	26.3	1.0	59.4	13.6	22602
Scenario 3: 50kg weight loss in morbidly obese (BMI ≥40kg/m^2)	26.3	1.0	58.4	19.2	9729
Scenario 4: A de-skew to resemble 1980 pop.	24.8	2.3	41.7	9.7	48501

* The relative risks (RRs) on which these calculations are based are those reported by the Prospective Studies Collaboration (2009), a recent study of 900, 000 adults in 57 prospective studies. The RRs used to calculate the excess number of deaths are as follows: BMI 15-17.5kg/m^2, RR 1.82 for males, 1.70 for females; BMI 17.5-20 kg/m^2, RR 1.27 for males, 1.18 for females; BMI 20-22.5 kg/m^2, RR 1.09 for males, 1.07 for females; BMI 22.5-25 kg/m^2, RR 1.00 for males; 1.00 for females; BMI 25-27.5 kg/m^2, RR 1.06 for males, 1.03 for females; BMI 27.5-30 kg/m^2, RR 1.17 for males, 1.17 for females; BMI 30-32.5 kg/m^2, RR 1.41 for males, 1.28 for females; BMI 32.5-35 kg/m^2, RR 1.57 for males, 1.46 for females; BMI 35-37.5 kg/m^2, RR 1.79 for males, 1.65 for females; BMI 37.5-40 kg/m^2, RR 1.94 for males; 1.91 for females; BMI 40-50 kg/m^2, RR 2.38 for males; 2.16 for females. These RRs are estimates for people aged 35 to 89. They are adjusted for smoking status [7].

Figure 1. Distribution of BMI in 2000 in urban adult Australians aged 25-64 years from capital cities

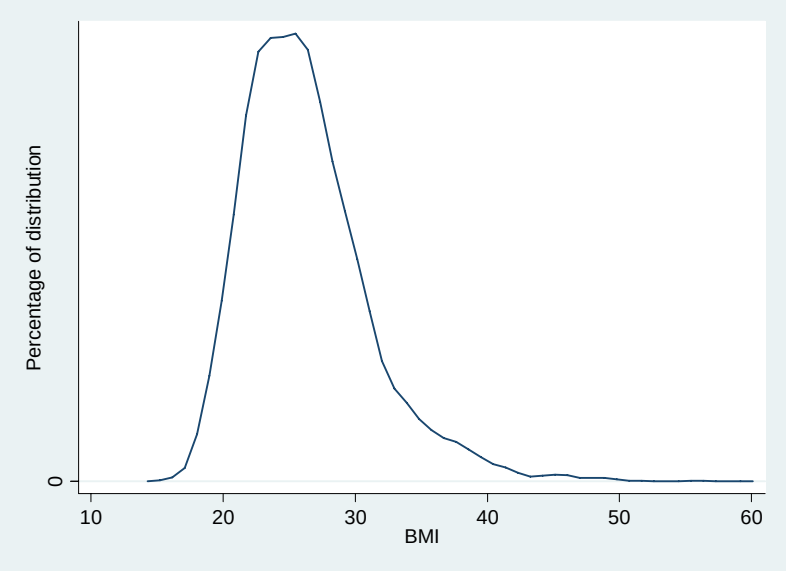


Figure 2. Mean-difference plot of BMI change between 1980 and 2000 in urban adult Australians aged 25-64 years from capital cities

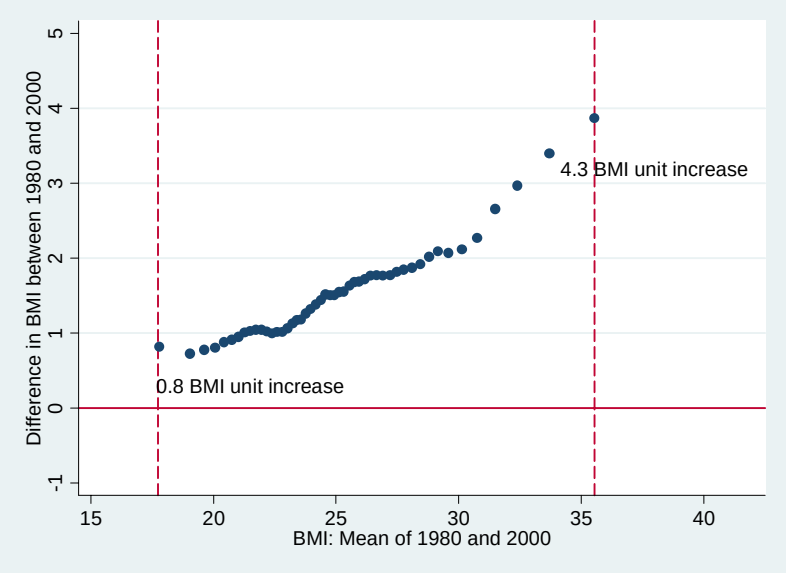
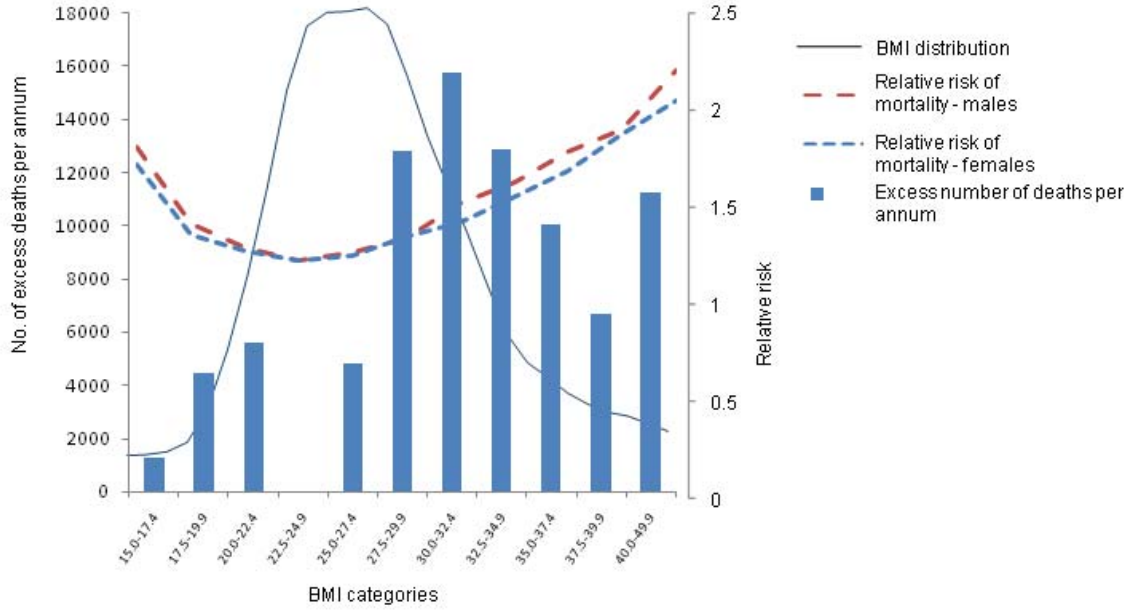


Figure 3. Excess number of deaths per annum due to body weight in Australians aged 25-64 years in 2000, based on recent estimates of mortality associated with body weight



Chapter 4: Discussion

This chapter summarises the major findings of the analyses presented in Chapter 3, describes the associated limitations, discusses the implications, and makes recommendations for future work in this area. Following this, two papers are presented that reflect on how to reduce the prevalence of obesity. The chapter finishes with concluding remarks.

4.1 Key results

The key results from each analysis are summarised below.

4.1.1 Trends in body weight

4.1.1.1 Trends in body mass index

Between 1980 and 2000 the age-adjusted mean increase in the BMI of urban Australian adults was 1.4 kg/m^2 in men and 2.1 kg/m^2 in women. However the increase was greater at the higher end of the distribution than at the lower end; and thus the distribution became increasingly skewed. The prevalence of obesity approximately doubled over this period. Yet as a percentage of total obesity, obesity class I (BMI 30.0-34.9 kg/m^2) decreased while obesity class III (BMI ≥40.0 kg/m^2) more than doubled.

4.1.1.1 Differential trends in different measures of excess body weight

Between 1988-94 and 2005-06 the mean BMI and WC of American adults increased by 1.8 kg/m^2 and 4.7cm, respectively (adjusted for sex, age, race-ethnicity and education). But the 4.7cm increase in WC was more than could be attributed simply to the 1.8 kg/m^2 increase in BMI. This independent increase in WC (of on average, 0.9cm) was consistent across the different weight groups, sexes, education levels and race-ethnicity groups. It

was present in the younger (age <50 years) but not the older (age ≥50 years) age groups. Overall in each BMI category, including normal-weight, the prevalence of having a low-risk WC decreased and the prevalence of having an increased-risk or substantially-increased-risk WC increased.

4.1.2 Obesity in a low-income country – the case of Vietnam

Underweight and overweight were both prevalent in 2004 in urban Hanoi adults. Based on Asian and Vietnamese-specific cut-points, overweight (including obesity) was found in 30% of males and 32% of females; and an increased-risk or substantially-increased-risk WC in 28% of males and 26% of females, respectively. Underweight was found in 15% of males and 13% of females. However based on the WHO cut-points – those commonly used but arguably more appropriate for Caucasian populations than Asian populations – the prevalence of underweight remains the same and the prevalence of overweight (including obesity) affects 14% of males and 13% of females. The prevalences based on Asian-specific cut-points are similar to those found by Cuong et al. (2007) in an analysis of body weight in HCMC.

4.1.3 Predictors of increasing waist circumference

Between 1999-2000 and 2005 an increase ≥5% of baseline WC occurred in approximately a third of Australian adults. Of the behavioural factors explored, poor diet quality was the key predictor of an increase ≥5% of baseline WC in this cohort. There were generally no significant associations with many expected predictors of weight gain such as low levels of physical activity and television viewing.

4.1.4 Predicting future obesity

Between 2000 and 2025 the total-population prevalence of obesity is predicted to increase from 20.5% to 33.9% in Australia. By the time those people aged 25-29 years in 2000 reach age 60-64 years, 22.1% will be normal-weight and 42.4% will be obese. On average, based on the current life expectancy, normal-weight females aged 25-29 years in 2000 will live another 56.2 years; 26.6 years normal-weight, 15.6 years overweight and 14.0 years obese. Normal-weight males aged 25-29 years in 2000 will live another 51.5 years; 21.6 years normal-weight, 21.1 years overweight and 8.8 years obese.

4.1.5 Obesity-reduction strategies

High-risk and population approaches to obesity reduction can both be relatively effective. However considering the risk of mortality associated with different body weights, high-risk strategies may be more appropriate until we are certain we are not creating harm at the lower end of the distribution by using population strategies. A population approach that de-skewed the population's body weight by reducing most the weight of the heaviest people and leaving largely unchanged the weight of the lightest people would be particularly safe and effective.

4.2 Limitations

The various limitations to the analyses conducted are discussed below. Some have already been addressed in the publications resulting from specific analyses, but here the limitations are discussed in greater depth.

4.2.1 Trends in body weight

4.2.1.1 Trends in body mass index

The major potential limitation to our analysis is the comparability of the surveys from which the results are derived.

The five studies used for this analysis were not exactly comparable; with slightly different populations and response rates, and thus different biases.

The three RFPS surveys were similarly conducted and had relatively high response rates (67%, 75% and 65%, respectively), but the NNS and AusDiab studies were methodologically different and had lower response rates (61% and 37 %, respectively) (217). These low response rates could lead to either an under- or an over-representation of BMI levels. However an under-representation is most likely, as non-response has been linked to having a sedentary lifestyle and lower socio-economic status, which is also associated with higher body weight, in developed countries (245, 246).

The target populations of the three RFPS surveys was residents of Australian capital cities, however Canberra and Darwin were only included in the 1989 RFPS survey (216). The target population of the NNS was residents of all Australian States and Territories (218), while the AusDiab target population was residents of six states and the Northern Territory (216).

Bias could also have been introduced from the different times at which data were collected: for example, the NNS data were collected in all regions between February 1995 and March 1996; whereas the AusDiab data were collected over a 21-month period in 1999 and 2000, with two months in each region.

To help overcome potential incomparability between surveys due to both differences in sampling and true changes in population demographics, we performed analyses stratified by key demographic variables. We found that differences in population demographics and the prevalence and intensity of smoking are unlikely explanations of the trends. This is because we observed similar trends in different age and sex groups, in the Australian-born

subgroup (the best available proxy measure for ethnicity) and in populations of never smokers, and for these subgroups between 1980 and 1995 as for 1980 to 2000.

The finding of a slowing of the previously increasing BMI trends between 1995 and 2000 may be affected by methodological issues. However, our main conclusions – increasing mean BMI, increasing right-skew and large increases in the more severe categories of obesity – also hold true for other time period comparisons (1980 to 1989 and 1980 to 1995). Although self-reported body weight trends continued increasing to 2005 (13), the recent slowing in the rate of increase in mean BMI has also been shown for some population groups in New Zealand and the USA (46, 122, 128) and in a slowing of the annual rate of overweight/obesity increase based on measured BMI in Australian children (247). The link between the rate of BMI increase and the degree of increase in distributional skew is also supported by New Zealand findings (46).

The data are sourced from five cross-sectional surveys and do not reflect a cohort of individuals, but rather describe changing population 'snapshots' of the BMI distribution between 1980 and 2000. A five-year longitudinal analysis of participants in the AusDiab study (1999/2000 to 2005) showed that gain in weight, BMI and WC have been greatest in individuals of normal weight and in younger people from 2000 to 2005 (248). However such an inference cannot be made from our analysis.

4.2.1.2 Differential trends in different measures of excess body weight

One of the limitations of the study is the short time period between the two more recent surveys, rendering it difficult to make clear inferences about changes over time. For this reason, the differences between the first and second surveys, and second and third surveys were not considered key results.

Inferences cannot be made about the trends in individuals over time. This is because, as explained above (Chapter 4.2.1.1), the results are based on data that are sourced from cross-sectional surveys and do not reflect a cohort of individuals.

That the mean WC values are close to the cut-off values for abdominal obesity could explain why a small increase in WC could lead to a large increase in the prevalence of

increased risk WC (14). However the analysis also used linear regression to explore the continuous changes in WC over and above BMI.

4.2.2 Obesity in a low-income country – the case of Vietnam

This study is based on a population health survey with a reasonably high response rate (73.9%) and measured height, weight and WC. However due to a relatively small sample size (978) and wide confidence intervals, the patterns by age and sex are generally not statistically significant.

The findings are similar to those for adults in urban areas of HCMC. However, these overweight/obesity estimates are likely to be larger, and the underweight estimates lower, than those occurring in rural Vietnam, a pattern shown by Tuan et al. (2008) and observed in a number of low-income countries (249-252).

Changes in trends over time were unable to be explored due to the unavailability of comparable surveys at different time points for the population studies.

4.2.3 Predictors of increasing waist circumference

The five-year follow-up period of the AusDiab study may be inappropriate for the identification of many predictors of WC gain. Five years may be too long, in that behaviours may have changed substantially over that length of time, limiting the relevance of the baseline behaviours. A longitudinal analysis of a shorter follow-up period may result in better identification of behavioural predictors of WC gain.

Analysis in a more representative study than AusDiab (for which response rates were low) would also be appropriate. Loss to follow-up may also mean that the proportion of people who underwent WC gain is not wholly reflective of population incidence rates. There may also be greater self-report bias for levels of some behavioural factors in people of higher body weights; which may explain, for example, our finding of a greater odds of WC gain amongst people with a substantially-increased-risk WC in people who viewed <2 hours/day of television than in people who viewed 4+ hours/day.

Poor diet quality was found to be the key predictor of WC gain in this population, however the composite nature of this dietary variable renders it difficult to understand what components of a diet are protective of a gain in WC. The dietary quality index (DQI) on which the variable is based reflects 10 dietary characteristics – total fat, saturated fat, dietary cholesterol, fruit, vegetables, grains, calcium, iron, dietary diversity and dietary moderation. 'Dietary diversity' reflects the desirability of consuming a variety of foods across and within the food groups to increase exposure to a range of dietary components. 'Dietary moderation' reflects the principle of limiting selected food constituents that contribute to excess risk such as fat, sodium and sugar (253). No one component of the DQI contributes disproportionately more than any other and thus it is difficult to explain the dietary components or patterns explaining the observed findings.

4.2.4 Predicting future obesity

As for the analysis above (Chapter 4.2.3) this analysis used incidence transitions in the AusDiab study, which had relatively low response rates. Future analysis in a more representative national cohort would be appropriate.

The analysis was limited to a projection of the future prevalence of normal-weight, overweight and obesity based on BMI. Future work should extend this analysis to explore the impact on morbidity, mortality and the health care and wider societal cost implications.

4.2.5 Obesity-reduction strategies

The major limitations of this analysis are the comparability of the survey data (1980 RFPS and AusDiab) from which the results are derived and the assumptions on which this analysis is based.

The survey comparability has been addressed in Chapter 4.3.1. The RFPS and AusDiab are methodologically different, and whereas the response fractions to the RFPS were relatively high (67%, 75% and 65%, respectively (217)), AusDiab had a low response fraction (37%) (220). This low response fraction could lead to either an under- or an over-representation

of BMI levels. However an under-representation is most likely, as people absent at follow-up were less likely to have completed tertiary education (254). Lower educational attainment is a proxy marker of low socio-economic status, associated with higher body weight in developed countries (245, 246, 255). The implications of this for our results would be that the reported prevalences of overweight and obesity and number of deaths prevented with each scenario are under-estimations. Most importantly, the actual relative comparison between scenarios would not be affected by an underestimation of body weight in 2000.

The calculation of premature deaths prevented relies on estimates of mortality associated with narrow BMI categories. The relative risks chosen are very influential in regard to the number of deaths prevented by different scenarios. The estimates in this analysis are from the association between BMI and mortality described by the Prospective Studies Collaboration (PSC) in early 2009. The risks of mortality associated with different BMIs in this study are derived from 900,000 adults in 57 prospective studies (30). The risk of mortality is increased at low body weights and high body weights; and the lowest risk is at the upper end of the normal-weight category.[1]

There was considerable debate regarding the association of BMI and mortality prior to the publication of the PSC study (30, 256). A notable study demonstrated what appeared to be a clear association between increasing mortality and BMI is that conducted by Calle et al. (1999) – an analysis of more than 1 million US adults aged 30 years and over in the Cancer Prevention Study II (257). In this study, an increased risk of mortality was observed from a BMI of 25 in never-smokers free of chronic disease. In late 2008, Pischon et al. (2008) released the results of a large cohort study in Europe (The EPIC Study), suggesting that the lowest risks of mortality were at a BMI of around 25 but that an increased risk was observed from a BMI of around 28 (258). The RRs reported by Calle et al. (1999) are less J-shaped than those reported by Pischon et al. (2008), which suggest a particularly high risk of mortality at low body weights and the lowest risk for the lower end of the overweight category.

[1] The RRs used to calculate the excess number of deaths are as follows: for BMI: 15.0–17.5 kg/m^2; RR: 1.82 for males, and 1.70 for females; BMI: 17.5–20 kg/m^2, 1.27, and 1.18; BMI: 20.0–22.5 kg/m^2, 1.09, and 1.07; BMI: 22.5–25.0 kg/m^2, 1.00, and 1.00; BMI: 25.0–27.5 kg/m^2, 1.06, and 1.03; BMI: 27.5–30.0 kg/m^2, 1.17, and 1.17; BMI: 30.0-32.5 kg/m^2, 1.41, and 1.28; BMI: 32.5–35.0 kg/m^2, 1.57, and 1.46; BMI: 35.0–37.5 kg/m^2, 1.79, and 1.65; BMI: 37.5– 40.0 kg/m^2, 1.94, and 1.91; BMI: 40.0–50.0 kg/m^2, 2.38, and 2.16. They are adjusted for smoking status and reportedly appropriate for adults aged 35-89 years.

Depending on which RRs of the association between BMI and mortality are chosen, the change in population body weight epidemic has substantially different impacts on mortality and the value of different intervention scenarios differs. However the PSC study appears to provide the best-estimates of the association between BMI and mortality to date.

4.3 Implications and recommendations for future work

The implications and recommendations relating to specific analyses are described below. However in general, there are four key recommendations that arise from this thesis.

First, countries such as Australia need to implement regular, comparable monitoring of the full population distribution of BMI and WC (and subsequent analysis). Such monitoring, as already takes places in the United States and New Zealand, is vital for understanding population changes in body weight, and informing our understanding of the causes of weight gain, the implications for future levels of obesity, and the most appropriate obesity-reduction responses.

Second, further research is needed to inform our understanding of the causes of weight gain, both at individual and population-levels.

Third, a considerable increase in the prevalence of obesity over coming decades (an approximate 70% increase in Australian adults between 2000 and 2025, for example) is predicted if nothing occurs to affect recent probabilities of transitioning between normal-weight, overweight and obese.

Fourth, it will be essential to include in the evaluation of the impact of obesity-reduction strategies the impact on the full population distribution of body weight, to better inform the overall impact an intervention is likely to have.

4.3.1 Trends in body weight

The trend towards a greater right-skewing of the BMI distribution has implications for the 'obese' BMI category. The overall prevalence of obesity has increased, but the type of obesity that has increased most is that at the extreme end of the distribution. This suggests that analyses focusing on the prevalence of total obesity (obesity classes I, II and III combined) are likely to underestimate the true burden of obesity. It also highlights the importance of regular, comparable survey monitoring of the whole population distribution of body weight, and subsequent analysis which is currently lacking in Australia. Such monitoring should use comparable methods of anthropometric measurement. The comparison of BMI and WC in this thesis was conducted for the United States rather than Australia, as the methods of WC measurement are inconsistent the different Australian surveys.

The findings also suggest that the environmental determinants of weight gain (such as changing dietary patterns due to the increasingly obesogenic environments in which we live) are not acting equally across the population and that population approaches to shift the BMI distribution leftwards may be insufficient on their own to substantially reduce the burden of obesity. Implications and recommendations regarding the individual-level determinants of WC gain are discussed in Chapter 4.2.3 and those regarding the most appropriate obesity-reduction response are discussed in Chapter 4.2.5. The right-skewing of the distribution could be rectified by an obesity-reduction strategy that de-skewed the population distribution – a strategy shown hypothetically in Chapter 4.2.5 to be particularly effective in terms of its effect on the distribution of body weight and the prevention of premature deaths amongst adults.

The differential trends in BMI and WC suggest that in each of the BMI categories the level of metabolic risk may have increased. Using BMI alone to monitor increasing body weight in the population may therefore lead to an underestimation of the associated health burden. The independent increase in WC is evident in people aged <50 but not in older people, suggesting that the changes in our environment have impacted more on younger adults, or that they are the result of more recent environmental change. Further research is needed to explore the cause of these differential trends. These findings also support the monitoring and further analysis of the differences in trends of both BMI and WC. In terms of trends in BMI and WC – and the comparison of these trends – a longitudinal analysis

would elucidate the individual-changes contributing to the observed findings in what have been cross-sectional analyses.

An important area for future monitoring and analysis will be in ethnic and social inequalities in these trends. In the Australian context, careful monitoring and analysis of body weight trends in Aboriginal and Torres Strait Islander people would also provide needed evidence regarding weight gain in this group. For example, in the AusDiab study the methods followed was for only those census collector districts (CDs – the smallest geographic unit defined by the Australian Bureau of Statistics at each census (220)) with less than 10% of the population Indigenous to be included. The rationale for this was to overcome the chance selection of a CD with a large proportion of Aboriginal and Torres Strait Islander people, potentially biasing national and state diabetes estimates. This approach was also considered important as 'aspects such as questionnaire design would have required extensive modifications to reflect the cultural differences' (259). While the acquirement of unbiased national estimates is important, the consequence of this approach was that the Indigenous population represent 2% of the total Australian population but only 0.8% of the total AusDiab population (259). This in turn means that first, a small group of Aborginal and Torres Strait Islander people were surveyed without modification to reflect cultural differences, and second, in Australia, analyses such as of body weight trends cannot be addressed by Aboriginal and Torres Strait Islander status due to small numbers. The seems a deplorable situation considering the extreme nature of health inequalities between Indigenous and non-Indigenous Australians (260).

4.3.2 Obesity in a low-income country – the case of Vietnam

Estimating the prevalence of overweight and obesity in Vietnam using Asian-specific cut-points suggests a much greater burden of overweight and obesity – one affecting almost a third of urban Hanoi adults – than that estimated using the WHO cut-points more appropriate in Caucasian populations. Considering the pace of social and economic change in Vietnam and the unfolding of the 'nutrition transition' (236), an increase in the burden of obesity can be expected. This increase is likely to be faster than that experienced in higher-income countries (236).

Regular and comprehensive monitoring of trends with regard to body weight in the population is essential, as is the development of appropriate interventions and policy responses (taking into account competing issues). Future analyses of BMI in Vietnam would be enhanced by greater power of the surveys on which they are based – by survey sample sizes that ensured significant differences by sex and age group would approach statistical significance.

Further analysis of body weight in rural areas is needed, as is analysis of whole-distribution changes in BMI and WC, and determining the most appropriate BMI and WC cut-points for people of Asian (and in this case, Vietnamese) ethnicity. A comparison of body weight between urban and rural areas would also be useful in terms of informing health policy in Vietnam regarding the future burden of chronic disease in the country. Analysis of social variations in body weight, such as by smoking status and socio-economic status, is also recommended.

4.3.3 Predictors of increasing waist circumference

The longitudinal analysis of behavioural predictors of a gain in WC in Australian adults revealed few predictors, and some counter-intuitive findings. Similar analyses in different populations and over a longer follow-up period will be important to determine whether the lack of the common behavioural predictors of television viewing and physical activity is a function of the longitudinal analysis (in contrast to the cross-sectional, for which high WC has been associated with a number of expected behavioural factors). Exploration of predictors of WC gain informed by life course epidemiology may be a useful avenue for future exploration. A life course approach would involve the measurement of variables likely to influence weight gain at a range of ages that combined, cover the life course. There is substantial evidence suggesting that early life exposures, such as birth weight and childhood socio-economic position, are influential in regard to one's propensity for weight gain in later life (137-139, 261). The entering of such exposure variables into a multivariate model must take into account the temporal relationship between variables and their interrelationships (262).

In a focus on the individual-level determinants of obesity, however, it is easy to overlook the wider context shaping these behaviours – the context in which individuals live their lives. This context is addressed further in Chapter 4.4.

4.3.4 Predicting future obesity

The prediction of the future obesity burden suggests a 70% increase in the prevalence of obesity amongst Australian adults over coming decades (from 2000 to 2025); and a high probability for contemporary normal-weight young adults of becoming overweight or obese.

The projections of the obesity burden are likely to be realised over coming decades if nothing occurs to affect the 2000-2005 transition rates. But as the trend over recent decades has been towards increasingly obesogenic environments and increasing rates of the incidence of overweight and obesity, our projections could easily be surpassed.

In 2008 the Council of Australian Governments (COAG) set a target (backed with financial support) to increase by 5% the proportion of Australian adults at a healthy body weight by 2017, over a 2009 baseline (263). However, based on our projection estimates, this target is unrealistic. Future work can, using the life tables developed, determine what change in current obesity incidence rates or what reduction in BMI units in Australia are required to meet the COAG targets, and provide an indication of more realistic targets by simulating realistic reductions in the projection estimates. Such simulations could also be tied to the effects of particular interventions. Finally, although it seems likely that using this incidence-based model, which captures the probabilities of moving between normal-weight, overweight and obese in Australia between 2000-2005, has significant advantages over a model based on extrapolations of past obesity prevalences of body weight distributions, it would be instructive to validate this claim.

4.3.5 Obesity-reduction strategies

Both high-risk and population approaches to obesity reduction can be relatively effective. A population approach that de-skewed the population's body weight – reducing most the

weight of the heaviest people and leaving largely unchanged the weight of the lightest people – would be particularly safe and effective, but it is unclear how to achieve such a change. In addition to monitoring and analysing changes in the body weight distribution over time, as suggested in Chapter 4.2.1, also important will be the monitoring and analysis of its response to interventions. The quality of such analyses would be enhanced in countries like Australia by the undertaking of regular, comparable nationally-representative biomedical population surveys. The benefits and risks associated with these changes also need to be better understood. Such work will also be informed by a better understanding of the association between body weight and morbidity/mortality, because the relative effectiveness of interventions is dependent on this relationship. Approaches to reducing the burden of obesity need to shift both the distribution of bodyweight leftward and target the higher BMI groups. Further research and evaluation are needed to elucidate the most effective approaches. Analysis of ethnic and social inequalities in the response to interventions will also be important.

4.4 Reflections on how to reduce the prevalence of obesity

4.4.1 Public health and obesity

The increasing prevalence of obesity is now the target of public health campaigns in most developed countries. However the public health targeting of obesity is associated with many limitations, and has the potential for harm.

Here, we discuss the limitations associated with the public health targeting of obesity, and suggest an alternative approach.

Walls HL, Peeters A, Proietto J, McNeil JJ. Public health campaigns and obesity – a critique. *Submitted*.

Declaration for Thesis Chapter 4.4.1

Walls HL, Peeters A, Proietto J, McNeil JJ. Public health campaigns and obesity – a critique. Submitted.

Declaration by candidate

In the case of Chapter 4.2.3, the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Conceptualisation and drafting of manuscript	80%

The following co-authors contributed to the work. Co-authors who are students at Monash University must also indicate the extent of their contribution in percentage terms:

Name	**Nature of contribution**	**Extent of contribution (%) (student co-authors only)**
A Peeters	Conceptualisation and drafting of manuscript	N/A
J Proietto	Conceptualisation and drafting of manuscript	N/A
JJ McNeil	Conceptualisation and drafting of manuscript	N/A

Candidate signature	**Date**
[redacted]	8 February 2010

Declaration by co-authors

The undersigned hereby certify that:

(1) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors;
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, expect for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (2) granting bodies, (b) the editor or publisher of journals or other publication, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s):
Department of Epidemiology & Preventive Medicine, Monash University

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant].

Co-author signatures	**Date**
[redacted]	7/2/2010
[redacted]	6 Feb 2010
[redacted]	10/2/10

Title

Public health campaigns and obesity – a critique

Authors

Helen L Walls, Anna Peeters, Joseph Proietto, John J McNeil

Introduction

Obesity is one of the highest priority issues in public health. There is a widely held view that the increasing rates of obesity will lead to an epidemic of diabetes and a subsequent reduction in life expectancy. Since the 1960s, however, 'all-cause' and cardiovascular-specific mortality rates have decreased steadily in most developed countries, and life expectancy has consistently increased. There are several reasons why obesity may not be strongly related to life expectancy, except at the extremes of the weight distribution. This calls into question the appropriateness of some current public health campaigns targeting obesity, particularly given the limited evidence of effectiveness of such campaigns and the potential harm from continuously reinforcing the undesirability of being overweight. Given these concerns, we advocate shifting the focus towards assisting those with established obesity (high-risk approach), and towards carefully constructed public health strategies focusing on diet and physical activity rather than body weight.

Standfirst

Community-wide public health campaigns targeting obesity have a number of limitations, including the limited evidence of effectiveness of such campaigns on lowering body weight and the negative effects of continuously reinforcing the undesirability of obesity. The focus needs to shift to assisting those with established obesity, and to public health programs that focus less on body weight and more on diet.

Main text

The increasing prevalence of obesity is now the target of public health campaigns in most developed countries. The cause is attributed to societal changes leading to reduced physical activity and increased consumption of calorie-dense foods. Health-promotion campaigns have been established emphasising the desirability of an ideal body weight, achieved by eating less, eating healthier foods, and exercising more. In general these campaigns have had a community-wide focus. There has been no explicit targeting of those with the highest BMIs, partly to avoid stigmatising these individuals and partly because of the limited evidence that any targeted interventions apart from surgery are effective at achieving long-term weight loss.

Population trends

The principal rationale for public health action is the deleterious effect of obesity on health. An increasing prevalence of obesity has been observed in most developed countries. This has been predicted to lead to an 'epidemic' of type II diabetes. This in turn is predicted to slow or reverse the decline in mortality that has been noted in most Western countries over the past 30-40 years (1).

The data accumulated to date have provided relatively little evidence in support of this view. Levels of obesity have been increasing since the 1950s (albeit slowly, initially) in the US and other developed countries (2). Over the same period, life expectancy has continued to increase at an undiminished rate, and cardiovascular-specific mortality rates have also decreased continually (3, 4).

Reasons for limited impact of obesity

A number of factors may explain the apparent discrepancy.

Firstly, it is possible that the deleterious impact of obesity is outweighed by other factors favourably influencing life expectancy. Under this scenario, the rate of decline of all-cause and CVD mortality might be faster still if it was not for the increasing prevalence of diabetes. Examples of factors driving mortality down include population-wide changes such as reductions in the prevalence and intensity of smoking.

Secondly, there is the possibility of improved medical interventions in some of the pathways linking obesity to CVD and all-cause mortality. For example, improved control of

hypertension and better management of hyperlipidaemia may blunt the impact of obesity on adverse health outcomes. Hypertension has been fairly well controlled in recent years and such control may be contributing to the limited effect of rising obesity levels. Overall, factors such as the improved control of hypertension may outweigh the effect of increasing levels of obesity and diabetes.

Thirdly, the impact of diabetes may have been overestimated because its principle adverse effects are experienced by a relatively small segment of the population. A number of cohort studies suggest that the mortality risk of excess body weight becomes substantial only when BMI exceeds 32-35(5). Only 15-25% percent of the US population have BMIs in this range. The risk for this group is thus likely vastly outweighed by the considerably less significant risks associated with body weight in the majority of the population.

Fourthly, it is possible that body weight is not a significant risk factor for mortality. Body weight may act simply as a surrogate for a constellation of attributes of lifestyle, in particular diet, physical activity level and genetic factors (6).

Finally, there is the mysterious 'obesity paradox' in which survival from acute events such as myocardial infarction, heart failure and dialysis for renal failure is improved in patients with mild to moderate obesity (7, 8). The mechanism for this observation is not known, and it may reflect that the more severely ill patients have lost weight on account of the severity of their illness.

These reasons explain why the impact of obesity on health may be more complex than is commonly recognised. This potentially complex relationship raises questions about the most appropriate approaches to controlling obesity at both individual and population-health levels.

Public health campaigns for obesity reduction

Most community-based campaigns to address obesity have adopted a whole-population approach. They emphasise the negative impact of obesity on health and fitness, and place substantial emphasis on the desirability of achieving an ideal body weight. They recommend achieving an ideal body weight by eating healthier food and increasing levels of physical activity.

Despite their logic, there is very limited evidence that such campaigns are effective at addressing obesity, and virtually none that they affect downstream health (9). The results of several programmes have been published. However the only substantially positive ones have been in children (10), in whom behaviour can be controlled more easily. Furthermore, social marketing campaigns that have targeted weight loss explicitly have generally shown poor weight loss outcomes (11). The long-term out-look for the maintenance of weight lost has been particularly disappointing, suggesting a difficulty with the maintenance of weight loss in the longer term. In combination, these issues call into question the value of current community-wide campaigns specifically targeting obesity.

A well recognised downside of the community-based programmes targeting obesity is their promotion of the social desirability of thinness. The desirability of 'normal' body weight is emphasised to an extent that overstates the public health evidence for benefit. The reinforcement by public health campaigns of the already socially entrenched attitudes regarding the undesirability of being overweight may be harmful to many people. The institutional and interpersonal mistreatment of obese people has been well documented (12). But there is little known about the psychological impact on people who are mild to moderately overweight, particularly in women, who face the strongest social pressure to be lean.

High-risk approaches to obesity reduction

The limited success of community-based programmes is matched by limited success in the high-risk approach to severely obese patients. Current drug treatments for obesity have very limited efficacy. Two of the most widely used agents have recently been removed from market because of adverse effects. Only surgery has demonstrated significant efficacy. But access to this intervention is always likely to be limited to the individuals at the very highest risk (13).

Proposed approach

Given the limited evidence of benefit, the possibility of harm, and the uncertainty of the balance between 'benefit' and harm associated with community-wide campaigns targeting obesity, the approach to obesity should shift towards evidence-based programmes likely to provide the greatest population impact.

The recommendation is:

1. Public health campaigns should avoid a focus on ideal body weight and weight loss. Instead, the efforts of public health should focus on improved diet quality, where substantial evidence exists of a health benefit, and increased physical activity levels.
2. Further research into surgical and medical approaches to treat those with established obesity.

Healthy diets are typically lower in caloric density and less likely to contribute to obesity. A more beneficial focus of community-wide public health campaigns and one far more easily followed than a focus on weight loss per se may be the adoption of particular diets or dietary components – a less processed diet, vegetarian diet, or diets low in saturated fat and/or sugar, for example.

There is good evidence from prospective studies of vegetarians and non-vegetarians to suggest that dietary factors have an important influence on weight and longevity. A 21-year follow-up study of Seventh-day Adventists in California found that those with more vegetarian diets had lower rates of hypertension, diabetes, coronary artery disease mortality, arthritis, colon cancer, prostate cancer, obesity and mortality than non-vegetarians (14, 15). Other studies have suggested similar benefits of the vegetarian diet; which appear to be due to a higher consumption of vegetables, fruit, nuts and grains, avoidance of meat, and lower saturated fat consumption (16, 17).

However we acknowledge the difficulties associated with the enactment of community-wide public health campaigns urging people to change their behaviour. Considering that individual behaviour is strongly influenced, if not determined, by the wider societal context in which we live, changes to this environment are a likely important ingredient. Thus, promotion of a healthy diet may be best achieved by a combination of health promotion and tax incentives or legislative changes that encourage healthier diets and increased activity levels. Policy and legislative changes to address the many unhealthy choices with which we are faced daily would render easier the adoption of a healthy lifestyle.

Conclusion

The public health and media targeting of obesity (more specifically, body weight) is associated with too many limitations, and may have the potential for harm.

We support a change in the focus of public health to promoting healthy lifestyles rather than a focus on obesity and weight loss. We also believe that further research into medical approaches to addressing established obesity must be prioritised, particularly given the limited options presently available. The focus of community-wide public health campaigns should shift to a focus on dietary changes known to be associated with improved health and increases in levels of physical activity. We also suggest the enactment of high-level policy and legislative changes to address the unhealthy environments in which we live – arguably the only sustainable method of addressing behaviour change (18).

Such a population-health focus has indeed already been chosen by some Governments (19). But for the most part, the attraction to obesity continues. Obesity has been an easier target than components of a healthy lifestyle – visible and more easily measured. However the dangers of stigmatisation may have been underestimated. Dew (2007) has postulated a strong argument for today's popularity of public health campaigns generally, describing public health in Durkheimian terms as today's 'cult of humanity' (20). A focus on obesity is in these terms particularly unsurprising, considering its role as a visual metaphor of the excesses of the Western lifestyle.

Provenance statement

HLW is a PhD candidate at Monash University and prior to this worked with the New Zealand Ministry of Health. AP is a public health researcher who holds positions on a number of obesity-related councils and committees. JP is Professor of Medicine at the Department of Medicine Austin Health, University of Melbourne. JJM is Head of the Monash University School of Public Health and Preventive Medicine. HLW wrote the article. All authors helped formulate concepts and reviewed drafts of the article. JJM is the article guarantor.

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4.4.2 The problem with education and choice

The previous paper (Chapter 4.4.1) explores solutions to the increasing burden of obesity from both individual and population perspectives. Individual choices and subsequent behaviours are part of the explanatory picture regarding body weight, however such a focus should not overlook that individual behaviours are shaped by the wider context in which individuals live. Much of this context is shaped by high-level laws and regulations; and by the incentive for constant growth (achieved through overproduction and overconsumption) in our economic system.

"*We live in an environment that has been dubbed "obesogenic", full of stimuli that encourage us to eat, to take less exercise, and to, above all, consume. This is very much a commercially run environment*". (Tim Lobstein in BBC's "If... we don't stop eating", April 2004) (264).

The pursuit of constant economic growth means that each year food and beverage companies must sell more (or more expensive) foods and beverages. A fast food chain, for example, may have a target of five per cent growth each year, which it can achieve in three ways[1]: paying less for produce, charging more for meals, and selling more meals overall. The most effective way, of course, is to implement all these options at once.

As food production is increasingly big business, the emphasis on growth, rather than well-being, renders consumer health (and environmental sustainability) an externality – outside the buyer-seller relationship – one that future taxpayers have to bear (142). Some companies have chosen to produce healthier foods, a change assisted by an increased awareness and demand for nutritious foods among some consumers (265), but for the most part, the availability of such products is swamped by the unhealthy options still available.

Policy and legislation are critical to addressing the unhealthy components of the food, agriculture, trade, media advertising, transport, urban design and built environments.

[1] Another way is to establish in as many countries as possible – the implementation of this is a likely contributor to the more recent increasing prevalence of obesity in low-income countries.

Here, we critique the paradigm, often perpetuated by the food and beverage industry itself, that the 'obesity problem' can be solved by better education of consumers and a greater number of healthy choices provided by food and beverage companies.

Walls HL, Peeters A, Loff B, Crammond BR. Why education and choice won't solve the obesity problem. Am J Public Health. 2009;49:590-2.

Declaration for Thesis Chapter 4.4.2

Walls HL, Peeters A, Loff B, Crammond BR. Why education and choice won't solve the obesity problem. Am J Public Health. 2009;49:590-2.

Declaration by candidate

In the case of Chapter 4.2.3, the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Conceptualisation and drafting of manuscript	70%

The following co-authors contributed to the work. Co-authors who are students at Monash University must also indicate the extent of their contribution in percentage terms:

Name	**Nature of contribution**	**Extent of contribution (%) (student co-authors only)**
A Peeters	Conceptualisation and drafting of manuscript	N/A
B Loff	Conceptualisation and drafting of manuscript	N/A
BR Crammond	Conceptualisation and drafting of manuscript	25%

Candidate signature	**Date**
[redacted]	8 February 2010

Declaration by co-authors

The undersigned hereby certify that:

(1) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors;
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, expect for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (2) granting bodies, (b) the editor or publisher of journals or other publication, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s):
Department of Epidemiology & Preventive Medicine, Monash University

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant].

Co-author signatures	**Date**
[redacted]	7/2/2010
[redacted]	10/2/10
[redacted]	9 February 2010

EDITORIALS

Why Education and Choice Won't Solve the Obesity Problem

The published industry commitments of the Australian Food and Grocery Council[1] state that members will:

- Expand the range of nutritious foods they produce
- Seek regulatory approval of health claims to encourage more companies to develop nutritious foods and deliver health messages into every home
- Promote information on nutrition and health to our consumers with our communication tools, such as nutrition information labels, in-store information, customer care lines, and Web sites
- Work with retailers, suppliers, advertising agencies, and the media to encourage broader promotion of nutrition and health information
- Encourage government to develop public education campaigns that raise awareness of the consequences of obesity, the benefits of being physically active, and the need to balance food intake and activity
- Advocate and participate in programs that promote nutrition education and physical activity in schools and in communities where food companies are located

Given that the *raison d'être* of industry is profit through increased consumption, it is significant that the Australian Food and Grocery Council should advance these health-promoting policies, which are similar to the goals of health professionals. Policies such as disseminating information about and increasing the availability of healthy food options are ultimately intended to reduce the consumption of unhealthy foods. These proposed solutions to the obesity epidemic have gained popularity with the media and the public as well as with health practitioners.[2–4] Information about good nutrition is primarily provided by health professionals and through social marketing campaigns to increase physical activity and consumption of nutritious food and to decrease portion sizes and consumption of sugary drinks. Thus far, the industry's response to these campaigns has been to increase the number of healthy choices available without reducing the selection of unhealthy foods.[5]

These popular approaches seek to provide consumers with the information and options necessary to make healthy choices; a failure to make a healthy choice, by implication, is a failure on the part of the individual (or possibly the campaign). Although education and access to information are fundamental rights and are important in a democracy, they have negligible impact on obesity. Further, increasing choice is little more than a ruse, more likely to increase consumer confusion than to promote healthy eating.

Changing behavior in any context is a challenge. Attempting to change behavior in an environment that reinforces contrary practice is quite a bold endeavor. Increasing levels of knowledge and awareness (through education or other means) does not always result in behavior change, let alone long-term behavior change.[6–9] This is intuitively obvious to those of us who already know how our physical activity levels and diet could be improved but who fail to act accordingly.

Most importantly, it has been demonstrated that there is great variation in the way people from different demographic groups respond to education and social marketing. Typically, people with already high levels of education respond best to further education; people with lower educational attainment are much less likely to change their behavior as a result of education efforts.[10–14] This is consistent across many areas of public health and is particularly relevant in the context of obesity, which, in Australia and other developed countries, is much more prevalent in minority and lower socioeconomic groups, whose members are also likely to have less education. Few quality evaluations of education campaigns addressing obesity have been conducted, but evidence from other areas of public health suggests that social marketing and education are only somewhat effective at best in changing behaviors in populations that have the lowest incidences of obesity—hardly characteristic of a well-targeted health program.

We have significantly more food and beverage choices than ever before, yet we also have an obesity epidemic.[15] The argument for increasing the number of options available to consumers rests on the assumption that providing more choice increases the likelihood that healthier options will be chosen, at least in an educated population. Research on

decision-making suggests that this is not the case.[16] Humans have a limited capacity to process and remember information, and as the number of choices increases, it becomes increasingly difficult to evaluate attributes and select the best option.[17] As more and more foods become available, it becomes nearly impossible to perform a full evaluation of the up to 15 nutritional attributes listed on packaged foods.

When faced with a bewildering variety of options, most people engage a simplified method of decision-making by choosing a primary attribute and evaluating only that attribute in a given food.[18] Even with this strategy, however, examining the side of every box to identify levels of the desired attribute is onerous—particularly when there may be up to 100 similar products on display. In these situations consumers are likely to rely heavily on front-of-package claims (e.g., low salt, high fiber, no added sugar). Even with the regulation of these claims, health-conscious consumers are likely to be confused if not misled.

In Australia we are witnessing a push to address the lack of a legal requirement that nutritional claims on packaged foods and drinks reflect the product's overall healthiness. Food Standards Australia New Zealand recently considered a proposal to prohibit health claims on products that fell below an overall level of healthiness.[19] Such a proposal, if made law, would prevent confectionary companies, for example, from advertising that a product is 99% fat free if it is also high in sugar. Traffic light food labeling (red, amber, green) is another method of influencing the nutritional quality of shoppers' food, through a simplification of the numerous nutritional attributes of a product. This method is applied to four nutritional areas of common concern—fat, saturated fat, sugar, and salt. The more green lights displayed, the healthier the product.[20] However, the traffic light method does not entirely alleviate the problem of choosing between multiple items with multiple attributes.

Both of these labeling measures focus on micronutrients: they rely on the belief that our diets can be improved by quantitative tinkering with the nutrient profile of foods, rather than by making more qualitative changes (such as switching from processed convenience foods to unprocessed whole foods).[21] Regardless, many of us are more or less aware of the relative healthiness of many foods but still regularly purchase products we know are unhealthy.

If more food options and additional education about nutrition will not substantially improve consumers' ability to identify and select healthy food, what are the alternatives? Measures that affect the accessibility, overwhelming ubiquity, and desirability of unhealthy food are essential. If we are serious about addressing the obesity epidemic, we could, to use an analogy from interventions to increase smoking cessation, ban the sale of all food designated as unhealthy (an appropriate definition would need to be developed), particularly in government venues and to children. However, less controversial options are also available, such as limiting the proportion of unhealthy products a food outlet can stock, increasing the price of unhealthy food (the so-called fat tax), and reducing the price of healthy food (through subsidies). Similarly interesting are proposals to establish calorie caps (a limit on the maximum number of calories a meal or snack can contain) and to reduce the density of fast-food outlets, particularly in disadvantaged areas.

Governments and public health researchers who are serious about tackling obesity must consider the toxic environment that has led to the current epidemic. Proposals for reform that focus solely on individuals—be it on their knowledge, willpower, or decision-making ability—will not be successful in reversing our society's body weight trajectory. We need measures aimed at changing the context, not the individual. Rather than simply encouraging consumers to make healthier choices, we must recast the environment so that healthy options are far more accessible, available, and desirable than unhealthy alternatives. A fruitful area for further exploration is corporate practice, because it is so influential in shaping consumption patterns and because it must be carefully regulated in new ways if we are to achieve any change in the epidemiology of obesity. ■

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H. L. Walls and B. R. Crammond wrote the original draft of the article. A. Peeters and B. Loff provided further interpretation. All authors helped formulate concepts and reviewed drafts of the article.

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4.5 Conclusions

This thesis contributes to a more detailed understanding of the trends in the population distribution of body weight, their causes, the likely trajectory of the future burden of obesity and the most appropriate obesity-reduction responses. Such information is essential for health policy decision-making regarding the most appropriate obesity-reduction response.

The growing burden of obesity provides a complex challenge, and one devoid of easy solutions. This thesis illustrates the need to consider trends in both parameters of the body weight distribution, such as means and prevalence, as well as the full population distribution of body weight when planning and evaluating obesity-reduction interventions and policies. It also emphasises the need for regular, comparable survey monitoring on which such analysis can be based.

Future work is needed to explore differences in our findings by ethnicity and socio-economic status, and the determinants of weight gain at both individual and population levels. Continued, regular monitoring of the population changes in obesity and rigorous evaluation of obesity-reduction strategies is also necessary; evaluation that takes into account potential changes to the whole distribution of body weight.

Obesity is now clearly on the map as a target for prevention and intervention. This thesis shows that unless current patterns of weight gain change, levels of adult obesity in Australia will increase by 70% by 2025. It is imperative that we improve current standards of monitoring and evaluation, and better understand the drivers of weight gain, if we are to make policy choices that will halt the current body weight trajectory.

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Appendices

Appendix 1: Published short reports

The report to follow is the published version of the full-length analysis presented in Chapter 3.5.

Walls HL, McNeil JJ, Peeters A. Population versus high-risk interventions for obesity. 2009. *Epidemiology*. 2009;20:929-30.

Population Versus High-risk Interventions for Obesity

To the Editor:

In 1985 Geoffrey Rose demonstrated that preventive strategies enacting population-wide shifts in the distribution of risk factors would most affect the population burden of disease.[1] Using plasma cholesterol and blood pressure as examples, Rose demonstrated that a small leftward shift in the population distribution could decrease morbidity and mortality more than a large decrease in risk in the high-risk individuals. This approach has become a fundamental tenet of prevention.

Obesity provides a more complex situation. The increased risk conferred by cholesterol or blood pressure increases progressively across the population distribution, but the risk conferred by body weight is J-shaped, affecting mainly the upper and lower ends of the weight distribution. Thus, a population approach that shifts the body mass index to lower levels may harm those at the lowest end of the distribution. Furthermore, the increasing prevalence of obesity in developed countries is a result of an increased skewing of the distribution of body weight rather than a simple uniform shift.[2–4]

The unequal shifting of BMI across the population and the J-shaped relationship between body weight and morbidity/mortality suggest that high-risk strategies may be the most appropriate obesity-reduction response.

We analyzed 4 theoretical responses to obesity-reduction strategies (Table). Scenario 1 is a population approach that reduces all levels of the distribution by 1.5 BMI units (as observed in Cuba during its 1989–2000 economic crisis[6]). Scenario 2 is a high-risk approach that reduces the weight of the obese individuals by 5 kg (the weight loss that may be expected by lifestyle and dietary interventions[7]). Scenario 3 is a more targeted high-risk approach that reduces the weight of the severely obese individuals by 50 kg (a figure achievable by surgical intervention[8]). Scenario 4 is a hypothetical population approach that acts unequally across the range of body weight such that it de-skews the distribution, achieving the 1980 distribution of BMI.

A uniform shirt of the BMI distribution comes close to achieving the 1980 distribution (Scenario 1). However based on the association between body weight and mortality, Scenario 1 is not the most effective strategy, preventing a similar number of premature deaths as one of the high-risk strategies (Scenario 2). Scenario 3 is relatively ineffective for the population as a whole. Scenario 4 achieves an approximation of the 1980 BMI distribution (by definition), and prevents the most premature deaths.

The scenarios demonstrate that both high-risk and population approaches can be relatively effective. High-risk strategies may be more appropriate until we are certain we are not creating harm at the lower end of the distribution by using population strategies. Population approaches have the potential to be most effective and perhaps cost-effective in the long-term. They are, as Rose explained, the ultimate answer to strongly environmentally determined health problems. Furthermore, weight loss from interventions targeting high-risk groups is generally not maintained (weight loss through bariatric surgery being an exception[9]).[10]

The most appropriate approach for reducing obesity is less clear than for some other prevention areas. Our results suggest a combination of high-risk and population-based approaches is needed.

TABLE. Population Impact of Theoretical Obesity-reduction Strategies

	Mean BMI	% Underweight (BMI ≤18.5)	% Overweight + Obese (BMI ≥25)	% Obese (BMI ≥30)	No. Deaths in Australians (Aged 25–64 years) Prevented Per Annum[a]
Australia in 1980	24.8	2.3	41.7	9.7	
Australia in 2000	26.7	1.0	59.4	21.0	
Australia in 2000, modified by the following intervention types:					
Scenario 1: 1.5 BMI unit (leftwards) uniform shift in pop	25.2	4.5	45.5	14.6	18,318
Scenario 2: 5 kg weight loss in obese (BMI ≥30 kg/m^2)	26.3	1.0	59.4	13.6	22,602
Scenario 3: 50 kg weight loss in severely obese (BMI ≥40 kg/m^2)	26.3	1.0	58.4	19.2	9729
Scenario 4: a de-skew to resemble 1980 pop	24.8	2.3	41.7	9.7	48,501

The RRs used to calculate the excess number of deaths are as follows: for BMI: 15–17.5 kg/m^2; RR: 1.82 for males, and 1.70 for females; BMI: 17.5–20, 1.27, and 1.18; BMI: 20–22.5, 1.09, and 1.07; BMI: 22.5–25, 1.00, and 1.00; BMI: 25–27.5, 1.06, and 1.03; BMI: 27.5–30, 1.17, and 1.17; BMI: 30–32.5, 1.41, and 1.28; BMI: 32.5–35, 1.57, and 1.46; BMI: 35–37.5, 1.79, and 1.65; BMI: 37.5–40, 1.94, and 1.91; BMI: 40–50, 2.38, and 2.16. These RRs are estimates for people aged 35–89. They are adjusted for smoking status.

The relative risks (RRs) on which these calculations are based are those reported by the Prospective Studies Collaboration (2009), a recent study of 900,000 adults in 57 prospective studies.[5]

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A population approach that de-skewed the population's body weight—reducing most the weight of the heaviest people and leaving largely unchanged the weight of the lightest people—would be particularly safe and effective, but it is not clear how to achieve such a change. We need to monitor changes over time in the body weight distribution and its response to interventions. We also need to better understand the benefits and risks associated with these changes, to successfully reverse the potential harm associated with the obesity epidemic.

ACKNOWLEDGMENTS

We thank Luc Bonneux for reviewing the manuscript.

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How Well do Clinical Researchers Understand Risk Estimates?

To the Editor:

In clinical trials and epidemiologic studies, statistical comparisons among groups of patients for a binary outcome are commonly expressed in terms of relative risk. Two measures of relative risk are widely used, namely the risk ratio (RR) and the odds ratio (OR). Briefly, the RR represents the ratio of the probability for an outcome to occur in the experimental group to the corresponding probability in the control group, whereas the OR refers to the ratio of odds.[1–3] We sought to evaluate the level of comprehension of OR and RR among biomedical researchers.

We conducted an international electronic survey among researchers indexed as corresponding authors for clinical studies that provided data in the abstract expressed as OR or RR. We used the e-mail addresses of the corresponding authors provided at the affiliation tags indexed in PubMed, and asked them to complete a Web-based questionnaire consisting of 2 multiple-choice questions.

The first question was, "If a studied outcome occurs in the first of 2 compared groups twice as often as in the second group (with the same number of patients and time of observation for both groups), then:" (i) "The odds ratio (OR) is 2"; (ii) "The risk ratio (RR) is 2" (correct answer); (iii) "None of the above"; (iv) "Both of the above"; "The values of both odds ratio (OR) and risk ratio (RR) depend on the probability of occurrence of the outcome."

The second question was, "If the outcome occurs in 30 of 100 patients in the first group and 10 of 100 patients in the second group, then:" (i) "The odds ratio (OR) is greater than 3" (correct answer); (ii) "The odds ratio (OR) is 3"; (iii) "The odds ratio (OR) is smaller than 3"; (iv) "The odds ratio (OR) cannot be calculated from the given data."

The online questionnaire was accessed 1532 times with 903 responses submitted (response rate, 59%). The first question was answered correctly by 60% of the respondents (541/903), and the second question was answered correctly by 65% (591/903). Fewer than half, 47% (420/903) answered correctly to both questions, while 21% (188/903) did not answer correctly to either question. Moreover, 5.4% (49/903) mistook the OR for the RR in both questions.

Misunderstanding of the most common measures of risk appears to be quite common among those who use them. Inadequate comprehension of the OR and RR terms among biomedical researches may compromise the communication of research findings in a clinically relevant manner. The latter is important because statistical significance does not always relate to clinical significance.[4]

Furthermore, the statistical terms chosen to present research findings considerably influence the clinicians' understanding of their clinical importance.[5,6] Several literature analyses have suggested that the OR is often misinterpreted as the RR.[7–9] Although this approximation is reasonable when the frequency of the outcomes is low, the OR substantially deviates from the RR as the frequency of the outcomes increases. Whether RR or OR should be preferred when reporting research findings remains a debated issue.[3]

Regardless, more emphasis should be given in on continuing medical education to the understanding and application of common biostatistical terms.

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Appendix 2: Letters to the editor

A letter to the journal *Epidemiology* was received in response to the article described in 3.5.1. Thus, to follow is this letter, followed by our response.

Walls HL, McNeil JJ, Peeters A. 2009, Population interventions for obesity. *Epidemiology*. 2010. In press.

***Title Page**

Type of manuscript: Letter

Manuscript title: Population interventions for obesity

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Population interventions for obesity

Reaction to: Walls, McNeil & Peeters. Population Versus High-risk Interventions for Obesity. Epidemiology 2009 Nov;20(6):929-930.

To the Editor:

In a recent letter in your journal, Walls et al write that "a population approach that de-skewed the population's body weight—reducing most the weight of the heaviest people and leaving largely unchanged the weight of the lightest people—would be particularly safe and effective, but it is not clear how to achieve such a change."[1]

We beg to differ, and believe that it is not at all difficult to think of population-wide interventions that affect the obese more than those of normal weight. For example, a tax on energy dense foods or sugared beverages[2] would automatically target the overweight, because this group consumes most of these products. Indeed, it is one of the causes of their overweight. Numerous studies have shown an association between the consumption of sugared beverages and BMI or energy dense foods and BMI.[3] This would make a tax on such drinks exactly be the kind of intervention Walls et al are looking for, ideally combined with subsidies on healthier foods to prevent purchasing power decreases among low-income earners. Other examples could be limiting the marketing of such foods, or banishing unhealthy foods from schools. An additional benefit is that, as opposed to explicitly targeted interventions, such changes to the 'obesogenic' environment do not risk to stigmatize obese persons.

We would posit, therefore, that safe and effective interventions that reduce the weight of the heaviest, but not of the lightest people, do exist. In contrast, we cannot think of realistic examples of a population-wide intervention that reduce the BMI of heavy and light people to an equal degree (Walls' scenario 1). The example of the economic collapse in an already poor country (Cuba) hardly counts as a realistic intervention to improve health – though it does seem to have had that effect. Furthermore, as Walls et al

themselves show, BMI distributions in western countries historically changed by increased skewness, not by shifting entire distributions up or down. It is unlikely that interventions that improve diet or increase opportunities for physical activity would do otherwise. The first scenario presented is therefore a bit of a red herring.

In conclusion, unlike Walls et al, we believe that it is well know what population-wide actions are likely to be effective. We do agree on the need for rigorous evaluation of the policy experiments that are undertaken, because exactly *how* effective such interventions are remains largely unknown.[4]

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Declaration for Thesis Appendix 2

Walls HL, McNeil JJ, Peeters A. 2009. Population interventions for obesity. *Epidemiology*. In press.

Declaration by candidate

In the case of Appendix 1, the nature and extent of my contribution to the work was the following:

Nature of contribution	**Extent of contribution (%)**
Conceptualisation and drafting of letter	70%

The following co-authors contributed to the work. Co-authors who are students at Monash University must also indicate the extent of their contribution in percentage terms:

Name	**Nature of contribution**	**Extent of contribution (%) (student co-authors only)**
JJ McNeil	Conceptualisation and drafting of letter	N/A
A Peeters	Conceptualisation and drafting of letter	N/A

Candidate signature	**Date**
[redacted]	8 February 2010

Declaration by co-authors

The undersigned hereby certify that:

(1) the above declaration correctly reflects the nature and extent of the candidate's contribution to this work, and the nature of the contribution of each of the co-authors;
(2) they meet the criteria for authorship in that they have participated in the conception, execution, or interpretation, of at least part of the publication in their field of expertise;
(3) they take public responsibility for their part of the publication, expect for the responsible author who accepts overall responsibility for the publication;
(4) there are no other authors of the publication according to these criteria;
(5) potential conflicts of interest have been disclosed to (2) granting bodies, (b) the editor or publisher of journals or other publication, and (c) the head of the responsible academic unit; and
(6) the original data are stored at the following location(s) and will be held for at least five years from the date indicated below:

Location(s):
Department of Epidemiology & Preventive Medicine, Monash University

[Please note that the location(s) must be institutional in nature, and should be indicated here as a department, centre or institute, with specific campus identification where relevant].

Co-author signatures	Date
[redacted]	10/2/10
[redacted]	7/2/2010

Title: Population Interventions for Obesity

Authors: Walls HL, McNeil JJ, Peeters A

Response to: Veerman JL, Barendregt JJ. Population interventions for obesity. *Epidemiology*.

To the Editor:

We agree with Veerman et al. [1] that there may be safe and effective interventions to reduce the weight of the heaviest people in the population but not the lightest. Examples may well include taxation of energy-dense foods and beverages (coupled with subsidies on healthier foods), and the removal of unhealthy foods and related marketing from schools and other settings [2].

Nonetheless, there is a dearth of evidence regarding the effect of population interventions, and particularly so in terms of their effect on the distribution of body weight. Obesity reduction is a complex challenge, and one devoid of easy solutions. The body appears to resist weight loss more than it resists weight gain [3], rendering weight loss and its maintenance particularly challenging (and even impossible according to some experts, surgery aside) [4]. Furthermore, there may be compensatory behavior (such as seen with physical activity[5]) that weaken population interventions for obesity. Would the unavailability of sugared beverages, for example, result in higher consumption of other energy-dense foods or beverages?

Common population approaches to reducing the burden of obesity, such as social marketing and other campaign falling under the umbrella of "health education" (including the contentious "traffic light" food labelling system [6]) are unlikely to reduce skew in the distribution of body weight. In public health, the best response to further education is consistently from those who already have high levels of education [7, 8]. This observation renders such obesity-reduction approaches ineffective at reducing skew, as obesity is more prevalent among minority and lower socioeconomic groups, whose members are also likely to have less education [6].

Our article provided simplistic scenarios to illustrate the importance of not taking a single approach to intervention for granted, and to emphasize the need to consider both the distribution and prevalence of obesity when planning and evaluating obesity-reduction interventions and policies.

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Appendix 3: Co-authored publications

Publications produced during candidature and for which the candidate has co-authorship, but not forming part of the thesis. The papers for which manuscripts are included are marked (***).

Abdullah A, Stoelwinder J, Shortreed S, Wolfe R, **Walls HL**, Stevenson C, de Courten M, Peeters A. The duration of obesity and risk of diabetes. Submitted. ***

Abdullah A, de Courten M, Stoelwinder J, Stevenson CE, **Walls HL**, Peeters A. The number of years lived with obesity and the risk of mortality. Submitted. ***

Ademi Z, **Walls HL**, Peeters A, Liew D, Hollingsworth B, Stevenson C, Deepak B, Steg G, Reid CM. Economic implications of obesity among people with atherothrombotic disease. *Int J Obes*. In press.

Backholer K, **Walls HL**, Magliano DJ, Peeters A. Setting population targets for measuring successful obesity prevention. Submitted. ***

The Duration of Obesity and the Risk of Type-2 Diabetes

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Abstract

The evidence for the association between obesity and the risk of type-2 diabetes has been derived mainly from the analysis of the degree of obesity. The role of the duration of obesity as an independent risk has not been fully explored. The objective of this study was to investigate the association between the duration of obesity and the risk of type-2 diabetes. We analyzed participants of Framingham Heart Study who were obese in at least two consecutive examinations during the 24 biennial examinations and measured their duration of obesity. Mortality was collected throughout the 50 years of follow-up of the study. The relation between duration of obesity and mortality was analysed using time-dependent Cox models, adjusting for a number of covariates. The unadjusted HR for the risk of type-2 diabetes was 1.11 (95% CI 1.10 – 1.13) per additional two years increase in the duration of obesity. Adjusted for social demographic variables, family history of diabetes, health behaviour and physical activity, the hazard ratio remained at 1.11 (95% CI 1.10 – 1.13). However, after adjustments for time-varying BMI and the age onset of obesity, the HR was 1.07 (95% CI 1.05 – 1.08) per additional 2 years of obesity duration. The hazard ratios associated with duration were significantly higher for males than for females and for those with and older age of onset of obesity.

Keywords: obesity, the duration of obesity, BMI, type-2 diabetes

Introduction

Obesity is clearly associated with type-2 diabetes. However, the estimation of the risk of diabetes associated with obesity has primarily focused on the analysis the severity of body weight at one point in time. The duration of obesity is not yet considered as an important risk factor in any risk-factor equations [1,2] as well as not being taken into account as a relevant variable in calculating the population burden of diabetes [3]. The potential impact of the duration of obesity as an

independent risk factor has been neglected. The risk of those who have been obese for one year and those who had been obese for 20 years is treated identically. We hypothesise that such approaches underestimate the association between obesity and risk of type-2 diabetes. With the age of onset, and consequently duration, of obesity in the community increasing, accurate estimates of these relationships are essential for accurate estimation of the future burden of diabetes.

The potential importance of the duration of exposure has been acknowledged in many epidemiology studies. For example, in 1970s several studies showed that the duration of smoking is more important than the intensity of smoking in causing lung cancer [4,5]. However, there is a lack of studies examining the association between the duration of obesity and the risk of several chronic diseases. Only six studies have specifically examined the association between the duration of obesity and type-2 diabetes [6-11]. Whilst four studies found a relationship between increased duration of obesity and increased risk of type-2 diabetes, two did not [10,11].

Most of these studies had limitations in the methodology of measuring the duration of obesity. In all but two studies, measurement of the years of exposure to obesity relied on recall of body weight, by asking the history of weight at certain ages [6,8,10,11]. It has been pointed out that the recalled body weight approach can induce significant bias [12,13]. Only two studies analyzed data using a longitudinal cohort study design [6,9]. The first of these two cohort studies was conducted in a very specific cultural population setting, the Gila River Indian community of Southern Arizona, where the prevalence of obesity and diabetes is relatively high compared with other populations [14]. In addition, the results were reported as incidence of diabetes per person-years of obesity adjusted only for sex, age and current BMI. Other important confounders such as the age of onset of obesity, and health behaviours related variables were not adjusted for. In the second cohort study [9], the duration of obesity was estimated only from observations at two five-year intervals. Clearly more accurate estimation of the relationship between the duration of obesity and the risk of type-2 diabetes is required.

The aim of this study was to examine whether the duration of obesity is a risk factor of type-2 diabetes, independent of degree or severity of BMI, age onset of obesity and other risk factors using a measured body-weight derived from a long-term cohort study with relatively short examination intervals.

Research design and methods

Data source

We used data from the original cohort study of the Framingham Heart Study (FHS) [15]. This cohort study followed up 5209 participants (aged 28 – 62 at the time of enrolment) for approximately 50 years. Body weight and height were measured every two years along with many other potential risk factors of chronic diseases [16]. For the purpose of our analysis, the inclusion criteria for participants were individuals who had a BMI of 30 kg/m^2 or greater on at least one examination during the study and were free from type-2 diabetes at baseline.

Measurement of the duration of obesity

The duration of obesity was defined as the length of the obesity assessment intervals during the study. The beginning of an individual's obesity interval was defined by the first exam of the period of two consecutive occurrences of obesity and the end point was defined at the first of two consecutive non-obese examinations or death, diagnosis with type-2 diabetes or the end of study for the participants alive until examination 24. Defining the onset of obesity as two consecutive obesity measurements (correlating with approximately four years of being continuously obese), accommodated potential miss-classification of body weight, particularly between the upper borderline of the 'overweight' BMI category and the lower borderline of the 'obesity' category.

Table 1, presents a hypothetical data-set to illustrate our definition of duration of obesity. The obesity interval for participant C begins at the 4^{th} examination and ends at the 14^{th}, their last examination, a total of 20 years. For participant E, the starting point was from examination 12, not from 6 since this participant was not obese in the following examination 7. In the case of participant F, the subject has

two starting and ending points according to our definition. The first stating point was at examination 4 and the second starting point was at examination 9. The duration of obesity was the sum of these periods of obesity.

Measurement of other variables

Methods of measurement of other variables are described briefly here. Baseline measurements were used for variables that did not vary, or varied constantly, with time. Age of participants was measured in years from date of birth to baseline of study. Education at baseline was grouped into four categories (8th grade or less, high school, college and post graduate). Ethnicity was categorized as born in the USA or outside of the USA. Family history of diabetes was measured at examination seven. Age of onset of obesity was determined as the age when a participant was categorized for the first time as obese. For participants who were already obese at baseline, the age of onset of obesity was their age at the baseline exam. Marital status, smoking, alcohol consumption, and physical activity were included as time varying variables. Marital status was grouped into three categories (single, married and widowed, divorced or separated). Current smoking status was determined as a smoker if the participant had smoked regularly in the past year. The average number of cigarettes smoked every day was also included. These variables were measured in all 24 examinations. Alcohol consumption was measured in ounces per month but it was measured but only in eight examinations (2, 4, 7, 19, 20-23). Physical activity was measured as a summary physical activity score, measured in four examinations (4, 11, 12, and 19). With the exception of BMI missing values were imputed by using the values in the previous examination or the values in the following examination, whichever exam was closer in time.

Measurement of the outcome and time to event

A person was defined as having type-2 diabetes, if the participant had taken insulin and/or oral hypoglycaemic agent or if an individual's fasting plasma glucose was greater than 200 mg/dl. This was the definition FHS used to categorize participants with diabetes and it is in line with the criteria for type-2 diabetes's from the World Health Organisation [17].

Time to incidence of type-2 diabetes was measured as the time until a participant was diagnosed as having type-2 diabetes. Individuals who died or reached the end of the follow-up (examination 24) before developing diabetes were censored at date of death or examination 24. We defined the date of diabetes incidence as the mid-point between the day of the first examination a participant was diagnosed with type-2 diabetes and the day of the previous attended examination.

Missing values and imputation

Missing values were present in the FHS data set as in any longitudinal study. The average number of missing values of BMI was 18.5%. Missing values of BMI were imputed using a multiple predictive linear regression model. The model was fit using age at prior exam, sex and several transformations of the previous BMI data (BMI, log BMI, BMI squared and BMI as a categorical variable).

Data analysis

The relationship between the duration of obesity and incidence of type-2 diabetes was estimated using time-dependent Cox proportional hazards regression [18]. Hazard ratios ware presented both as crude hazard ratios and multi-adjusted hazard ratios. Hazard ratios were adjusted for the time-independent variables of sex, age of participant at baseline of study, age of onset of obesity, education, ethnicity, family history of diabetes and for the time varying variables of marital status, body mass index (per unit BMI), smoking, physical activity and alcohol consumption. We used a model allowing for an interaction term between gender, age of onset of obesity and duration of obesity to test if there is an additional effect of duration of obesity for men or women. The analysis was performed using STATA Version 10 software [19].

Sensitivity analyses

A sensitivity analysis was performed to examine whether the results of the association between the duration of obesity and type-2 diabetes incidence might be influenced by the imputation methods of the missing values. To test this

assumption, the result was compared with the model of including only participants with no missing values of BMI in any examinations. A sensitivity analysis was also performed to test the impact of unknown BMI values prior to baseline by analysing only those without obesity at baseline. The effect of defining duration using two consecutive occurrences of obesity was tested by analysing the crude counting of exams with obesity as a measure of duration.

Results

From the total of 5,209 study participants, 1,256 were identified as ever obese in at least one examination during the 48 years of study follow-up. Forty-seven percent of participants were obese at study baseline and 2% of them were continuously obese in all 24 examinations. 11% of participants were obese for some periods and then became not obese for at least two examinations, but obese again for several subsequent examinations. The average duration of follow-up was 31 years and the average duration of obesity was approximately 15 years. Out of the 1,256 participants in this study, 279 (22%) were diagnosed as having type-2 diabetes during follow-up. The characteristics of the participants included in this study at baseline can be seen in Table 2.

The unadjusted hazard ratio for type-2 diabetes incidence for each additional two years of obesity duration was 1.11 (95% CI 1.10 – 1.13). Hazard ratios for males were higher than females, 1.14 (95% CI 1.12 – 1.17) and 1.09 (95% CI 1.08 – 1.11) respectively (Table 3). This indicates that there is a different effect of duration of obesity for men and women. Adjustment for the key demographic behavioural variables did not change the hazard ratios. Hazard ratios changed after adjustment for BMI at each exam and age of onset of obesity. After adjustment for all the measured confounding variables, the hazard ratio (model D) for males was 1.11 (95% CI 1.08 – 1.15) while for females it was 1.04 (95% CI 1.02 – 1.06) per extra two years of obesity duration.

In addition to sex, the effect of the duration of obesity on type-2 diabetes differed significantly according to age of onset of obesity categories, with a particularly high

risk of duration in those with an age of onset over 60 years of 1.18 (95% CI 1.11 – 1.26) per two years increase in obesity duration (Table 4).

When obesity duration was analysed categorically, there was a significant difference in the risk of incident type-2 diabetes between the different categories of obesity duration (log-rank test for equality of survivor functions, P value < 0.001) (Table 3). The dose-response relationship was very clear for males. The adjusted hazard ratio in males who were obese for 5 – 15 years was more than three times higher than in those obese for less than 5 years. For males who were obese for 15 – 25 years the risk of diabetes was almost five times higher and almost seven times higher in those obese for more than 25 years compared with males obese for <5 years. For females, there was no clear dose-response relationship

The overall results of association between the duration of obesity and the risk of type-2 diabetes were not influence by the methods of imputation of missing values of BMI or by including/excluding the people who obese at baseline. The results of sensitivity analyses showed that adjusted hazard ratios derived from the analysis of only the participants who had no missing values of BMI (84.5% of participants) during 24 examinations of study follow-up was 1.12 (95% CI 1.08 – 1.18) per two year obesity duration which is relatively similar to hazard ratio that derived from imputation of missing value. Excluding those already obese at baseline (n=586, 47%) also showed relatively similar results, with a hazard ratio of 1.07 (95% CI 1.03-1.10) per additional two years increase in the duration of obesity. Sensitivity analysis comparing effect of using two consecutive occurrences of obesity approach of calculating the duration of obesity and the crude counting of exams with obesity as a measure of duration of obesity also demonstrated a similar finding (data not shown).

Discussion

This study has found that the duration of obesity is significantly associated with an increased risk of developing type-2 diabetes. The association was still significant even after adjusting for current body mass index and age of onset of obesity, with

each additional two years increase in the duration of obesity associated with an approximately seven percent increased risk of diabetes. In this study, the effect of the duration of obesity on type-2 diabetes appeared to be stronger for males than females and for those with an older age of onset of obesity.

Nowadays, people are exposed to obesity far longer than in previous decades as results of increasing prevalence of obesity at younger ages [20]. Our results suggest that increasing exposure to obesity might accelerate the incidence of type-2 diabetes in the future. Although the biological mechanism that underlies the association between the duration of obesity and risk of chronic diseases is still unclear, several experimental studies have reported that the duration of obesity might be associated with partial exhaustion of beta cells [21,22], and resultant insulinopenia may cause depressed glucose oxidation and impaired glucose tolerance [21]. Whether the effect of the duration of obesity is mediated through insulin resistance and/or through effect on insulin secretion is largely unclarified [23]. Resistance to glucose disposal is strongly associated with obesity and results in high fasting and post-load serum insulin concentrations. Prolonged duration of obesity could conceivably worsen this resistance and result in even higher serum insulin concentration. However, study of residents of the Gila River Indian community of southern Arizona showed a different result [6] and the authors suggested that a long duration of obesity might be associated with a low insulin response rather than with insulin resistance.

Our study was the third study examining the association between the duration of obesity and risk of type-2 diabetes using measured height and weight derived from a prospective cohort study. A key strength of our study was the measurement of weight and height at two year intervals for almost 50 years. A further strength of our study was that we were able to adjust for a large number of potential confounding variables including health behaviour related variables, age of onset of obesity and time varying BMI.

Our study found a slightly stronger relation between duration of obesity and risk of diabetes than the two other cohort studies [6,9]. In Everhart et al [6] the subjects obese for more than ten years had a risk of diabetes more than double those who were obese for less than five years. In our study, the risk was almost 5 times as high (recalculated using similar cut-off points for duration of obesity to make the results comparable). If our results compared to Wannamethee and Shaper [9] study, the risk of diabetes for those who were obese for more than five years compared to the subjects obese for less than five years was three times higher in our study. Differences in our findings are likely to be due to the differences in the study populations and in analytical methods used.

An important limitation of this study relates to the sample population. The original Framingham cohort study began in 1948 and the prevalence rates of obesity and type-2 diabetes were relatively low at that time. The prevalence of obesity in the 1950s was below 10% [24]. It could be argued that the results of this study might not reflect the current population, where the prevalence rate of obesity is higher than fifty years ago. In 2008, the prevalence obesity among adults in the United States reached approximately 30% [25]. Future research using contemporary, regularly observed body weight from a long-term cohort study is recommended.

This study analyzed the duration of obesity separately from the degree of obesity. Further research analyzing the interaction term between both degree and the duration of overweight and obesity is needed - for example estimating the risk of type-2 diabetes for every additional excess “BMI-years” analogous to the “pack-years” concept used for smoking. It will be important to also determine whether duration of obesity is an independent predictor of an increased risk of other chronic diseases such as musculoskeletal disease, cardiovascular disease, and cancer. In the future, the duration of obesity needs to be taken into account in calculating the burden of disease and in estimating the future burden of type-2 diabetes.

Conclusion

This analysis shows that the duration of obesity is a relevant predictor of type-2 diabetes risk, independent of the actual level of BMI and the age onset of obesity. This suggests that in the future we need to take into account the duration of obesity in our obesity prevention strategies to delay the onset of obesity as well as to limit its duration.

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Disclosure

We declare no conflict of interest.

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Table 1. Illustration of method used to identify start and end points of obesity and consequent calculation of duration of obesity in seven example subjects

Sub-jects	Examinations (exam 1 to exam 24)																							Starting Point	Ending Point	Duration of Obesity
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22	23	24			
A	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	Exam 1	Exam 24	46 years
B	2	2	2	2	2	2	2	1	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	Exam 9	Exam 24	30 years
C	2	2	2	3	3	3	3	3	3	3	3	3	3	3											Exam 4	Exam 14	20 years
D	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	2	2					Exam 1	Exam 18	34 years
E	2	2	2	2	2	3	2	2	2	2	2	3	3	3	3	3	3	3	3	3	3	3	3	2	Exam 12	Exam 23	22 years
F	2	2	2	3	3	3	2	2	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	3	Exam 4 Exam 9	Exam 6 Exam 24	32 years
G	2	2	2	2	2	2	2	2	2	2	2	2	2	2	3	3	2	2	2	2	2	2			Exam 15	Exam 16	2 year

Note: 3 indicated the subject was obese, 2 overweight and 1 normal weight

Table 2. Characteristics of participants who were obese for at least one exam during study follow-up

Variables and characteristic	
Sample (n)	1256
Population characteristics	
Age at baseline	mean 43 years, range (28 – 62)
Age onset of obesity	mean 52 years, range (29 – 88)
Sex	males (n=494; 39%), females (n=762; 60%)
Country of birth	
	born in USA (n= 993; 79%) not born in USA (n=263; 21%)
Marital status at baseline	
	single (n=99; 8%) married (n=1,076; 86%) Widowed/divorced (n=81; 6%)
Educational level at baseline	
	8th grade or less (n =458; 38%) high school (n=497; 41%) college (n=120; 10%) postgraduate/school (n=130; 11%)
Health behaviour	
Smoking status at baseline	yes (n=618; 49%)
Number cigarettes smoked per day at baseline	mean 8.3, range 1 cigar. – 3 packs
Physical activities score at exam 4	mean of score 32, range 26 – 83
Alcohol drinking (ounces/month) at exam 2	mean 14 ounces, range 0–360
Biochemical characteristics	
Systolic Blood Pressure at baseline	mean 142, range 94 – 300
Diastolic Blood Pressure at baseline	mean 89, range 58 – 155
Hypertension at baseline	yes (n=318; 25%)
Serum cholesterol baseline	mean 224, range 121 – 534
Body weight characteristic	
BMI at baseline	mean 29.9, range 18.6 – 46.3 normal weight (n=135; 11%) overweight (n=534; 42%) obese (n=586; 47%)
Obesity duration	mean 15 years (SD=11 years) <5 years, (n=264; 21%) 5 – 15 years (n=445; 35%) 15 – 25 years (n=290; (23%) ≥25 years (n=257; 21%)
Diagnosed with type 2 diabetes	
	yes (n=279; 22%) no (n=979; 78%)

Table 3. Univariate and multivariate hazard ratios of type 2 diabetes per two-year increase in duration of obesity and per categories of duration of obesity.

Model	% increased HR	HR per categorical duration of obesity			
Hazard ratio (HR)	**Per two years**	**<5 years**	**5–14.9 years**	**15–24.9 years**	**>=25 years**
Univariate					
– Males	1.14 (1.12–1.17)	1	4.16 (2.45–7.07)	7.28 (4.24–12.50)	11.05 (6.42–19.05)
– Females	1.09 (1.07–1.11)	1	5.30 (3.40–8.26)	6.33 (3.93–10.19)	4.95 (2.85– 8.60)
– Males + Females	1.11 (1.10–1.13)	1	4.81 (3.43–6.74)	6.67 (4.67– 9.53)	7.08 (4.82–10.39)
Multivariate (Model A)					
– Males	1.15 (1.12–1.18)	1	3.91 (2.30–6.65)	6.82 (3.92–11.89)	10.55 (5.89–18.89)
– Females	1.09 (1.07–1.11)	1	4.83 (3.04–7.62)	5.84 (3.59– 9.49)	4.56 (2.61– 7.95)
– Males + Females	1.11 (1.10–1.13)	1	4.49 (3.18–6.34)	6.31 (4.37– 9.10)	6.82 (4.59–10.13)
Multivariate (Model B)					
– Males	1.13 (1.09–1.16)	1	3.48 (1.98–6.10)	5.67 (3.05–10.55)	8.09 (4.15–15.79)
– Females	1.05 (1.03–1.07)	1	3.43 (2.08–5.66)	3.67 (2.15– 6.29)	2.62 (1.44– 4.79)
– Males + Females	1.08 (1.06–1.10)	1	3.44 (2.37–4.99)	4.28 (2.84– 6.44)	4.14 (2.65–6.45)
Multivariate (Model C)					
– Males	1.12 (1.09–1.15)	1	3.24 (1.74–6.02)	5.27 (2.74–10.13)	7.59 (3.79–15.21)
– Females	1.05 (1.03–1.07)	1	3.60 (2.16–6.02)	3.99 (2.34– 6.82)	2.80 (1.53– 5.12)
– Males + Females	1.08 (1.06–1.10)	1	3.44 (2.33–5.07)	4.41 (2.93– 6.66)	4.30 (2.76– 6.70)
Multivariate (Model D)					
– Males	1.11 (1.08–1.15)	1	3.04 (1.67–5.54)	4.82 (2.51–9.23)	6.65 (3.34–13.25)
– Females	1.04 (1.02–1.06)	1	3.17 (1.92–5.22)	3.32 (1.95–5.66)	2.32 (1.28– 4.20)
– Males + Females	1.07 (1.05–1.08)	1	3.09 (2.11–4.51)	3.75 (2.49–5.66)	3.52 (2.27– 5.48)

In the model A, HR adjusted for social demographic variables of sex, age at baseline, marital status, educational levels, ethnicity (born in the USA or outside of the USA), family history of diabetes (at examination 7) and health behaviour variables of time varying of smoking, alcohol consumption, and physical activity.

In the model B, HR adjusted for all variables as in the model A, plus adjusted for time varying body mass index (per unit BMI)

In the model C, HR adjusted for all variables as in the model A, plus adjusted for age of onset of obesity.

In the model D, HR adjusted for all variables as in the model A, plus adjusted for time varying body mass index (per unit BMI) and age of onset of obesity.

Table 4. Crude and multivariate hazard ratios of type 2 diabetes per two-year increase in duration of obesity according to age of onset of obesity and sex

Variables	n–sample (cases)	Hazard ratio per additional year the duration of obesity	
		Crude	Adjusted[a]
Age onset of Obesity <50 years			
– Males	235 (74)	1.12 (1.09–1.15)	1.12 (1.08–1.16)
– Females	277 (78)	1.06 (1.04–1.09)	1.04 (1.01–1.07)
– Males + Females	512 (152)	1.09 (1.07–1.11)	1.07 (1.05–1.09)
Age onset of Obesity 50–59.9 years			
– Males	147 (37)	1.19 (1.13–1.25)	1.13 (1.05–1.21)
– Females	268 (56)	1.07 (1.03–1.11)	1.04 (0.99–1.08)
– Males + Females	415 (93)	1.10 (1.06–1.13)	1.07 (1.03–1.11)
Age onset of Obesity >=60 years			
– Males	112 (10)	1.29 (1.18–1.41)	1.19 (1.04–1.35)
– Females	217 (24)	1.32 (1.24–1.39)	1.19 (1.10–1.29)
– Males + Females	219 (34)	1.30 (1.24–1.37)	1.18 (1.11–1.26)

[a)] HR adjusted for social demographic variables of sex, age at baseline, marital status, educational levels, ethnicity (born in the USA or outside of the USA), family history of diabetes (at examination 7) and health behaviour variables of time varying of smoking, alcohol consumption, and physical activity, time varying body mass index (per unit BMI)

Title

The Number of Years Lived with Obesity and the Risk of Mortality

Running Title

The Duration of Obesity and Mortality

Authors

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Abstract

Background: As the prevalence of obesity increases at younger ages the number of years that obese adults have lived with obesity is also increasing. This may affect the health risks associated with obesity. There has been no examination of the relationship between the number of years lived with obesity and the risk of mortality.

Objective: To investigate the association between the years of exposure to obesity and the risk of mortality

Design: Prospective cohort study

Setting: The Framingham Heart Study (FHS), follow-up from 1948–1998.

Participants: 677 FHS participants who were not obese and free from pre-existing disease at baseline, but were obese for at least two consecutive examinations during follow-up.

Measurement: The duration of obesity was measured from the first of two consecutive examinations at which a person was classified as obese. The outcomes were all-cause mortality, cardiovascular mortality and cancer mortality. The analyses were performed using Time-Dependent Cox models adjusted for socio-demographic variables, health behavior variables, body mass index and age of onset of obesity.

Results: The adjusted Hazard Ratios (HR) for all-cause mortality, cardiovascular mortality and cancer mortality were 1.19 (95% CI 1.16–1.21), 1.16 (1.13–1.19) and 1.18 (1.13–1.22), respectively per additional two-year increase in the duration of obesity. The effect of the duration of obesity appeared to be stronger in non-smokers and in females. In non-smoker, there was a clear dose-response relation between years of duration of obesity and risk of mortality.

Conclusions: In those with obesity, the risk of mortality doubles for each additional ten years of exposure. This will affect the future health burden associated with current obesity trends.

Background

There has been a worldwide increase in the prevalence of overweight and obesity over the past two decades (1-3). In recent years, the prevalence of childhood obesity has also been increasing at accelerating rates. For example, in the USA, 25% percent of children are overweight and 11% obese (4). In Australia, the prevalence of overweight and obesity in children is increasing by approximately one percentage point per year (5). Consequently, the number of years that obese adults have lived with obesity is continually increasing.

We hypothesise that the increasing duration of obesity is associated with an increased risk of chronic diseases and mortality over and above the risk associated with a given level of body mass index (BMI). Current estimates of the association between obesity and the risk of mortality are derived from the analysis of the degree of body weight at one point in time. The risk for those who have been obese for one year and those who have been obese for 20 years, for example is treated identically. The potential impact of the duration of obesity as an independent risk factor has been neglected. It is likely that such approaches underestimate the association between obesity and the risk of mortality.

There are a number of pathways through which an increasing duration of obesity might increase mortality risk, independent of the level of BMI achieved. Longer exposure to obesity might be expected to lead to a endogenous production of reactive oxygen species and oxidative DNA damage, alterations in carcinogen-metabolizing enzymes, alteration in endogenous hormone metabolism (6) and partial exhaustion of beta cells, with the resultant insulinopenia causing depressed glucose oxidation and impaired glucose tolerance (7-9). Consequently, any association between duration of obesity and mortality might be expected to be partially explained by intermediate variables in the causal pathway to mortality such as blood pressure, serum cholesterol, and serum glucose as well as chronic diseases such as diabetes (10), cardiovascular disease and cancer.

This study has two main objectives. The first objective is to examine whether the duration of obesity is associated with the risk of mortality independent of the level of BMI attained. The second objective is to examine to what extent any observed association is mediated by intermediate variables, including biological intermediates (11) and by incident diseases during the study follow-up. We investigated these objectives using a long-term prospective longitudinal study where BMI and other covariates were measured regularly in 24 biennial examinations, spanning 50 years (12).

Methods

Data Source and Inclusion Criteria

We used data from the original cohort study of the FHS (12). This cohort study followed 5209 participants (aged 28 – 62 at the time of enrolment in 1948–50). In the present study, we restricted the analysis to participants who were not obese and free from pre-existing diseases at baseline, but were obese in at least two consecutive examinations during study follow-up. 677 participants met these inclusion criteria.

Measurement of Obesity

In the FHS, body weight and height were measured every two years along with many other potential risk factors for chronic diseases (13). Body weight was measured to the nearest pound and height was measured in partial inches. We calculated BMI by dividing weight by height squared and multiplied the result by 703 to convert to the appropriate units. A

participant is included in the obese category if their BMI is greater than or equal to 30 kg/m^2.

Measurement of the Duration of Obesity

The duration of obesity was defined as the total length of the obesity intervals during the study. The beginning of an individual's obesity interval was defined by the first examination of two consecutive examinations with measured obesity. The end point was defined at the first of two consecutive non-obese examinations, or death, or the end of the study follow-up for the participants alive until examination 24. Defining the onset of obesity as two consecutive obesity measurements (representing at least 2 years of being continuously obese) accommodated potential miss-classification of body weight, particularly between the upper borderline of the 'overweight' BMI category and the lower borderline of the 'obesity' category.

Measurement of Other Variables

Methods of measurement of other variables are described briefly here. Baseline measurements were used for variables that did not vary, or varied constantly, with time. Age of participants was measured in years from date of birth to baseline of study. Education at baseline was grouped into four categories (8th grade or less, high school, college and post graduate). Ethnicity was categorized as born in the USA or outside of the USA. Age of onset of obesity was determined as the age when a participant was categorized for the first time as obese. Marital status, smoking, alcohol consumption, and physical activity were included as time varying variables. Marital status was grouped into three categories (single, married and widowed, divorced or separated). Current smoking status was determined as a smoker if the participant had smoked regularly in the past year. The average number of cigarettes smoked per day was also included. Alcohol consumption was measured in ounces per month but it was measured only in eight examinations (2, 4, 7, 19, 20–23). Physical activity was measured as a summary physical activity score (14) at four examinations (4, 11, 12, and 19). For the examinations where it was not directly measured, the imputed values were used.

A panel of three physicians reviewed each cardiovascular event according to pre-established criteria. CVD included CHD, as well as angina, coronary insufficiency (unstable angina with ischemic electrocardiogram changes), congestive heart failure, and

atheroembolic brain infarction; CVD death included deaths attributable to these causes. Details regarding the methods of risk factor measurement and laboratory analysis have been described elsewhere (15). Each examination included CVD assessment, 12-lead electrocardiogram, and blood testing. A person was defined as having type-2 diabetes, if the participant had taken insulin and/or oral hypoglycemic agent or if an individual's fasting plasma glucose was greater than 200 mg/dl. For cancer, the basis diagnosis criteria was 1) microscopically confirmed, 2) clinically diagnosed, or 3) indicated on death certificate only. These CVD, diabetes and cancer variables were recorded in all examinations during the study follow-up.

Measurement of Events

The outcomes of interest were all-cause, CVD and cancer mortality. All deaths were adjudicated by a panel of 3 investigators using previously described criteria (15). Information on cause of death was obtained from death certificates, hospital admission records, medical records, and family members. CVD was identified as the cause of death if any of the following conditions were responsible for the death: coronary heart disease, intermittent claudication, congestive heart failure, stroke, or transient ischemic attack. Cancer was identified through three types of basic diagnosis -- microscopically confirmed, clinically diagnosed, or death certificate. Time to event (survival time) was calculated in days from exam 1 (baseline) to survival date, which was either date of death, loss to follow-up or end of follow-up at exam 24.

Missing Values and Imputation

15.7 % of participants had at least 1 examination with missing values. For those with missing values, the median number of examinations containing a missing value was 7, with 75% having missing values at fewer than 6 examinations. With the exception of BMI, missing values were imputed using the values in the previous examination, or the values in the following examination, whichever examination was closer in time. BMI was imputed differently, using a regression model. The model was filled using age at prior examination, sex and several transformations of the previous BMI data (BMI, log BMI, BMI squared and BMI as a categorical variable).

Data Analysis

The relationship between the duration of obesity and risk of mortality was estimated using time-dependent Cox proportional hazards regression (16, 17). Hazard ratios were presented both as crude hazard ratios and as multi-adjusted hazard ratios. In the first adjusted model, hazard ratios (HRs) were adjusted for the time-independent socio-demographic variables of sex, age at baseline, marital status, educational level, ethnicity (born in the USA or outside of the USA), and the time varying health behavior variables of smoking, alcohol consumption, current body mass index (per unit BMI), and age of onset of obesity. In the second model, adjustment was also made for time varying intermediate variables: physical activity, blood pressure, and serum cholesterol, as well as incident cardiovascular disease, type-2 diabetes or cancer during study follow-up. The variables used in the time-dependent Cox analysis are listed in Table 1. The statistical analysis was performed using STATA software Version 10 (18).

Sensitivity Analyses

Sensitivity analyses were performed to examine whether the results might be influenced by the methods of analysis of the duration of obesity, methods of imputing missing values, or the effect of selecting our sample by excluding the participants with obesity at baseline. The effect of defining duration using two consecutive occurrences of obesity was tested by analyzing the crude counting of examinations with obesity as a measure of duration. The effect of imputing missing values was tested by comparing the results with an analysis including only participants with no missing values of BMI in any examinations (85%, n=573). The effect of our sample selection was tested by including those with obesity at baseline in the analysis (n=1227).

Results

The eligible participants were followed-up for an average of 38 (9 to 48) years with the average duration of obesity being approximately 13 years (SD = 10, range 2 – 44 years). During the 50 years of study follow-up, 415 (61%) died and 40% of the deaths were caused by cardiovascular disease, 24% were due to cancer and 35% due to other causes. The characteristics of the participants included in the analysis can be viewed in Table 2.

All-cause mortality

The unadjusted hazard ratio for all-cause mortality for each additional two years lived with obesity was 1.14 (95% CI 1.12–1.16). Adjusted for the social demographic variables of age,

sex, marital status, education and country of birth, the hazard ratio was 1.17 (95% CI 1.15 – 1.19). Additionally adjusted for the health-related behavior variables of smoking and alcohol consumption, the hazard ratio was 1.16 (95% CI 1.14 – 1.18) and further adjusted for baseline BMI, current BMI and age of onset of obesity, the hazard ratio slightly increased to 1.19 (95% CI 1.16 – 1.21). After adjusting for biological intermediate variables of blood pressure, cholesterol and blood sugar level, the hazard ratio changed marginally to 1.15 (95% CI 1.12 – 1.18). Additional adjustment for pre-existing diseases had varying effects: with adjustment for onset of type2 diabetes, the hazard ratio remained at 1.15 (95% CI 1.12 – 1.18) per two-year increase in obesity duration. After also adjusting for onset of cancer, the HR slightly dropped to 1.13 (95% 1.10 – 1.16) and the hazard ratio dropped further to 1.09 (95% CI 1.06 – 1.12) after adjusting for onset of cardiovascular diseases (Table 3).

A significant interaction was found between the duration of obesity and the age of onset of obesity, smoking status, sex and age at baseline (Table 3). The effect of each additional two-year increase in the duration of obesity was higher in those with an older age of onset of obesity (age ≥55 vs. age <45 years). For those whose age of onset of obesity was above 65 years, the hazard ratio was 1.35 (95% CI 1.27–1.42). The effect of every additional two-year increase in the duration of obesity on mortality also differed by smoking status. The hazard ratio per additional two-year increase in the duration of obesity was 1.23 (95% CI 1.19–1.28) for non-smokers. The effect of each additional two-year increase in obesity duration on the risk of mortality was higher in females compared males with a hazard ratio of 1.17 (95% CI 1.13 – 120) for males and 1.21 (95% CI 1.18 – 1.25) for females.

When interactions were analyzed in males and females separately, significant interactions were again found for smoking status (for both males and females), age at baseline (only in females), and age of onset (for both males and females) (Table 4). In non-smoking males, the effect of each additional two-year increase in the duration of obesity was almost 2 times higher compared to male non-smokers, even after adjusting for all potential confounders, including pre-existing diseases. In addition, the effect of each additional two-year increase in the duration of obesity was approximately 2 times higher in females who age at baseline above 50 years compared to females aged below 40 years at baseline. In females, the hazard ratio was also approximately 2 times higher for those whose aged

onset of obesity was above 65 years compared with those whose age of onset of obesity was below 45 years.

Cardiovascular and cancer mortality

The duration of obesity was also associated with an increased risk of cardiovascular and cancer mortality. The unadjusted risk of cardiovascular and cancer mortality per additional two-year increase in the duration of obesity was similar to the risk of all-cause mortality (Table 5). Adjusted for socio-demographic variables, health behavior variables, BMI and age of onset of obesity, the hazard ratios for cardiovascular and cancer mortality were 1.16 (95% CI 1.13 – 1.20) and 1.18 (95% CI 1.14 – 1.22) per additional two-year increase in the duration of obesity, respectively (Table 5). However, after also adjusting for physical activity, biological intermediates and incident diseases, the hazard ratio for each additional two year duration of obesity was 1.16 (95% CI 1.11 – 1.21) for CVD mortality and 1.11 (95% CI 1.08 – 1.16) for cancer mortality, slightly higher than the hazard ratio of all-cause mortality.

For CVD mortality, a significant interaction was only found between the duration of obesity and age of onset of obesity, with a hazard ratio of 1.31 (95% CI 1.20 – 1.43) for those whose age of onset of obesity was above 65 years, double compared with those whose age at onset of obesity was below 45 years. For cancer mortality, no significant interaction was found between the duration of obesity and age at baseline, age of onset of obesity, sex or smoking status.

A Dose-Response Relationship

The association between the duration of obesity and risk of all-cause mortality followed a dose-response relationship both in crude and adjusted models (Figure 1). For those who were obese for 5–14.9 years, 15–24.9 years, and longer than 25 years, the relative risk of all-cause mortality was 4, 8 and 10 times higher compared to those who were obese for less than five years, respectively and the trend was statistically significant (P value <0.00001)

A dose-response relationship was particularly clear in the group that were non-smokers during study follow-up (n=248, 37%). For those non-smokers who were obese for 5–14.9 years, 15–24.9 years, and longer than 25 years, the relative risk of all-cause mortality was 5,

10 and 20 times higher compared to those who were obese for less than five years, respectively and the trend was statistically significant (P value <0.00001). In male non-smokers who were obese for longer than 25 years, the risk of overall mortality was more than 50 times higher compared to those whose exposure to obesity was less than 5 years. A clear dose-response was also evident for CVD and cancer mortality.

Sensitivity Analysis

Results analysing only the participants with no missing BMI values (n=573, 85%) were similar to the results already presented. The adjusted hazard ratio of all-cause mortality was 1.18 (95% CI 1.16–1.21), CVD mortality was 1.16 (95% CI 1.12 – 1.21) and cancer mortality was 1.17 (95% CI 1.12 – 1.21). The results of analyzing the crude counting of examinations with obesity as a method of measuring the duration of obesity also showed relatively similar figures to the results based on defining the duration of obesity using two consecutive occurrences of obesity (data not shown). The hazard ratio associated with increasing obesity duration was slightly lower when the participants who were obese at baseline (n = 567) were included in the analysis (total n=1244). The adjusted hazard ratios for all-cause mortality, CVD and cancer mortality were 1.14 (95% CI 1.13 – 1.15), 1.13 (95% CI 1.11–1.15), and 1.14 (95% CI 1.11 – 1.16), respectively.

Discussion

This study found that the risk of mortality increases strongly with increasing number of years lived with obesity, independent of the level of BMI attained. For every additional two-year increase in the duration of obesity, the risk of all-cause mortality increased by almost 20 percent. In non-smokers, a dose-response relation between years of duration of obesity and risk of mortality was clear, with an approximate doubling of risk for every ten-year increase in the duration of obesity. The association was still significant after adjusting for all possible confounding covariates including current BMI, age of onset of obesity and smoking. Some of the effect was explained by incident disease, particularly CVD and cancer. Some differences in effect size were seen in different population sub-groups. The effect appeared to be stronger in non-smokers, in females and in those with a later age of onset of obesity.

This is the first study to examine the association between the duration of obesity and the risk of mortality. This study has several strengths, in term of the quality of the data, the measurement of the variables and the statistical analysis. The association was derived from the analysis of long-term prospective cohort study dataset where body weight was regularly measured, mortality and causes of death was also measured and confirmed. In addition, most of the variables included in the model were analyzed as time-varying variables, using a time-dependent Cox proportional hazard model to capture the changing of variables over time.

Body weight and other variables were measured regularly and comparably for 24 examinations over two-year intervals for approximately 48 years of study follow-up. To reduce the residual possibility of body-weight misclassification at a given exam impacting on our definition of obesity duration, we used two consecutive examinations at which a participant was classified as obese as the starting point for the measurement of the duration of obesity.

We adjusted for all available potential confounding factors, including current BMI, smoking and age of onset of obesity. The subjects with existing cardiovascular diseases, cancer, and diabetes at baseline, as well as those dying in the first four years of follow-up, were excluded from the analysis to minimize the impact of reverse causation.

The confounding effect by smoking that has been a concern generally in analyzing the association between obesity and mortality (19) has been addressed in this analysis. Smokers tend to weigh less than non-smokers, but have higher mortality rates (20) which can combine to produce an artifact of an elevated mortality risk among leaner individuals (19). To minimize this effect, the analyses were adjusted for time-varying current smoking status and the number of cigarettes smoked. The analysis was also conducted separately for smokers and non-smokers. The analysis suggested that the risk of mortality per additional two-year increase in the duration of obesity was stronger in non-smokers compared with smokers.

The effect of potential intermediate variables in the causal relationship between the duration of obesity and mortality was analyzed. Adjusting for the available intermediate variables, in particular the pre-existing diseases of CVD and cancer, led to a decrease in the

estimated hazard ratios of approximately 10%, indicating that some, but not all, of the added risk associated with increasing duration of obesity is acting through these diseases.

Age is another variable that has been shown to confound and modify the relation between increasing weight and mortality (21, 22). In the current analysis, age was adjusted for, both as age at baseline and as age of onset of obesity. In the current study age of onset of obesity was found to be an important variable that must be considered in the future analysis of the effect of obesity duration. The effect of the duration of obesity was higher for those with older age of onset of obesity.

An important limitation of this study relates to the sample population. The original Framingham cohort study began in 1948 and the prevalence rates of obesity were relatively low at that time. The prevalence of obesity in the 1950s in the USA was below 10% (23) while in 2008, the prevalence of obesity among adults in the United States reached approximately 30% (24). However, there is no *a priori* reason to expect that the effect of increasing obesity duration would differ in today's populations.

For future research, it is recommended that the association between the number years of obesity and the risk of mortality be re-analyzed using a contemporary and a larger long-term cohort study dataset where body weight and other covariates are regularly measured. This study analyzed the duration of obesity separately from the degree of obesity. For future research, analysis of an overall measure combining both the degree and duration of overweight and obesity is suggested - for example estimating the risk of mortality for every additional "BMI-year", analogous to the "pack-years" concept used for smoking.

The results of this study suggest that the duration of obesity needs to be taken into account in estimating the future population burden of diseases related with obesity. Whilst a number of reports have suggested that the relation between obesity and mortality has decreased a little recently, possibly due to improved treatment and management (25), it would be expected to increase again in the future as the duration of obesity increases. In this study, the average age of onset of obesity was approximately 50 years and the average number of years lived with obesity was approximately 13 years. Today, the average age of onset of obesity is likely to be more than 10 years earlier than in previous decades. The results of the current study demonstrated that for every additional 10-years lived with

obesity, the risks of all-cause mortality, CVD and cancer mortality more than doubled, implying that the risk of mortality associated with current obesity in adults might be double that in previous decades.

Conclusion

This analysis shows that the duration of obesity is a strong predictor of mortality, independent of the actual level of BMI, the age of onset of obesity and current smoking status. As the onset of obesity occurs earlier and the number years of exposure to obesity is longer, the risk of mortality associated with adult obesity in contemporary populations is expected to increase compared to previous decades. Obesity prevention strategies need to be focused on delaying the onset of obesity. In addition, it is necessary to take the duration of obesity into consideration when estimating the future health burden associated with current obesity trends.

Competing interests

The authors declare that we have no competing interests.

Authors' contribution

Author AA developed the analytical techniques of study, performed data analysis, interpretation, drafting of the article and prepared the final version for publication; MdeC contributed to the study analysis design, interpretation of data, and reviewing of the article; JS helped review the data analysis, reviewed the article, and prepared the final version for publication; CS contributed to the data analysis design and reviewed the article, HW contributed to data analysis, and reviewed the article; AP supervised the implementation of the study, designed the study's analytic strategy, reviewed the article and prepared the final version for publication.

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Table 1: List of Variables and Covariates Used in the Time-Dependent Cox Analysis

Covariates included in the models	Covariate type (continuous/ categorical)	Fixed time dependent covariate or time-dependent covariates
1. Duration of obesity	continuous	Time-dependent covariate
2. BMI & obesity related variables		
BMI at baseline	categorical	Fixed covariate
Current BMI	continuous	Time-dependent covariate
age of onset of obesity	continuous	Fixed covariate
3. SES variables		
age at baseline	continuous	Fixed covariate
Sex	categorical	Fixed covariate
marital status	categorical	Time-dependent covariate
educational level	categorical	Time-dependent covariate
country of birth (ethnicity)	categorical	Fixed covariate
4. Health behaviour variables		
present of smoking	categorical	Time-dependent covariate
number of cigarettes smoking	continuous	Time-dependent covariate
total alcohol consumption	continuous	Time-dependent covariate
physical activity score	continuous	Time-dependent covariate
5. Physiological variables		
blood pressure	continuous	Time-dependent covariate
Cholesterol	continuous	Time-dependent covariate
6. Pre-existing diseases		
Diabetes	categorical	Time-dependent covariate
cancer	categorical	Time-dependent covariate
CVD	categorical	Time-dependent covariate

Table 2. Characteristics of participants included in analysis

Variables and characteristic	
Total participants in FHS	5203
Participants who were ever obese in at least two consecutive examinations	1,287
Participants who were obese at baseline	567
Participants who had pre-existing diseases at baseline	43
*Eligible participants**	677
The characteristics of eligible participants	
Age at baseline	mean 40 years, range (28 - 62)
Age of onset of obesity	mean 58 years, range (32 – 88)
Sex	males n=277 (41%)
	females n=396 (59%)
Country of birth	born in USA (n= 566; 84%)
	not born in USA (n=107, 16%)
Marital status at baseline	100%
Educational level at baseline	8th grade or less (n =203; 30%)
	high school (n=313; 46%)
	college (n=70;10%)
	postgraduate/school (n=87; 13%)
Health behavior	
Smoking status	Ever smoking (n=427; 63%)
Number cigarettes smoked per day at baseline	mean 9.5, range 1 cigar. – 3 packs
Physical activities score at exam 4	mean of score 33, range 26 – 83
Alcohol drinking (ounces/month) at exam 2	mean 15 ounces, range 0–360
Biochemical characteristics	
Systolic Blood Pressure at baseline	mean 132, range 94 – 240
Diastolic Blood Pressure at baseline	mean 84, range 58 – 138
Hypertension at baseline	yes (n=82;18%)
Serum cholesterol baseline	mean 219; range 121 – 392
Obesity duration	mean 13; range 2 – 44
	<5 years, (n=180; 27%)
	5 – 15 years (n=113; 17%)
	15 – 25 years (n=120; 17%)
	≥25 years (n=260; 38%)
Outcome Event (Death)	
All causes mortality	n=415 (61%) of total sample
CVD caused mortality	n=166 (40%) of eligible sample
Cancer caused mortality	n=102 (24%) of eligible sample
Other caused mortality	n=147 (35%) of eligible sample

**Eligible participants were those who were ever obese in at least two consecutive examinations but not obese and free from pre-existing diseases at baseline*

Table 3. Association of the duration of obesity with the risk of all-cause mortality*

Variable Categories	n sample (death)	Hazard Ratios (95% CI)		
		Crude	Model A	Model B
All sample	677 (415)	1.14 (1.12–1.16)	1.19 (1.16–1.21)	1.09 (1.06–1.12)
Age at baseline				
<40	369 (154)	1.15 (1.12–1.17)	1.15 (1.11–1.18)	1.06 (1.02–1.11)
40-49.9**	218 (174)	1.21 (1.18–1.24)	1.24 (1.20–1.28)	1.15 (1.10–1.20)
≥50	90 (87)	1.20 (1.15–1.24)	1.23 (1.16–1.31)	1.13 (1.03–1.23)
Gender**				
Males	277 (193)	1.14 (1.12–1.16)	1.17 (1.13–1.20)	1.05 (1.01–1.09)
Females	400 (222)	1.15 (1.13–1.16)	1.21 (1.18–1.25)	1.13 (1.09–1.18)
Age of onset of obesity**				
<45	99 (65)	1.11 (1.08–1.15)	1.17 (1.12–1.23)	1.09 (1.01–1.16)
45 – 64.9	174 (119)	1.16 (1.14–1.18)	1.21 (1.18–1.24)	1.13 (1.10–1.17)
≥65	206 (111)	1.40 (1.34–1.46)	1.35 (1.27–1.42)	1.22 (1.13–1.32)
Smoking Status**				
Never Smoking	248 (148)	1.14 (1.11–1.16)	1.23 (1.19–1.28)	1.16 (1.11–1.21)
Ever Smoking	429 (267)	1.14 (1.12–1.16)	1.15 (1.12–1.18)	1.06 (1.02–1.09)

Model A, HR adjusted for social demographic variables of sex, age at baseline, marital status, educational levels, ethnicity (born in the USA or outside of the USA), and health behavior variables of time varying smoking, alcohol consumption, BMI at baseline, current body mass index (per unit BMI), and age of onset of obesity.
Model B, Adjusted for all variables in model A, plus physical activity, blood pressure, serum cholesterol, type 2 diabetes, CVD and cancer.
*The analysis is presented per additional two-year increase in the duration of obesity; when the analysis performed per 10 years increase in the duration of obesity, the HR in all sample for adjusted model A was 2.34 (95% CI 2.12– 2.59).
**The variables have a significant interaction with the duration of obesity.

Table 4a. Association of the duration of obesity with the risk of all-cause mortality in males

	n sample (death)	Hazard Ratios (95% CI)	
		CRUDE	ADJUSTED
All sample	277 (193)	1.14 (1.12–1.16)	1.15 (1.13–1.16)
Age at baseline			
<40	153 (82)	1.15 (1.11–1.18)	1.15 (1.09–1.21)
40-49.9	87 (74)	1.19 (1.15–1.24)	1.20 (1.14–1.27)
>=50	37 (37)	1.17 (1.09–1.24)	1.23 (1.12–1.38)
Age of onset of obesity**			
<45	48 (33)	1.13 (1.08–1.18)	1.21 (1.12–1.30)
45 – 64.9	151 (101)	1.16 (1.13–1.19)	1.21 (1.15–1.26)
>=65	78 (49)	1.44 (1.34–1.54)	1.32 (1.19–1.46)
Smoking Status**			
Never Smoking	38 (25)	1.21 (1.14–1.28)	1.31 (1.15–1.48)
Ever Smoking	239 (168)	1.13 (1.11–1.15)	1.15 (1.11–1.19)

* HR adjusted for social demographic variables of sex, age at baseline, marital status, educational levels, ethnicity (born in the USA or outside of the USA), and health behavior variables of time varying of smoking, alcohol consumption, BMI at baseline and time varying of current body mass index (per unit BMI), and Age of onset of obesity.
**Significant interactions with smoking status and age of onset.

Table 4b. Association of the duration of obesity and risk of all-cause mortality in females

	n sample (death)	Hazard Ratios (95% CI)	
		CRUDE	ADJUSTED
All sample	400 (222)	1.15 (1.13–1.16)	1.21 (1.18–1.25)
Age at baseline**			
<40	216 (72)	1.15 (1.12–1.19)	1.14 (1.10–1.20)
40-49.9	131 (100)	1.23 (1.20–1.27)	1.26 (1.21–1.32)
>=50	53 (50)	1.21 (1.16–1.27)	1.30 (1.20–1.41)
Age of onset of obesity**			
<45	51 (32)	1.12 (1.06–1.17)	1.18 (1.10–1.26)
45 – 64.9	221 (128)	1.16 (1.13–1.19)	1.22 (1.18–1.26)
>=65	190 (62)	1.41 (1.34–1.49)	1.41 (1.30–1.53)
Smoking Status**			
Never Smoking	210 (123)	1.13 (1.10–1.15)	1.24 (1.19–1.28)
Ever Smoking	190 (99)	1.16 (1.14–1.19)	1.16 (1.10–1.21)

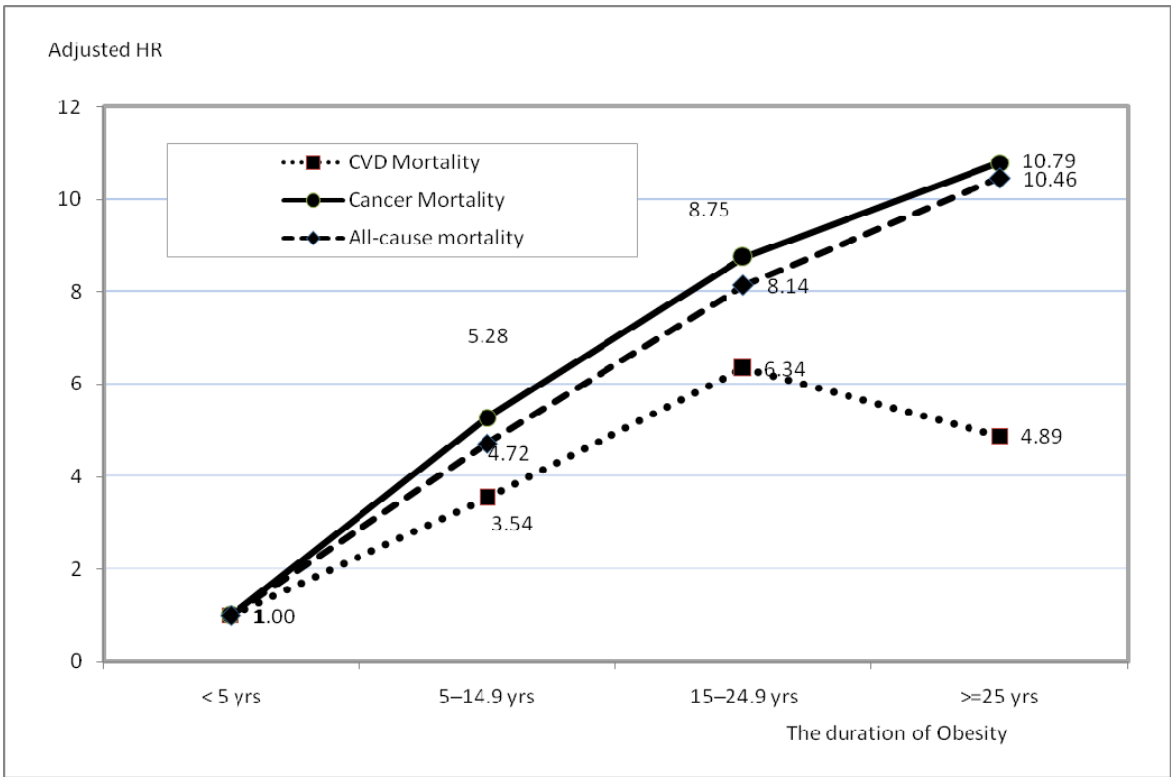
* HR adjusted for social demographic variables of sex, age at baseline, marital status, educational levels, ethnicity (born in the USA or outside of the USA), and health behavior variables of time varying of smoking, alcohol consumption, BMI at baseline and time varying of current body mass index (per unit BMI), and Age of onset of obesity.
**Significant interactions with smoking status, age at baseline and age of onset.

Table 5. The association the duration of obesity and risk of CVD and cancer mortality**

Variables Categories	Sample	Adjusted Hazard Ratios (95% CI)			
		(cases)	CVD Mortality	(cases)	Cancer Mortality
All sample	677	(166)	1.16 (1.13–1.20)	(102)	1.18 (1.14–1.22)
Age at baseline					
<40	369	(57)	1.13 (1.07–1.18)	(49)	1.15 (1.09–1.22)
40-49.9	218	(75)	1.19 (1.14–1.25)	(37)	1.27 (1.19–1.36)
>=50	90	(34)	1.16 (1.07–1.25)	(16)	1.22 (1.08–1.38)
Gender					
Males	277	(82)	1.15 (1.09–1.21)	(49)	1.16 (1.09–1.23)
Females	400	(84)	1.19 (1.14–1.24)	(53)	1.20 (1.14–1.27)
Age of onset of obesity*					
<45	99	(30)	1.13 (1.06–1.20)	(17)	1.27 (1.15–1.42)
45 – 64.9	372	(96)	1.20 (1.15–1.24)	(56)	1.20 (1.14–1.26)
>=65	206	(40)	1.31 (1.20–1.43)	(29)	1.35 (1.22–1.49)
Smoking Status					
Never Smoking	248	(55)	1.20 (1.15–1.26)	(27)	1.24 (1.17–1.33)
Ever Smoking	429	(111)	1.13 (1.09–1.18)	(75)	1.13 (1.08–1.19)

Model A, HR adjusted for social demographic variables of sex, age at baseline, marital status, educational levels, ethnicity (born in the USA or outside of the USA), and health behavior variables of time varying of smoking, alcohol consumption, BMI at baseline and time varying of current body mass index (per unit BMI), and age of onset of obesity.
*In CVD mortality, there was a significant interaction with the duration of obesity and age onset of obesity.
**The analysis was presented per additional two-year increase in the duration of obesity; when the analysis performed per 10 years increase in the duration of obesity, HR for CVD and cancer mortality in all sample for adjusted model A were 2.11 (95% CI 1.83–2.44) and 2.29 (95% CI 1.89–2.76).

Figure 1: The relationship between mortality (cause specific and all causes) and the duration of obesity.*



*Adjusted for social demographic variables of sex, age at baseline, marital status, educational levels, ethnicity (born in the USA or outside of the USA), and health behavior variables of time varying of smoking, alcohol consumption, BMI at baseline and time varying of current body mass index (per unit BMI), and age of onset of obesity.

Title: Setting population targets for measuring successful obesity prevention

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Kathryn Backholer, Helen Walls, Dianna Magliano *and* ***Anna Peeters*** *demonstrate the critical importance of utilising a reliable model of future obesity prevalence to assess and monitor national body weight targets.*

Many developed countries worldwide have set future targets on the prevalence of normal weight, overweight and/or obesity in an attempt to express their political will to tackle the obesity epidemic. In 2008 The Council of Australian Governments (COAG) set a target to increase by 5% the proportion of Australian adults at a normal body weight by 2017, over a 2009 baseline (1). No target was made in reference to overweight and obese body weight categories. This is a significant target, particularly as it is backed with financial support. However, there is currently no context within which to decide whether such a target is achievable, nor whether it is the best measure of successful obesity prevention. As the

prevalence of normal weight is expected to plummet in the coming decade (2-4), even maintenance of current levels of normal weight might be considered a marker of success. We are concerned that without realistic and practical obesity targets, the general public and the policy makers tasked with enacting these targets, may become disillusioned and question our ability to tackle the current obesity epidemic. Insufficient public and political confidence will undermine the drive needed to address the current body weight trajectory.

Herein we determine what change in current levels of population weight gain would be required to meet these national targets. In doing so, we demonstrate the critical importance of using a reliable model of future body weight prevalence as the context for target setting.

Obesity projection models as a context for national target setting

The transition based multi-state life table models used in this study have been described previously, demonstrating that the prevalence of normal weight in Australian adults is likely to drop from 35% in 2010 to 30% in 2020 if current trends persist (3). In our model, the future prevalence of normal weight, overweight and obesity is dependent on the age-specific probabilities of moving from one body mass index (BMI) category to another as observed in the Australian Diabetes, Obesity and Lifestyle cohort study (AusDiab) between 2000 and 2005.

Using this model we estimate the impact of hypothetical scenarios on the future prevalence of normal weight. First we analysed the effect of reducing the degree of weight gain over time for the whole population. This was done by incrementally reducing the observed increase in BMI in individuals within the AusDiab cohort over the five year follow-up. Second, we analysed the effect of changes restricted to those with normal weight, by decreasing their rate of progression to overweight. Changes in incidence of overweight were analysed by making

incremental changes to the probability of becoming overweight from normal weight observed in the AusDiab cohort. These altered transition probabilities were applied to our model from the year 2010 and flow-on effects on future prevalence of normal weight, overweight, and obesity were then estimated to 2025.

What measures are needed to meet national (government) targets?

In the first instance, we analysed what changes would be required to achieve the Australian national target. We demonstrate that a 75% reduction in 5-year BMI gain would be necessary to meet the national targets for normal weight prevalence by 2020, decreasing the mean increase in adult BMI over 5 years from 1.66 kg/m^2 to 0.50 kg/m^2. Similarly, a 80% decrease in the probability of becoming overweight from normal weight would be required to meet the target, changing the overall probability of incident overweight over five years from around 15% to 4% (Figure 1).

What will it take to halt the declining prevalence of normal weight?

Given the improbability of reaching the target prevalence for normal weight we next analysed which scenarios would halt the continuing decrease in the prevalence of normal weight, simply holding it steady at around 35% from 2010.

Stabilising the prevalence of normal weight from 2010 to 2020 would require a whole of population reduction in BMI gain over the 5 year period of 45%, changing the mean increase in BMI over 5-years from 1.66 kg/m^2 to 0.91 kg/m^2. Alternatively, this result could be achieved with a 45% reduction in the 5 year probability of becoming overweight from normal weight, shifting the probability of incident overweight from 15% to 8%.

What is the likely impact on future prevalence of normal weight following current "successful" obesity prevention interventions?

Finally, we modelled the effects of a 0.5 BMI unit lesser weight gain than observed over the 5 year period on the future prevalence of normal weight, overweight and obesity. Whilst very optimistic, a 0.5 BMI unit reduction in weight gain was chosen to represent the most effective single adult obesity prevention intervention detailed in a recent systematic review (5).

This scenario slowed the current decreasing trends in the prevalence of normal weight, and resulted in a decline of normal weight prevalence from 35% in 2010 to 32% in 2020 (compared to 30% with current trends; Table 1). In addition to a positive effect on the prevalence of normal weight, this scenario also resulted in a lesser increase in the prevalence of adult obesity, with the prevalence increasing only 1% between 2010 and 2020 rather than 5% with current trends. The lesser increase in obesity was offset by an increase over current projections in the prevalence of overweight from 38% in 2010 to 40% in 2020 (compared to 38% with current projections).

The importance of a reliable model for target setting

In an environment with a growing number of obesity prevention initiatives, it is becoming increasingly important to base policy decisions on the soundest evidence available to ensure the greatest chance of success. This requires knowledge of the effectiveness of interventions at a population level compared to the consequences of a 'do nothing' scenario. For example, one might conclude from our analysis, that a collection of obesity prevention strategies that result in a prevalence of normal weight greater than 32% in Australian adults in 2020 would be

deemed successful. Projecting forward the impact of such scenarios on the prevalence of body weight categories requires reliable obesity projection models.

Recent evidence suggests that the rate of increase in BMI may be slowing (6-9), presenting a serious limitation to projection models that are based on past prevalence (2). The prevalence of obesity is a result of decades of varying environmental influences and behavioural patterns, and projection models based on past prevalence are likely to capture trends which are no longer applicable. Our incidence based model incorporates the most recent probabilities of transitioning between BMI groups, thereby producing a robust future projection of BMI groups assuming no change in current patterns of weight gain. A similar population level incidence-based model has recently been validated against the observed trends in the USA (10). Nevertheless, all models have limitations and any attempt to predict the future is an inherently uncertain task. The further out the projection, the greater the uncertainty. In our model, the forecast prevalence is conditional on current transition rates between BMI categories, and does not take into account future changes in other factors that may affect these rates. Additionally, when using such a model (or any model) as the context for target setting it is assumed that the only factor affecting these BMI transitions will be policy measures, but this is evidently not the case. In light of these limitations we advocate the funding of repeated cohort studies and encourage detailed analysis of population changes in weight gain. When setting obesity targets, policy makers should make the best use of the most contemporary cohort studies available, as we have in our analysis.

Conclusion

In this analysis we demonstrate that in order to meet current national targets for normal weight, the rate of 5-year weight gain in Australia would need to be 75% less than it was between 2000 and 2005. Even to stabilise current levels of normal weight, current rates of 5 year BMI progression must be reduced by almost half. Given the modest success of obesity prevention interventions in adults to date (a recent systematic review reports a mean BMI change of 0.06(5)), a stabilisation, let alone a reversal of current adult normal weight prevalence by 2020, is a highly unlikely consequence of newly introduced policy initiatives. As our theoretical modeling of a best practice obesity prevention intervention illustrates, *a slowing of the current projected decline in adult normal weight trends may indeed be the best marker of successful obesity prevention*.

Our analysis further indicates that whilst a reduction in 5-year weight gain by 0.5 BMI units is not sufficient to halt or reverse the projected rise in obesity prevalence, it would markedly slow the escalating levels of obesity (with obesity prevalence increasing only 1% between 2010 and 2020 rather than 5% with current trends). This significant slowing of the prevalence of obesity should be considered a marker of success, given the burden of obesity on the health care system. Nevertheless, the same simulated reduction in BMI gain still resulted in a decline of normal weight prevalence from 35% in 2010 to 32% in 2020, far from the proposed national target. *We advocate that national targets additionally focus on a slowing of the current projected increase in obesity prevalence*.

Obesity is a multi-faceted condition with few successful intervention options. Thus, curbing current body weight trends to meet government targets will be challenging. Governments

should set realistic milestones which focus, at least initially, on slowing current projected trends. Targets should indeed offer an inspirational role demonstrating political commitment and providing the impetus for change, however they also need to be realistic and focused around the acheivement of tangible results. The formulation and evaluation of such targets requires the use of a reliable incidence-based projection model. Use of such a model is critical to provide a meaningful context for the setting of targets, the choice of strategies by policy makers and the evaluation of the long-term impact of the chosen obesity prevention strategies.

Contributors and sources: KB is a research fellow. HW is a PhD student. DM is a senior epidemiologist. AP holds a Vic Health senior research fellowship with a research interest in the burden of overweight and obesity and has over 10 years experience in epidemiological modelling. All authors were involved in the conception, design, analysis, and interpretation of data, drafting the article and revising it critically for important intellectual content. KB executed the analyses and wrote the article. All authors gave final approval of the version to be published. The views are those of the authors and not necessarily those of these departments. AP is the guarantor.

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Provenance and peer review: Not commissioned; externally peer reviewed.

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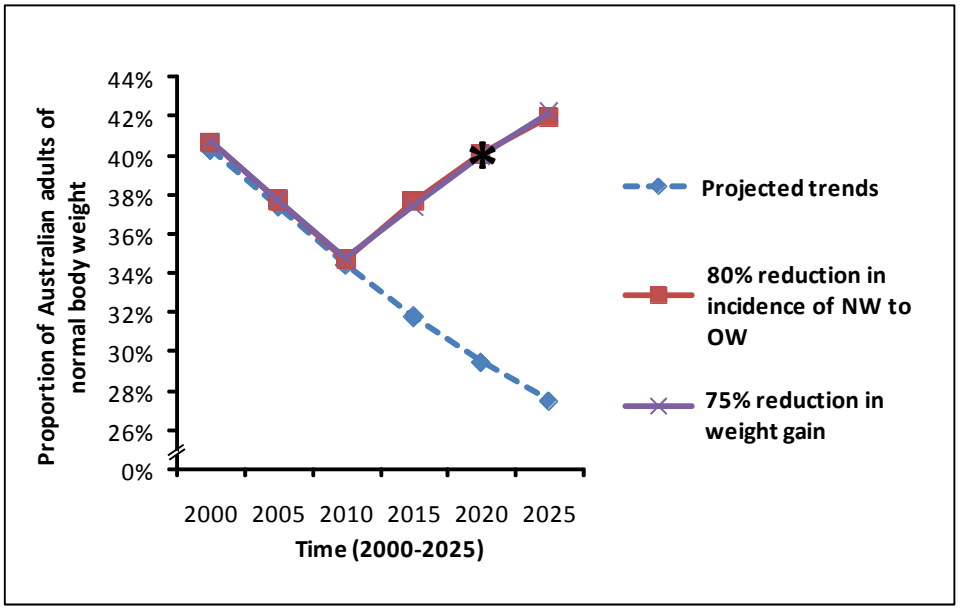


Figure 1: Projected prevalence of normal weight for Australian adults; comparing projected trends assuming a continuation of current rates of BMI progression (dotted line) with scenarios that achieve the national targets for normal weight by 2020. * Approximate national target for normal weight prevalence. NW: normal weight, OW: overweight.

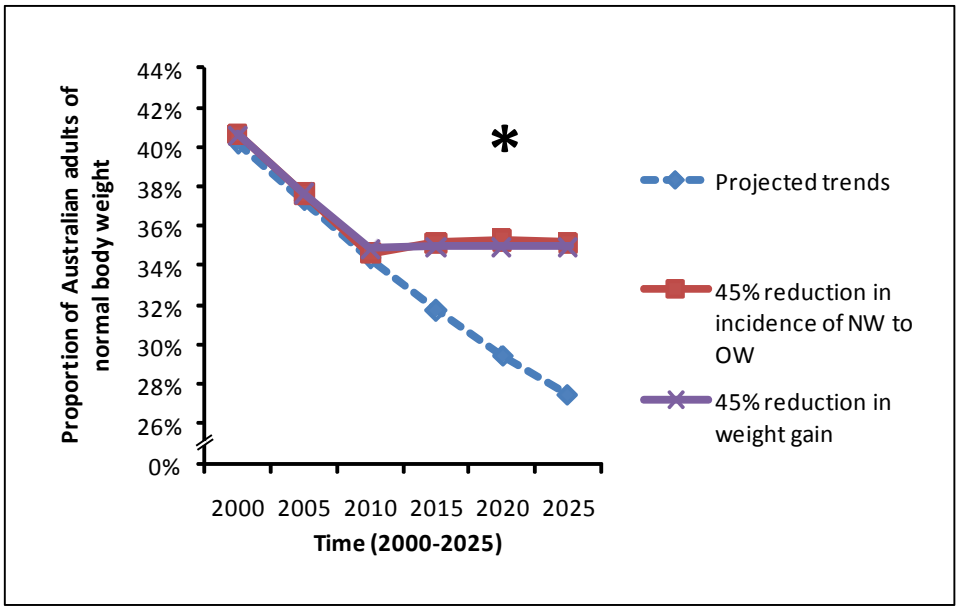


Figure 2: Projected prevalence of normal weight for Australian adults; comparing projected trends assuming a continuation of current rates of BMI progression (dotted line) with scenarios that stabilise the trends for normal weight between 2010 and 2020. * Approximate national target for normal weight prevalence. NW: normal weight, OW: overweight.

Table 1. Projected prevalence of normal-weight, overweight and obesity from 2000 to 2025; comparing projected trends assuming a continuation of current rates of BMI progression with a hypothetical reduction in 5 year BMI progression by 0.5 BMI units.

	Normal weight		Overweight		Obese	
	current trends	0.5BMI reduction	current trends	0.5BMI reduction	current trends	0.5BMI reduction
2000	41%	41%	39%	39%	20%	20%
2005	38%	38%	38%	38%	24%	24%
2010	35%	35%	38%	38%	27%	27%
2015	32%	33%	38%	39%	29%	27%
2020	30%	32%	38%	40%	32%	28%
2025	28%	31%	38%	41%	34%	28%

Appendix 4: Amendments

ERRATA

- p51, paragraph 3, last sentence: should read 'They are not in a position...' rather than 'They are in a position...'.

APPENDUM

- p13, section '1.2.3 Economic': the following should be added to the end of section 1.2.3: 'Colditz et al. (1992) estimated for US adults the cost attributable to obesity for a range of medical conditions. Of the total cost, which amounted to a reported 5.5% of the cost of illness in the US in 1986, cardiovascular disease contributed to 56.5%, type 2 diabetes mellitus to 28.8%, gall bladder disease to 6.1%, breast and colon cancer to 4.8% and hypertension to 3.8% (*). This can be considered a conservative estimate, considering that the study did not include the impact of obesity on musculoskeletal disorders or on cancers with a lower incidence than colon and breast cancer (*). These studies were also conducted some years ago, at a time when the prevalence of obesity, and particularly severe obesity, was considerably lower than today.
  In a German study (2002) exploring the direct and indirect costs of overweight and obesity, the direct cost burden was driven by outpatient drug costs for circulatory and endocrinological diseases (which accounted for one third of costs), and the indirect cost burden was driven by paid work for males and unpaid work for females (#). The cost burden is reportedly, at least for direct costs, considerably greater for severe obesity than moderate obesity (^).'
  *The reference for this is: Colditz GA. Economic costs of obesity. *Am J Clin Nutr*. 1992;55:503S-7S.
  # The reference for this is: Konnopka A, Bodemann M, Konig HH. Health burden and costs of obesity and overweight in Germany. *Eur J Health Econ*. 2010 [Epub ahead of print].
  ^ The reference for this is: von Lengerke T, John J, Mielck A, KORA Study Group. Excess direct medical costs of severe obesity by socioeconomic status in German adults. Psychosoc Med. 2010;7:Doc01.
- p14, section '1.3.1 International trends': the following should be added to the end of section 1.3: 'However a slowing in the rate of increase in the prevalence of obesity has been shown for some population groups in New Zealand and the US (ref: 46, 122, 128) and there has been an apparent slowing of the annual rate of overweight/obesity increase based on measured BMI in Australian children (*)'.
  * The reference for this is: Booth ML, Dobbins T, Okely AD, Denney-Wilson E, Hardy LL. Trends in the prevalence of overweight and obesity among young Australians, 1985, 1997 and 2004. *Obesity*. 2007;15:1089-95.

This new paragraph should be followed by the last two paragraphs of section 1.3.2 (p16), which should be removed from this later section.

* All references listed above should be included in the reference section of the thesis.