Overweight and obesity in the young and old

Prevalence, prevention and eating behavior

Gabriele Eiben

GÖTEBORG UNIVERSITY

Department of Public Health and Community Medicine, Primary Health Care, The Sahlgrenska Academy, Göteborg University, Sweden

Göteborg 2007

Printed by Kompendiet, Göteborg, Sweden

ISBN 978-91-628-7101-7

Für meine Eltern
Georg und Elvira

“Wenn jemand sucht, dann geschieht es leicht, dass sein Auge nur noch das Ding sieht, das er sucht, dass er nichts zu finden, nichts in sich einzulassen vermag, weil er nur immer an das Gesuchte denkt, weil er ein Ziel hat, weil er vom Ziel besessen ist. Suchen heißt: Ein Ziel haben. Finden aber heißt: frei sein, offen stehen, kein Ziel haben.”

Herman Hesse,
Siddharta

ABSTRACT

Background: During the last 25 years the prevalence of overweight and obesity has increased dramatically, both in developed and developing countries. The World Health Organization is declaring it an epidemic. In Sweden the increase was almost 50% between 1980 and 2005. The largest increases occurred in the 16-24-year age group. The highest prevalence can be observed in older age groups.

The general aim of this thesis is to study obesity and its determinants in the young and the elderly. The specific aims are to document secular trends in overweight and obesity and lifestyle determinants in the elderly, to investigate the influence of genetic and environmental factors in body shape and behavioral aspects of weight in teenagers and to conduct an intervention study to prevent overweight and obesity in young high-risk women.

Methods: Five cross-sectional samples of 3 702 70-year-olds born between 1901 and 1930 in Göteborg, Sweden were examined in the Gerontological and Geriatric Population Studies (H70). Cohort differences in anthropometric parameters and lifestyle determinants such as diet, physical activity, smoking habits and education were assessed.

The Twin Study of Child and Adolescent Development (TCHAD) is a population-based longitudinal study including all twin pairs born in Sweden between 1985 and 1986. The 15-17-year-old twins reported their anthropometric data and answered a questionnaire about body image and disordered eating. Respective genetic and environmental influences were estimated.

Young women, aged 18-28, with severely obese parents, were randomized to control or intervention groups in which they participated in a year-long individualized behavioral program (Health Hunters) focusing on diet and physical activity.

Results: The prevalence of overweight and obesity increased in 70-years-olds and was about 65% and 22% respectively in 2000. BMI increased in all lifestyle groups, but the increase was lowest among the physically active. Many secular changes in food selection patterns were observed in the elderly and the nutritional content of their diets was generally adequate.

In the young twins, genetic factors had a major effect on anthropometric parameters while environmental factors were most important for body image and eating behavior.

Young women with a familial predisposition to obesity were at high risk for weight gain; however the Health Hunters prevention program succeeded in preventing weight gain.

Conclusion: Both the young and the elderly are afflicted by the obesity epidemic. Research activities and prevention initiatives must draw attention to both age groups. Our results indicate the importance of focusing on body image in the young, while diet and physical activity are relevant for all age groups. Working with prioritized groups is one component of managing the obesity epidemic.

SVENSK SAMMANFATTNING

Bakgrund: Under de senaste 25 åren har förekomsten av övervikt och fetma ökat dramatiskt i de flesta länder. WHO talar om en global fetmaepidemi. Även i Sverige har ökningstakten varit hög, närmare 50% från 1980-2005 och främst i de yngre åldersgrupperna (16-25 år). Den högsta prevalensen av övervikt ses hos äldre.

Syftet med avhandlingen är att studera övervikt och fetma och dess bakomliggande faktorer hos yngre och äldre. Delsyften är att dokumentera sekulära överviktstrender och fetmarelaterade livsstilfaktorer hos äldre, att analysera hur arv och miljö påverkar kroppsform, kroppsuppfattning och ätbeteende hos tonåringar samt att genomföra en interventionsstudie för att förebygga viktuppgång hos unga högriskkvinnor.

Metod: Fem tvärsnittsstudier inkluderande 3702 70-åringar ur Göteborgs befolkning, födda mellan 1901 och 1930, undersöktes i de gerontologiska och geriatriska populationsstudierna i Göteborg (H70-studien). Kohortskillnader i antropometriska mått och livsstilsfaktorer såsom kost, fysisk aktivitet, rökning och utbildning studerades.

The " Swedish Twin Study of Child and Adolescent Development" (Tchad) är en populationsbaserad longitudinell studie som inkluderar alla tvillingpar födda 1985-1986 i Sverige. När tvillingarna var 15-17 år ombads de att själva rapportera sina antropometriska mått och svara på frågor om kroppsuppfattning och ätbeteende. Den relativa betydelsen av arv och miljö analyserades.

Unga kvinnor, 18-28 år, med gravt överviktiga föräldrar, randomiserades till interventions- eller kontrollgrupp i "Health Hunters" programmet. Under ett år fick interventionsgruppen en individualiserad information baserad på beteendeförändringar som fokuserade på kost och fysisk aktivitet.

Resultat: Bland 70-åringar var prevalensen av övervikt ca 65% och av fetma ca 22%. BMI ökade med tiden i alla aktivitets-, rök- och utbildningsgrupper, men ökningen var lägst hos de fysiskt aktiva. De äldre uppvisade en tillfredsställande näringssituation och kostintaget hade förändrats på flera sätt.

Hos de yngre hade hereditet störst betydelse för vikt, BMI och midjeomfång medan miljöfaktorer hade störst betydelse för kroppsuppfattning och ätbeteende.

Unga kvinnor, predisponerade för övervikt, utgjorde en högriskgrupp för viktuppgång men i "Health Hunters" programmet kunde viktuppgång förebyggas.

Slutsats: Både yngre och äldre drabbas av den pågående fetmaepidemin. Forskningen och de preventiva åtgärderna måste rikta mer uppmärksamhet på yngre och äldre åldersgrupper. Dessa grupper måste integreras i preventiva åtgärder där det är viktigt att arbeta med kroppsuppfattning hos de yngre, medan kost och fysisk aktivitet är relevant för alla åldersgrupper. Att arbeta med dessa prioriterade grupper är betydelsefullt när fetmaepidemin ska bekämpas.

DEUTSCHE ZUSAMMENFASSUNG

Hintergrund: In den letzten 25 Jahren ist die Übergewichts- und Adipositasprävalenz in vielen Ländern dramatisch gestiegen. Die WHO spricht inzwischen von einer Epidemie. In Schweden stieg die Zahl zwischen 1980 und 2005 um fast 50%. Der stärkste Anstieg vollzog sich in der jüngeren Altersgruppe (16 - 25 Jahre). Nach wie vor ist die höchste Übergewichtsprävalenz jedoch in den älteren Altersgruppen zu beobachten.

Ziel dieser Doktorarbeit ist die Analyse von Übergewicht und Adipositas sowie deren Determinanten in den beiden genannten Altersgruppen. In Bezug auf die ältere Altersgruppe werden die säkularen Trends der Adipositasprävalenz und die damit assoziierten Lebensstilfaktoren dokumentiert und in Bezug auf die Jüngeren werden Analysen des genetischen Einflusses und dem der Umwelt auf die Körperform, Körperauffassung und Ernährungsverhalten durchgeführt. Eine Interventionsstudie untersucht zusätzlich Präventionsmöglichkeiten, um der Gewichtszunahme bei jungen, für Adipositas prädisponierten Frauen entgegenzuwirken.

Methoden: Im Rahmen der „Gerontologischen und geriatrischen Bevölkerungsstudien in Göteborg“ (H70) wurden 3 702 Probanden im Alter von 70 Jahren in fünf Querschnittsstudien im Zeitraum 1971 - 2000 untersucht. Dabei wurden Kohortenunterschiede in Anthropometrie, Ernährung, körperlicher Aktivität, Rauchverhalten und Ausbildung dokumentiert.

Die schwedische Zwillingsstudie zur Entwicklung von Kindern und Heranwachsenden (TCHAD) ist eine bevölkerungsbasierte Langzeitstudie, die alle Zwillinge einbezieht, die in den Jahren 1985/1986 in Schweden geboren wurden. Im Alter von 15 bis 17 Jahren wurden die Zwillinge gebeten, einen Fragebogen zu ihrem Essverhalten und anthropometrischen Maßen auszufüllen. Genetische und umweltbedingte Einflüsse auf Anthropometrie, Körperauffassung und Essverhalten wurden analysiert.

Im Rahmen des „Health Hunters“- Programms wurden Frauen, mit adipösen Eltern, im Alter von 18 bis 28 Jahren in eine Interventions- bzw. Kontrollgruppe randomisiert. In einem Untersuchungszeitraum von 12 Monaten wurde die Interventionsgruppe einem intensiven Beratungsprogramm unterzogen, welches auf eine vorteilhafte Änderung ihres Ernährungs- und Aktivitätsverhaltens abzielte.

Resultate: Bei den 70-jährigen Probanden lag die Prävalenz des Übergewichts bei 65% und die der Adipositas bei ca. 22%. Der BMI stieg in allen Aktivitäts-, Rauch- und Ausbildungsgruppen an. Allerdings war der Anstieg in der körperlich aktivsten Gruppe am geringsten. Der Ernährungszustand der 70-jährigen war zufriedenstellend und die Ernährung veränderte sich in vielerlei Hinsicht.

Das Körpergewicht, der BMI und der Taillenumfang in Jugendlichen waren größtenteils genetisch determiniert. Hingegen spielten Umweltfaktoren für die Körperauffassung und das Essverhalten die größte Rolle.

Junge Frauen, die eine erbliche Prädisposition für Adipositas haben, zeigten ein erhebliches Risiko für Gewichtszunahme. Das „Health Hunters“ - Programm zeigte die Möglichkeit einer erfolgreichen Prävention vor Gewichtszunahme in dieser Risikogruppe.

Schlussfolgerung: Sowohl junge als auch ältere Menschen sind von der Adipositasepidemie betroffen. Folglich müssen sich Forschung und Prävention auch auf diese Altersgruppen konzentrieren. Die Resultate dieser Doktorarbeit zeigen, dass bei jungen Altersgruppen die Sensibilisierung der eigenen Körperauffassung wichtig ist. Ernährung und körperliche Aktivität wirken sich in allen Gruppen positiv aus. Ein Teil der Eindämmung der Adipositasepidemie, sollte daher spezielle Präventionsprogramme für Problemgruppen sein.

ORIGINAL PAPERS

This thesis is based on the following papers, which will be referred to in the text by their Roman numerals:

I Eiben G, Dey DK, Rothenberg E, Steen B, Björkelund C, Bengtsson C, Lissner L. Obesity in 70-year-old Swedes: Secular changes over 30 years. International Journal of Obesity (2005) 29, 810–817.

II Eiben G, Andersson CS, Rothenberg E, Sundh V, Steen B, Lissner L. Secular trends in diet among elderly Swedes – cohort comparisons over three decades. Public Health Nutrition 2004;7(5):637-644.

III Eiben G, Lissner L, Lichtenstein P. Genetic and environmental factors in relation to body shape, body image and disordered eating in 15-17-year-old Swedish twins. Manuscript.

IV Eiben G, Lissner L. Health Hunters - an intervention to prevent overweight and obesity in young high-risk women. International Journal of Obesity 2006 Apr;30(4):691-6.

CONTENTS

ABBREVIATIONS

A	additive genetic
B	Bulimia
BD	Body dissatisfaction
BF	body fat
BMI	Body Mass Index
BMR	basic metabolic rate
BW	body weight
C	shared environment
CHD	coronary heart disease
DH	diet history
DT	Drive for thinness
DXA	dual X-ray absorptiometry
DZ	dizygotic
E	non-shared environment
E%	energy percent, percent of total energy intake
EDI	Eating Disorder Inventory
EI	energy intake
H70	The Gerontological and Geriatric Population Studies in Gothenburg
HC	hip circumference
MZ	monozygotic
PPSW	The Prospective Population Study of Women in Göteborg
sd	standard deviation
SE	standard errors
SOS	Swedish Obese Subjects
TCHAD	Swedish Twin Study of Child and Adolescent Development
WC	waist circumference
WHO	World Health Organization
WHR	waist-hip ratio

INTRODUCTION

The World Health Report 2002 identified the ten most important risks to human health in the developed countries; overweight and related factors such as inactivity and low fruit and vegetable intake are among the top ten (see Figure 1). During the last 25 years the prevalence of overweight and obesity has increased dramatically both in developed and developing countries. This development has been called an epidemic and, recently, a pandemic. In Sweden, this increase was almost 50% between 1980 and 2005; however, the situation is different in different age groups. The prevalence of overweight and obesity is highest in the elderly and increasing most in the younger age groups.

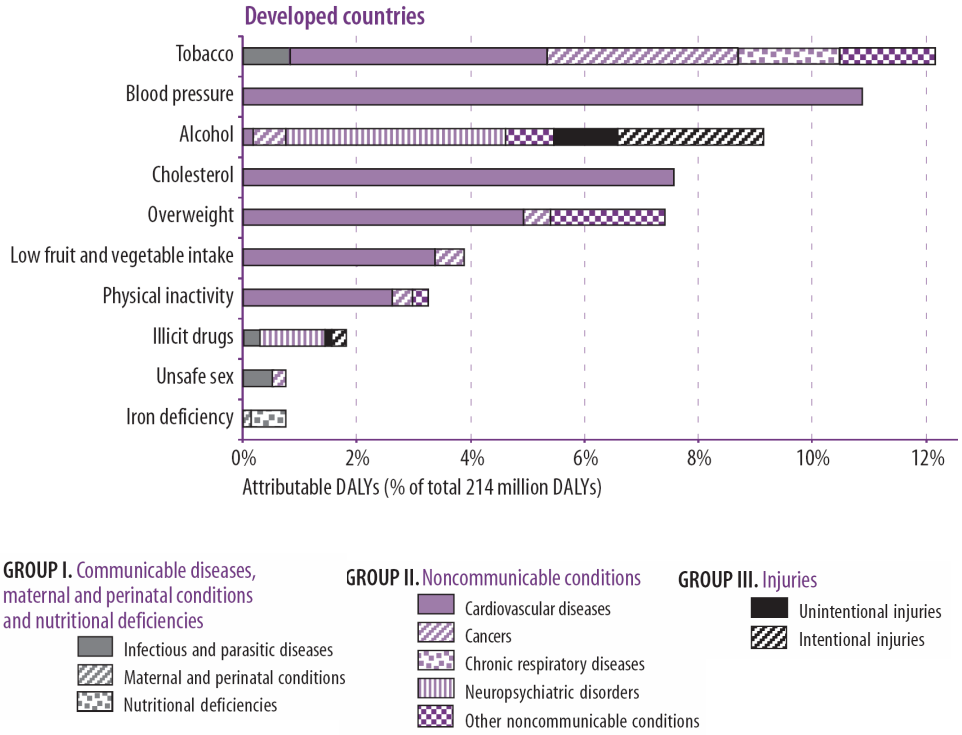


Figure 1 Burden of disease attributable to 10 selected leading risk factors, by level of development and type of affected outcome. Source: WHO 2003.

Life expectancy among the elderly has increased over recent decades. Thus, the elderly constitute a rapidly growing segment of the population in Sweden, as in most Western countries. This changing demographic structure makes it increasingly important to focus on older individuals with respect to overweight and its lifestyle determinants.

Growing body size in children and young adults in Sweden and most other countries could reverse life expectancy gains. Researchers fear that today's younger generation will have shorter and less healthy lives than their parents for the first time in modern history unless society stops this development. Prevention of overweight and obesity must become a priority. Planning and implementing community prevention requires an understanding of the genetic and environmental determinants.

This thesis reports on secular trends in overweight in the elderly and lifestyle determinants such as diet, physical activity, smoking and education. Moreover, genetic and environmental influences on body size, body image and eating behavior in teenagers are explored and, finally, the implementation of a unique primary prevention in young women with a strong family history of obesity is described.

1 BACKGROUND

1.1 Defining overweight and obesity

1.1.1 BMI, WC and WHR

According to the World Health Organization (WHO), obesity is defined as a disease in which excess body fat has accumulated to the extent that health is impaired. The most frequently used measure for estimating overall body fat at present is the body mass index (BMI), which is calculated from the body weight (kg) divided by the height squared (m^2). According to WHO, the definition of overweight in adults is BMI ≥ 25 while obesity is defined as BMI ≥ 30 (Table 1) (WHO 2000). This classification is primarily based on the association between BMI and mortality in adults and is independent of age and sex.

Table 1 Classification of overweight in adults based on BMI (WHO 2000).

Classification	BMI	Risk of co-morbidities
Underweight	< 18.5	Low
Normal range	18.5 – 24.9	Average
Overweight	≥ 25	
Pre-obese	25 -29.9	Increased
Obese Class I	30 – 34.9	Moderate
Obese Class II	35 – 39.9	Severe
Obese Class III	≥ 40	Very severe

It is important to measure not only overall obesity; the accumulation of abdominal fat is essential as well. Abdominal, particularly visceral, fat has been shown to be associated with an increased risk of cardiovascular disease and metabolic disorders. Therefore, waist circumference (WC) and the waist-hip circumference ratio (WHR) are used to estimate the amounts of abdominal fat. An increased risk of obesity-associated metabolic complications has been seen at a WC ≥ 102 cm in men and ≥ 88 cm in women. WHR measurements above 1.0 in men and 0.88 in women identify individuals with abdominal fat accumulation (WHO 2000).

1.1.2 Elderly populations

BMI has limitations for some groups. It is not a good estimator of body fat for people who have either elevated muscle mass (e.g. athletes) or diminished muscle mass (e.g. elderly) relative to their body fat. With normal aging, body composition changes and differs from that of younger persons. Age depend-

ent loss of lean body mass and shrinking leads to a higher percentage of total body fat (Hughes et al. 2002). Fat tends to be redistributed with age towards a more abdominal (particularly visceral) location. BMI becomes a poorer indicator of both overall and abdominal overweight in older individuals since body fat becomes increasingly located in the abdominal cavity; thus WC is more useful in this age group (Visscher et al. 2001). The issue of whether the WHO cutoffs for overweight and obesity are appropriate for elderly populations is being discussed. The classification is primarily based on the association between BMI and mortality in middle-aged adults. However, the mortality risk is less pronounced in older populations with an increasing BMI (Janssen and Mark 2007). In one review it was indicated that the optimal BMI range for elderly people could be increased to 24-29 kg/m^2 (Beck and Ovesen 1998).

1.1.3 Younger populations

In order to classify overweight and obesity in children (aged 2-18), an international BMI reference has been developed by Cole et al, based on data from children in the USA, the UK, Hong Kong, the Netherlands, Singapore and Brazil. Age- and sex-specific BMI cutoff points for overweight and obesity have been defined (Cole et al. 2000).

1.2 Causes of obesity

Weight gain occurs when energy intake exceeds energy expenditure, implying that the solution should be to eat less and be more physically active. But there is no simple solution for this major health issue. There is a lack of knowledge about the regulation of food intake and about which genes are involved in which processes. The etiology of obesity is multifactorial and includes genetic and environmental factors as well as gene-environment interactions.

1.2.1 Genetics

It is well understood that obesity runs in families and that BMI and body size are genetically determined to a major extent (Agras et al. 2004). An individual with a family history of obesity has a two to three times higher risk of developing obesity, compared to an individual with no heredity (Bouchard 1997). Twin studies have shown that approximately 70% of the variation in relative body weight (BW) between individuals is due to genetic differences (Schousboe et al. 2003; Wade et al. 2001). Adoption studies, on the other hand, have shown considerably lower genetic influence, about 40% (Schousboe et al. 2004). The truth may lie somewhere in between these figures. It is important to interpret these substantial genetic influences correctly. Many people believe that if there is strong genetic predisposition to

obesity, behaviors such as food intake and activity levels do not matter, a common misconceptation. Predisposition is not synonymous with predetermination. Strong genetic effects on a phenotype increase the probability that the phenotype will occur, but occurrence is dependent on other, environmental, factors, some of which can be modified. Awareness of individuals´ susceptibility to environmental factors should be heightened.

1.2.2 Environment

The obesity pandemic has developed during the last three decades, whereas it takes centuries and many generations for gene pools to change. Obesity can thus not be explained by changes in human genes; environment must be pinpointed as the main cause of obesity. During the last decades, the environment has changed tremendously and is now called obesogenic, i.e. favoring high energy intake and low energy expenditure.

1.2.2.1 Diet

Excess energy is stored as fat in order to provide for our needs during times of crisis, a necessary evolutionary mechanism in order to survive famine. Times have changed, at least in the developed countries, but human physiology has not. We perceive strong signals when we are hungry but less when we are overeating. The modern human would require just the opposite signals in order to maintain normal weight. Living in an environment where energy-dense food, often poor in nutrients, is available 24 hours a day, inevitably leads to weight gain. There is convincing evidence that high intake of energy-dense foods increases the risk of weight gain. There is some evidence that intake of high-sugar-content drinks, advertisement of energy-dense food and fast food outlets and large portion sizes promote weight gain. There is also evidence that intake of high dietary non-starch polysaccharide fiber, home and school support of healthy food choices and breastfeeding decrease the risk of weight gain (Swinburn et al. 2004).

An inconsistency between self-reported energy intake and BMI, especially in women, can be found in many studies (Jebb 1997; Weber et al. 2001). This discrepancy might be due to inadequacy of the diet assessment methods used and/or underreporting of the energy intake. Another explanation might be that overweight and obesity are results of minor overeating over a long period of time, difficult to measure at one particular timepoint. In an Australian study, the authors estimated that the average weight gain for adults is one gram daily (Bennett and Magnus 1994) which implies an overeating of less than 10 kcal/day.

1.2.2.2 Physical activity

People are shifting towards an increasingly sedentary lifestyle, e.g. spending hours in front of different screens and taking the car instead of walking (Prentice and Jebb 1995). But it is not only leisure activities that tend to be more sedentary; work-related energy expenditure has decreased due to labor-saving technology (Fogelholm et al. 1996). There is convincing evidence that regular physical activity decreases the risk of weight gain, a risk increased by a sedentary lifestyle (Swinburn et al. 2004). Physical activity also decreases the risk of morbidity and mortality (Blair et al. 1989; Grundy et al. 1999). A consensus meeting on how much physical activity is sufficient to prevent unhealthy weight gain recommended moderate-intensity activity for about 45–60 minutes daily, while 60-90 minutes daily is required to prevent weight regain. However, 30 minutes of moderate physical activity daily should limit the risk of related chronic diseases (Saris et al. 2003).

1.3 Prevalence and secular trends

More than one billion adults in the world are overweight and at least 300 million are obese (WHO), a situation currently described as a pandemic, rather than an epidemic, encompassing the developed and developing countries. According to Popkin, there are now more overweight than undernourished people in the world (speech at the International Association of Agricultural Economists 26th Annual Conference, Sydney, Australia, 2006). Obesity has become one of the most important global health problems during the last 25 years and it is still increasing. WHO has collected prevalence data from all over the world in a Global Info database (WHO Global Infobase) which is available on the Internet. Figures 2 and 3 show the prevalence of overweight and obesity in European and Swedish men and women aged >15 in 2002 and 2005, as well as the estimates for 2010 according to this database. All European countries reporting their respective 2005 prevalence to the database are included in these estimates. More than half of the adult European population is overweight and about one in six individuals is obese. Compared to Europe as a whole, the prevalence of overweight and obesity in Sweden is about 4 percentage units lower in men and about 7 percentage units lower in women; however, the slope over time is approximately the same. The projected overweight prevalence in 2010 suggests that Swedish men may attain the same levels as other Europeans did in 2005. Similarly, it will probably take another five years for Swedish women to reach the 2005 level of their European counterparts.

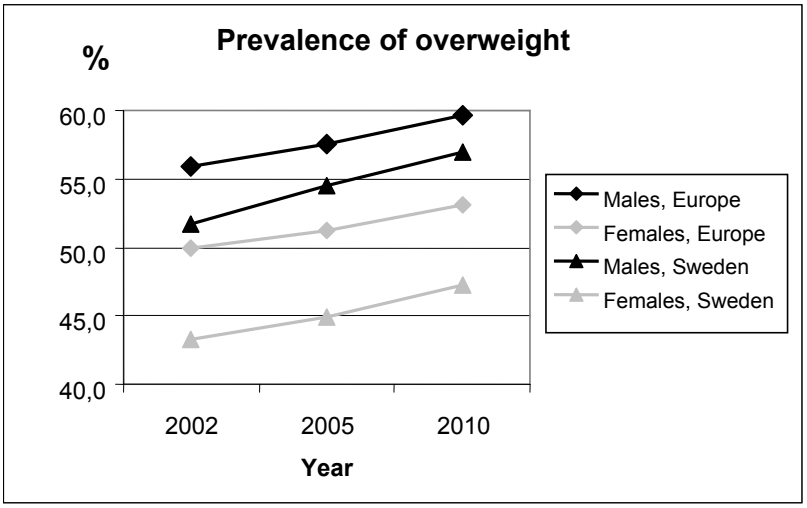


Figure 2 Estimated prevalence of overweight in Europe and Sweden for men and women, 2002-2010. Source: WHO Global Infobase.

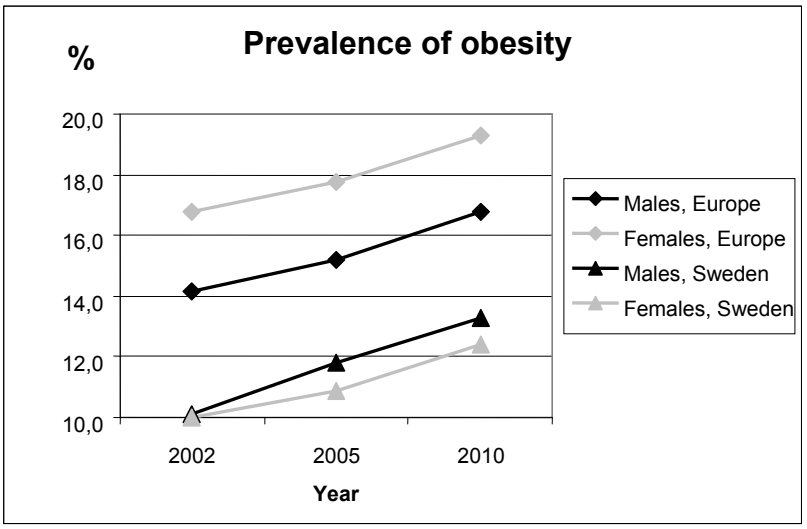


Figure 3 Estimated prevalence of obesity in Europe and Sweden for men and women, 2002-2010. Source: WHO Global Infobase.

1.3.1 Elderly populations

Elderly people represent a large and constantly growing part of the population in Sweden, as in most developed countries. Several studies have shown that the prevalence of overweight and obesity is increasing in elderly populations (Flegal et al. 2002; Gutierrez-Fisac et al. 2004; Mokdad et al. 2001; Nooyens et al. 2005). The National Statistics in Sweden recently showed an increasing prevalence of overweight and obesity in the Swedish population,

including in the elderly. The increase from 1988 to 2005 was 20% in 65-75-year age group. In 2005, about 60% of the men and 54% of the women in that age group were overweight or obese, according to self-reports (see Figure 4) (Statistiska Centralbyrån 2006). Studies from Sweden and other Nordic regions have reported that the obesity epidemic has affected middle-aged and elderly men, with a relatively limited impact on women (Dey and Lissner 2003; Lahti-Koski et al. 2001; Lissner et al. 1998a). Major secular increases in BW and height between 1971 and 1992 have been reported in 70-year-olds in the Gerontological and Geriatric Population Studies in Gothenburg (H70) (Rinder et al. 1975; Steen and Djurfeldt 1993); increases in BMI were only detected in men (Cabrera et al. 2003; Dey et al. 2001b).

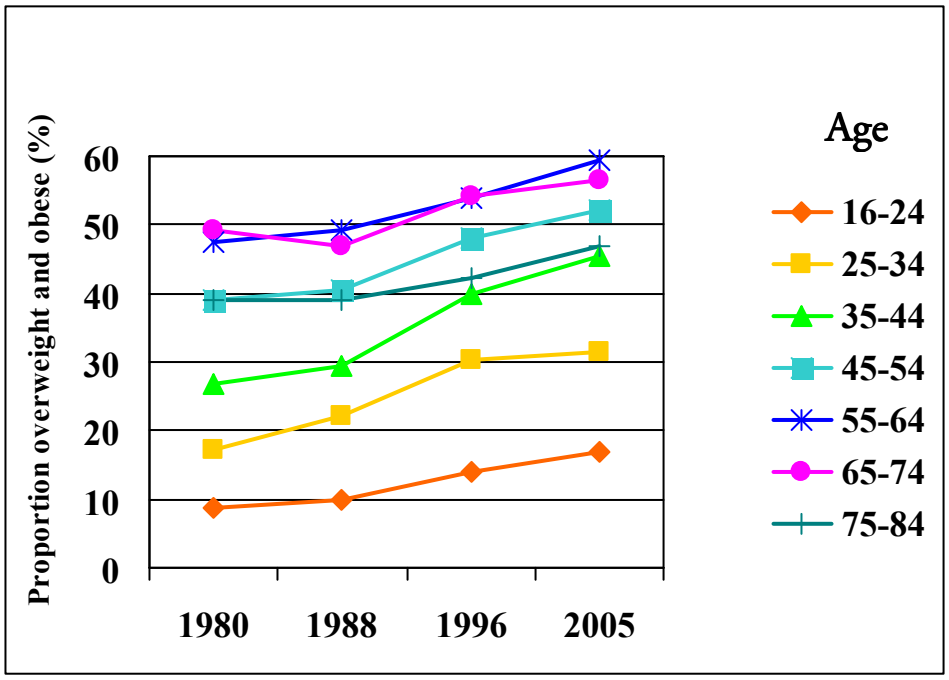


Figure 4 Prevalence of overweight and obesity, according to self reported BMI in Sweden in different age categories between 1980 and 2005. Source: Statistiska Centralbyrån 2006.

1.3.2 Younger populations

Young adulthood is recognized as a high-risk period for developing weight problems (Dietz 1997; Lissner et al. 2000; Rasmussen et al. 1999). Dramatic increases in the obesity prevalence in US adolescents between ages 13-19 and 20-26 were detected in the National Longitudinal Study of Adolescent Health. The prevalence increased from 11% in 1999 to 22% in 2001 (Gordon-Larsen et al. 2004). In Sweden, the National Statistics estimated the prevalence of overweight and obesity in 16-24-year old Swedes at about

17% in 2005, which is an increase of 93% since 1980/81 (see Figure 5). The 16-24-year age group is that with the most rapidly increasing prevalence of overweight and obesity, followed by the next age group (25-34-year-olds) in which prevalence has increased by 84% since 1980/81. Furthermore, there is a major gender difference. The prevalence of overweight and obesity in 16-24-year-old men increased by about 65% while the prevalence in young women in the same age range almost tripled (Statistiska Centralbyrån 2006).

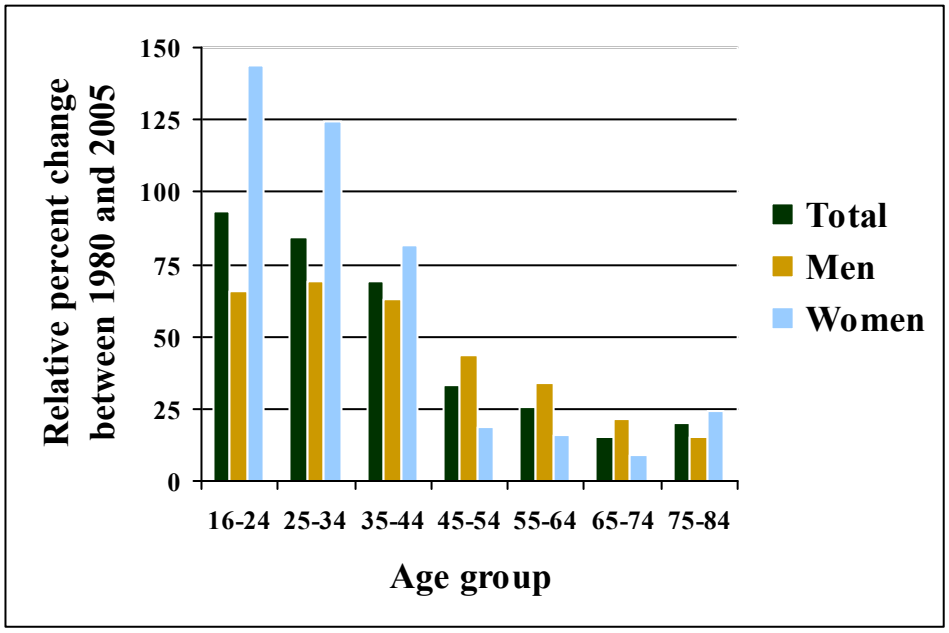


Figure 5 Relative percent increase in prevalence of overweight and obesity in Sweden for men and women in different age categories between 1980 and 2005. Source: Statistiska Centralbyrån 2006.

1.4 Health consequences

In the fourth century BC Hippocrates wrote, "Corpulence is not only a disease itself, but the harbinger of others", recognizing the enormous impact of obesity on health. The health, economic and psycho-social consequences of increasing obesity are substantial (WHO 2000). Obesity is strongly related to morbidity and mortality. Obesity is related to numerous non-communicable diseases such as type-2 diabetes, cardiovascular disease, hypertension, dyslipidemia, gallbladder disease, sleep apnea and certain types of cancers (Conway and Rene 2004; Haslam and James 2005). The increase in the prevalence of obesity is one of the reasons for the forecasts that diabetes will become an epidemic, most dramatically in newly industrialized and de-

veloping nations. As in the rest of the world, diabetes is increasing in Europe, a trend that is predicted to continue (WHO Global Infobase).

The psychological consequences of obesity are numerous and include lowered self-esteem and clinical depression. Obese subjects report worse health-related quality of life, compared to a reference population (Sullivan et al. 1993). Obese children's stigmatization starts at early age (Warschburger 2005).

Recent estimates suggest that 2-8% of the total health care costs in Western countries are attributable to obesity. The chronic diseases and poor psychosocial functioning related to obesity impose a substantial economic burden on both society and the individual in terms of health care expenditures and productivity loss. Obesity is associated with an increased risk of disability pension and an excess number of restricted activity days and lost workdays (Narbro et al. 1999). Moreover, obesity is related to an increase in the total consumption of health care. An estimated 1-6% of direct health care expenditures in affluent countries is attributable to obesity (Agren et al. 2002).

1.4.1 Elderly populations

The health implications of obesity in the elderly are not necessarily the same as in the middle-aged and the effect of age on optimal BW is controversial. Relationships between anthropometry and health change with aging. There is a reported U-shaped relationship between BMI and mortality as well as morbidity for the middle-aged (Stevens et al. 1998). In the elderly this relation, shown as a curve, has a large flat bottom and rises first at BMI 31-32 (Heiat et al. 2001; Janssen and Mark 2007). Previous analyses of 70-year-old Swedes indicate that high BMI ($\geq$ 28 kg/m^2) is protective for total mortality, albeit being a risk factor for coronary heart disease (CHD) and stroke in men. BMI was unrelated to CHD risk in female H70 cohorts (Dey and Lissner 2003; Dey et al. 2002). A recent review (Janssen and Mark 2007) concluded that overweight in women and men over 65 is not associated with an increased risk of mortality and that obesity is only associated with a modestly increased risk. On the other hand, low BMI and weight loss in the elderly is associated with increased mortality (Dey et al. 2001a; Visscher et al. 2000).

WC may potentially be more useful than BMI in detecting associations between increasing BW and mortality in the elderly (Lindqvist et al. 2006; Visscher et al. 2001). High WC ($\geq$ 99 cm) has been shown to be a risk factor for cardiovascular endpoints in men but WC was not related to CHD risk in female H70 cohorts (Dey and Lissner 2003; Dey et al. 2002). In the Prospective Population Study of Women in Göteborg (PPSW), Lindqvist et al

showed that higher WC was associated with an increased risk of mortality both in younger (38-46) and in older (50-60) women (Lindqvist et al. 2006).

Sarcophenic obesity is a relatively new category of obesity in the elderly. The term sarcophenia is defined as loss of muscle mass < 2 SD below the young-normal mean and increased fat mass > than the median value for each gender group (Baumgartner et al. 1998). Sarcophenic obesity has been shown to be associated with disability and physical impairment (Baumgartner 2000; Janssen et al. 2002). It may thus be of clinical importance to identify elderly individuals with sarcophenic obesity (Zamboni et al. 2005).

In a recent review (Zamboni et al. 2005), the authors stated that the risk of obesity in the elderly may be underestimated due to the effects of certain confounders such as survival effect, competing causes of mortality, smoking, weight change, unintentional weight loss and relatively shortened life expectancy in the elderly, which could underlie the existing controversy about the health consequences of overweight and obesity in the elderly (Zamboni et al. 2005).

1.4.2 Younger populations

Adolescence has been identified as one of the high-risk periods that may be critical in the development of obesity (Wing 1995). The probability of overweight and obese adolescents becoming obese adults or maintaining a high BMI during adulthood is well known (Kindblom et al. 2006; Kvaavik et al. 2003; Oren et al. 2003). A 40-year follow-up of overweight children admitted to hospital between 1921 and 1947 has shown that overweight and obesity during puberty is associated with higher morbidity and mortality in adults (Mossberg 1989). In a review on health consequences of childhood obesity (Reilly et al. 2003), the authors emphasized that childhood and adolescent obesity is not only a cosmetic matter as there is evidence that obesity affects short- and long-term health. It has been shown that cardiovascular risk factors, type-2 diabetes, chronic inflammation, asthma, orthopedic abnormalities and liver disease were related to pediatric and adolescent obesity (Kiess et al. 2001; Lobstein et al. 2004; Reilly et al. 2003). Young obese women have difficulties becoming pregnant and, if they succeed, suffer more complications during pregnancy and delivery (Cnattingius and Lambe 2002; Linne 2004).

In addition to the physical and psychological health consequences, there are many other problems. Overweight young adults are less likely to marry, compared to their normal-weight counterparts, they are less likely to obtain a university education and they have lower socioeconomic status (Gortmaker

et al. 1993). They are more often unemployed and, if employed, they earn less (Rosmond and Bjorntorp 1999).

In a large prospective study including 6 000 subjects in Germany, it has been shown that the greatest obesity-associated excess mortality is among young adults (Bender et al. 1999). Fontaine and colleagues estimated that a 20-year-old man with a BMI > 45 will lose 13 years of life, compared to a normal-weight man; the corresponding loss of life for women at that age and BMI is 8 years (Fontaine et al. 2003). Today's younger generation will have shorter and less healthy lives than their parents for the first time in modern history unless this trend is arrested (Fontaine et al. 2003; Olshansky et al. 2005).

1.5 Body image and disordered eating

As BMI increases, a growing part of the population is dissatisfied with its body weight and shape (Derenne and Beresin 2006). These individuals struggle towards an ideal body shape, presented by the media, which is getting thinner and thinner for women (Katzmarzyk and Davis 2001) and more and more muscular for men (Pope et al. 2001). The discordance between the ideal body shape and reality can result in weight cycling and eating disorders (Neumark-Sztainer et al. 2006a; Schofield et al. 1985). Eating disorders can be quantified with the extensively used Eating Disorder Inventory-2 (EDI-2) Drive for thinness (DT), Bulimia (B) and Body dissatisfaction (BD) subscales (Garner 1991).

EDI-2 is used as an instrument for examining the prevalence of eating disorders in certain populations. However, in recent years twin researchers have used it to estimate genetic and environmental influences on eating disorders. These studies have shown that body image and disordered eating in females is mainly influenced by genetic determinants (Kamakura et al. 2003; Klump et al. 2000; Rutherford et al. 1993), while less is known about genetic and environmental determinants in males. There is some evidence indicating a purely environmental determinant in males, in contrast to females (Keski-Rahkonen et al. 2005). Strong gender differences in global body image dissatisfaction have also been identified, leading to recommendations that this issue should be studied separately in females and males (Kostanski et al. 2004).

1.6 Prevention

In 1997 the WHO gathered obesity experts from all over the world for a consultation on obesity. The report "Obesity - Preventing and managing the

global epidemic" was a major breakthrough for recognition of the importance of obesity prevention (WHO 2000). Prevention is believed to be the only feasible public health strategy for dealing with the growing problem of obesity.

There are several arguments for prioritizing prevention rather than treatment. Obesity is a chronic condition and treatment is often ineffective, requiring long-term weight maintenance strategies. A literature review found that only 15% of dieters had maintained a significant proportion or all of their weight loss after five years (Ayyad and Andersen 2000). Surgical treatment of severe obesity produced the best long-term weight maintenance results (Sjostrom 2003) but it is not feasible to offer this expensive treatment to every obese patient. Obesity and overweight currently cost Swedish society about 16 billion SEK per year (The National Food Administration and Swedish National Institute of Public Health 2005). When the increasing requirements for any kind of obesity treatment due to the rising numbers of patients, the relatively limited impact of treatment effectiveness and the enormous expenses for society are taken into consideration, the importance of prevention becomes clear.

Previous reviews of obesity prevention studies have shown that interventions have moderate effects (Douketis et al. 1999). A recent review report from the Swedish Council on Technology Assessment in Health concluded that there is evidence that obesity can be prevented by interventions which improve diet and physical activity. Nonetheless, there were no observable effects in almost half of the studies included in the review, which shows the difficulties entailed in changing lifestyle (SBU Report from the Swedish Council on Technology Assessment in Health Care 2005).

Prevention of obesity, as outlined by the WHO, can occur at different levels (see Figure 6). While universal prevention on the population level is believed to be the only realistic long-term strategy for slowing the global epidemic, this approach requires major political, environmental and technological changes. To achieve prevention in the shorter term, it may be necessary to employ a targeted strategy in which high-risk individuals and groups are identified and offered professional support to prevent excessive weight gain. This selective prevention could be offered to groups with known risk factors related to obesity, e.g. family history. The inner circle represents the targeted and secondary prevention focusing on individuals who are already overweight but not obese. In these cases, the primary purpose is to prevent further weight gain, thus avoiding obesity-related co-morbidities (WHO 2000).

Figure 6 Levels of preventive action for dealing with weight gain and obesity. Source: WHO 2000.

1.6.1 Prevention in elderly populations

The 55-64- and 65-74-year-old age groups have the highest prevalence of overweight and obesity in Sweden (see Figure 4). However, there is relatively little discussion about prevention in population groups aged over 65, presumably due to the controversial health implications of overweight and obesity in the older age groups. There are few published reports on obesity prevention in the elderly, although there are studies in which body composition was improved by increasing physical activity (Campbell et al. 1994; Hunter et al. 2002). Furthermore, there are a few studies concerning whether voluntary weight loss is beneficial in the elderly. Zamboni et al concluded in a review that intentional weight loss may have beneficial effects on health (Zamboni et al. 2005). Another argument for the prevention of obesity in the elderly has been brought forward by Seidell et al (Seidell et al. 2005); in the authors' opinion, cost-effectiveness improves with age up to 65 or 70. There is a growing demand for developing strategies to prevent obesity, particularly the accumulation of visceral fat, in elderly men and women. Intervention programs might focus on increasing physical activity aimed at reducing visceral fat and maintaining muscle mass and strength. It is believed that weight loss in the elderly could reduce morbidity from arthritis, diabetes, cardiovascular disease and sleep apnea and increase well-being. Increased physical activity could produce beneficial effects on muscle strength, endurance and fat distribution (Elia 2001; Kennedy et al. 2004; Rossner 2001; Seidell et al. 2005; Zamboni et al. 2005).

1.6.2 Prevention in young adult high-risk groups

Young adulthood is recognized as a high-risk period for developing weight problems (Lissner et al. 2000; Rasmussen et al. 1999) and obesity is known to run in families (Bouchard 1996). Thus, prevention in young adults with a parental history of obesity should be a matter of course. Observational evidence supports the hypothesis that a high-risk, predisposed group might gain particular benefit from reducing dietary fat (Heitmann et al. 1995) and, furthermore, that the obesity-promoting effects of fat may be dependent on physical activity (Lissner et al. 1997). Growing up in the Internet era, today's youth is probably the most inactive generation in history. Consequently, the importance of prevention in a young high-risk group, focusing on diet behavior and physical activity, should have high priority.

Prevention in young high-risk groups is often not only a matter of primary, but also of secondary, prevention. A targeted approach must also address already overweight high-risk groups in which the primary goal is to prevent further weight gain and the development of co-morbidities in the long run. In its "National action plan against obesity", the Danish National Board of Health placed great importance on target groups, especially children and adolescents who are at high risk of developing obesity or obesity-related conditions. Prevention is recommended on the private, community and public sector levels. The most important task is to develop systems in the health care sector capable of detecting children and adolescents at risk (Danish National Board of Health 2003). Campaigns directed at high-risk groups should not detract attention and financial support from prevention on the population level; instead, they should intensify ongoing efforts (Yoon et al. 2003).

1.6.3 Prevention of body dissatisfaction and eating disorders

Both normal- and overweight young adolescents, influenced by media reports about the perfect body shape and which diets and exercise to use, try to transform their bodies (Groesz et al. 2002; Neumark-Sztainer et al. 2006a). Media-presented tools to achieve these goals are often unhealthy. Studies have shown that more than half of female and one- third of male teenagers in Western societies exhibit unhealthy weight control behaviors such as self-induced vomiting, use of diet pills, skipping meals, fasting and taking laxatives (Neumark-Sztainer et al. 2002). Preoccupation with thinness and body dissatisfaction predict the development of eating disorders (Kotler et al. 2001; Tyrka et al. 2002). Longitudinal studies have also shown a relationship between unhealthy weight control behaviors and the development of obesity (Neumark-Sztainer et al. 2006b). However, prevention should target both aspects, i.e. eating disorders and obesity.

2 AIMS OF THE THESIS

The general aim of this thesis is to study obesity and its determinants in the young and the elderly.

The specific aims were:

- to document secular trends in overweight and obesity and lifestyle determinants in the elderly
- to investigate the influence of genetic and environmental factors on body shape and behavioral aspects of weight in teenagers
- to conduct an intervention study to prevent overweight and obesity in young high-risk women

3 STUDY POPULATIONS

3.1 Gerontological and Geriatric Population Studies in Gothenburg

The H70 studies were initiated in 1971 with the general objectives of studying the normal aging processes and diseases in old age, thereby identifying clinical reference values, disease criteria and risk factors. The surveys of the medical and social conditions were intended to facilitate the planning of care for the elderly and the prevention of disease. Gothenburg (Göteborg, in Swedish) is the second largest city in Sweden, located on the west coast. The Revenue Office of Göteborg maintains a registry of all inhabitants, from which representative study groups of specific ages may be selected. Using this registry, 70-year-olds in Göteborg were recruited for health examinations, starting in 1971-72 based on day of birth. Subsequently, in 1976, 1981, 1992 and 2000, 70-year-old men were sampled the same way. Women were sampled similarly for health examinations in 1971, 1976 and 1981. In the latter two examinations, women born in 1922 and 1930 were partly identified on the basis of previous inclusion in the PPSW (see 3.2). One hundred and forty women in 1992 and 182 women in 2000 participated in both the PPSW and H70. To make the 1922 and 1930 female birth cohorts representative in 1992 and 2000, the samples were supplemented with additional women, born on selected dates, who had moved to Göteborg after the original study was started. The participation rates were high at the first examination in 1971 (84%) and at the second in 1976 (81%) and subsequently decreased to 77% in 1981 and about 65% in 1992 and 2000. The general design, procedures, data collection methods and representativeness of these cohorts have been reported elsewhere (Berg 1980; Eriksson et al. 1987; Rinder et al. 1975; Steen and Djurfeldt 1993). The examination year typically ran from September to June. Paper I is based on data from all five cohorts (n=3702) while Paper II includes only the cohorts born 1901, 1911, 1922 and 1930 (n=1306).

3.1.1 Diet sub-sample

Participants in the diet studies in 1971, 1981 and 1992 were randomly chosen from the complete H70 cohorts. One hundred and eighty-one women and 179 men in 1971, 129 women and 132 men in 1981 and 130 women and 59 men in 1992 completed the dietary survey. In the 2000 survey all 70-year-olds were scheduled for the dietary part of the examination and a total of 318 women and 232 men took part in diet history (DH) interviews. A small fraction of participants in the main health survey in 2000 (n = 19) did not complete the diet interview for various reasons, e.g. impaired cognitive

function, language difficulties, leaving before the interview or unavailability of a dietician (data not shown).

3.2 Prospective Population Study of Women in Göteborg

The PPSW started in 1968 (Bengtsson et al. 1973). A general initial objective was to study menopause but the objectives were extended to cover most aspects of women's health as the examinations proceeded. A representative sample, according to date of birth, was obtained from the Revenue Office Register. Five age strata were selected: ages 38, 46, 50, 54 and 60. A total of 1 462 women participated in the first examination, resulting in a participation rate of 90%. Follow-ups were carried out in 1974, 1980, 1992 and 2000. As described earlier, women examined 1992 and 2000 were also part of the H70 study.

3.3 Swedish Twin Study of Child and Adolescent Development

In 1994 the longitudinal Twin Study of Child and Adolescent Development (TCHAD) was started at Karolinska Institutet (KI) in Stockholm, Sweden, aiming at studying the role of genetics and environment in children's health and behaviors (Lichtenstein et al. 2002). The initial sample consisted of 1480 twin pairs born in Sweden between May, 1985 and December, 1986. Initially, the parents were asked to answer different questionnaires about their twins' health and behavior. The follow-up studies in 1999 and 2002 included questionnaires, not only to parents, but also to the children and their teachers. A total of 1 254 girls and 1 113 boys responded to the 2002 follow-up study, a response rate of 82%. Zygosity was determined by questionnaires and validated in a sub-sample (see Methods).

3.4 Health Hunters

Women aged 18–28 with at least one obese parent were identified via their parents who were enrolled in the ongoing Swedish Obese Subjects (SOS) study (Lissner et al. 1998b; Sjostrom et al. 1992). The SOS study included women and men with a BMI > 40 and > 37 kg/m^2, respectively, who were offered surgical or nonsurgical treatment. Based on data collected at the initial SOS screening examination, the subjects' young adult offspring could be identified. With the SOS subjects' consent, the offspring were contacted and invited to participate in a health program. Inclusion criteria were: living in Göteborg or vicinity, BMI over 18.5 and the ability to fill in the questionnaires unaided. Pregnancy, evidence of eating disorders, serious diseases that could affect compliance and plans to move far from Göteborg within the

next year were pre-defined exclusion criteria. Eighty-six SOS participants could be contacted, of which ten did not want their offspring to participate. Of 76 offspring we excluded 21 who were living outside Göteborg. Invitation letters were sent to 55 offspring; 41 accepted participation and 40 were included. One woman was excluded due to inability to fill in the questionnaires unaided (see Figure 7).

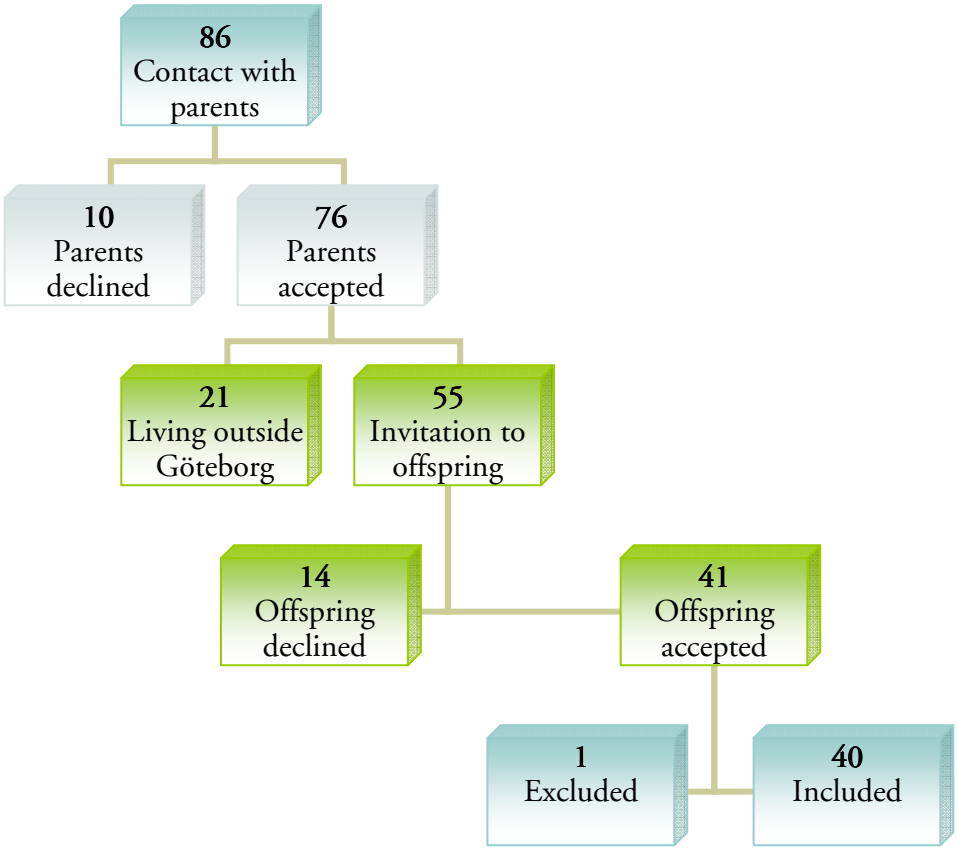


Figure 7 Inclusion and drop-out procedure in the Health Hunters study.

4 METHODS

4.1 Anthropometric data

4.1.1 H70 and Health Hunters

Measurements were made in the morning with fasting subjects wearing light clothing. BW was recorded to the nearest 0.1 kg and standing height was measured to the nearest cm. BMI was calculated. WC was measured to the nearest cm in the recumbent position using a tape measure placed halfway between the lower rib margin and the iliac crest. Hip circumference (HC) was measured to the nearest cm with the same tape measure, at the widest point between hip and buttock. WHR was calculated from the waist (cm) divided by hip (cm) circumferences. In Paper IV, total body composition was estimated by dual X-ray absorptiometry (DXA).

4.1.2 TCHAD

Weight and height were self-reported. BMI was calculated. Because the subjects were under age 18, the prevalence rates of overweight and obesity were calculated with Cole´s international reference BMI values (Cole et al. 2000). Four age intervals, 15.75-16.25, 16.25-16.75, 16.75-17.25 and 17.25-17.75, were used to correspond to ages 16.0, 16.5, 17.0 and 17.5, respectively. The twins were asked to measure WC at the smallest part of their waist and HC at the widest point of their buttocks using the survey measuring tape.

4.2 Dietary assessment methods

4.2.1 Diet history - H70

In Paper II, the DH method was used to estimate food, energy and nutrient intakes during the previous three months. The interviews lasted for about one hour, during which participants were asked to report what they usually eat and drink for each meal and, as far as possible, to specify the food items and amounts consumed. The interviews started with open-ended questions about usual food pattern and continued in a structured format. The same DH method has been used in all earlier H70 cohorts. Methodological details on the three earlier examinations have been published previously (Rothenberg et al. 1996). In the 2000 DH examinations, four dieticians were trained by one of the dieticians who had participated in the previous surveys; two of them entered the data from all coded interviews. In contrast to the 1971 and 1981 examinations, during which dietary interviews had been performed in the participants' homes, the DH interviews in 1992 and

2000 were performed in a clinical research unit. The method has been validated and found to yield comparable energy values to those predicted by the heart rate method. However, it underestimated usual energy intake by 12%, compared with the doubly-labelled water method, in 12 subjects and some obesity-related underreporting was observed (Rothenberg et al. 1998).

4.2.2 Food frequency questionnaire - Health Hunters

The dietary questionnaire used in the Health Hunters study was a self-administered semi-quantitative food frequency questionnaire with emphasis on portion size. The questionnaire inquires about the usual food intake during the last three months. It has been validated and is considered to provide good estimates of energy intake in both overweight and normal-weight adult subjects (Lindroos et al. 1993).

4.3 Physical activity assessment methods

4.3.1 Interview - H70

In 2000, physical activity levels were assessed by a physician-administered questionnaire using a 4-point scale ranging from almost completely sedentary to extremely active. The physical activity questions and scales used over the years have varied; details have been reported elsewhere (Dey et al. 2001b).

4.3.2 Questionnaire - Health Hunters

Physical activity questions describing activity during work, commuting and leisure were transformed into a point-based physical activity scale (kcal/week) (Persson et al. 1998).

4.3.3 VO2 max - Health Hunters

In Study IV, a VO2-max test was performed on the treadmill for measuring oxygen uptake and carbon dioxide production using a Medical graphics CPX analysis system. Participants started with a warm-up period at a speed of 1.5 km/hour for 2 min, followed by an increase to 12% elevation for one min. The speed was subsequently increased by 0.1 km/hour every 10 s until the participant was unable to continue.

4.4 Health Hunters Intervention

The one year intervention program was designed with the objectives of increasing energy expenditure, modifying obesity-related dietary practices and promoting behavioral skills that support these lifestyle changes. Previous

exploratory interviews conducted in the high-risk target population strongly indicated a need for individualized programs. At baseline the intervention group underwent an initial face-to-face examination and counseling session with the staff in order to identify their individual expectations and interests, including their stage of health consciousness (Prochaska et al. 1992). The intervention group received a customized support package divided into three main theme areas: physical activity, diet and weight control. Within each theme area, relevant informational and self-help materials were prepared, according to the individuals' expressed needs, preferences and health consciousness stage. To use an example from the nutrition theme, the prepared materials were relevant to different stages of change, ranging from never having contemplated changes in certain intake patterns, on the one hand, to maintenance of long-term improvement, on the other. While all subjects received some basic core materials from each theme area, they themselves decided when to focus on each section of the program. The initial counseling was followed by regular personalized contact via telephone, e-mail and 'booster' visits with dieticians. Every other week the participants received a letter with information material of their own choice. This information material was followed up by telephone about once a month. The need for 'booster' visits varied; participants decided on their own how often they wanted to see the dietician, ranging from every other week to only once. Occasional group sessions on food preparation and weight control topics and special interest lectures were offered throughout the intervention period. Together, subjects worked with the support staff to develop individual strategies for overcoming barriers to more healthy lifestyles. After finishing the study the control group was given access to the information material, which is now available on the Internet (Health Hunters).

4.5 Eating Disorder Iventory - TCHAD

The Eating Disorder Inventory 2 (Garner 1991) has been translated into Swedish and validated by Norring (Nevonen et al. 2006; Norring and Sohlberg 1988). The EDI-2 consists of 91 items and 11 subscales. The first three subscales, DT, B and BD, form an eating disorder symptom index (see Table 2). The other eight subscales in the EDI-2, e.g. ineffectiveness and perfectionism, were not used in this study. The DT subscale contains questions measuring preoccupation with weight and dieting, the B subscale contains questions measuring binging and purging behaviors and the BD subscale contains questions measuring satisfaction with specific body parts such as waist, thighs and buttocks. The test has a 6-point scale answer format: "always", "almost always", "frequently", "sometimes", "rarely" and "never", where 1 is equivalent to "always".

Table 2 Items in the subscales Drive for thinness, Bulimia and Body dissatisfaction subscales.

Drive for Thinness
I eat sweets and carbohydrates without feeling nervous.
I think about dieting.
I feel extremely guilty after overeating.
I am terrified of gaining weight.
I exaggerate or magnify the importance of weight.
I am preoccupied with the desire to be thinner.
If I gain a pound, I worry that I will keep gaining.
Bulimia
I eat when I am upset.
I stuff myself with food.
I have gone on eating binges where I felt that I could not stop.
I think about bingeing (overeating).
I eat moderately in front of others and stuff myself when they are gone.
I have the thought of trying to vomit in order to lose weight.
I eat or drink in secrecy.
Body dissatisfaction
I think that my stomach is too big.
I think that my thighs are too large.
I think that my stomach is just the right size.
I feel satisfied with the shape of my body.
I think my hips are too big.
I think that my thighs are just the right size.
I think my buttocks are too large.
I think that my hips are just the right size.

4.6 Zygosity determination - TCHAD

Zygosity for same-sex twins was determined by a series of five questions answered by the parents concerning the twins' physical similarity. The questionnaire has been validated in a sub-sample in which zygosity was determined by analysis of 16 polymorhic DNA markers. Twin pairs were classified as monozygotic (MZ) or dizygotic (DZ). In cases with contradictions between any of the five answers zygosity was recorded as unknown (Lichtenstein et al. 2006). This study only includes same-sex MZ and DZ twin pairs.

4.7 Statistics

4.7.1 H70 - anthropometry

Mean values and standard deviations were calculated for anthropometric parameters at all examinations. Two-sample t-tests were used to test the differences between the two latest-born cohorts. Tests of differences across all cohorts were conducted as tests of trend in linear least-square regression models using year of birth as the independent variable and continuous anthropometric indicators as time-dependent variables. Logistic models were used to compare dichotomous outcomes. Interactions in linear regression models were tested in order to investigate the influences of gender, smoking, physical activity and education on obesity trends. P-values of less than 0.05 were considered to be significant. Although we have described true longitudinal changes in two cohorts of women, terminology suggestive of longitudinal measurements (increases, decreases) is occasionally used in other parts of the paper, reflecting secular trends across H70 cohorts.

4.7.2 H70 - diet

The main results in Paper II are based on tests for linear trends across the four examination years. Tests of linear-by-linear association were used to assess cohort trends in mean intakes of nutrients and foods (g/day) and proportions (%). All results are reported separately for females and males. Values are expressed as means and standard deviation. The Goldberg method (Goldberg et al. 1991), in which energy intake (EI) is divided by an estimate of basal metabolic rate (BMR), was used to compare energy intakes reported by the different cohorts. BMR is predicted from weight (W) by means of standard equations for subjects aged 60–74 (males 0.0499 x W + 2.93, females 0.0386 x W + 2.875) (Department of Health 1991; Schofield et al. 1985). An EI/BMR ratio of 1.35 has previously been used to identify probable underreporting (Black and Cole 2001). For the physical activity comparisons reported here, the category described as 'almost completely inactive' was considered comparable across all cohorts. Subjects describing themselves as moderately or very active (according to various definitions over the years) are pooled and considered together to be the comparison group. Nutrient intakes have been compared with the Nordic Nutrition Recommendations (Sandström et al. 1996) and are described as the percentage of subjects with a nutrient intake above the recommended level and below the safe level, the latter suggesting a risk of nutrient deficiency.

4.7.3 TCHAD

Standard methods were used to calculate mean values and standard deviations. Two-sample t-tests were used to test the hypothesis of no difference in mean differences between the MZ and DZ group. Pearson's correlation (i.e.

intra-class correlation) was used to assess the within-pair similarity among MZ and DZ twin pairs. These analyses were applied to BW, BMI and WC. Studies have been shown that WC is a better predictor of abdominal adiposity in adolescents than HC or WHR (Taylor et al. 2002).

The twin design makes it possible to distinguish genetic, shared and non-shared influences. The models assume that MZ twins have 100% of their genes in common; the average corresponding figure for DZ twins is 50%. Genetic influence (A) is indicated by a higher intra-class correlation in MZ than in DZ twins. Shared environment (C) is implied if the MZ and DZ twin correlations are similar and statistically significant. If both MZ and DZ twin correlations are small, non-shared environment will have a strong influence. The non-shared environment (E) contributes to differences between family members, including measurement errors. Figure 8 illustrates the relationship between the observed phenotypes in two members of a pair. The genetic correlation is 1.0 in MZ and 0.5 in DZ twin pairs. The shared environment correlation is set at 1.0 in both groups. There is no within-pair correlation set between E terms. Structural equation modeling using the MX computer program (Neale and Maes 2004) was performed to evaluate the quantitative contributions of genetic and environmental (shared and non-shared) components. Parameters for the full ACE model, which includes A (genetic), C (shared environment) and E (non-shared environment), were estimated. Using the raw data maximum likelihood estimation in MX enabled the inclusion of unmatched twin pairs. Bivariate Cholesky models were used, as described in Neale et al (Neale and Maes 2004), in order to examine whether the genetic and environmental effects on the different EDI-2 scores were mediated by BMI. P-values of less than 0.05 were considered to be significant.

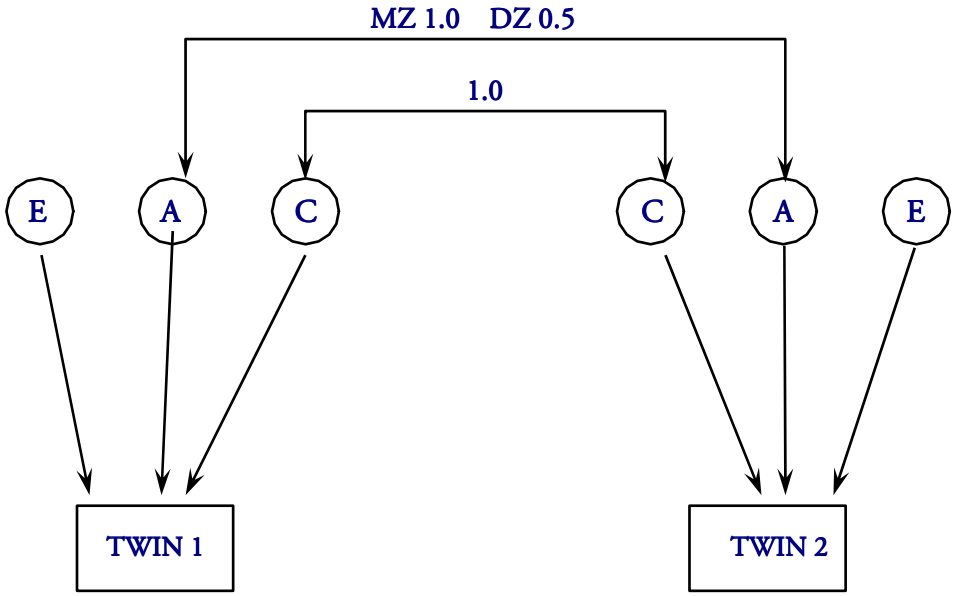


Figure 8 Univariate model of twin data. Additive genetic (A), shared environmental (C) and non-shared environmental (E) influences on the measured variable are shown for both members of the twin pair.

4.7.4 Health Hunters

Standard methods were used to calculate mean values and standard deviations. Two-sample t-tests and ANOVA were used to test the hypothesis of no difference in mean changes between the control and intervention groups. Linear regression models were used to analyze the weight change in all participants in relation to changes in diet, physical activity and selected behaviors. P-values of less than 0.05 were considered to be significant. An intention-to-treat analysis was conducted in which dropouts in the intervention and control groups were assigned the average weight change values observed in the control group.

5 RESULTS AND DISCUSSION

5.1 Papers I and II - H70

5.1.1 Participation

Participation rates were higher in the earlier-born cohorts (85% in 1971) than in the later-born study groups (65% in 2000). Although response rates decreased, they remained higher than in some other studies in elderly populations, e.g. (van't Hof and Burema 1996). However, participation was stable among the two latest-born cohorts (66% in 1992, 65% in 2000). Because participation rates were lower in the two most recent surveys, it was considered important to investigate whether non-participants differed in any measurable ways from participants and the former group's responses were thus compared with those of the latter group. An analysis was conducted comparing all H70 subjects attending the 2000 examination with those who did not attend but did complete the non-respondent questions (47% of all non-participants). This group was found to be similar with respect to self-rated health, history of myocardial infarction, smoking and diabetes, although the unmarried state was significantly under-represented among participating men (Bergh et al. 2003). Nevertheless, increasing non-participation rates must be considered a potential source of bias in observed secular differences.

In 1992/1993, the male sub-sample in the diet examination was small, compared to the other samples. Due to organization and financing problems, the diet examination did not start before the second half of the study. Because the majority of the men were examined in the first part of the study period, only a small number were interviewed about their diet.

Most of the women born 1922 and 1930 are also part of the longitudinal PPSW. Therefore, it is possible that the later examinations captured a more self-selected and healthier cohort, which could theoretically explain some of the apparent dietary improvements. On the other hand, it may be noted that the trends reported in these cohorts are consistent with food disappearance data from the Department of Agriculture (Statistiska Centralbyrån 2006), suggesting that the changes are likely to be real as well as being comparable with those in younger age groups.

Since 1968, 116 women in the 1922 and 1930 cohorts moved away from Göteborg but are still part of the PPSW; most of them live in nearby regions. A comparison between women living outside Göteborg and women in the same cohort living in Göteborg did not reveal any differences in BW,

height, BMI, WC, HC or WHR (see Table 3) and these women thus remain in the H70 analyses.

Table 3 Comparison of women living in Göteborg with those who moved away, mean values.

	Women born 1922			Women born 1930		
	Living in Göteborg n=241	Living outside Göteborg n=51		Living in Göteborg n=195	Living outside Göteborg n=65	
Height (cm)	161.6	162.2	ns	161.7	162.9	ns
BW (kg)	68.4	68.4	ns	70.5	71.8	ns
BMI (kg/m^2)	26.2	26.0	ns	27.0	27.0	ns
WC (cm)	83.6	83.7	ns	87.2	87.6	ns
HC (cm)	101.0	100.9	ns	102.9	103.8	ns
WHR	0.827	0.828	ns	0.847	0.844	ns

5.1.2 Overweight and obesity

5.1.2.1 Prevalence of overweight and obesity

During the last three decades, the prevalence of obesity has increased in 70-year-old men and women in Göteborg. In 2000, 24% of the women and 20% of the men had BMI ≥ 30 kg/m^2 (see Table 4), corresponding to a 50% increase in women and more than a doubling in men over three decades. The BMI trends were detected earlier in men than in women, while secular increases also became apparent in women during the final 8 years. The secular increases in obesity are in general agreement with recent studies of younger Göteborg cohorts (Berg et al. 2005), as well as nationally representative statistics on 65–74-year-old Swedes recorded between 1980 and 1997, although the national survey data are based on self-reported weights and heights (Lissner et al. 2000). The larger increases observed in 70-year-old men, compared to women, are also similar to the secular trends reported among 60–64-year-old Finns between 1972 and 1997. Specifically, BMI increased 1.96 units in Finnish men during this period, compared to 0.32 BMI units in women (Lahti-Koski et al. 2001). The apparent gender difference in secular trends in Nordic populations may also be compared with data from the NHANES surveys in the US, in which obesity increased from 10.5% to 35.8% in 60–70-year-old men and from 23.4% to 39.6% in women of the same age between 1971 and 2000 (Flegal et al. 2002). Thus, the obesity prevalence in men seems to be catching up with rates in women in many parts of the world.

A lower participation rate is always a limitation in studies and the issue of how it might possibly influence the reported prevalence of obesity in the

elderly must be discussed. It has been shown that more health-conscious individuals with higher socioeconomic status are likely to participate which may result in an underestimation of the true obesity prevalence and trend, assuming that they are leaner than individuals with low socioeconomic status (van't Hof and Burema 1996). This may apply to our study, especially considering the differences in BMI between individuals with low and high levels of education. On the other hand, the upward trend in overweight prevalence over time might instead be overestimated if the growing numbers of non-participants among the elderly are the ones who are suffering from diseases resulting in weight loss.

In the 2000 H70 study, unmarried men were underrepresented and the question is whether their diets are likely to differ from those of married men. An analysis comparing the participating married and unmarried men showed that there was no difference in BMI. Furthermore, the unmarried men reported a significantly higher EI, corresponding to a higher intake of most nutrients. Thus, our conclusions that elderly men are consuming nutritionally adequate diets and getting heavier cannot be explained by under representation of single men.

Table 4 Cohort differences in anthropometric parameters among 70-year-old men and women.

Year of birth	1901		1906		1911		1922		1930		p*
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	
Men											
Height (cm)	173	6.6	174	6.1	173	6.2	175	6.2	176	7.1	<.0001
BW (kg)	76.4	11.7	77.8	12.5	78.0	12.6	82.7	11.9	83.1	13.8	<.0001
BMI (kg/m^2)	25.4	3.5	25.8	3.7	26.0	3.7	26.9	3.7	26.9	3.9	<.0001
BMI≥25 (%)	54.6		57.0		61.5		67.5		67.0		<.0001
BMI≥30 (%)	8.5		12.3		11.3		20.6		20.0		<.0001
WC (cm)	90	10	93	10.5	-	-	95	10.1	98	10.9	<.0001
HC (cm)	101	7.2	102	7.6	-	-	103	9.3	102	8.4	0.53
WHR	0.93	0.06	0.92	0.06	-	-	0.93	0.06	0.96	0.06	<.0001
Women											
Height (cm)	160	5.8	161	5.8	160	5.8	162	5.9	162	6.1	<.0001
BW (kg)	66.9	11.4	65.8	10.8	68.1	12.0	68.4	11.7	71.2	12.5	<.0001
BMI (kg/m^2)	26.2	4.2	25.5	4.0	26.5	4.5	26.2	4.2	27.1	4.6	<.0001
BMI≥25 (%)	58.3		51.3		60.2		56.6		64.3		0.02
BMI≥30 (%)	16.6		12.6		19.1		16.2		23.8		.002
WC (cm)	83	9.0	87	11.3	-	-	84	11	88	11.5	.001
HC (cm)	105	9.5	102	8.98	-	-	101	9.5	103	9.8	0.27
WHR	0.81	0.05	0.81	0.06	-	-	0.83	0.06	0.85	0.06	<.0001

*Trends across cohorts

5.1.2.2 Secular trends in WC and WHR

Between 1992 and 2000, BW and BMI in 70-year-old men were stable, while both WC and WHR increased significantly during the same period. These upward trends might indicate a change in body fat distribution towards a larger amount of visceral fat. HC increased less than WC both in men and women, which may be an indication of increasing cardiovascular risk. Previous analyses of earlier-born 70-year-old cohorts described here indicate that a high BMI is protective for total mortality in men, albeit a risk factor for CHD and stroke. WC is also a risk factor for these cardiovascular endpoints in men (Dey and Lissner 2003; Dey et al. 2002). In female H70 cohorts, however, neither BMI nor WC was related to CHD risk. The health implications seem to be much clearer for men than for women. It has been argued that increased abdominal obesity is better reflected by increased WC than by BMI (Molarius and Seidell 1998; Visscher et al. 2001), and that WHR is not a good predictor of overweight (Molarius and Seidell 1998; Molarius et al. 1999). On the other hand, WC has in fact been found to be more closely related to total body fat than to visceral fat in the elderly (Harris et al. 2000).

5.1.2.3 BMI trends stratified by lifestyle factors

After stratifying for the physical activity groups, a significant upward trend in BMI was found across the cohorts in both sexes, but secular increases were significantly smaller in the more active individuals (interaction: birth year × physical inactivity, $p<0.0001$ in men and $p=0.01$ in women) (see Figures 9 and 10). After stratifying for smoking, significant upward trends in BMI were observed across the cohorts in all three smoking groups (smoker, ex-smoker and never-smoker), with the exception of never-smoking men, who displayed a relatively stable mean BMI. Finally, stratification by education indicated a secular upward trend in BMI of similar dimensions both in more and less educated men. Among women, on the other hand, BMI increases were most pronounced in the subgroup with more than basic education in which significant evidence of an interaction was found.

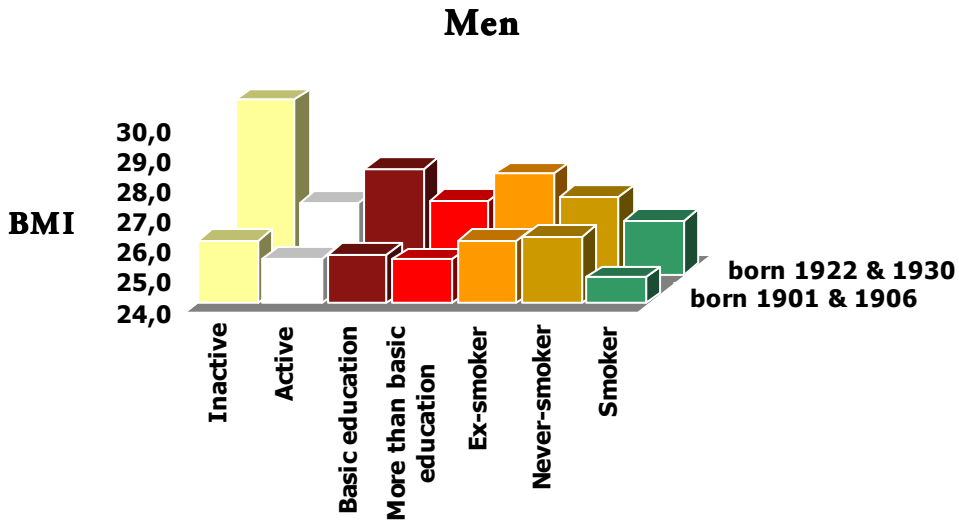


Figure 9 Cohort differences in BMI among 70-year-old men related to physical activity, education and smoking habits. (For simplicity the cohort born 1911 is removed.)

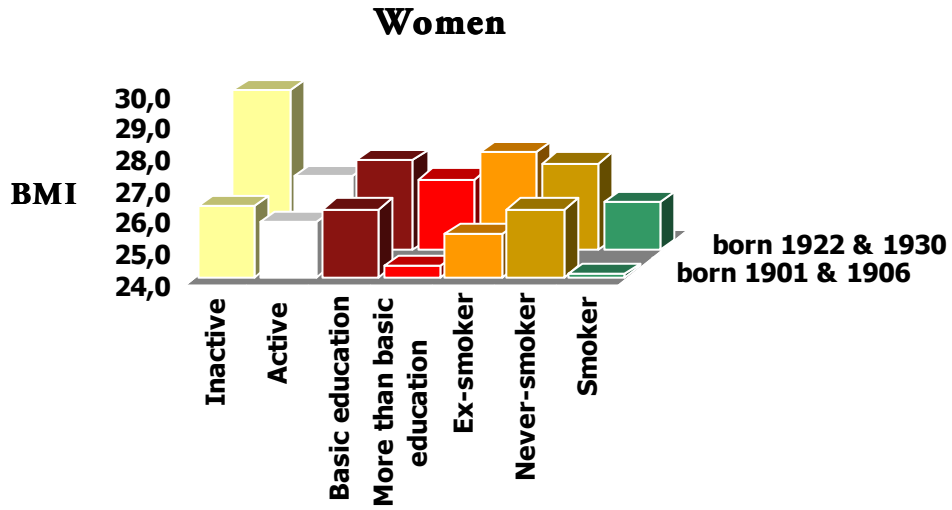


Figure 10 Cohort differences in BMI among 70-year-old women related on physical activity, education and smoking habits. (For simplicity the cohort born 1911 is removed.)

It is encouraging to note that the percentage of sedentary individuals decreased from 18% to 10% in men and from 23% to 8% in women, suggesting that some form of physical activity has increased among the elderly during the last 30 years. However, the higher proportion of physically active

individuals of both sexes did not correspond to an improvement in BMI or WHR during the same time period. BMI did increase faster in the sedentary groups in both sexes, which suggests that regular physical activity may have inhibited the secular trend in BMI in the more active group. In the latest-born male and female cohorts, the mean BMI in the sedentary group exceeded 30, which implies a health risk (WHO 2000). Examining other obesity-related factors indicated, for instance, that the prevalence of smoking decreased in both sexes (for the first time in women) in the two last cohorts and that BMI in the ex-smokers increased most, which could explain some of the recent weight increases in women. In women, the observed effect modification in the educated groups showed clearly that BMI increased much faster in the more educated than in the less educated women. The educational gradient in obesity appeared to be narrowing in women and widening in men, which is a continuation of a trend observed in earlier cohorts (Cabrera et al. 2003). The significant increases in energy intake detected in men may partly explain their large increase in obesity (Eiben et al. 2005).

5.1.2.4 WC trends stratified by physical activity

WC exhibited a significant upward trend across the cohorts in both sexes after stratifying for the physical activity groups (see Figure 11). Secular increases were significantly higher in the inactive male group (interaction: birth year × physical inactivity, $P<0.01$). No interaction could be seen in women ($p= 0.12$). However, the differences in WC between the activity groups in the last cohort in both male and females were highly significant ($p<0.0001$). These results provide more evidence of the importance of physical activity in preventing abdominal fat accumulation in the elderly.

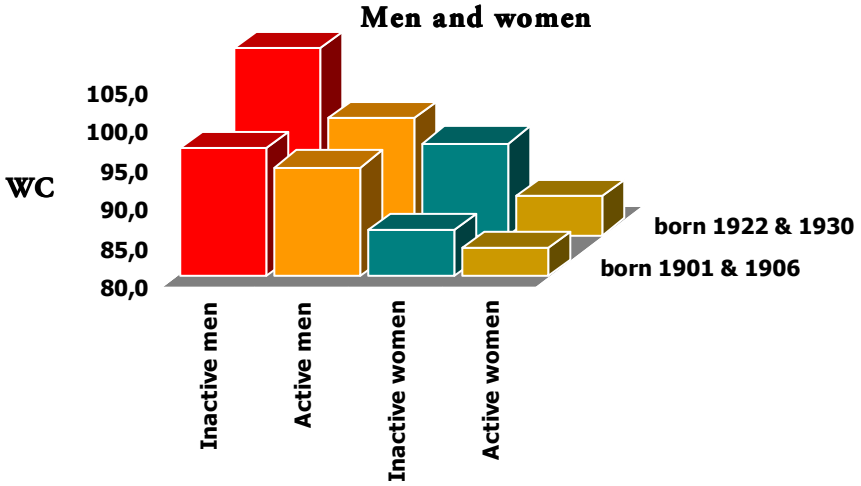


Figure 11 Cohort differences in WC among 70-year-old men and women according to level of physical activity. (For simplicity the cohort born 1911 is removed.)

5.1.2.5 Longitudinal changes in BMI and WHR

Figure 12 shows individual changes in BMI related to age for sub-samples of women born in 1922 and 1930, participants in the prospective study since 1968. Consistent with the total analysis, women born in 1930 had a significantly higher BMI at age 70 than women born in 1922. However, a large fraction of this difference was already apparent when the women were in their mid-40s. Similarly, WHR at age 70 was significantly higher in women born in 1930, compared to the 1922 cohort, and this difference was already present in middle age. This indicates that the differences in BMI observed in 1992 and 2000 had earlier origins (Bengtsson et al. 1979), which may relate to social and environmental conditions in early life.

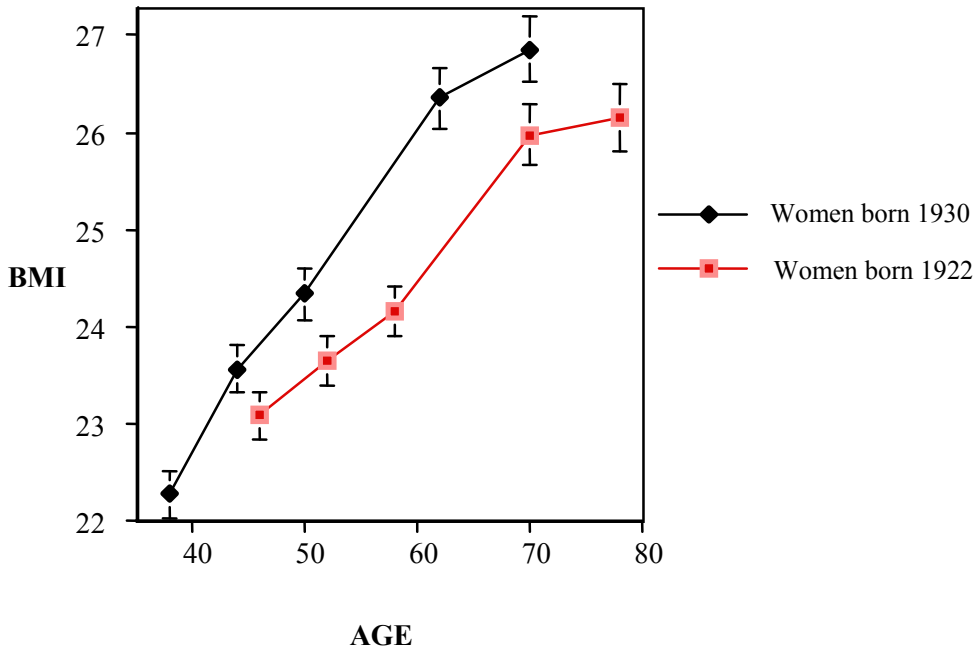


Figure 12 Longitudinal BMI, mean and SE, in females born in 1922 and 1930.

5.1.3 Diet

5.1.3.1 Food consumption trends

Table 5 presents a complete list of secular differences in consumption of selected products by the four cohorts. It highlights the products consumed in increasing amounts throughout the four examinations, items that became

consumed less frequently and those that stayed stable during the observation period.

We observed many secular changes in food selection patterns when comparing the different 70-year-old cohorts. The content of main meals shifted towards more meat, poultry and vegetables, and potatoes were partly exchanged for pasta and rice. Traditional breakfast foods such as porridge, gruel and more refined bread products were replaced with cereals, fruit yoghurt and whole-grain breads. Later-born women and men consumed less high-fat spread ('spread' is defined here as table fat, mostly used on bread) and the intake of low-fat spread, cheese and milk alternatives increased. Later-born cohorts of both sexes ate more fresh fruit as well as less canned fruit and fruit soup than earlier-born cohorts. As the consumption of extra sugar and sweet bakery products decreased, increases could be observed in candy consumption. Finally, although alcohol consumption could not be estimated in the original (1971) survey, large increases in wine consumption were seen between 1981 and 2000 in both sexes. Beer consumption trends seem to have mirrored changes in the availability of stronger beer types in Swedish grocery stores. There was a clear gender difference in the consumption of sugar-sweetened soft drinks. Men increased their consumption by 47% and women decreased their consumption by 36%, both beginning with the same amount in 1971. The consumption of soft drinks increased about 200% in Sweden as a whole from 1980 to 2004, which means that men followed the general trend to a greater extent. In women, soft drink consumption had already decreased in the cohort examined 1981, in which there were no PPSW participants, which might be one explanation of a more health-conscious diet. However, other studies have shown the extent to which women are influenced by health messages, which might be another explanation (Fagerli and Wandel 1999).

Dietary quality has improved in a number of ways, and these findings in the elderly are consistent with national food consumption trends in the general population (Jordbruksverket 2000).

Table 5 Linear trends in amounts of foods (g) consumed daily by different 70-year-old female and male cohorts examined between 1971 and 2000.

	Women				Test of	Men				Test	
	1971	1981	1992	2000	trend	1971	1981	1992	2000	trend	
Spread 40%	-	7	9	11	***	-	5	23	15	***	
Fruit yoghurt	0.3	8	12	29	***	0.2	6	3	17	***	
Cheese >17% fat			See below			30	36	39	43	***	
Cheese <17% fat	4	4	10	18	***	3	3	8	15	***	
Breakfast Cereals	3	5	10	16	***	2	7	16	19	***	
Wholemeal bread	9	41	27	25	***	9	35	38	35	***	i
Meat	51	64	67	61	**	58	79	100	81	***	n
Poultry	8	7	8	12	***	7	6	6	13	***	c
Fish	46	31	52	48	**			See below			r
Pasta	3	6	8	13	***	3	7	12	15	***	e
Rice	5	11	14	17	***	4	7	12	19	***	a
Vegetables, total	54	57	145	146	***	44	47	95	128	***	s
Vegetables, fresh	28	37	96	97	***	22	25	54	79	***	e
Fresh fruit	151	120	176	197	***	127	118	143	171	***	d
Juice	30	37	55	67	***	12	24	33	84	***	
Light beer	16	29	30	34	**	44	75	79	83	***	
Wine	-	11	17	29	***	-	23	35	46	**	
Candy	6	5	9	13	***	6	6	10	17	***	
Soft drinks, syrup			See below			117	99	118	172	*	
Cheese >17% fat	25	28	33	28	0.22			See above			
Fish			See above			57	43	68	57	0.27	s
Liquor	-	-	3	2	0.94	-	12	8	13	0.62	t
Coffee	441	490	508	473	0.34	490	572	526	499	0.90	a
Tea	147	163	127	135	0.23	163	191	137	171	0.95	b
Milk 1.5% fat	-	-	93	104	0.55	-	-	116	124	0.84	l
Milk 0.5% fat	146	117	138	136	0.45	117	171	160	145	0.41	e
White bread			See below			30	40	29	28	0.33	
Soft drinks, syrup	116	69	54	74	*			See above			
Spread ≥ 60% fat	18	16	9	6	***	25	26	11	9	***	
Milk 3.0% fat	204	243	96	77	***	268	307	96	132	***	d
Porridge	60	76	44	22	***	67	69	71	30	***	e
Gruel	25	13	6	3	***	33	26	10	6	***	c
Sweet rye bread	53	29	29	40	*	77	59	48	48	***	r
White bread	26	27	20	17	***			See above			e
Egg	26	20	20	17	***	31	32	25	24	**	a
Potatoes	129	124	92	95	***	202	209	151	134	***	s
Canned fruit	63	64	37	39	***	59	66	34	36	***	e
Beer ≥2.8%	31	8	5	18	*	139	54	39	86	**	d
Buns, cakes	54	51	37	36	***	64	54	46	49	***	
Sugar	13	9	4	3	***	24	20	11	11	***	

*** = p <0.001, ** = p<0.01, * = p<0.05

5.1.3.2 Energy and nutrients

Energy intake increased significantly in men and marginally in women (p=0.06) (see Table 6). Protein intake increased in both sexes while carbohydrate intake only increased in men. There was a non-significant upward trend in fat intake.

Table 6 Energy and nutrient intakes for women and men. Values expressed as mean and sd (standard deviation).

Nutrient	1971		1981		1992		2000		Test of
	mean	sd	mean	sd	mean	sd	mean	sd	trend
Women									
EI/BMR*	1.40	0.4	1.49	0.4	1.41	0.4	1.45	0.4	0.44
Energy, kcal	1819	504	1967	509	1847	435	1931	468	0.06
Protein, g	67	17	71	19	76	18	77	19	<0.0001
Fat, g	75	25	84	26	74	24	76	26	0.39
Carbohydrates, g	217	65	227	65	213	59	226	62	0.26
Alcohol, g**	_	_	2.4	4	3.5	7.1	4.8	6.2	<0.01
Men									
EI/BMR*	1.59	0.39	1.84	0.50	1.67	0.52	1.77	0.48	<0.01
Energy, kcal	2218	530	2553	645	2425	708	2561	651	<0.0001
Protein, g	80	19	86	22	96	25	98	25	<0.0001
Fat, g	92	27	109	35	96	32	101	36	0.056
Carbohydrates, g	264	69	289	77	277	99	290	83	<0.01
Alcohol, g**	_	_	8.9	12.6	8.9	11.7	13.1	15.5	<0.0001

*Ratio of reported energy intake to predicted basal metabolic rate
**Trends in alcohol from wine, beer and liquor based on data from 1981 – 2000 only. Alcohol from beer in 1971 (mean 0.3g) is included in energy and where applicable nutrient group means.

In the 2000 survey, more than 65% of the subjects had intakes of vitamin C, riboflavin, thiamine, calcium, iron and vitamin A above the Nordic Nutrition Recommendations levels (Sandström et al. 1996). None of the subjects failed to achieve the recommended safe intake of vitamin C, potassium or thiamine. Less than 3% had intakes of vitamin D, calcium, iron, riboflavin (mostly men) and vitamin A (mostly women) below the recommended safe levels. In general, the majority of the 70-year-olds examined in 2000 consumed nutritionally adequate diets.

5.1.4 Methodological considerations

5.1.4.1 Paper I

A limitation of this research, common to many repeated cross-sectional studies, involves changes in procedures. The examiners at each investigation were trained similarly throughout the years, with the aim of collecting comparable anthropometric data at all examinations. However, slightly different questions were used on each occasion, concerning physical activity, smoking and education. In order to compare these variables across cohorts, it was necessary to reduce the number of categories which may have caused loss of information (Dey et al. 2001b).

It must be emphasized that all of the H70 secular trends reflect comparisons between different examined birth cohorts. Cohort differences may be strongly related to nutritional status; particularly height might be varyingly affected by childhood nutrition and living conditions, environmental factors and social factors throughout life (Dey et al. 2001b). The differences in BMI between the two latest-born female cohorts did not appear for the first time within the final 8 years. Inspection of longitudinal data in sub-samples of women born in 1922 and 1930 reveals consistent differences in BMI and WHR which were already present when the women were in their mid-40s. The estimated average life expectancy of Swedes born in 1930 is about 6 years longer than that of Swedes born in 1901 (Statistiska Centralbyrån 2006), which may imply that the later-born cohort is biologically younger than the earlier-born cohort. It may even be speculated that the heavier BWs reflect a new generation of elderly individuals with a longer survival prognosis. Longitudinal data are necessary to understand the nature of these changes.

5.1.4.2 Paper II

The main challenge of this study involves using the same dietary instrument in cohorts examined across three decades. It is obvious that the availability of foodstuffs in shops and cooking methods may change in many ways over such long intervals, reflecting new consumption patterns and practices. Thus, the instrument had to evolve over time, both in terms of listed food choices and assumed preparation methods and recipes (Rothenberg et al. 1996). Whether the instrument is equally valid throughout the years remains an important methodological question, one we were only partially able to assess. Systematic trends were observed in the EI/BMR ratio among men, who were also getting heavier with similar activity levels. Among women, the ratio was stable but there was some indication of changes in physical activity levels, suggesting that the ratio should have increased. Therefore, we cannot rule out the possibility of a decreasing degree of completeness in reported

intake for women, even in the situation with an apparently stable EI/BMR ratio. For men, the increases in EI/BMR are consistent with a strong secular increase in relative weight during that period. The estimated EI/BMR values in this study are a bit lower that those obtained with the doubly-labelled water method at that age and BMI (Roberts and Dallal 2005).

Another set of problems arises from changes in the nutrient database which has been improved by new analytical techniques, making nutrient values in earlier databases obsolete. We used the 2000 database to decrease the probability that observed changes in dietary intake are due to changes in the database. This creates several problems. First, 'old' foods must be added to the new database and this can re-introduce errors. The new database cannot be used for certain nutrients such as iron, due to changes in fortification policies. Finally, using the same database masks real changes in nutrient composition for the same foods. However, we concluded that, on the whole, the benefits of using the new database outweigh the disadvantages.

When making comparisons between food intake data from different birth cohorts over long periods of time, it is necessary to achieve high precision in the standardization of nutrient databases and to analyze and express intake data so that observed differences are as unbiased as possible. Despite a number of important limitations, the present study is unique in its ability to monitor population-based cohorts over three decades using a comparable dietary method.

Finally, the large number of foods analyzed increases the risk of statistical mass significance. However, our data showed high levels of significance in most of the analyses which reduces the risk of chance findings.

5.2 Paper III - TCHAD

5.2.1 Anthropometry

Descriptive statistics on the anthropometric data from the sample are presented in Table 7. The overall percentage for overweight (BMI ≥ 25) in girls and boys was 6.7% and 10.3%, respectively, and for obesity (BMI ≥ 30) 1.5% and 1.6%, respectively. Swedish measured data has been presented for girls and boys (non-twins) of similar ages and at about the same time (Neovius et al. 2005). The authors reported higher mean values for weight, height and BMI compared to ours (Neovius et al. 2004). There are several potential explanations for this discrepancy. Self-reported weight and BMI are often lower than the same parameters when measured (Nieto-Garcia et al. 1990). Furthermore, another explanation might be that twins are smaller at

birth, both in terms of weight (30% deficit) and height (17% deficit), although they reach the singleton norm at age 8, according to Wilson (Wilson 1979). An earlier analysis of a twin sample of the same age revealed that the discrepancy in size persisted until the end of puberty, especially in males (Pietilainen et al. 1999).

Table 7 Self-reported anthropometric data from the study population, divided in MZ and DZ, and measured anthropometric data in Swedish adolescents (Neovius et al. 2004). Values are expressed as means.

	Girls			Boys		
	MZ	DZ	Neovius et al	MZ	DZ	Neovius et al
Age, years	16.7	16.7	16.8	16.7	16.7	16.9
BW, kg	55.2	56.2	59.7	64.5	65.3	68.7
Height, cm	164.8	165.9	167	176.6	176.7	180
BMI, kg/m^2	20.4	20.5	21.5	20.7	20.9	21.1
WC, cm	70.3	71.9	71.4	76.4	77.7	75.4
HC, cm	92.4	93.8	92.3	93.0	93.8	92.9
BMI ≥ 25	7.4%	5.7%	-	9.6%	11.2%	-
BMI ≥ 30	1.5%	1.6%	-	2.5%	0.6%	-

5.2.2 Genetic and environmental influences on BW, BMI and WC

The results of the univariate structural equation models for BW, BMI and WC are shown in Table 8. The ACE model estimated major genetic influences for all anthropometric parameters in girls (56% for BW, 60% for BMI and 72% for WC). Shared environmental effects could be detected for BW and BMI (28% and 24%, respectively), and the estimated influences of non-shared environment were 16% for BW, 15% for BMI and 28% for WC. In boys, genetic effects on the anthropometric parameters were found to account for 72% of the variance in BW, 74% in BMI and 67% in WC. No significant effects of the shared environment were found in boys; influences of the non-shared environment accounted for 17% for BW, 26% for BMI and 33% for WC.

Table 8 Estimates of genetic and environmental effects based on ACE model-fitting, including additive genetics (A), shared environment (C), and non-shared environment (E), for BW, BMI and WC in girls and boys.

	Parameter estimates (95%CI)		
	A	C	E
Girls			
BW	.56 (.38 -.78)	.28 (.06 - .46)	.16 (.13 - .20)
BMI	.60 (.42 - .83)	.24 (.01- .43)	.15 (.12 - .19)
WC	.72 (.54 - .78)	.00 (0 - .17)	.28 (.22 - .35)
Boys			
BW	.72 (.51 - .86)	.11 (0 - .31)	.17 (.13 - .21)
BMI	.74 (.51 - .79)	.00 (0 - .22)	.26 (.21 - .32)
WC	.67 (.40 -.74)	.00 (0 - .24)	.33 (.33 - .26)

In summary, the ACE model suggested a high genetic component in body weight and weight-for-height in boys and girls. These findings are consistent with many studies in the literature. However, differences have been reported in the magnitude of the genetic influences on BMI, ranging from 45% to 81% in young women and men aged 20-29 (Schousboe et al. 2003). The few available studies on adolescents show genetic influences on BMI in about the same range as in adults (Faith et al. 1999; Jacobson and Rowe 1998). The corresponding estimates in the current study were 60% and 74% for girls and boys, respectively. The relatively low estimates for genetic influences in girls, compared to boys, can be explained by shared environmental influences for BW and BMI in girls. Most studies do not find shared environmental influences with twin models including both MZ and DZ twins, which may partly be due to insufficient power. Segal and Allison included virtual twins (same-age unrelated siblings reared together since infancy) in their analysis and could detect shared environment influences in BMI of the same magnitude as we found in girls (Segal and Allison 2002). Jacobson and Rove found results similar to ours as well; they detected shared environmental influences in female, but not in male, adolescents (Jacobson and Rowe 1998). They explain the difference between males and females with the larger interest taken by females in weight control behaviors resulting in stronger environmental influences. We documented this greater female interest in weight control behaviors in our study. Shared environmental influences are more easily detected in childhood and decrease during puberty as adolescents become more and more independent.

5.2.3 Prevalence of DT, B and BD

The prevalence of DT and BD was many times higher in girls (21% and 29%, respectively) than in boys (3% and 7%, respectively); however, the B diagnosis was equally prevalent in both sexes (2%). The large differences in

the prevalence of diagnosed DT and BD between girls and boys are in line with other studies (Keski-Rahkonen et al. 2005; Kjelsas et al. 2004). However, the similar prevalence of B in girls and boys was unexpected. Most of the questions in the EDI-2 scale for B (see Table 2) deal with binge eating, which raises the possibility that it may be normal for a 15-17-year-old growing boy to consume great amounts of food. The use of EDI-2 scales in men is not very common, since they focus more on conventional women's concerns such as body weight and shape, while the areas to which boys pay attention, e.g. muscularity and stature, are not mentioned (McCabe et al. 2002). Consequently, the validity of EDI-2 is limited in men (Spillane et al. 2004). The subscales are possibly not suited to boys' eating behavior, and the results must thus be regarded with caution. Further studies aimed at developing tools to measure body image and disordered eating in males are needed.

5.2.4 Genetic and environmental influences on EDI-2 subscales scores

The results of the univariate model-fitting analyses for all EDI-2 scores are presented in Table 9. In girls, both genetic and environmental influences were important for EDI-2 scores. The low prevalence among boys indicates limited power, and the model could thus not elucidate whether genetic and/or shared environmental effects were responsible for the familiality, as shown by the confidence intervals.

Table 9 Estimates of genetic and environmental effects based on ACE model-fitting, including additive genetics (A), shared environment (C) and non-shared environment (E), for DT, B and BD in girls and boys.

	Parameter estimates (95%CI)		
	A	C	E
Girls			
DT score	.41 (.16 - .67)	.21 (0 - .43)	.38 (.31 - .46)
B score	.43 (.11 - .56)	.04 (0 - .30)	.54 (.44 - .64)
BD score	.58 (.33 - .72)	.09 (0 - .31)	.34 (.27 - .41)
Boys			
DT score	.23 (0 - .53)	.30 (.04 - .52)	.47 (.38 - .58)
B score	.22 (0 - .51)	.19 (0 - .42)	.59 (.48 - .71)
BD score	.37 (.07 - .61)	.16 (0 - .41)	.47 (.38 - .57)

To our knowledge, there is only one study analyzing the genetic and environmental influences on EDI-2 sub-scores in males. The results of Keski-Rahkonen et al's study of 22-27 year old Finish men differ from ours (Keski-Rahkonen et al. 2005). They found that more than 80% of the influences on DT and BD were from the shared environment while the genetic and the non-shared environmental influences were of little consequence. In contrast,

our results showed strong influences of the non-shared environment while the shared environment accounted for 30% of the influences on DT and was not significant for BD. These contrasting results may be explained by the different age groups (the Finnish twins were an average of eight years older), which might be a critical factor for the dimensions of shared environment influence. However, the pattern of influence on girls was about the same in the Finnish and the Swedish twins, further confirmed by Klump´s results from Minnesota in the same age group (Klump et al. 2000). In summary, both genetic and environmental influences are important for the development of body image and disordered eating in our female sample. In the case of the male sample, our analyses did not distinguish between the genetic and shared environmental influences, especially for the EDI-2 B score, for which it was not possible to conclusively determine whether familial factors arise from genetic or shared environmental influences. The influences of the non-shared environment seem to be most important for the EDI-2 B score in boys; these influences include experiences unique to the individual and not shared by his co-twin, e.g. different peer groups, as well as differential treatment within the family (but also measurement error).

5.2.5 Associations between BMI and EDI-2 subscales

The Pearson correlation coefficients between BMI and the EDI-2 scores for DT and BD were all significant. In contrast, the correlations between BMI and B were weak or non-significant, suggesting less power for carrying out bivariate analyses for this score. As a result of the low correlations between BMI and the B score, further bivariate analyses were not performed for B.

In girls, genetic influences were the most important factors in explaining the relationship between BMI and the EDI-2 DT and BD scores (see Figure 13). Thirteen percent and 25% were contributed from genetic effects in common with BMI to DT and BD, respectively. Genetic influences on DT in boys were exclusively in common with BMI (21%), while the majority of the genetic influences on BD were unique to BD, and only a smaller part (6%) in common with BMI. Influences of the shared environment in common to BMI could be seen in boys (31% for DT and 16% for BD). Non-shared environmental effects on EDI-2 scores in common with BMI were not found or negligible in both sexes.

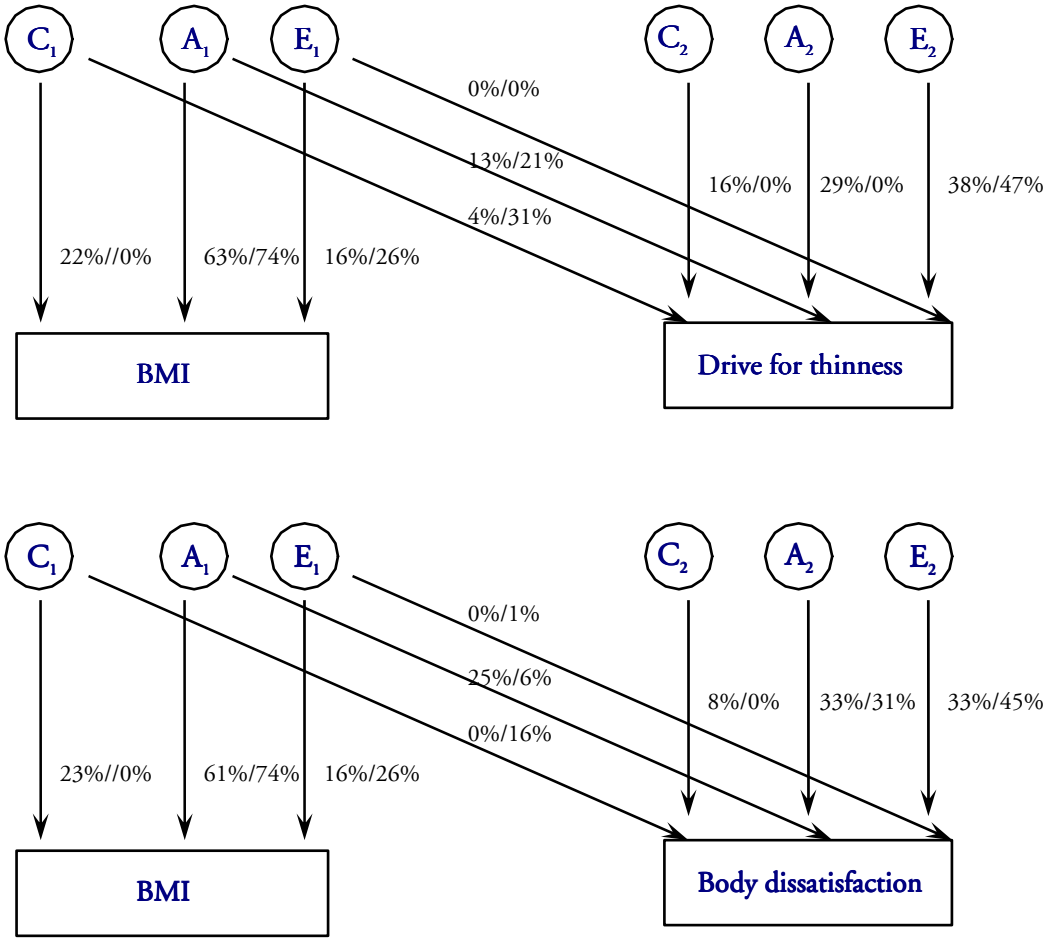


Figure 13 Bivariate analysis of twin data. The model estimates parameters unique to BMI and the EDI-2 DT and BD scores and those that are in common with BMI and the EDI-2 DT and BD scores in girls/boys.

For both DT and BD, about 75% of the genetic and environmental variance in girls was specific to the scores and independent of BMI. The relationship between EDI-2 scores and BMI in boys showed different patterns for the different scores. A substantial proportion of both genetic and environmental variances for the DT score in boys were in common with BMI. The fact that the DT score is driven by BMI seems to be obvious and requires no further explanation. The magnitude of the associations between BMI and the BD score in boys is modest and due to shared environment. Overall BMI only explains a minor part of the estimates influencing the different EDI-2 scores, except DT in boys.

Some studies have shown that unhealthy weight-control behaviors increase the risk of weight gain (Neumark-Sztainer et al. 2006a). However, in the current study, the estimates for genetic and environmental influences on body image and disordered eating were in common with BMI to a lesser extent. Longitudinal studies might answer the question of whether weight problems influence body image and disordered eating or vice versa.

One limitation of this study is that no gene-environment interactions have been considered. Environmental influences on individuals may affect their expression of genotype, while different genetic influences can result in different reaction to similar environmental occurrences.

The strength of this study is the use of the nationwide twin registry, covering all of Sweden, and the high response rate which minimizes biases associated with selected twin samples.

5.3 Paper IV – Health Hunters

5.3.1 Subjects

Baseline examinations were conducted on 40 women. Follow-up examinations were carried out on 14 women in the intervention group and 16 in the control group. The causes for dropout were pregnancy (6), moving out of Göteborg (2) and other reasons (2). The small sample size is the major limitation of this study. The study was originally designed to include a larger number of subjects but this plan was altered due to circumstances. The mean duration of follow-up was 13.7 and 13.5 months in the intervention and control groups, respectively (n.s.). A follow-up period exceeding one year would have been desirable, although extending the observation period would probably have resulted in even greater dropout from pregnancies and relocation.

5.3.2 Anthropometry

When the changes occurring among women in the intervention and control groups were compared, significant differences were found for BW (see Table 10). This significance remained in a re-analysis controlling for baseline BW as well as age and smoking. Additionally, changes in BW were analyzed with the intention-to-treat principle, in which the worst case was assumed for the dropouts; i.e. that they would increase in BW by the same amount as the control group. Results showed that the intervention group still had a significant BW change, compared to the control group. As a consequence of the wide range of changes in BW, which may have indicated heavy influences of

outlying values, non-parametric analyses were carried out with consistent results.

During one year, the BW in the control group increased by 2.6 kg, almost as much as BW decreased in the intervention group, an alarming situation since this weight gain may continue in the absence of an intervention. It is urgent to start prevention programs in high-risk groups as early as possible.

Significant effects were also found for WC and WHR, when the intervention and control groups were compared (see Table 10). Although changes in body composition were not statistically significant, the intervention tended to be associated with improved body composition.

Table 10 Mean values and standard errors (SE) in changes in BW, BMI, WC, HC, WHR and body fat (BF) (%) for completers.

Changes in	Intervention n=14		Control n=16		p*
	mean	SE	mean	SE	
BW, kg	-3.2	2.0	2.6	1.9	0.046
BMI (kg/m^2)	-1.3	0.75	0.9	0.7	0.046
WC, cm	-7.0	2.7	-3.0	2.5	0.011
HC, cm	-2.7	1.7	0.9	1.6	0.138
WHR	-0.05	0.02	0.02	0.02	0.011
BF, %	-3.0	1.4	0.9	1.4	0.063

* Comparison of parameter changes in intervention and control groups

5.3.3 Lifestyle factors

The intervention resulted in increased physical activity in the intervention group, as reported by participants, but this was not confirmed by more objective measures of fitness (see Table 11). However, both fitness measures were significantly associated with concurrent weight loss in the two treatment groups combined. It should be mentioned that self-reported physical activity was significantly associated with both VO2 max and time spent on the treadmill (not shown), suggesting that these three variables contain some of the same information. Thus, on the whole, these results are fairly consistent with the conclusion that the physical activity component of the intervention worked as intended. However, we cannot exclude the possibility that part of the apparent intervention effect was a consequence of subjects' over-reporting their compliance.

After one year, smoking was stable in the intervention group while it tended to increase in the control group, although the difference was not significant

(see Table 11). This could have resulted in an underestimation of the weight change effect.

Table 11 Mean values and standard errors (SE) in changes in diet, physical activity and smoking for completers.

Changes in	Intervention n=14		Control n=16		p*
	mean	SE	mean	SE	
Physical activity points	1464	96	200	383	0.030
Time on the treadmill, min	0.7	0.4	-0.3	0.4	0.080
Watching TV, hours	-5.0	1.8	-0.1	1.9	0.072
Energy intake, kcal	-259	191	110	178	0.169
Fat intake, E%	-1.3	1.4	-0.1	1.3	0.524
Carbohydrate intake, E%	0.5	1.3	0.4	1.2	0.938
Protein intake, E%	0.8	0.6	-0.3	0.6	0.192
Fiber, g/1000 kcal	0.6	0.7	0.4	0.7	0.838
Smoking after one year	21%		44%		0.2

* Comparison of parameter changes in intervention and control groups

The results of the dietary intervention were somewhat variable. There was no statistically significant intervention effect on any of the studied dietary indicators (see Table 11). Presumably, the semi-quantitative food frequency questionnaire was too crude to detect changes on the individual level, which would, on the other hand, probably have been observable if doubly-labelled water methods had been used. However, analysis of concurrent dietary changes in relation to weight change yielded strong associations in both intervention groups combined (see Table 12). Specifically, there was a significant positive association between weight loss and decreased fat intake and even stronger inverse associations with protein and fiber intake. These results are in line with current knowledge from experimental (Skov et al. 1999) and intervention (Sherwood et al. 2000) research. Controlling for treatment group had no major effect on the associations between behavioral and weight changes.

Table 12 Changes in BW in relation to simultaneous changes in diet, physical activity and fitness.

Δ Body weight (kg) (n=30)	ß coeff	SE ß	p	ß coeff*	SE ß*	p*
Δ Physical activity points	-51.2	36.9	0.177	-25.6	37.9	0.504
Δ Time on treadmill	-0.10	0.03	0.002	-0.09	0.03	0.009
Δ VO2	-0.25	0.12	0.049	-0.26	0.13	0.058
Δ Energy intake, kcal	26,2	16,6	0.126	20.3	17.9	0.269
Δ Fat intake, E%	0.26	0.11	0.024	0.27	0.12	0.033
Δ Carbohydrate intake, E%	-0.08	0.12	0.485	-0.09	0.13	0.48
Δ Protein intake, E%	-0.15	0.05	0.002	-0.15	0.05	0.007
Δ Fiber intake, g/1000 kcal	-0.18	0.06	0.002	-0.21	0.06	0.002

* adjusted for intervention vs. control group

5.3.4 Comparisons with the literature

This is, to our knowledge, a unique study of obesity prevention in young adult high-risk individuals with a familial predisposition to obesity. Most previous high-risk studies have been conducted in children (Alexandrov et al. 1992; Caballero et al. 2003; Epstein et al. 2001) and many have not yielded positive intervention effects. Weight gain prevention studies with targeted adult groups such as pregnant (Polley et al. 2002) and menopausal women (Kuller et al. 2001) have shown significant results, providing further evidence that interventions can be effective in high-risk female groups. A number of previous obesity prevention studies were directed to whole populations or groups. In 1988 Foster and Jeffrey reported successful weight gain prevention in their study of individuals who had been screened for cardiovascular risk factors in the Minnesota Heart Health Program (Forster et al. 1988). About 219 men and women were enrolled in the study and randomly assigned to treatment or control. After one year BW had decreased one kg in the treatment group, while BW in the control group was almost stable. The treatment method was similar to Health Hunters in some ways: the intervention group received regular newsletters and took a four-session educational course, but there was no individual contact. In other prevention studies published in the 1990s or earlier, the primary purpose was to prevent cardiovascular disease and one of the secondary aims was to prevent obesity. The majority of these studies were directed at whole populations. Most studies succeeded in modifying risk factors such as smoking, high blood pressure and high cholesterol (Brannstrom et al. 1993; Jeffery et al. 1995; Taylor et al. 1991; Tudor-Smith et al. 1998; Vartiainen et al. 1994) but not in preventing weight gain. During recent years more and more studies have been published reporting positive effects on weight gain prevention in different populations (Andersen et al. 2002; Engberg et al. 2002; Lingfors et al. 2003;

Lupton et al. 2002; Peel and Booth 2001). Nonetheless, about half of the published studies cannot show any intervention effect on weight, which demonstrates the difficulties of preventing weight gain (Cook et al. 2001; Lamb et al. 2002; Miller et al. 2001; Rosamond et al. 2000).

6 SUMMARY AND CONCLUSIONS

In summary, this thesis has highlighted selected aspects of the obesity epidemic in the young and the elderly:

Elderly populations

- During three decades, the prevalence of overweight and obesity has increased.
- BMI increased in all lifestyle groups but the physically active had the lowest increases.
- Many secular changes in food selection patterns have been occurred and diets are generally nutritionally adequate.

Younger populations

- Genetic factors had a major effect on anthropometric parameters.
- Environmental factors were most important for body image and disordered eating.
- Young women with severely obese parents constitute a high-risk group for weight gain.
- The Health Hunters prevention program succeeded in preventing weight gain in young high-risk women with a familial predisposition to obesity.

In these studies research activities and prevention initiatives have drawn attention to elderly and younger age groups. Our results indicate that it is important to focus on body image in the young, while diet and physical activity are relevant for all age groups. Working with prioritized groups is one part of managing the obesity epidemic.

7 IMPLICATIONS

When it comes to the implications of our results in the wider context of obesity prevention in society, I will move from the strictly research level to a more personal and speculative level.

In 1997 the opening lecture of the European Conference on Obesity in Dublin was about prevention of obesity, an unusual and challenging topic at that time. For us, just starting the preparatory work for a prevention study, it was a great support to know that we were not alone on that track. Almost ten years later, ministers and their national delegates from 53 member states of the WHO European Region came together for the WHO European Ministerial Conference on Counteracting Obesity in Istanbul, Turkey. They adopted the European Charter on Counteracting Obesity, recognizing the importance of immediate action. Even if this step was taken late, considering the dimensions of the obesity problem, it is still very important, because now the issue has been raised from the individual level to the political level. A paradigm shift has been achieved so that obesity is no longer considered an individual problem, i.e. everyone must blame themselves for gaining weight, but rather a "normal physiology within a pathological environment" (Egger and Swinburn 1997). It will require strong leadership on the part of governments and broad cross-sector actions to reverse one of the biggest public health threats of the 21st century; all parts of society must be involved.

What can this thesis contribute to addressing the huge challenge society faces? When it comes to interventions to prevent overweight and obesity it is always necessary to begin on a smaller scale in order to test different strategies. Our study on high-risk young adults resulted in different important outcomes. First of all, we now have a complete program for weight gain prevention. The annual costs of the Health Hunters program have been roughly estimated to about 3 h of work for a dietician to prevent or decrease 1 kg BW. This calculation uses an average weight gain of 2.6 kg as a reference. At present, it is feasible to continue the obesity prevention program on a larger scale, at a lower economical level, since the program's routines are established. The costs for prevention must be weighed against the health consequences of obesity later in life. The efficacy of the Health Hunters program has been shown in a randomized controlled trial and it is important to study its effectiveness in a community-implemented trial. It could easily be implemented in other settings, as has already been undertaken. We developed the program primarily for a high-risk group and the participants were very self-selected but now, considering the increasing prevalence of obesity, there are increasing numbers of young adults with obese parents. In the US about

30% of adults are obese, which means about every third child could qualify for our high-risk study. This transforms our definition of high risk into a normal situation for young adults.

Another important finding in our study is the poor prognosis for young, adults with obese parents, who are not offered help. They are already generally overweight, and some are obese, but the most alarming observation is the speed of the weight gain. The control group gained an average of more than 3 kg, in one case 10 kg, in one year. By increasing their BW, they are increasing their risk of co-morbidities. Our results showed that this scenario can be changed with appropriate intervention.

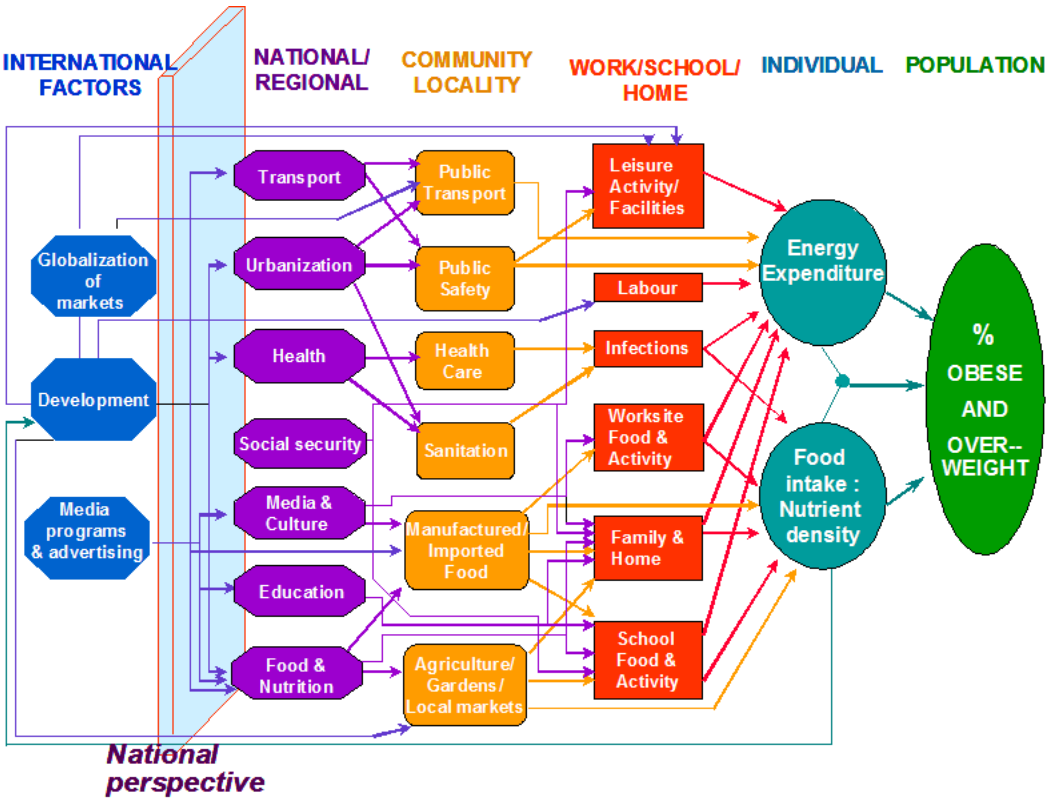
Furthermore, this thesis has confirmed that although BW, BMI and WC are to a large extent inherited, the increasing prevalence of overweight and obesity must be an indication of the environment interacting with genetics. Genetic influences largely determine whether a person can become obese but it is the environment that determines whether such a person does become obese, and the extent of that obesity. Awareness of individuals´ susceptibility to environmental factors should be heightened. Identifying individuals at risk of obesity is one approach, but it might be simpler to intensify the current efforts to motivate people to exercise and eat healthily, regardless of genetic susceptibility and environmental exposure. If the high-risk approach is taken, offspring of individuals with obesity and/or eating disorders could be a target group since they are at greater risk than the normal population. Focusing on environmental influences is, of course, equally important when developing prevention programs. The familial environment is as important as other parts of the living environment such as schools, friends, media etc. Preventive actions should focus on increasing self-esteem in children and young adults. Parents and teachers must discuss the ideal of beauty presented by the media with their children, and they should discourage dieting. These efforts should be combined with the provision of a healthy environment with good, nutritious food and entertaining physical activities, feasible steps toward preventing eating disorders and overweight.

The elderly population is very much part of the obesity epidemic, as has been shown in this thesis. In addition to the health implications of obesity in the elderly, which are as yet incompletely elucidated and thus the object of focus for future gerontological research, the accumulation of abdominal fat increases the risk of morbidity and mortality. The elderly population is clearly a group to which attention should be devoted in terms of prevention. “After work” prevention could start right after retirement; retirees would benefit from increasing physical activity which has been shown to prevent both abdominal fat accumulation and decrease weight gain speed. Regarding diet, we have shown that most of the elderly consume nutritionally adequate

diets, which enables prevention programs to focus mainly on physical activity. However, age-specific diet recommendations should also be considered, according to the existing evidence.

In conclusion, although our studies were small and limited, the results can be used in an ecological approach. We focused on younger and elderly populations but we must clearly reach the middle-aged as well as very young individuals. Obesity has a multifactorial etiology, requiring a multidimensional approach as described in the "European Charter on counteracting obesity". Prevention must take place at all levels of society (see Figure 14) and involve stakeholders such as parents, teachers, medical staff, health administrators, food producers and processors, retailers and caterers, advertisers and the media, recreation and sport planners, urban architects, city planners, politicians and legislators.

Figure 14 Societal policies and processes influencing the population prevalence of obesity. Source: (Kumanyika et al. 2002)



Obesity is one of the most interesting research fields, one that is deeply rooted in reality. Unfortunately, we are in a hurry; the epidemic has been called a "runaway weight gain train: too many accelerators, not enough brakes" (Swinburn and Egger 2004). Overweight and obesity are issues with many aspects; we only could focus on a few in this thesis which nevertheless hopefully contributed to the understanding and knowledge of some accelerators and some brakes.

7.1 Future perspective

What next? The implementation of a multi-center obesity prevention trial in pre-schools and primary schools in western Sweden will represent further steps toward prevention. The University of Göteborg will be involved in the integrated project (IP) "Identification and Prevention of Dietary and Lifestyle-induced Health Effects In Children and Infants" ("IDEFICS"), within the EU Commission's 6th framework program, which started in September 2006 and will continue for 5 years (Ahrens et al. 2006; Bammann et al. 2006). The strategic objectives are to increase knowledge of the health effects of changes in diet, social environment and lifestyle in infants and children and to develop, implement and validate specific intervention approaches in order to reduce the prevalence of diet- and lifestyle-related diseases and disorders in Europe. Surveys and interventions will be carried out and implemented in 16 000 children (aged 2-10) in eight European countries. The overall intervention program will consist of modules operating at different levels: communities (including environmental, societal and political dimensions), pre-schools and primary schools (including educational programs, catering services, and school environment modifications), households and individuals. The main messages will focus on diet and physical activity. The intervention modules will be developed according to an ecological model of health behavior in which processes influencing lifestyle take place simultaneously at different and multi-dimensional levels. Environmental, cultural, social, psychological, economical and other elements play a role and interact with each other in this context. The content of the intervention itself is currently under development by the IDEFICS planning team. An intervention and survey protocol will be developed centrally and adapted to each country's language, food culture and other local circumstances. The intervention and associated survey will be administered by the Department of Pediatrics and the Department of Public Health and Community Medicine, Sahlgrenska Academy. I will serve as the intervention program manager in the Swedish arm of the project.

8 ACKNOWLEDGEMENTS

I wish to express my sincere gratitude to all who have contributed to this thesis in different ways, especially to:

Lauren, the best imaginable supervisor. For providing me with an excellent possibility to conduct research, always encouraging me, pushing me forward gently and respectfully, overlooking my faults and bringing out my strengths, for leading and guiding me into the exciting world of research, epidemiology and statistics. Mostly I appreciate your never- ending great ideas.

Paul, always there to help me out of the jungle of MX statistics. Without you I would have been devoured by the matrices.

Debu, for being such a supportive and generous co-worker, and for always sharing your knowledge.

Elisabet R, for providing constantly support during my excursions into the diet database and for your invaluable contributions and comments.

Cissi, my colleague and friend, for all our discussions about the purposes of research and life. Thank you for great collaboration and, most important, for the laughter!

Valter, thank you for great statistical support and nice music in the late evening working hours.

Cilla and Bengt, for welcoming me to the department and for your constant support, important comments and great interest in my research. Primary Health Care became a second home for me.

Calle, for generously sharing your great knowledge and for your encouragement, both when it came to research and running long-distance race.

Bertil, for giving me the opportunity to work with all these marvelous and inspiring 70-year-olds.

Kristina, Maud and Jenny, for the fantastic collaboration in the Health Hunters study. Group work has never been more effective and fun.

Lolo, our PhD mom, spoiling us with your warmth, care, accuracy and great knowledge of every detail.

Agneta, Ann, Anna, Bernhard, Bertil, Christina, Claudia, Dag, Dimitri, Dorota, Eja, Elisabet A, Elisabet B, Elisabeth O, Elisabeth S, Gunilla, Helen, Jörgen, Karin, Karolina, Kate, Kerstin L, Kerstin R, Kirsten, Kristian, Lena, Leyla, Lilian, Lolo, Madlen, Mats, Peter, Staffan, Tine, Vibeke, Östen and the colleagues from Social Medicine, for all the productive discussions both at research meetings and at the coffee table, for all the help with administrative matters, for providing me with such a enjoyable working atmosphere and, most important, such a good time.

The staff at the Department of Body Composition and Metabolism, Health Care Research Unit, Osteoporosis Center and Metabolic Laboratory, for performing all the examinations and being helpful and co-operative with such kindness.

NEON, BODIS and colleagues at Clinical Nutrition for all stimulating discussions.

All participants in the H70, KVUS, Twin Study and, last but definitely not least Health Hunters. Meeting participants was always a great pleasure, coming down from the research sky to the reality ground and understanding why I'm doing research.

Joy, for highly professional and careful language revision.

Monica, for your creative help in the Health Hunters study and for saving my back from getting stiff every Tuesday evening.

Sintra, my dear friend and colleague, following each other through our studies in nutrition and studies in life. Thank you for your permanent invaluable support and being there whenever I need you in what matter it may be.

Conchi, for the professional guidance in graphic design and most for being my friend.

Elsie, thank you for teaching me Swedish food culture, which is an important condition for nutrition research in Sweden.

Alex , Anita, Betina, Bo Stefan, Gerda, Lisa, Liza, Petra, Susanne, Tommy, Ulrike and all my friends both in Sweden and Germany. Thank you for being part of my life and drawing my attention from research to more important experiences in life.

Ursel, Rüdiger, Gitte, Wolfgang, Ute, Micha, Angela, Kalle, Thomas, Susi, Armin, Matthias, Ina, Paul und alle meine Nichten, Neffen, Tanten und Onkel, meine große, phantastische Familie in Deutschland, die immer für mich da ist, mir zur Seite steht und mir eine Sicherheit im Leben gibt die kleine und große Lebenssprünge ermöglicht.

Meine geliebten Eltern, die mir meine Ausbildungen ermöglicht haben und mich immer in allen meinen Entscheidungen unterstützt haben. Eure Liebe und euer Vertrauen in mich gaben und geben mir die besten Vorraussetzungen für das Leben. Papa, ich wünschte, du wärest dabei!

Catti, my beloved daughter, who always gives me the right view of what is important in life. Thank you for your love, help, great support, patience and releasing me from cooking.

9 REFERENCES

Agras WS, Hammer LD, McNicholas F, Kraemer HC. 2004. Risk factors for childhood overweight: a prospective study from birth to 9.5 years. J Pediatr 145(1):20-5.

Agren G, Narbro K, Naslund I, Sjostrom L, Peltonen M. 2002. Long-term effects of weight loss on pharmaceutical costs in obese subjects. A report from the SOS intervention study. Int J Obes Relat Metab Disord 26(2):184-92.

Ahrens W, Bammann K, de Henauw S, Halford J, Palou A, Pigeot I, Siani A, Sjostrom M. 2006. Understanding and preventing childhood obesity and related disorders--IDEFICS: a European multilevel epidemiological approach. Nutr Metab Cardiovasc Dis 16(4):302-8.

Alexandrov AA, Maslennikova GY, Kulikov SM, Propirnij GA, Perova NV. 1992. Primary prevention of cardiovascular disease: 3-year intervention results in boys of 12 years of age. Prev Med 21(1):53-62.

Andersen LB, Klausen K, Nisbeth O. 2002. [One-year effect of health counseling on life style and risk factors of heart disease]. Ugeskr Laeger 164(13):1814-8.

Ayyad C, Andersen T. 2000. Long-term efficacy of dietary treatment of obesity: a systematic review of studies published between 1931 and 1999. Obes Rev 1(2):113-9.

Bammann K, Peplies J, Sjöström M, Lissner L, De Henauw S, Galli C, Iacoviello L, Krogh V, Marild S, Pigeot I and others. 2006. Assessment of diet, physical activity and biological, social and environmental factors in a multi-centre European project on diet- and lifestyle-related disorders in children (IDEFICS). J Public Health (14):279-289.

Baumgartner RN. 2000. Body composition in healthy aging. Ann N Y Acad Sci 904:437-48.

Baumgartner RN, Koehler KM, Gallagher D, Romero L, Heymsfield SB, Ross RR, Garry PJ, Lindeman RD. 1998. Epidemiology of sarcopenia among the elderly in New Mexico. Am J Epidemiol 147(8):755-63.

Beck AM, Ovesen L. 1998. At which body mass index and degree of weight loss should hospitalized elderly patients be considered at nutritional risk? Clin Nutr 17(5):195-8.

Bender R, Jockel KH, Trautner C, Spraul M, Berger M. 1999. Effect of age on excess mortality in obesity. Jama 281(16):1498-504.

Bengtsson C, Blohme G, Hallberg L, Hallstrom T, Isaksson B, Korsan-Bengtsen K, Rybo G, Tibblin E, Tibblin G, Westerberg H. 1973. The study of women in Gothenburg 1968-1969--a population study. General design, purpose and sampling results. Acta Med Scand 193(4):311-8.

Bengtsson C, Hallberg L, Noppa H, Tibblin E. 1979. Anthropometric data in middle-aged women. The population study of women in Goteborg 1968-1969. Acta Morphol Neerl Scand 17(2):133-43.
Bennett SA, Magnus P. 1994. Trends in cardiovascular risk factors in Australia. Results from the National Heart Foundation's Risk Factor Prevalence Study, 1980-1989. Med J Aust 161(9):519-27.
Berg C, Rosengren A, Aires N, Lappas G, Toren K, Thelle D, Lissner L. 2005. Trends in overweight and obesity from 1985 to 2002 in Goteborg, West Sweden. Int J Obes (Lond) 29(8):916-24.
Berg S. 1980. Psychological functioning in 70- and 75-year-old people. A study in an industrialized city. Acta Psychiatr Scand Suppl 288:1-47.
Bergh I, Steen G, Waern M, Johansson B, Oden A, Sjostrom B, Steen B. 2003. Pain and its relation to cognitive function and depressive symptoms: a Swedish population study of 70-year-old men and women. J Pain Symptom Manage 26(4):903-12.
Black AE, Cole TJ. 2001. Biased over- or under-reporting is characteristic of individuals whether over time or by different assessment methods. J Am Diet Assoc 101(1):70-80.
Blair SN, Kohl HW, 3rd, Paffenbarger RS, Jr., Clark DG, Cooper KH, Gibbons LW. 1989. Physical fitness and all-cause mortality. A prospective study of healthy men and women. Jama 262(17):2395-401.
Bouchard C. 1996. Can obesity be prevented? Nutr Rev 54(4 Pt 2):S125-30.
Bouchard C. 1997. Genetics of human obesity: recent results from linkage studies. J Nutr 127(9):1887S-1890S.
Brannstrom I, Weinehall L, Persson LA, Wester PO, Wall S. 1993. Changing social patterns of risk factors for cardiovascular disease in a Swedish community intervention programme. Int J Epidemiol 22(6):1026-37.
Caballero B, Clay T, Davis SM, Ethelbah B, Rock BH, Lohman T, Norman J, Story M, Stone EJ, Stephenson L and others. 2003. Pathways: a school-based, randomized controlled trial for the prevention of obesity in American Indian schoolchildren. Am J Clin Nutr 78(5):1030-8.
Cabrera C, Wilhelmson K, Allebeck P, Wedel H, Steen B, Lissner L. 2003. Cohort differences in obesity-related health indicators among 70-year olds with special reference to gender and education. Eur J Epidemiol 18(9):883-90.
Campbell WW, Crim MC, Young VR, Evans WJ. 1994. Increased energy requirements and changes in body composition with resistance training in older adults. Am J Clin Nutr 60(2):167-75.
Cnattingius S, Lambe M. 2002. Trends in smoking and overweight during pregnancy: prevalence, risks of pregnancy complications, and adverse pregnancy outcomes. Semin Perinatol 26(4):286-95.
Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. 2000. Establishing a standard definition for child overweight and obesity worldwide: international survey. Bmj 320(7244):1240-3.

Conway B, Rene A. 2004. Obesity as a disease: no lightweight matter. Obes Rev 5(3):145-51.
Cook C, Simmons G, Swinburn B, Stewart J. 2001. Changing risk behaviours for non-communicable disease in New Zealand working men--is workplace intervention effective? N Z Med J 114(1130):175-8.
Danish National Board of Health. 2003. National action plan against obesity. Recommendations and perspectices. Short version. Copenhagen: National Board of Health.
Department of Health. 1991. Report on health and social subjects; 41. Dietary reference values for food energy and nutrients for the United Kingdom. HMSO: London.
Derenne JL, Beresin EV. 2006. Body image, media, and eating disorders. Acad Psychiatry 30(3):257-61.
Dey DK, Lissner L. 2003. Obesity in 70-year-old subjects as a risk factor for 15-year coronary heart disease incidence. Obes Res 11(7):817-27.
Dey DK, Rothenberg E, Sundh V, Bosaeus I, Steen B. 2001a. Body mass index, weight change and mortality in the elderly. A 15 y longitudinal population study of 70 y olds. Eur J Clin Nutr 55(6):482-92.
Dey DK, Rothenberg E, Sundh V, Bosaeus I, Steen B. 2001b. Height and body weight in elderly adults: a 21-year population study on secular trends and related factors in 70-year-olds. J Gerontol A Biol Sci Med Sci 56(12):M780-4.
Dey DK, Rothenberg E, Sundh V, Bosaeus I, Steen B. 2002. Waist circumference, body mass index, and risk for stroke in older people: a 15 year longitudinal population study of 70- year-olds. J Am Geriatr Soc 50(9):1510-8.
Dietz WH. 1997. Periods of risk in childhood for the development of adult obesity--what do we need to learn? J Nutr 127(9):1884S-1886S.
Douketis JD, Feightner JW, Attia J, Feldman WF. 1999. Periodic health examination, 1999 update: 1. Detection, prevention and treatment of obesity. Canadian Task Force on Preventive Health Care. Cmaj 160(4):513-25.
Egger G, Swinburn B. 1997. An "ecological" approach to the obesity pandemic. Bmj 315(7106):477-80.
Eiben G, Dey DK, Rothenberg E, Steen B, Bjorkelund C, Bengtsson C, Lissner L. 2005. Obesity in 70-year-old Swedes: secular changes over 30 years. Int J Obes (Lond) 29(7):810-7.
Elia M. 2001. Obesity in the elderly. Obes Res 9 Suppl 4:244S-248S.
Engberg M, Christensen B, Karlsmose B, Lous J, Lauritzen T. 2002. General health screenings to improve cardiovascular risk profiles: a randomized controlled trial in general practice with 5-year follow-up. J Fam Pract 51(6):546-52.
Epstein LH, Gordy CC, Raynor HA, Beddome M, Kilanowski CK, Paluch R. 2001. Increasing fruit and vegetable intake and decreasing fat and sugar intake in families at risk for childhood obesity. Obes Res 9(3):171-8.

Eriksson BG, Mellstrom D, Svanborg A. 1987. Medical-social intervention in a 70-year-old Swedish population. A general presentation of methodological experience. Compr Gerontol [C] 1:49-56.
Fagerli RA, Wandel M. 1999. Gender differences in opinions and practices with regard to a "healthy diet". Appetite 32(2):171-90.
Faith MS, Pietrobelli A, Nunez C, Heo M, Heymsfield SB, Allison DB. 1999. Evidence for independent genetic influences on fat mass and body mass index in a pediatric twin sample. Pediatrics 104(1 Pt 1):61-7.
Flegal KM, Carroll MD, Ogden CL, Johnson CL. 2002. Prevalence and trends in obesity among US adults, 1999-2000. Jama 288(14):1723-7.
Fogelholm M, Mannisto S, Vartiainen E, Pietinen P. 1996. Determinants of energy balance and overweight in Finland 1982 and 1992. Int J Obes Relat Metab Disord 20(12):1097-104.
Fontaine KR, Redden DT, Wang C, Westfall AO, Allison DB. 2003. Years of life lost due to obesity. Jama 289(2):187-93.
Forster JL, Jeffery RW, Schmid TL, Kramer FM. 1988. Preventing weight gain in adults: a pound of prevention. Health Psychol 7(6):515-25.
Garner DM. 1991. Eating disorder inventory 2, professional manual.
Goldberg GR, Black AE, Jebb SA, Cole TJ, Murgatroyd PR, Coward WA, Prentice AM. 1991. Critical evaluation of energy intake data using fundamental principles of energy physiology: 1. Derivation of cut-off limits to identify under-recording. Eur J Clin Nutr 45(12):569-81.
Gordon-Larsen P, Adair LS, Nelson MC, Popkin BM. 2004. Five-year obesity incidence in the transition period between adolescence and adulthood: the National Longitudinal Study of Adolescent Health. Am J Clin Nutr 80(3):569-75.
Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. 1993. Social and economic consequences of overweight in adolescence and young adulthood. N Engl J Med 329(14):1008-12.
Groesz LM, Levine MP, Murnen SK. 2002. The effect of experimental presentation of thin media images on body satisfaction: a meta-analytic review. Int J Eat Disord 31(1):1-16.
Grundy SM, Blackburn G, Higgins M, Lauer R, Perri MG, Ryan D. 1999. Physical activity in the prevention and treatment of obesity and its comorbidities. Med Sci Sports Exerc 31(11 Suppl):S502-8.
Gutierrez-Fisac JL, Lopez E, Banegas JR, Graciani A, Rodriguez-Artalejo F. 2004. Prevalence of overweight and obesity in elderly people in Spain. Obes Res 12(4):710-5.
Harris TB, Visser M, Everhart J, Cauley J, Tylavsky F, Fuerst T, Zamboni M, Taaffe D, Resnick HE, Scherzinger A and others. 2000. Waist circumference and sagittal diameter reflect total body fat better than visceral fat in older men and women. The Health, Aging and Body Composition Study. Ann N Y Acad Sci 904:462-73.
Haslam DW, James WP. 2005. Obesity. Lancet 366(9492):1197-209.
Health Hunters. http://www.neon.sahlgrenska.gu.se/Health_hunters.htm.

Heiat A, Vaccarino V, Krumholz HM. 2001. An evidence-based assessment of federal guidelines for overweight and obesity as they apply to elderly persons. Arch Intern Med 161(9):1194-203.
Heitmann BL, Lissner L, Sorensen TI, Bengtsson C. 1995. Dietary fat intake and weight gain in women genetically predisposed for obesity. Am J Clin Nutr 61(6):1213-7.
Hughes VA, Frontera WR, Roubenoff R, Evans WJ, Singh MA. 2002. Longitudinal changes in body composition in older men and women: role of body weight change and physical activity. Am J Clin Nutr 76(2):473-81.
Hunter GR, Bryan DR, Wetzstein CJ, Zuckerman PA, Bamman MM. 2002. Resistance training and intra-abdominal adipose tissue in older men and women. Med Sci Sports Exerc 34(6):1023-8.
Jacobson KC, Rowe DC. 1998. Genetic and shared environmental influences on adolescent BMI: interactions with race and sex. Behav Genet 28(4):265-78.
Janssen I, Heymsfield SB, Ross R. 2002. Low relative skeletal muscle mass (sarcopenia) in older persons is associated with functional impairment and physical disability. J Am Geriatr Soc 50(5):889-96.
Janssen I, Mark AE. 2007. Elevated body mass index and mortality risk in the elderly. Obes Rev 8(1):41-59.
Jebb SA. 1997. Aetiology of obesity. Br Med Bull 53(2):264-85.
Jeffery RW, Gray CW, French SA, Hellerstedt WL, Murray D, Luepker RV, Blackburn H. 1995. Evaluation of weight reduction in a community intervention for cardiovascular disease risk: changes in body mass index in the Minnesota Heart Health Program. Int J Obes Relat Metab Disord 19(1):30-9.
Jordbruksverket. 2000. Konsumtionen av livsmedel m m 1996-1999. Rapport 2000:13.
Kamakura T, Ando J, Ono Y, Maekawa H. 2003. A twin study of genetic and environmental influences on psychological traits of eating disorders in a Japanese female sample. Twin Res 6(4):292-6.
Katzmarzyk PT, Davis C. 2001. Thinness and body shape of Playboy centerfolds from 1978 to 1998. Int J Obes Relat Metab Disord 25(4):590-2.
Kennedy RL, Chokkalingham K, Srinivasan R. 2004. Obesity in the elderly: who should we be treating, and why, and how? Curr Opin Clin Nutr Metab Care 7(1):3-9.
Keski-Rahkonen A, Bulik CM, Neale BM, Rose RJ, Rissanen A, Kaprio J. 2005. Body dissatisfaction and drive for thinness in young adult twins. Int J Eat Disord 37(3):188-99.
Kiess W, Reich A, Muller G, Meyer K, Galler A, Bennek J, Kratzsch J. 2001. Clinical aspects of obesity in childhood and adolescence--diagnosis, treatment and prevention. Int J Obes Relat Metab Disord 25 Suppl 1:S75-9.
Kindblom JM, Lorentzon M, Norjavaara E, Lonn L, Brandberg J, Angelhed JE, Hellqvist A, Nilsson S, Ohlsson C. 2006. Pubertal timing is an

independent predictor of central adiposity in young adult males: the Gothenburg osteoporosis and obesity determinants study. Diabetes 55(11):3047-52.
Kjelsas E, Bjornstrom C, Gotestam KG. 2004. Prevalence of eating disorders in female and male adolescents (14-15 years). Eat Behav 5(1):13-25.
Klump KL, McGue M, Iacono WG. 2000. Age differences in genetic and environmental influences on eating attitudes and behaviors in preadolescent and adolescent female twins. J Abnorm Psychol 109(2):239-51.
Kostanski M, Fisher A, Gullone E. 2004. Current conceptualisation of body image dissatisfaction: have we got it wrong? J Child Psychol Psychiatry 45(7):1317-25.
Kotler LA, Cohen P, Davies M, Pine DS, Walsh BT. 2001. Longitudinal relationships between childhood, adolescent, and adult eating disorders. J Am Acad Child Adolesc Psychiatry 40(12):1434-40.
Kuller LH, Simkin-Silverman LR, Wing RR, Meilahn EN, Ives DG. 2001. Women's Healthy Lifestyle Project: A randomized clinical trial: results at 54 months. Circulation 103(1):32-7.
Kumanyika S, Jeffery RW, Morabia A, Ritenbaugh C, Antipatis VJ. 2002. Obesity prevention: the case for action. Int J Obes Relat Metab Disord 26(3):425-36.
Kvaavik E, Tell GS, Klepp KI. 2003. Predictors and tracking of body mass index from adolescence into adulthood: follow-up of 18 to 20 years in the Oslo Youth Study. Arch Pediatr Adolesc Med 157(12):1212-8.
Lahti-Koski M, Jousilahti P, Pietinen P. 2001. Secular trends in body mass index by birth cohort in eastern Finland from 1972 to 1997. Int J Obes Relat Metab Disord 25(5):727-34.
Lamb SE, Bartlett HP, Ashley A, Bird W. 2002. Can lay-led walking programmes increase physical activity in middle aged adults? A randomised controlled trial. J Epidemiol Community Health 56(4):246-52.
Lichtenstein P, De Faire U, Floderus B, Svartengren M, Svedberg P, Pedersen NL. 2002. The Swedish Twin Registry: a unique resource for clinical, epidemiological and genetic studies. J Intern Med 252(3):184-205.
Lichtenstein P, Tuvblad C, Larsson H, Carlström E. 2006. The Swedish Twin study of CHild and Adolescent Development – the TCHAD-study. In press.
Lindqvist P, Andersson K, Sundh V, Lissner L, Bjorkelund C, Bengtsson C. 2006. Concurrent and separate effects of body mass index and waist-to-hip ratio on 24-year mortality in the Population Study of Women in Gothenburg: Evidence of age-dependency. Eur J Epidemiol 21(11):789-94.
Lindroos AK, Lissner L, Sjostrom L. 1993. Validity and reproducibility of a self-administered dietary questionnaire in obese and non-obese subjects. Eur J Clin Nutr 47(7):461-81.

Lingfors H, Lindstrom K, Persson LG, Bengtsson C, Lissner L. 2003. Lifestyle changes after a health dialogue. Results from the Live for Life health promotion programme. Scand J Prim Health Care 21(4):248-52.
Linne Y. 2004. Effects of obesity on women's reproduction and complications during pregnancy. Obes Rev 5(3):137-43.
Lissner L, Bjorkelund C, Heitmann BL, Lapidus L, Bjorntorp P, Bengtsson C. 1998a. Secular increases in waist-hip ratio among Swedish women. Int J Obes Relat Metab Disord 22(11):1116-20.
Lissner L, Heitmann BL, Bengtsson C. 1997. Low-fat diets may prevent weight gain in sedentary women: prospective observations from the population study of women in Gothenburg, Sweden. Obes Res 5(1):43-8.
Lissner L, Johansson SE, Qvist J, Rossner S, Wolk A. 2000. Social mapping of the obesity epidemic in Sweden. Int J Obes Relat Metab Disord 24(6):801-5.
Lissner L, Lindroos AK, Sjostrom L. 1998b. Swedish obese subjects (SOS): an obesity intervention study with a nutritional perspective. Eur J Clin Nutr 52(5):316-22.
Lobstein T, Baur L, Uauy R. 2004. Obesity in children and young people: a crisis in public health. Obes Rev 5 Suppl 1:4-104.
Lupton BS, Fonnebo V, Sogaard AJ, Langfeldt E. 2002. The Finnmark Intervention Study. Better health for the fishery population in an Arctic village in North Norway. Scand J Prim Health Care 20(4):213-8.
McCabe MP, Ricciardelli LA, Finemore J. 2002. The role of puberty, media and popularity with peers on strategies to increase weight, decrease weight and increase muscle tone among adolescent boys and girls. J Psychosom Res 52(3):145-53.
Miller SL, Reber RJ, Chapman-Novakofski K. 2001. Prevalence of CVD risk factors and impact of a two-year education program for premenopausal women. Womens Health Issues 11(6):486-93.
Mokdad AH, Ford ES, Bowman BA, Nelson DE, Engelgau MM, Vinicor F, Marks JS. 2001. The continuing increase of diabetes in the US. Diabetes Care 24(2):412.
Molarius A, Seidell JC. 1998. Selection of anthropometric indicators for classification of abdominal fatness--a critical review. Int J Obes Relat Metab Disord 22(8):719-27.
Molarius A, Seidell JC, Sans S, Tuomilehto J, Kuulasmaa K. 1999. Waist and hip circumferences, and waist-hip ratio in 19 populations of the WHO MONICA Project. Int J Obes Relat Metab Disord 23(2):116-25.
Mossberg HO. 1989. 40-year follow-up of overweight children. Lancet 2(8661):491-3.
Narbro K, Agren G, Jonsson E, Larsson B, Naslund I, Wedel H, Sjostrom L. 1999. Sick leave and disability pension before and after treatment for obesity: a report from the Swedish Obese Subjects (SOS) study. Int J Obes Relat Metab Disord 23(6):619-24.

Neale M, Maes HH. 2004. Methodology for genetic studies of twins and families. Dordrecht, Nederländerna.
Neovius M, Linne Y, Rossner S. 2005. BMI, waist-circumference and waist-hip-ratio as diagnostic tests for fatness in adolescents. Int J Obes Relat Metab Disord 29(2):163-9.
Neovius MG, Linne YM, Barkeling BS, Rossner SO. 2004. Sensitivity and specificity of classification systems for fatness in adolescents. Am J Clin Nutr 80(3):597-603.
Neumark-Sztainer D, Levine MP, Paxton SJ, Smolak L, Piran N, Wertheim EH. 2006a. Prevention of body dissatisfaction and disordered eating: What next? Eat Disord 14(4):265-85.
Neumark-Sztainer D, Story M, Hannan PJ, Perry CL, Irving LM. 2002. Weight-related concerns and behaviors among overweight and nonoverweight adolescents: implications for preventing weight-related disorders. Arch Pediatr Adolesc Med 156(2):171-8.
Neumark-Sztainer D, Wall M, Guo J, Story M, Haines J, Eisenberg M. 2006b. Obesity, disordered eating, and eating disorders in a longitudinal study of adolescents: how do dieters fare 5 years later? J Am Diet Assoc 106(4):559-68.
Nevonen L, Clinton D, Norring C. 2006. Validating the EDI-2 in three Swedish female samples: eating disorders patients, psychiatric outpatients and normal controls. Nord J Psychiatry 60(1):44-50.
Nieto-Garcia FJ, Bush TL, Keyl PM. 1990. Body mass definitions of obesity: sensitivity and specificity using self-reported weight and height. Epidemiology 1(2):146-52.
Nooyens AC, Visscher TL, Schuit AJ, van Rossum CT, Verschuren WM, van Mechelen W, Seidell JC. 2005. Effects of retirement on lifestyle in relation to changes in weight and waist circumference in Dutch men: a prospective study. Public Health Nutr 8(8):1266-74.
Norring C, Sohlberg S. 1988. Eating Disorder Inventory in Sweden: description, cross-cultural comparison, and clinical utility. Acta Psychiatr Scand 78(5):567-75.
Olshansky SJ, Passaro DJ, Hershow RC, Layden J, Carnes BA, Brody J, Hayflick L, Butler RN, Allison DB, Ludwig DS. 2005. A potential decline in life expectancy in the United States in the 21st century. N Engl J Med 352(11):1138-45.
Oren A, Vos LE, Uiterwaal CS, Gorissen WH, Grobbee DE, Bots ML. 2003. Change in body mass index from adolescence to young adulthood and increased carotid intima-media thickness at 28 years of age: the Atherosclerosis Risk in Young Adults study. Int J Obes Relat Metab Disord 27(11):1383-90.
Peel GR, Booth ML. 2001. Impact evaluation of the Royal Australian Air Force health promotion program. Aviat Space Environ Med 72(1):44-51.
Persson LG, Lindstrom K, Lingfors H, Bengtsson C, Lissner L. 1998. Cardiovascular risk during early adult life. Risk markers among partici-

pants in "Live for Life" health promotion programme in Sweden. J Epidemiol Community Health 52(7):425-32.
Pietilainen KH, Kaprio J, Rissanen A, Winter T, Rimpela A, Viken RJ, Rose RJ. 1999. Distribution and heritability of BMI in Finnish adolescents aged 16y and 17y: a study of 4884 twins and 2509 singletons. Int J Obes Relat Metab Disord 23(2):107-15.
Polley BA, Wing RR, Sims CJ. 2002. Randomized controlled trial to prevent excessive weight gain in pregnant women. Int J Obes Relat Metab Disord 26(11):1494-502.
Pope HG, Jr., Olivardia R, Borowiecki JJ, 3rd, Cohane GH. 2001. The growing commercial value of the male body: a longitudinal survey of advertising in women's magazines. Psychother Psychosom 70(4):189-92.
Prentice AM, Jebb SA. 1995. Obesity in Britain: gluttony or sloth? Bmj 311(7002):437-9.
Prochaska JO, DiClemente CC, Norcross JC. 1992. In search of how people change. Applications to addictive behaviors. Am Psychol 47(9):1102-14.
Rasmussen F, Johansson M, Hansen HO. 1999. Trends in overweight and obesity among 18-year-old males in Sweden between 1971 and 1995. Acta Paediatr 88(4):431-7.
Reilly JJ, Methven E, McDowell ZC, Hacking B, Alexander D, Stewart L, Kelnar CJ. 2003. Health consequences of obesity. Arch Dis Child 88(9):748-52.
Rinder L, Roupe S, Steen B, Svanborg A. 1975. Seventy-year-old people in Gothenburg. A population study in an industrialized Swedish city. Acta Med Scand 198(5):397-407.
Roberts SB, Dallal GE. 2005. Energy requirements and aging. Public Health Nutr 8(7A):1028-36.
Rosamond WD, Ammerman AS, Holliday JL, Tawney KW, Hunt KJ, Keyserling TC, Will JC, Mokdad AH. 2000. Cardiovascular disease risk factor intervention in low-income women: the North Carolina WISEWOMAN project. Prev Med 31(4):370-9.
Rosmond R, Bjorntorp P. 1999. Psychosocial and socio-economic factors in women and their relationship to obesity and regional body fat distribution. Int J Obes Relat Metab Disord 23(2):138-45.
Rossner S. 2001. Obesity through the ages of man. Int J Obes Relat Metab Disord 25 Suppl 4:S29-33.
Rothenberg E, Bosaeus I, Lernfelt B, Landahl S, Steen B. 1998. Energy intake and expenditure: validation of a diet history by heart rate monitoring, activity diary and doubly labeled water. Eur J Clin Nutr 52(11):832-8.
Rothenberg E, Bosaeus I, Steen B. 1996. Food habits in three 70-year-old free-living populations in Gothenburg, Sweden. A 22-year cohort study. Scandinavian Journal of Nutrition 40:104-10.

Rutherford J, McGuffin P, Katz RJ, Murray RM. 1993. Genetic influences on eating attitudes in a normal female twin population. Psychol Med 23(2):425-36.
Sandström B, Aro A, Becker W, Lyhne N, Pedersen J, Tórsdóttir I. 1996. Nordic Nutrition Recommendations 1996. Copenhagen: Nordic Council of Ministers.
Saris WH, Blair SN, van Baak MA, Eaton SB, Davies PS, Di Pietro L, Fogelholm M, Rissanen A, Schoeller D, Swinburn B and others. 2003. How much physical activity is enough to prevent unhealthy weight gain? Outcome of the IASO 1st Stock Conference and consensus statement. Obes Rev 4(2):101-14.
SBU Report from the Swedish Council on Technology Assessment in Health Care. 2005. Interventions to Prevent Obesity A Systematic Review. Report No 173E, ISBN 91-85413-04-6, Stockholm:SBU
Schofield W, Schofield C, James P. 1985. Basal metabolic rate: review and prediction. Human nutrition: Clinical nutrition 39(Suppl. 1):1-96.
Schousboe K, Willemsen G, Kyvik KO, Mortensen J, Boomsma DI, Cornes BK, Davis CJ, Fagnani C, Hjelmborg J, Kaprio J and others. 2003. Sex differences in heritability of BMI: a comparative study of results from twin studies in eight countries. Twin Res 6(5):409-21.
Schousboe K, Visscher PM, Erbas B, Kyvik KO, Hopper JL, Henriksen JE, Heitmann BL, Sorensen TI. 2004. Twin study of genetic and environmental influences on adult body size, shape, and composition. Int J Obes Relat Metab Disord 28(1):39-48.
Segal NL, Allison DB. 2002. Twins and virtual twins: bases of relative body weight revisited. Int J Obes Relat Metab Disord 26(4):437-41.
Seidell JC, Nooyens AJ, Visscher TL. 2005. Cost-effective measures to prevent obesity: epidemiological basis and appropriate target groups. Proc Nutr Soc 64(1):1-5.
Sherwood NE, Jeffery RW, French SA, Hannan PJ, Murray DM. 2000. Predictors of weight gain in the Pound of Prevention study. Int J Obes Relat Metab Disord 24(4):395-403.
Sjostrom CD. 2003. Surgery as an intervention for obesity. Results from the Swedish obese subjects study. Growth Horm IGF Res 13 Suppl A:S22-6.
Sjostrom L, Larsson B, Backman L, Bengtsson C, Bouchard C, Dahlgren S, Hallgren P, Jonsson E, Karlsson J, Lapidus L and others. 1992. Swedish obese subjects (SOS). Recruitment for an intervention study and a selected description of the obese state. Int J Obes Relat Metab Disord 16(6):465-79.
Skov AR, Toubro S, Ronn B, Holm L, Astrup A. 1999. Randomized trial on protein vs carbohydrate in ad libitum fat reduced diet for the treatment of obesity. Int J Obes Relat Metab Disord 23(5):528-36.
Spillane NS, Boerner LM, Anderson KG, Smith GT. 2004. Comparability of the Eating Disorder Inventory-2 between women and men. Assessment 11(1):85-93.
Statistiska Centralbyrån. 2006. Swedish Statistics, Stockholm.

Steen B, Djurfeldt H. 1993. The gerontological and geriatric population studies in Gothenburg, Sweden. Z Gerontol 26(3):163-9.
Stevens J, Cai J, Pamuk ER, Williamson DF, Thun MJ, Wood JL. 1998. The effect of age on the association between body-mass index and mortality. N Engl J Med 338(1):1-7.
Sullivan M, Karlsson J, Sjostrom L, Backman L, Bengtsson C, Bouchard C, Dahlgren S, Jonsson E, Larsson B, Lindstedt S and others. 1993. Swedish obese subjects (SOS)--an intervention study of obesity. Baseline evaluation of health and psychosocial functioning in the first 1743 subjects examined. Int J Obes Relat Metab Disord 17(9):503-12.
Swinburn B, Egger G. 2004. The runaway weight gain train: too many accelerators, not enough brakes. Bmj 329(7468):736-9.
Swinburn BA, Caterson I, Seidell JC, James WP. 2004. Diet, nutrition and the prevention of excess weight gain and obesity. Public Health Nutr 7(1A):123-46.
Taylor CB, Fortmann SP, Flora J, Kayman S, Barrett DC, Jatulis D, Farquhar JW. 1991. Effect of long-term community health education on body mass index. The Stanford Five-City Project. Am J Epidemiol 134(3):235-49.
Taylor RW, Jones IE, Williams SM, Goulding A. 2002. Body fat percentages measured by dual-energy X-ray absorptiometry corresponding to recently recommended body mass index cutoffs for overweight and obesity in children and adolescents aged 3-18 y. Am J Clin Nutr 76(6):1416-21.
The National Food Administration and Swedish National Institute of Public Health. 2005. Kostnadsberäkningar och finansieringsförslag för underlag till handlingsplan för goda matvanor och ökad fysisk aktivitet.
Tudor-Smith C, Nutbeam D, Moore L, Catford J. 1998. Effects of the Heartbeat Wales programme over five years on behavioural risks for cardiovascular disease: quasi-experimental comparison of results from Wales and a matched reference area. Bmj 316(7134):818-22.
Tyrka AR, Waldron I, Graber JA, Brooks-Gunn J. 2002. Prospective predictors of the onset of anorexic and bulimic syndromes. Int J Eat Disord 32(3):282-90.
Wade TD, Bulik CM, Heath AC, Martin NG, Eaves LJ. 2001. The influence of genetic and environmental factors in estimations of current body size, desired body size, and body dissatisfaction. Twin Res 4(4):260-5.
van't Hof MA, Burema J. 1996. Assessment of bias in the SENECA study. Eur J Clin Nutr 50 Suppl 2:S4-8.
Warschburger P. 2005. The unhappy obese child. Int J Obes (Lond) 29 Suppl 2:S127-9.
Vartiainen E, Puska P, Jousilahti P, Korhonen HJ, Tuomilehto J, Nissinen A. 1994. Twenty-year trends in coronary risk factors in north Karelia and in other areas of Finland. Int J Epidemiol 23(3):495-504.

Weber JL, Reid PM, Greaves KA, DeLany JP, Stanford VA, Going SB, Howell WH, Houtkooper LB. 2001. Validity of self-reported energy intake in lean and obese young women, using two nutrient databases, compared with total energy expenditure assessed by doubly labeled water. Eur J Clin Nutr 55(11):940-50.
WHO. http://www.who.int/bmi/index.jsp.
WHO. 2000. Obesity: preventing and managing the global epidemic. Report of a WHO consultation. World Health Organ Tech Rep Ser 894:i-xii, 1-253.
WHO Global Infobase. (http://infobase.who.int/).
Wilson RS. 1979. Twin growth: initial deficit, recovery, and trends in concordance from birth to nine years. Ann Hum Biol 6(3):205-20.
Wing RR. 1995. Changing diet and exercise behaviors in individuals at risk for weight gain. Obes Res 3 Suppl 2:277s-282s.
Visscher TL, Seidell JC, Menotti A, Blackburn H, Nissinen A, Feskens EJ, Kromhout D. 2000. Underweight and overweight in relation to mortality among men aged 40-59 and 50-69 years: the Seven Countries Study. Am J Epidemiol 151(7):660-6.
Visscher TL, Seidell JC, Molarius A, van der Kuip D, Hofman A, Witteman JC. 2001. A comparison of body mass index, waist-hip ratio and waist circumference as predictors of all-cause mortality among the elderly: the Rotterdam study. Int J Obes Relat Metab Disord 25(11):1730-5.
Yoon PW, Scheuner MT, Khoury MJ. 2003. Research priorities for evaluating family history in the prevention of common chronic diseases. Am J Prev Med 24(2):128-35.
Zamboni M, Mazzali G, Zoico E, Harris TB, Meigs JB, Di Francesco V, Fantin F, Bissoli L, Bosello O. 2005. Health consequences of obesity in the elderly: a review of four unresolved questions. Int J Obes (Lond) 29(9):1011-29.