Epidemiological studies of weight gain and its determinants

The studies presented in this thesis were conducted at the National Institute for Public Health and the Environment (RIVM), Bilthoven, the Netherlands, the Department of Public and Occupational Health and EMGO Institute (www.emgo.nl), VU University Medical Center Amsterdam, the Netherlands, and at the Institute of Health Sciences at the VU University Amsterdam, the Netherlands. The EMGO Institute participates in the Netherlands School of Primary Care Research (CaRe) which was re-acknowledged in 2005 by the Royal Netherlands Academy of Arts and Sciences.

The studies presented in this thesis were supported by a grant from the Netherlands Heart Foundation (NHF – 2000T202).

Financial support by the Netherlands Heart Foundation for the printing of this thesis is gratefully acknowledged.

Painting on front by Thomas Nooyens (2003).

ISBN 9789086592753

Printed by: Koninklijke Drukkerij Em. de Jong BV, Baarle-Nassau, the Netherlands.

VRIJE UNIVERSITEIT

Epidemiological studies of weight gain and its determinants

ACADEMISCH PROEFSCHRIFT

ter verkrijging van de graad Doctor aan
de Vrije Universiteit Amsterdam,
op gezag van de rector magnificus
prof.dr. L.M. Bouter,
in het openbaar te verdedigen
ten overstaan van de promotiecommissie
van de factulteit der Aard- en Levenswetenschappen
op donderdag 8 januari 2009 om 15.45 uur
in de aula van de universiteit,
De Boelelaan 1105

door

Astrid Cornelia Johanna Nooyens
geboren te Baarle-Nassau

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	dr. T.L.S. Visscher

Voor ons pa en ons ma

Contents

General introduction

The NHF-NRG project

The "Netherlands Research programme weight Gain prevention" (NHF-NRG) was initiated by the Netherlands Heart Foundation (NHF). The focus of this programme was to study determinants of weight gain, and to develop, carry out and evaluate comprehensive weight gain prevention programmes for different target groups. In the studies that were part of the NHF-NRG programme, both food intake and physical activity behaviours were integrated, rather than focusing on one of these categories of behaviours only.[1] Therefore, a multidisciplinary research team was composed including epidemiologists, nutritionists, exercise physiologists and health promotion scientists. The NHF-NRG programme group identified three major risk groups with elevated risk of weight gain, who can be reached easily in an already existing setting: adolescents (12–16 y) at secondary school, young adults (20–40 y) at the workplace, and recently retired people (55–65 y) at home.[1] The combination of high risk and the relatively easy way to approach these groups, make them of particular interest for intervention programmes.

Most (overweight) people can lose weight on a short-term basis. However, it is difficult to maintain weight loss in the long run. Most people regain weight when the weight loss program stops.[2] In addition, prevention of weight gain has more potential in controlling the incidence of obesity and to halt the obesity epidemic than obesity treatment alone.[3-5] Therefore, weight gain prevention programmes are needed.

The aim of the NHF-NRG programme was to develop, carry out and evaluate three age-specific weight gain prevention programmes. To support evidence-based weight gain prevention programmes, two projects were included with the aim to identify important age-specific determinants of weight gain in already ongoing longitudinal studies. The first project focused on determinants of behaviours related to energy balance. The second project focuses on life-style predictors of weight gain, and is presented in this thesis.

The following introduction briefly summarizes the current status of overweight and obesity in the Netherlands and addresses the importance of weight gain prevention. Furthermore, it provides background on mechanisms of weight gain, and it describes possible lifestyle determinants of weight gain. Finally, it ends with an outline of the present thesis.

Definitions of overweight and obesity

In most cases, overweight and obesity are defined on the basis of body mass index (BMI). BMI is calculated as body weight (in kg) divided by body height squared (in m^2). For adults, a BMI of 25 or higher is referred to as overweight. A BMI of 30 or higher is severe overweight, referred to as obesity. Waist circumference (WC) is also used to define (abdominal) overweight and obesity. Cut-off values for waist circumference are different for men compared to women.[6] Table 1.1 shows current criteria for overweight and obesity based on both BMI and waist circumference in adults. Subjects having a BMI or waist circumference above these levels are at increased risk of mortality or developing morbidity and disability.[6,7]

TABLE 1.1 Classification of overweight and obesity by BMI, waist circumference and associated disease risk of type 2 diabetes, hypertension and cardiovascular disease (CVD). *Adapted from National Institutes of Health, 1998.*[8]

	Disease Risk relative to normal weight and waist circumference		
		Waist circumference	
	Body mass index (kg/m²)	*Men ≤102 cm Women ≤88 cm*	*Men >102 cm Women >88 cm*
Underweight	<18.5	*	*
Normal	18.5 – 24.9	*Reference category*	+
Overweight	25 – 29.9	Increased	High
Obesity	≥30	High	Very high

* Underweight persons have lower disease risk of type 2 diabetes, hypertension and CVD, but higher risk of mortality from chronic respiratory disease.[9,10]
+ Increased waist circumference can also be a marker for increased risk even in persons of normal weight.

In children and adolescents, overweight and obesity are defined based on BMI. Age- and gender-specific cut-off points to define overweight and obesity are suggested by Cole, based on normal gender-specific growth curves and reference values of 25 and 30 kg/m^2 at age 18 years (adult age)[11] as shown in Table 1.2. These cut-off points are meant to compare childhood and adolescent overweight and obesity rates between countries and over time, but are also used in the clinical setting[12] and in obesity related research.

Prevalence and trends of overweight and obesity

Over the last few decades, the prevalence of overweight and obesity has increased dramatically in many different parts of the world. This has also been observed in the Netherlands, based on measured body weight.[13-15]
In 2007, the prevalence of adult (self-reported) overweight (including obesity) was 51% in Dutch men and 40% in Dutch women.[16] In 1982, these figures were 36 and 29%, respectively.[17] To interpret these figures, one has to put in mind that obesity prevalence based on self-reported data is underestimated by 25-30% due to over-reporting height and underreporting body weight.[18]

TABLE 1.2 International cut-off points for body mass index for overweight and obesity by sex between 12 and 18 years.* *Adapted from Cole et al, 2000.*[11]

Age (y)	Overweight		Obesity	
	Boys	**Girls**	**Boys**	**Girls**
12	≥ 21.22	≥ 21.68	≥ 26.02	≥ 26.67
12.5	≥ 21.56	≥ 22.14	≥ 26.43	≥ 27.24
13	≥ 21.91	≥ 22.58	≥ 26.84	≥ 27.76
13.5	≥ 22.27	≥ 22.98	≥ 27.25	≥ 28.20
14	≥ 22.62	≥ 23.34	≥ 27.63	≥ 28.57
14.5	≥ 22.96	≥ 23.66	≥ 27.98	≥ 28.87
15	≥ 23.29	≥ 23.94	≥ 28.30	≥ 29.11
15.5	≥ 23.60	≥ 24.17	≥ 28.60	≥ 29.29
16	≥ 23.90	≥ 24.37	≥ 28.88	≥ 29.43
16.5	≥ 24.19	≥ 24.54	≥ 29.14	≥ 29.56
17	≥ 24.46	≥ 24.70	≥ 29.41	≥ 29.69
17.5	≥ 24.73	≥ 24.85	≥ 29.70	≥ 29.84
18	≥ 25	≥ 25	≥ 30	≥ 30

* These BMI cut-off points exist for boys and girls from age 3 up to 18 years.
Only those for 12-18 year olds are shown here, because in the present thesis no younger children were studied.

The prevalence of measured overweight (including obesity) in children and adolescents has increased at an even faster rate than the adult prevalence rates between 1980 (boys 3.9%, girls 6.9%) and 2002-2004 (boys 14.5%, girls 17.5%), with increases between 1997 (boys 9.7%, girls 13.0%) and 2002-2004 being steeper than the increases between 1980 and 1997.[19]

The public health impact of overweight and obesity

Overweight and obesity are associated with elevated levels of cardiovascular risk factors (e.g. elevated blood pressure, adverse blood lipid profile, decreased glucose tolerance) and for several chronic diseases and conditions, such as diabetes, cardiovascular disease, cancer, gallstones, and sleep apnoea.[7,20] Because these obesity related health outcomes are mostly chronic conditions, prevention of weight gain is important.

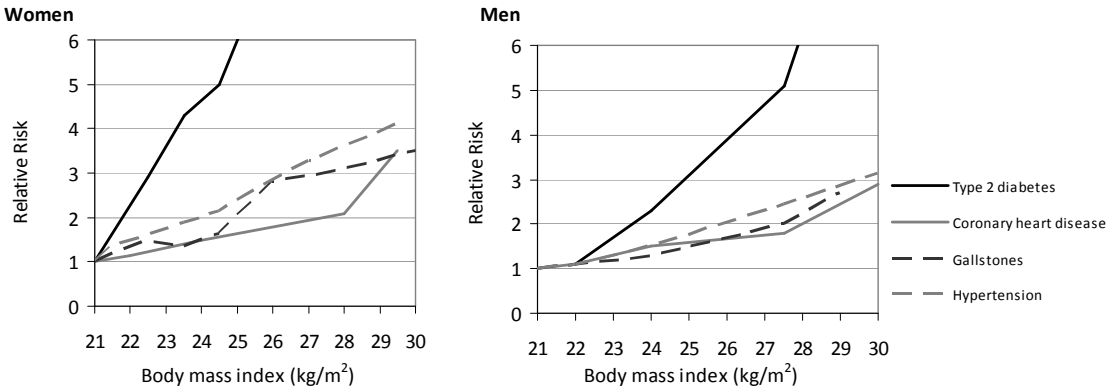

FIGURE 1.1 Association between body mass index up to 30 and the relative risk of type 2 diabetes, hypertension, coronary heart disease, and gallstones in adult women with 18 years of follow-up and in adult men with 10 years of follow-up. *Figures adapted from Willett et al 1999.*[20]

The risk of disease does not increase at a certain cut-off point of BMI, but the increase in risk already starts at levels of BMI below 25 kg/m^2,[21] as can be observed in Figure 1.1. In addition, not only weight status, but also weight gain is a predictor of disease risk, independent of initial weight status (Figure 1.2), indicating that weight gain prevention is of Public Health relevance across the whole BMI distribution including those within the normal weight range.

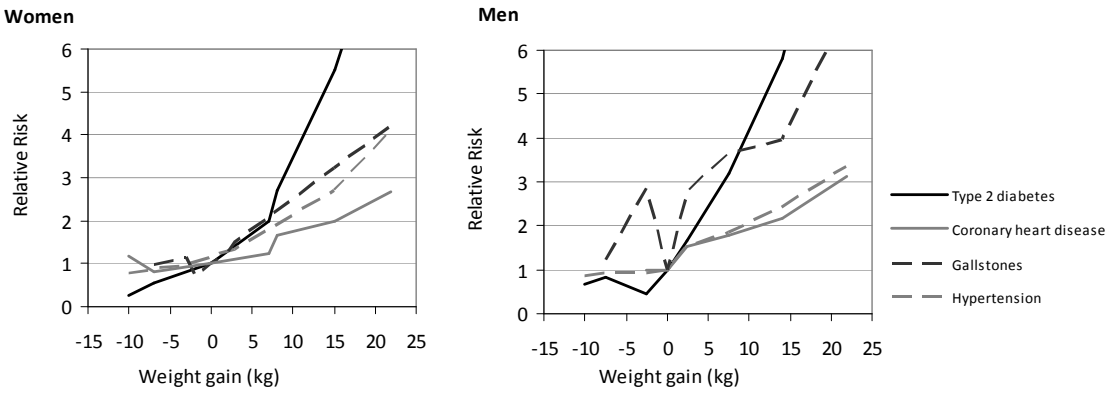

FIGURE 1.2 Association between weight gain during adulthood and the relative risk of type 2 diabetes, hypertension, coronary heart disease, and gallstones in adult women with 18 years of follow-up and in adult men with 10 years of follow-up. *Figures adapted from Willett et al 1999.*[20]

Concept of energy balance

Weight gain is always a result of a positive energy balance, meaning that the total energy intake exceeds the total energy expenditure (see figure 1.3). Excessive energy intake is stored in the body, mostly as fat mass. The two most important energy balance related behaviours are diet (for energy intake) and physical activity (as part of the energy expenditure). If not counterbalanced, changes in dietary intake and/or physical activity level therefore result in changes in energy balance. Minor changes in food intake or physical activity over longer periods can result in a change of several kilos in body weight and the prevalence of obesity in the population.[22] To get an impression of the order of magnitude, one additional can of soda each day will result in about 8 kilograms weight gain over a 5-years period.[23] The energy content of one can of soda (130 kcal)[24] is equal to the energy expenditure during about 35 minutes of walking the dog.[25]

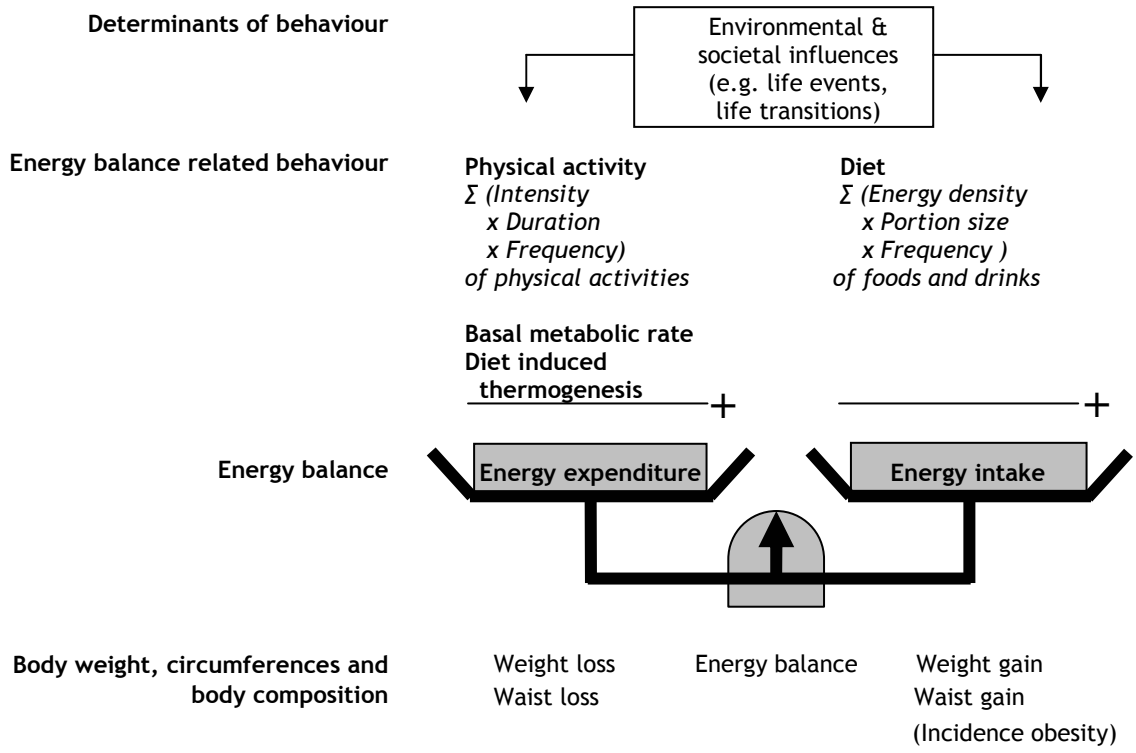

FIGURE 1.3 Systematic overview of the concept of energy balance.

Daily fluctuations in body weight mostly reflect changes in water content of the body and intestinal content. Sustained changes of larger magnitude and over longer periods of time are more certain to reflect change in fat or changes in muscle mass, as a result of resistance training, old age or sickness. Unintentional weight gain will nearly always reflect an increase in fat mass, accompanied by some increase in fat free mass. As a result, the body will need more energy to maintain the increased body weight. Therefore, when the changed behaviour, which caused the weight gain, is sustained over time, a new energy balance will be reached at a higher body weight. The same is true for losing body weight: a new energy balance is found at a lower body weight, with a lower energy requirement.

Although genetic variation may explain differences in susceptibility for weight gain between individuals,[26] genes cannot explain the increase in prevalence of overweight and obesity over the past decades, since the gene pool has not changed in this relatively short period of time. Whether or not the association between obesity and genes is considered strong, weight *gain* is always caused by a positive energy balance, resulting from changes in behaviour (e.g. as a result

of life events or life transitions). Such changes in behaviour are often supported by an environment making it easier to ingest large amounts of food and at the same time become less physically active, also referred to as an “obesogenic environment”.

Energy intake

Total energy intake is determined by three components of dietary intake: the energy density of the food, the portion size, and the frequency of eating moments. Energy density reflects the amount of energy (in kilocalories (kcal) or kilojoules (kJ); 1 kcal ≈ 4.187 kJ) per gram food and is determined by the macronutrients fat, carbohydrate, and protein, water content and fibre content. One gram of fat contains 9 kcal (37 kJ), which is more energy than one gram of carbohydrates (4 kcal (17 kJ)) or proteins (4 kcal (17 kJ)).[24] Therefore a fatty diet will contain more energy than a carbohydrate-rich diet, when portion sizes are equal. Another source of energy is alcohol, which contains 7 kcal (29 kJ) per gram.[24] Water contains no energy and will lower the energy density of foods and drinks. Dietary fibre can lower the energy density of the (solid) food, since the human body is not capable to digest dietary fibre and therefore cannot use its energy.

Energy expenditure

Total energy expenditure (TEE) consists of the resting metabolic rate (RMR; for maintenance of the body and thermoregulation), the thermic effect of feeding and the energy expenditure of physical activities. Resting metabolic rate is primarily determined by fat-free body mass, age, and gender, and (to a lesser extend) fat body mass and other determinants. Because of differences in body build and physical activity, total energy expenditure can vary considerably between persons. For an inactive subject, about 75-80% of TEE is resting metabolic rate, 10 % is diet-induced thermogenenis and 15-20% of TEE is expended through physical activity.[27,28]

Total energy spent on physical activities is determined by three components of physical activity: duration of the physical activities, intensity of the physical activities and frequency of the physical activities. A change in any of these components will change the total energy spent on physical activities. Cycling at a speed of 15 km/hour (energy cost about 5 times RMR) is more intensive than walking (3.5 times RMR), which is in turn more intensive than sitting at a desk (1.5 times RMR).

Activities with a low and moderate intensity are contributing more to total energy expenditure than highly intense activities, since low and moderate intense activities can be performed for a longer period.[29] But off course, with equal time spent, more energy is expended on higher intensity physical activities than on lower intensity physical activities.

Epidemiology

In the present thesis, lifestyle determinants of weight gain are described using observational data and epidemiological analyses. Groups of people (cohorts) were followed over time. Data on lifestyle behaviour were gathered through questionnaires or interviews, and body weight, waist circumference and other anthropometrical indices were measured during a physical examination. Therefore, (changes in) self-reported lifestyle could be studied in relation to changes in body weight over time.
Lifestyle behaviours associated with weight gain, however, are not necessarily (causal) determinants of weight gain. A specific behaviour may coexist with another behaviour that in fact is a determinant of weight gain. In this example the behavioural factor is a so-called confounder. In addition, associations may be different in different subgroups of the cohort, for example men and women. This is called effect modification. In epidemiological studies adjustments for confounding variables can be made. By studying subgroups, effect modification can be studied.

Cross-sectional versus longitudinal analyses
Until now, most studies on determinants of overweight and weight gain have been based on cross-sectional data. Cross-sectional analyses investigating determinants of body weight have several drawbacks. First, it is not possible to disentangle cause and effect since both factors are measured at the same time. Second, information bias is likely to occur: overweight subjects tend to underreport their dietary intake[30-33] and to over-report their physical activities[34,35] more than normal weight subjects. Using longitudinal data analyses to study determinants of overweight or weight gain is thought to be more valid, since the determinants are assessed before the outcome occurs. Moreover, information bias is thought to be less pronounced when studying changes in diet and physical activities, rather than the baseline behaviours only, under the assumption that reporting errors are similar at baseline and at follow-up.

Methods

In the Netherlands, only a few studies exist in which body weight, body height, and waist circumference are measured over a longer period of time and in which also information on energy intake and physical activity are collected in the same group of subjects. Such cohorts are crucial to study lifestyle determinants of weight gain in a real life setting. In this thesis we have used data of two Dutch cohort studies containing both objective measures of body weight, body height and waist circumference as well as self-reported lifestyle measures: i.e. the Amsterdam Growth and Health Longitudinal Study (adolescents and young adults; 13 years at baseline

followed for 24 years)[36] and the Doetinchem Cohort Study (young adults, recently retired subjects, and adults in general; age 20-59 years at baseline followed for 11 years).[37,38]

Aims of the present thesis

The main objectives of this thesis were to 1) identify and describe high-risk groups of weight gain and 2) to describe modifiable determinants of weight gain within the selected age groups (adolescents, young adults and recently retired persons).
High-risk groups were identified based on personal circumstances (such as household composition/work etc.), life events or life transition, and on anthropometric indices. Modifiable determinants of weight gain that we studied were (changes in) the main energy balance related behaviours, e.g. total dietary intake and physical activity level, or components of these two main behaviours, i.e. energy density of the food, portion size and eating frequency, and intensity, duration and frequency of physical activities.

Outline of the thesis

This thesis comprises studies that are related to the above described objectives. In chapter 2, BMI during adolescence is compared with skinfold thickness during adolescence as predictor of high percentage body fat at adult age. Chapter 3 reports on the relationship between aging and weight gain in a general adult population. Chapters 4 to 7 describe studies investigating determinants of weight gain in different age groups. In chapter 4, the associations of (changes in) energy intake and physical activity with changes in skinfold thickness and in body weight from adolescence into adulthood are described. In chapter 5, the impact of life transitions during young adulthood is reported in relation to changes in several anthropometrical indices. Then, baseline and changes in (aspects of) diet and physical activity are described in relation to weight gain in young adults (chapter 6) and in recently retired men (chapter 7).
Finally, in the general discussion in chapter 8, the methodology used in the presented studies is discussed and results are integrated into overall conclusions, implications for (clinical) practice and suggestions for future research.

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Adolescent skinfold thickness is a better predictor of high body fatness in adults than is body mass index – the Amsterdam Growth and Health Longitudinal Study

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Published in: *American Journal of Clinical Nutrition* 2007; **85**: 1533–1539.

ABSTRACT

Background: Body mass index (BMI) during adolescence is predictive of BMI at adult age. However, BMI cannot distinguish between lean and fat body mass. Skinfold thickness may be a better
predictor of body fatness.
Objective: The objective of this study was to evaluate the relations between BMI and skinfold thickness during adolescence and body fatness during adulthood.
Design: We included 168 men and 182 women from the Amsterdam Growth and Health Longitudinal Study, a prospective study that conducted 8 measurements of BMI and skinfold thickness between 1976 and 2000. BMI and skinfold thickness during adolescence were analyzed in relation to adult body fatness measured at a mean age of 37 years with dual-energy X-ray absorptiometry.
Results: None of the boys and 1.7% of the girls were overweight at baseline, whereas the prevalence of high body fatness during adulthood was 29% in men and 32% in women. At the ages of 12–16 years, skinfold thickness was more strongly associated with adult body fatness than was BMI. Age-specific relative risks for a high level of adult body fatness varied between 2.3 and 4.0 in boys and between
2.1 and 4.3 in girls in the highest versus the lowest tertile of the sum of 4 skinfold thicknesses. For the highest tertile of BMI, the relative risk varied between 0.8 and 2.1 in boys and between 1.3 and 1.8 in girls.
Conclusion: Skinfold thickness during adolescence is a better predictor of high body fatness during adulthood than is BMI during adolescence.

INTRODUCTION

Overweight during adolescence is a risk factor for overweight in adulthood[1-8] and for several chronic diseases, such as cardiovascular disease,[4,6,7] type 2 diabetes,[4] certain forms of cancer,[9] and adult mortality.[9,10] Over the past decades, the mean body mass index (BMI) and the prevalences of overweight and obesity in adolescents have increased dramatically.[11,12] The prevention of excessive weight gain during adolescence is crucial for the reduction of the pandemic of overweight and its related consequences.

Adolescence appears to be a critical period for the development of obesity.[13] Still, little is known about the identification of adolescents who are at increased risk of becoming overweight or obese at an adult age, and it is uncertain which measures should be used to identify adolescents at high risk of adult obesity. The widely used definitions of overweight and obesity in adolescents are based on age-specific BMI (in kg/m^2) cut-off points for overweight and obesity that correspond, cross-sectionally, with overweight (BMI ≥25 kg/m^2) and obesity (BMI ≥30 kg/m^2) at age 18 years.[14]

Several studies have shown tracking of overweight from adolescence to adulthood.[2,6-8,15] Overweight in these studies was invariably defined by BMI levels. Obesity, however, is defined as an excess of body fat, and it is the amount of this fatness that is associated with morbidities,[16] more strongly so than BMI.[17] Therefore, the assessment of obesity should ideally be based on measurement of body fatness.[18,19] Because BMI does not distinguish between fat mass and lean body mass,[16,20] a high tracking of BMI from adolescence to adulthood can also represent a high tracking of body build rather than fatness.[21] Skinfold thickness (i.e., a proxy for subcutaneous fat) is likely to be a better alternative for determining body fatness in children and adolescents[22-25] and for monitoring obesity in children.[18]

In the present longitudinal study, we will answer the following research questions: 1) How has BMI and the sum of 4 skinfold thicknesses (S4SF) developed during and since adolescence in adults with high body fatness and in adults with lower body fatness? and 2) Can high body fatness at an adult age be better predicted from BMI or from skinfold thickness during adolescence?

SUBJECTS AND METHODS

Amsterdam Growth and Health Longitudinal Study

The Amsterdam Growth and Health Longitudinal Study (AGAHLS) is an observational, longitudinal study with a total inclusion of 698 subjects. The initial goal of the AGAHLS was to describe the natural development of growth, health, and the lifestyle of adolescents and to investigate longitudinal relations between biological and lifestyle variables.[26-28] The study started in 1976 with boys and girls (mean age: 13 years) from the first and second form from 2 secondary schools in the Netherlands:
one in Amsterdam and one in Purmerend. Informed consent was obtained from the children and their parents, and all subjects agreed to participate in the study. The AGAHLS was approved by the Medical Ethics Committee of the VU University Medical Center in Amsterdam, Netherlands. The most recent measurement took place in the year 2000 when the subjects' mean age was 37 years. In the adolescent period (mean ages: 13–16 years), 4 annual measurements took place in autumn, followed by measurements at mean ages of 22, 28, 33, and 37 years in the spring. Pupils from the school in Amsterdam were invited for all 8 measurements. Pupils from the school in Purmerend were not invited for the measurements at mean ages of 22 and 28 years. At each measurement, anthropometric variables (body height, body weight, and skinfold thickness) were assessed. During the last examination, all participants were invited for a body-composition measurement by dual-energy X-ray absorptiometry (DXA).

Inclusion

For the present study, only participants who underwent a DXA scan in the year 2000 (*n* = 355) were eligible for inclusion, because we used this percentage body fat (PBF) measurement as our central outcome variable. Subjects who suffered from chronic diseases (*n* = 5) were excluded. Women who reported being pregnant during the measurement in 2000 underwent no DXA-scan and, therefore, were not included in the present study. If women had reported being pregnant during a previous measurement, the data from that particular measurement were excluded from the analyses. In total, 168 men and 182 women were included in the present study, from whom data for 2-8 (mean: 6.1) measurements were available.

Anthropometric measures

At each measurement, body height [with a wall-mounted stadiometer (Holtain, Crymych, United Kingdom), to the nearest 0.1 cm], body weight [with a spring balance (van Vucht, Amsterdam, Netherlands), to the nearest 0.1 kg], and S4SF [biceps, triceps, subscapula, and suprailiac to the nearest 0.1mm with a Harpenden caliper (Holtain)] were assessed according to standard procedures.[29] BMI was calculated as body weight (kg) divided by body height squared (m^2). The assessment of overweight during adolescence was based on BMI levels as recommended by the

International Obesity Task Force (IOTF).[14] Overweight at an adult age was defined as a BMI ≥25 kg/m^2.[30] S4SF was calculated and expressed in mm. A whole-body DXA scanner (Hologic QDR-2000, software version V5.67A; Hologic Inc, Waltham, MA) was used to assess PBF at the mean age of 37 years. The PBF variable was dichotomized: participants were classified as having high body fatness if their PBF was equal to or exceeded 25% for men and 35% for women. We chose these particular cut-off values for high body fatness because they are simple and within the ranges proposed in earlier publications on this topic.[31-33] For clarity, the term *overweight* is used when referring to BMI level, and the terms high and low body fatness when referring to the PBF measured by DXA.

Analyses

The development of BMI and S4SF from mean age 13 to 37 years was calculated for the group of subjects with high and low body fatness at an adult age by using the generalized estimating equations. This method adjusts for the correlation between repeated observations taken in the same subject and has the advantage of handling longitudinal data of varying numbers of subjects and observations unequally spaced in time. In the generalized estimating equations analyses, an exchangeable correlation structure was assumed, and analyses were adjusted for age. To estimate the development of each variable investigated from mean age 13 to 37 years, time was treated as a categorical variable, and the results were plotted. To investigate whether the development of

BMI and of the S4SF was different for men and women between adolescence and adulthood and between groups with a high and a lower PBF, we tested the interaction of age and sex and of age and PBF group in a longitudinal regression model. Additionally, at the mean age of 37 years, correlations were calculated between BMI, S4SF, and (the continuous) PBF. Finally, the absolute and relative risk of having high body fatness at an adult age was calculated for age- and sex-specific tertiles of BMI, S4SF, and separate skinfold thicknesses (biceps, triceps, subscapula, and suprailiac) at calendar ages 12-16 years. To analyze whether the relative risks, based on BMI and S4SF tertiles during adolescence, of becoming an adult with high body fatness varied with age during adolescence, we tested the interactions of BMI and age and S4SF and age in a logistic regression analysis. All analyses were performed for men and women separately with the use of SAS version 9.1 (SAS Institute Inc, Cary, NC).

RESULTS

None of the boys and 3 of the girls (1.7%) were overweight on the basis of current international BMI cut-off points when first measured at the mean age of 13 years (Table 2.1). At the last follow-up measurement, at the mean age of 37 years, 41.7% of the men and 23.6% of the women had a BMI ≥25 kg/m^2, and 29.2% of the men had a PBF ≥25% and 32.4% of the women had a PBF ≥35%. The interactions of age and sex on the development of BMI and S4SF were highly statistically significant (p <0.001), which indicated that the increase in both BMI and S4SF from adolescence to adulthood is steeper in men than in women. After stratification for sex, the increase in BMI and S4SF from adolescence to adulthood was higher in subjects with a high PBF than in subjects with a lower PBF at an adult age (p for interaction <0.001) in both men and women.

TABLE 2.1 General characteristics of the study population

	Men	Women	p for difference[1]
Baseline, 1976			
N	163	172	
Age (years)	12.9 ± 0.6[2]	12.9 ± 0.6	0.77
BMI (kg/m^2)	17.0 ± 1.3	17.7 ± 2.0	<0.01
Overweight (%)[3]	0.0	1.7	
S4SF (mm)[4]	26.9 ± 8.6	36.3 ± 12.5	<0.01
Last follow-up, 2000			
N	168	182	
Age (years)	36.5 ± 0.6	36.6 ± 0.6	0.47
BMI (kg/m^2)	24.7 ± 2.8	23.4 ± 3.2	<0.01
Overweight (%)[3]	41.7	23.6	
S4SF (mm)	47.2 ± 15.8	55.2 ± 19.2	<0.01
Body fat (%)	21.5 ± 6.3	32.2 ± 6.7	<0.01
High body fatness (%)[5]	29.2	32.4	

1 *t*-test.
2 mean ± SD (all such values).
3 The percentage of subjects who were overweight at baseline was derived from age-dependent International Obesity Task Force BMI cut-offs for overweight in adolescents.[14] At follow-up, overweight was defined as a BMI ≥25 kg/m^2.
4 S4SF, sum of 4 skinfold thicknesses.
5 Percentage of subjects with high body fatness at the last follow-up: ≥25% for men and ≥35% for women.

The longitudinal development of BMI and S4SF in men and women from mean age 13 to 37 years is shown in Figure 2.1. The mean BMI of men and women who were classified as having high body fatness at a mean age 37 years was higher than the mean BMI of the men and women with lower body fatness at the mean age of 37 years, at each year of measurement. Also, at each year of measurement, the mean S4SF was significantly higher in both the men and women with high body fatness than in the group with lower body fatness at the mean age of 37 years. Differences in BMI and S4SF between the groups became more pronounced with increasing age, which was confirmed by significant interactions (p <0.0001) of age and PBF group on BMI and S4SF. The group of men with high adult body fatness had a greater increase in body weight (47.3 compared with 38.3 kg), BMI (9.7 compared with 6.9), and S4SF (33.3 compared with 15.0 mm) than the group of men with lower adult body fatness (p <0.0001). Also, women with a high PBF had a greater increase in body weight (27.2 compared with 20.2 kg), BMI (7.5 compared with 4.8), and S4SF (29.5 compared with 14.1 mm) between the mean ages of 13 and 37 years (p <0.0001) than women with a lower PBF at an adult age.

In general, the correlations of BMI and skinfold thicknesses at baseline and BMI and skinfold thicknesses at later measurements became lower with a longer follow-up. In men, correlations for body weight decreased from 0.93 between the mean ages of 13 and 14 years to 0.33 between the mean ages of 13 and 37 years. These correlations for BMI and S4SF decreased from 0.89 to 0.40 and from 0.81 to 0.35, respectively. In women, these correlations decreased from 0.94 to 0.44 for body weight, from 0.93 to 0.51 for BMI, and from 0.87 to 0.35 for S4SF. At the mean age of 37 years, correlation coefficients between S4SF and PBF for men (r = 0.84) and women (r = 0.79) were higher than the correlation coefficients between BMI and PBF (r = 0.67 and 0.72 for men and women, respectively).

The risk of having high adult body fatness was not always higher in adolescents with a BMI in the highest tertile than in those with a BMI in the lowest tertile (Table 2.2 and Table 2.3). However, in the same adolescents, having an S4SF in the highest tertile was always associated with a higher risk of having high adult body fatness. In women, having a BMI or S4SF in the highest tertile was always associated with a higher risk of becoming an adult with high PBF, although statistical significance was not always reached in analyses regarding BMI. The relative risks of the high versus the low S4SF tertile ranged from 2.3 to 4.0 in boys and from 2.1 to 4.3 in girls aged 12-16 years. For BMI, these relative risks were smaller (0.8 to 2.1 in boys and 1.3 to 1.8 in girls) and not statistically significant most of the time.

Relative risks for the high versus the low tertiles of separate skinfold thicknesses were highest for subscapula skinfold thickness in boys (range: 2.4-8.5) and for biceps skinfold thickness in girls (range: 2.3-4.5) (data not shown). The interaction of BMI tertiles and age was significant (p

<0.10) in boys, which indicated that the relative risks of BMI tertiles with high body fatness at an adult age decreased from age 12 to 16 years. The interactions of BMI and age in girls (p = 0.90) and of S4SF and age in both boys (p = 0.90) and girls (p = 0.61) were not statistically significant.

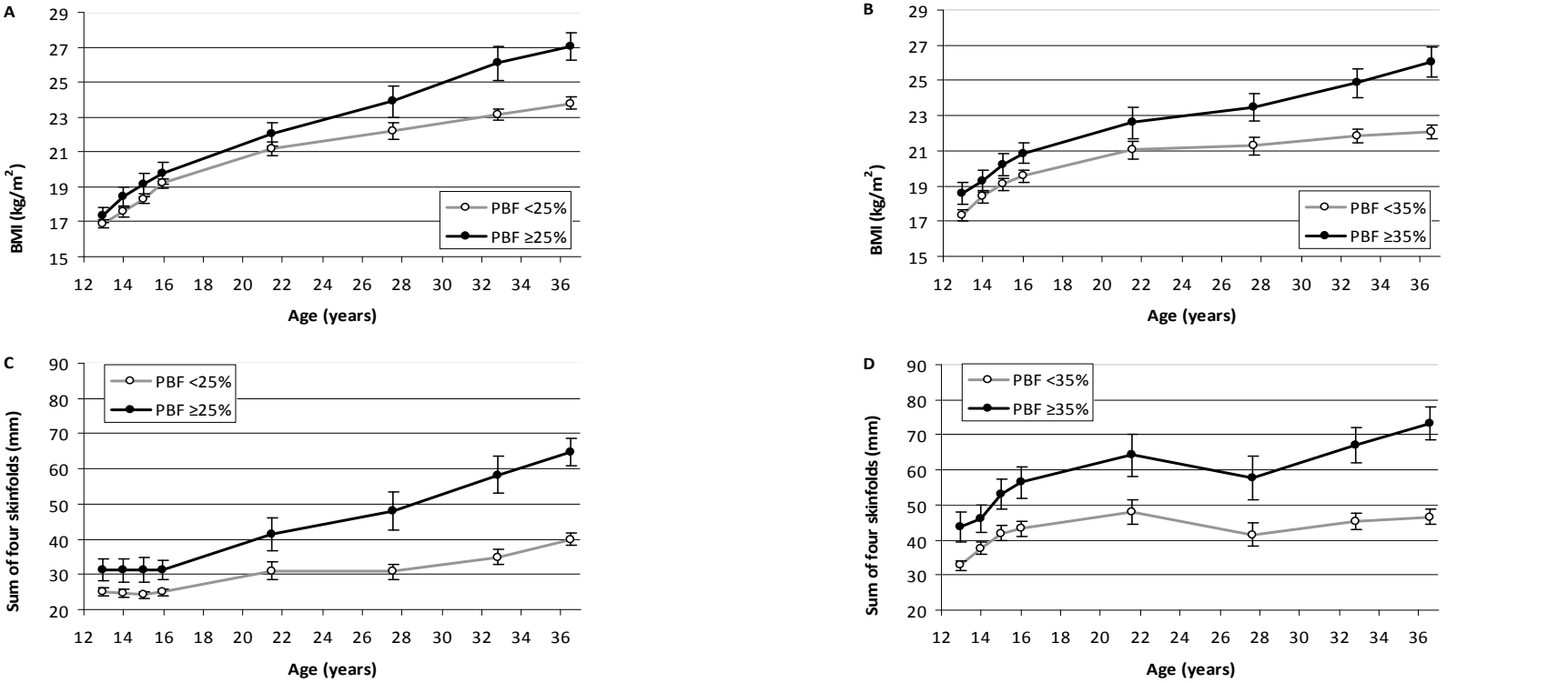

FIGURE 2.1 Mean longitudinal development of BMI and sum of 4 skinfold thicknesses (S4SF) in men (A and C) and women (B and D) at mean ages ranging from 13 to 37 years, stratified by high and low percentage body fat (PBF) at a mean age of 37 years. Error bars represent 95% confidence limits. The data were derived by using generalized estimating equations and were adjusted for age. The number of subjects at each age is as follows: 13 years (*n* = 163 M and 172 F), 14 years (*n* = 134 M and 156 F), 15 years (*n* = 130 M and 152 F), 16 years (*n* = 131 M and 154 F), 22 years (*n* = 64 M and 72 F), 28 years (*n* = 68 M and 74 F), 33 years (*n* = 152 M and 173 F), and 37 years (*n* = 168 M and 182 F). Over all of the years, the proportion of adults with high PBF was 28–31% for men and 30–32% for women, except for years 1985 and 1991 (mean ages: 22 and 28 years), at which time the values were 38% and 37%, respectively. The sex × age × PBF interaction was significant (*P* <0.0001) for both the development of BMI and S4SF. The age x sex interaction for both variables was also significant (*P* <0.001). Within each sex, the PBF × age interaction was significant for both variables (*P* <0.001).

TABLE 2.2 Absolute risk (AbR) and relative risk (RR) of having a high percentage body fat (≥25% for men) at the age of 37 years for tertiles of adolescent BMI and adolescent sum of 4 skinfolds thicknesses (S4SF), stratified by age.

	Age 12 years (n_{BMI}=101, n_{S4SF}=100)			Age 13 years (n=131)			Age 14 years (n=132)			Age 15 years (n=130)			Age 16 years (n=60)		
			95% CL			95% CL			95% CL			95% CL			95% CL
	AbR	RR	of RR	AbR	RR	of RR	AbR	RR	of RR	AbR	RR	of RR	AbR	RR	of RR
Low BMI[1]	0.21	Ref		0.20	Ref		0.23	Ref		0.26	Ref		0.30	Ref	
Medium BMI	0.24	1.18	0.48;2.88	0.21	1.02	0.45;2.33	0.27	1.20	0.58;2.48	0.23	0.89	0.42;1.87	0.20	0.67	0.22;2.01
High BMI	0.44	2.14	1.00;4.59	0.41	2.00	1.01;3.96	0.41	1.80	0.94;3.45	0.33	1.27	0.65;2.48	0.25	0.83	0.30;2.29
p for trend[2]	0.04			0.03			0.07			0.47			0.72		
Low S4SF	0.12	Ref		0.16	Ref		0.22	Ref		0.14	Ref		0.15	Ref	
Medium S4SF	0.29	2.43	0.84;6.98	0.16	0.98	0.37;2.55	0.19	0.88	0.38;2.01	0.14	0.98	0.34;2.79	0.15	1.00	0.23;4.37
High S4SF	0.48	4.00	1.50;10.70	0.50	3.07	1.47;6.43	0.50	2.30	1.23;4.29	0.53	3.83	1.73;8.47	0.45	3.00	0.95;9.48
p for trend[2]	<0.01			<0.01			<0.01			<0.01			0.03		

CL, confidence limit.

1 Low, medium, and high designations for BMI and S4SF indicate age-specific tertiles of distribution.

2 Logistic regression analysis.

The interaction of BMI tertiles and age was significant (P <0.10). The interaction of S4SF tertiles and age was not significant (P = 0.90).

TABLE 2.3 Absolute risk (AbR) and relative risk (RR) of having a high percentage body fat (≥35% for women) at the age of 37 years for tertiles of adolescent BMI and adolescent sum of 4 skinfold thicknesses (S4SF), stratified by age.

	Age 12 years (n=104)			Age 13 years (n=152)			Age 14 years (n=151)			Age 15 years (n=147)			Age 16 years (n=74)		
			95% CL			95% CL			95% CL			95% CL			95% CL
	AbR	RR	of RR	AbR	RR	of RR	AbR	RR	of RR	AbR	RR	of RR	AbR	RR	of RR
Low BMI[1]	0.26	Ref		0.31	Ref		0.22	Ref		0.24	Ref		0.24	Ref	
Medium BMI	0.29	1.14	0.53;2.46	0.22	0.70	0.36;1.36	0.27	1.25	0.63;2.48	0.31	1.25	0.65;2.39	0.21	0.87	0.30;2.47
High BMI	0.46	1.78	0.91;3.47	0.41	1.31	0.78;2.21	0.40	1.82	0.98;3.39	0.45	1.83	1.03;3.28	0.40	1.67	0.71;3.89
p for trend[2]	0.08			0.29			0.05			0.03			0.21		
Low S4SF	0.17	Ref		0.24	Ref		0.18	Ref		0.22	Ref		0.12	Ref	
Medium S4SF	0.30	1.82	0.74;4.45	0.22	0.92	0.45;1.88	0.16	0.87	0.37;2.08	0.22	0.98	0.47;2.05	0.21	1.74	0.47;6.48
High S4SF	0.54	3.26	1.48;7.19	0.50	2.13	1.21;3.75	0.56	3.11	1.64;5.90	0.56	2.51	1.41;4.46	0.52	4.33	1.40;13.37
p for trend[2]	<0.01			<0.01			<0.01			<0.01			<0.01		

CL, confidence limit.

1 Low, medium, and high designations for BMI and S4SF indicate age-specific tertiles of distributions.

2 Logistic regression analysis.

The interaction of BMI tertiles and age ($P = 0.90$) and of S4SF tertiles and age ($P = 0.61$) were not significant.

DISCUSSION

To our knowledge, this is the first longitudinal study that relates adolescent BMI and skinfold thickness to adult body fatness, measured by DXA. Significant differences were observed in mean BMI and S4SF values during adolescence, and in the development of BMI and S4SF into adulthood, between groups of subjects with high and low adult body fatness. Both S4SF and separate skinfold thicknesses for adolescents were better predictors of high body fatness at an adult age than was adolescent BMI.

Many articles have been written on the topic of overweight and obesity in children, adolescents, and adults. In 1997, Power et al[34] presented a review of studies that related adolescent adiposity to adult adiposity. In this review, all studies related adolescent BMI (or weight or relative weight) to adult BMI or adolescent skinfold to adult skinfold thickness. No comparisons have been made between BMI and skinfold thickness in their ability to predict adult adiposity. Also, several studies have been conducted on the same topic since 1997. In contrast with the present study, most of the published studies were *1*) only cross-sectional,[35-44] *2*) longitudinal and related childhood or adolescent BMI to adult overweight (based on BMI),[2,6,45,46] or *3*) longitudinal and related childhood or adolescent BMI to adult body fatness (or adiposity) based on either skinfold thickness or DXA.[21,47-50] The overall conclusion of these studies is that adolescent BMI is strongly associated with adiposity measures (BMI, skinfold thickness, or DXA) during adolescence and adulthood. However, to our knowledge, no studies have been published that compared adolescent BMI with adolescent skinfold thickness in relation to adult PBF measured by DXA. We found one study in which both adolescent BMI and skinfold thickness were related to adult adiposity.[50] The results of that study showed a slightly stronger association between childhood (and adolescent) triceps skinfold thickness and adult adiposity. However, adult adiposity was based on skinfold thickness and not on DXA.

Although it is not new that skinfold thicknesses relate better to fatness than does BMI, the present study does add new information on this topic. That is, in a longitudinal fashion, the relative risk of becoming an adult with high body fatness based on adolescent BMI was not significant in most of the age- and sex-specific strata. This is in contrast with the mostly significant relative risks based on S4SF in adolescents.

On the basis of our study, it is too early to conclude that the measurement of skinfold thickness should be the standard and the only procedure in clinical practice. Other measures, such as waist circumference, may be useful in predicting health outcomes, and innovative measurements of body fat, such as DXA and 3-dimensional laser techniques, are worthy of study. Ideally, measurements and cut-off points are distinctive in predicting an increased risk of morbidity,

disability, or mortality. Such studies in children are rare, because such health outcomes are to be expected decades after baseline measurements are made. Because waist circumference and DXA measurements were introduced only recently for use in adolescents, we will have to wait another few decades for longitudinal relations between these measures during adolescence and PBF at an adult age.

The development of triceps skinfold thickness during adolescence is dependent on sex, but BMI also develops differently in boys and girls during adolescence. This was one of the reasons that we conducted our analyses in boys and girls separately. Of course, on the basis of this fact, one should create guidelines for skinfold thickness cut-off points for boys and girls separately, but the same is true for BMI. Note that it is not our intention to conclude whether a single skinfold thickness measure is a better measure than is another skinfold thickness. Instead, we wanted to demonstrate that even a single skinfold thickness during adolescence is already a better predictor of PBF at an adult age than is BMI during adolescence.

At baseline (mean age: 13 years), none of the boys and only 3 of the girls were classified as overweight (or having a high risk of becoming overweight in adulthood) on the basis of the IOTF cut-offs,[14] whereas the prevalence of high body fatness at an adult age in our population was 29% in men and 32% in women. Indeed, all 3 girls who were classified as being overweight at baseline became overweight as adults with a high PBF. However, most of the adults with a high PBF at age 37 years were not classified as overweight at age 13 years on the basis of IOTF BMI cut-off points. This finding agrees with several other studies, which showed a high specificity but a low sensitivity for BMI during adolescence as a predictor of overweight.[22,51,52]

Sardinha et al[23] conducted a cross-sectional study of the predictability of body fatness (by DXA) from BMI, triceps skinfold thickness, and upper arm girth in adolescent boys and girls. Using receiver operator characteristic analysis, they concluded that, of these 3 measures, triceps skinfold thickness was the best measure for predicting body fatness in both boys and girls between 10 and 15 years of age.[23] In the present study, at age 37 years, S4SF was a better predictor of body fatness than was BMI. When we looked at separate skinfolds, body fatness was strongest correlated with the subscapula skinfold in men and the triceps skinfold in women (data not shown). But more interestingly, adolescent skinfold thickness showed, longitudinally, also to be a better predictor of high adult body fatness, in comparison to adolescent BMI: adolescents in the highest tertile of the S4SF distribution had about 2 times the relative risk of becoming an adult with high body fatness, in comparison with adolescents in the highest tertile of the BMI distribution. Moreover, in boys aged 16 years, a higher BMI was not associated with a higher risk to become an adult with high body fatness.

In our study population, both S4SF and adolescent skinfold thickness were better predictors of adult body fatness than was adolescent BMI. Adolescent subscapular skinfold thickness was the best predictor of adult body fatness in boys, and adolescent biceps skinfold thickness was the best predictor of adult body fatness in girls. Both of these single skinfold thickness measures were better predictors of adult body fatness than was S4SF. This could indicate that only one skinfold thickness needs to be measured in adolescents to obtain a good prediction measure for adult body fatness. However, this has not been investigated before. Generally, if only one skinfold is measured, it is the triceps skinfold; in other cases, triceps skinfold thickness is measured in combination with subscapular skinfold thickness to determine body fatness at the time of measurement. As far as we know, single skinfold thicknesses have not been tested to predict body fatness later in life. Therefore, more and larger studies are needed to confirm our results before any recommendations can be made about the measurement of only one single skinfold in adolescents to predict adult body fatness.
Hughes et al[18] had already recommended in 1997 that skinfold thickness measurements should be used to monitor obesity in children, because of its higher sensitivity. The advantage of using BMI as a screening tool to assess overweight in adolescence is that weight and height are readily available and easy to measure.[53] However, the measurement of skinfold thickness is also feasible for use in the field, is relatively inexpensive, and is generally acceptable to the child; furthermore, the equipment needed for such measurement is portable.[19]

A possible drawback of the present study was the relatively low representativeness of the study population for the general Dutch population. Most of the subjects (96%) reported to have Western European parents. The remaining 4% of the subjects had 1 or 2 parents of Asian (Indonesia, Java, and China) or Caribbean (Suriname, Curaçao, and Netherlands Antilles) ethnicity. The educational level of the participants was rather high compared with that of the general Dutch population: 53.6% completed a higher vocational or university education. In comparison, 26.4% of the general Dutch population of the same birth years had completed a higher vocational or university education.[54] This difference can be ascribed to the selection of 2 schools in Amsterdam and Purmerend, which were both schools for higher secondary education. Furthermore, the subjects who participated during adolescence, but not at an adult age, had, on average, a higher BMI and S4SF at a mean age 13 years than did subjects who did participate at a mean age 37 years. For women, these differences at adolescence were statistically significant. However, none of our data indicate that these drawbacks influenced our results or conclusions. Because we looked at tertiles of BMI and the sum of skinfold thicknesses, only the thresholds for the tertiles would have shifted upward when more overweight adolescents were included. This would have no or only a small effect on the relative risks, because overweight during adolescence predicts overweight at adulthood. Therefore, the associations found between

adolescent BMI and S4SF and adult body fatness in our study population would probably not have been different in the original cohort or in the general population.

Prevention of excessive (fat) weight gain in critical periods of obesity development could prevent, or temper, adult overweight and its complications. In the present study, we showed that both male and female adults with high body fatness had higher skinfold thicknesses during adolescence. Therefore, the measurement of adolescent skinfold thickness would yield a simple risk indicator for high adult body fatness, better than the measurement of adolescent BMI. Adolescents with large skinfold thicknesses could be a good target group for secondary weight-gain prevention programs. Finally, as early as the age of 13 years, a difference in mean skinfold thickness was present between adolescents who did and did not become adults with high body fatness, which indicates that weight gain prevention should start before adolescence.

In conclusion, high adult body fatness is better predicted by adolescent skinfold thickness than by adolescent BMI. Skinfold thickness should therefore be used as the preferred screening tool to determine which adolescents are at increased risk of becoming adults with high body fatness. Weight-gain prevention programs should therefore focus on adolescents with large skinfold thicknesses. Appropriate cut-off values for skinfold thickness still need to be assessed in larger populations.

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Age, period and cohort effects on body weight and body mass index in adults – the Doetinchem Cohort Study

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Published in: *Public Health Nutrition* 2008; **24**: 1–9.

ABSTRACT

Objective To study the development of body weight with aging, in a general adult population, taking into account possible period and cohort effects.
Design A prospective cohort study with 11 years of follow-up. At baseline and after 6 and 11 years, body weight and height were measured.
Setting The Doetinchem Cohort Study, consisting of inhabitants of Doetinchem, a town in a rural area of The Netherlands.
Subjects In total 4070 healthy men and women aged 20-59 years at baseline.
Results Increase in BMI with ageing was less profound based on cross-sectional data than based on longitudinal data. More recent born cohorts had a higher BMI at a given age than cohorts that were born earlier. Increase in mean BMI with ageing was observed in all age groups and was similar for groups with a different educational level. Highest increase in BMI over 11 years was observed in the youngest group, aged 20-29 year at baseline (2.2 [95%CL: 2.0;2.3] kg/m^2) and lowest increase in the oldest group, aged 50-59 year at baseline (1.1 [1.0;1.3] kg/m^2).
Conclusions Findings of this study using longitudinal data suggest that increase in BMI with ageing is underestimated in all age groups by studying cross-sectional data only. Further, weight gain is present at all educational levels and does not stop at middle age.

INTRODUCTION

It is often observed that levels of body mass index (BMI) and prevalence of obesity increase with age until age 60-70 years and decline thereafter. These observations are usually based on cross-sectional studies.[1-5] However, such age-BMI relationships may result from mixed effects of ageing per se, characteristics of subsequent birth-cohorts and selective survival. Studies based on successive cross-sectional surveys, show that younger birth cohorts have a higher mean BMI at the same age compared to older cohorts,[2,6-10] and that the prevalence of overweight and obesity increases over time, independent of age.[11-15]

Increases in BMI with ageing in cross-sectional studies may be the result of age and cohort effects, while increases in BMI with ageing in longitudinal studies may include period effects. Age effects on body weight and BMI are due to behavioural and physiological changes within individuals[16] that occur as a result of ageing, e.g. the lowering in energy requirement at rest with ageing. Period effects are due to population-wide changes in behaviours and other exposures among individuals independent of ageing[16] during the study period and affect all birth cohorts simultaneously, e.g. a change in legislation during the study period that promotes active transport. Cohort effects are influences of population-wide exposures that took place before the study period, e.g. secular differences in environment that may affect behaviour during later life. Disentangling age, period, and cohort effects on BMI is crucial to assess future trends in BMI and accompanying health disorders.[17]

Evaluation of age, period and cohort effects on BMI requires longitudinal assessment with repeated measures in the same individuals. There are only a few studies that allow this evaluation. We found four studies that have evaluated age and/or cohort effects on BMI on longitudinal data in recent time periods. These studies show that people who are born later have higher BMI at the same attained age[18,19] and that younger people gain more weight as they become older compared to older people.[18-21] These studies were conducted on data from either only young (18-30 years)[20] or middle-aged (45-64 years)[18] adults, based on self-reported data on body weight,[19,21] or in one gender only.[19]

In the present study, impact of age, period and cohort effects on BMI will be evaluated in the Doetinchem Cohort Study (DCS). This is a population-based cohort study in the Netherlands, in which subjects aged 20 to 59 years at baseline are followed over a period of 11 years between 1987-1991 and 1998-2002. For all subjects, body height and weight were measured by trained staff.

METHODS

Sample

Based on an age- and sex- stratified sample survey from the civil registries of the Dutch town Doetinchem, 20 155 inhabitants aged 20-59 years were invited to visit the municipal health centre to participate in the 'Monitoring Project on Cardiovascular Disease Risk Factors' between 1987 and 1991.[22] From the participants in this first examination (n=12 405, participation rate 62%), a random sample of 7769 was invited for a second examination (1993-1997) for budgetary reasons.[23] This random sample is considered as the basis for the prospective Doetinchem Cohort Study (DCS). Participants who actively refused to participate in the second examination were not invited for the third examination (1998-2002). In the second examination, 6118 subjects were examined (participation rate 79%)[24] and 6579 participants were again invited for a third examination between 1998 and 2002, of whom 4917 were examined (participation rate 75%). The cohort profile of the DCS is described in detail elsewhere.[23]

In total, 4636 subjects fully completed all three examinations. For the purpose of the present study, subjects who suffered from cancer (n=220), diabetes (n=117) and/or cardiovascular diseases (n=158) were excluded, as were women who reported to be pregnant during any of the examinations (n=116). This left a total of 4070 subjects, 1988 men and 2082 women, for the present study.

Measures

Body weight and height were measured at the municipal health centre at all three examinations, wearing light indoor clothing with emptied pockets and without shoes. Height was measured with a wall-mounted stadiometer to the nearest 0.5 cm. Body weight was measured with a balance beam scale to the nearest 0.5 kg. To adjust for light indoor clothing, 1 kg was subtracted from the measured weight. Body mass index (BMI) was calculated as weight divided by height squared (kg/m^2).

Demographic characteristics and medical history of chronic diseases were collected using standardized questionnaires,[24] by which also educational level and smoking status were recorded. Educational level was assessed as the highest level of completed education at follow-up and classified into three categories: low (intermediate secondary education or less), moderate (intermediate vocational or higher secondary education) and high (higher vocational education or university). Smoking status was defined as being a non-smoker (i.e. smoking less than one cigarette per month), an ex-smoker or a smoker at the time of measurement, based on a question about current cigarette smoking. Never smokers were defined as subjects who reported to be a non-smoker at all three examinations. We have no information on country of birth or ethnicity per se, but we do have information on nationality. Since over 98% of the participants were Dutch, we did not take nationality into account in our analyses.

Analyses

To evaluate age, period and cohort effects on BMI, four approaches were used.

Cross-sectional approach

The mean BMI of subjects in 10-years age groups was compared *within* the cross-sectional surveys. In these analyses, age groups (20-29, 30-39, 40-49, 50-59 and 60-69 years) were not included when the range of the age group was not full within the survey, e.g. the age group 20-29 years was not included in the second measurement round, since youngest participants were aged 26 years. In the figures based on this (multiple) cross-sectional approach, average BMI in the 10-years age groups within the measurement rounds was plotted by measurement round.

Time series

The mean BMI of subjects within the 10-years age groups was compared *across* periods. In these analyses, age groups were only included when all ages within the range of the age group (20-29, 30-39 40-49 50-59 and 60-69 years) were present in all three surveys, with one exception: the youngest age group in 1998-2002 was aged 31-39 years.

Longitudinal approach

The mean BMI of subjects within different cohorts was followed over time using longitudinal data. This third approach was also applied for mean body weight. Cohorts were defined based on age at baseline (20-29, 30-39, 40-49, and 50-59 years). In the figures based in this longitudinal approach, average BMI or body weight in these four cohorts was plotted against the average age in the cohorts during the measurements.

Random intercept models In order to quantify the independent associations of age, period and cohort with BMI, random intercept model analyses were used. BMI and age were entered as continuous variables. Period (baseline, 6 and 11 years follow-up) and cohort (age groups at baseline: 20-29, 30-39, 40-49 and 50-59 years) were entered as categorical variables. First, age and period were related to BMI, representing a cross-sectional perspective. Second, age and cohort were related to BMI, representing a longitudinal perspective. In these models, age was entered as both a linear and a quadratic term, to take into account a potentially exponential relation between age and BMI. If the quadratic term was not statistically significant (two sided at $p = 0.10$), it was excluded from the model. To evaluate potential differences in BMI development with age within different periods, an interaction term of age and period was entered in the age-period model. To evaluate potential differences in BMI development with age for different birth cohorts, an interaction term of age and cohort was entered in the age-cohort model. If these interaction terms were not statistically significant (two sided at $p = 0.10$), they were excluded from the model.

The increase in BMI with ageing, adjusted for, respectively, period and cohort effects, was plotted. For these figures, the average BMI level of the group aged 20-29 years in 1987-1991 was used as reference point for the intercept at age 20: BMI 23.4 kg/m^2 for men and BMI 22.5 kg/m^2 for women.

All analyses were carried out for men and women separately. Analyses on longitudinal data were additionally stratified for level of education in order to study potential influences of the level of education on changes in BMI with ageing. Differences in (changes in) BMI between the different levels of education were tested by analyses of variance at p=0.05. Analyses on longitudinal data were also performed for never-smokers only, in order to study changes in BMI with ageing independent of influences of (changes in) smoking behaviour. All analyses were carried out using SAS version 9.1 (SAS Institute Inc, Cary, NC).

RESULTS

At baseline, mean age was 39.8 years and mean BMI was 24.4 kg/m^2. In total, 37.9% of the participants had a BMI ≥25 kg/m^2, and 5.0% had a BMI ≥30 kg/m^2. After 11 years of follow-up, the mean BMI had increased to 26.0 kg/m^2, and the prevalence of BMI ≥25 kg/m^2 and BMI ≥30 kg/m^2 to 57.8% and 13.1%, respectively. Cumulative incidence of BMI ≥30 kg/m^2 was 8.3% and 9.7% over the 11-years follow-up period among men and women, respectively, and was highest for the men and women aged 50-59 years at baseline (9.5% and 14.1%, respectively). Overall, 31.7% of the participants were smokers at baseline, at the second examination the proportion smokers was 29.3% and at the third examination 26.4%. In older age groups, participants were less tall, were more often non-smokers, had more often a lower educational level and were more often overweight and obese than participants in younger age groups (Table 3.1).

TABLE 3.1 General baseline characteristics of the study population

Age at baseline	20-29 years		30-39 years		40-49 years		50-59 years	
	Men	Women	Men	Women	Men	Women	Men	Women
N	332	365	667	706	619	630	370	381
Age (years), mean	25.6	25.5	35.1	35.0	44.2	44.1	54.3	54.4
Height (cm), mean	182.0	168.1	180.5	166.7	178.2	165.6	176.9	164.3
Non smokers*, %	63.0	64.7	65.2	66.4	67.5	70.6	77.8	78.2
Level of education**								
Low, %	28.6	35.1	34.6	50.6	40.2	61.7	47.6	73.0
Moderate, %	49.4	49.0	34.3	24.9	30.4	18.6	24.3	14.2
High, %	22.0	15.9	31.0	24.5	29.4	19.7	28.1	12.9
Overweight (25≤BMI<30	24.4	12.6	36.0	21.1	48.1	28.3	50.8	42.0
Obesity (BMI≥30 kg/m^2),	1.8	2.5	3.0	3.1	5.5	7.5	6.2	10.8

* Non smoking in all three examinations

** Highest attained level during follow-up

Multiple cross-sectional data

Mean BMI was higher in older age groups in all periods (Figure 3.1), except for men aged 60-69 years, for whom the mean BMI was not different from the mean BMI of men aged 50-59 y in the period 1998-2002 (Figure 3.1a).

Time series

When the mean BMI of the 10-year age groups was compared across periods, the mean BMI was higher in more recent periods for every age category and in both men and women (Table 3.2, Figure 3.3).

Longitudinal data

Highest increase in body weight and in BMI during the 11-years follow-up was observed in the youngest age category (7.3 kg or 2.1 kg/m^2 for men and 6.7 kg or 2.2 kg/m^2 for women). Smallest increases in mean body weight and mean BMI were observed in the oldest group of adults, aged 50-59 y at baseline (2.3 kg or 0.9 kg/m^2 for men and 2.8 kg or 1.3 kg/m^2 for women over the 11-years follow-up period, Figures 3.2 and 3.3).

The mean BMI of subjects with the lowest educational level was always higher than the mean BMI of the highest educated subjects, except for young adult men (Figure 3.4). The mean BMI of the moderate educational level was always in between the mean BMI of the lowest and highest groups. The increase in BMI over the 11-year follow-up was similar for all levels of education within cohorts, except for the group of men aged 30-39 years, in which the lowest educated men increased statistically significantly less in BMI than the highest educated men (1.5 and 1.9 kg/m^2, respectively, over 11-year follow-up, p<0.01), and the men aged 50-59 years at baseline, in which the lowest educated men increased more in BMI than moderate educated and the highest educated men (1.2 *vs*. 0.7 (p=0.04) and 0.6 kg/m^2 (p<0.01), respectively, over 11-year follow-up). Increase in BMI over time among never-smokers was similar to the increase observed for the total cohort (data not shown).

Random intercept models

The results of the mixed-model analysis for age and period in relation to BMI showed that the relation between age and BMI was curvilinear (p for quadratic term <0.001) for both men and women (Table 3.3). In men, the increase in BMI with ageing was similar over the periods. BMI increased 0.8 units from the first to the third period independent of age. In women, the increase in BMI with ageing was stronger in the first period and in younger women.

The results of the mixed-model analysis for age and cohort in relation to BMI also showed a curvilinear relation between age and BMI. The increase in BMI with ageing was similar for all cohorts (no statistical significant interaction of age and cohort) and comparable for men and women (Table 3.3). Mean BMI at a given age was higher for more recent-born cohorts. The most recent-born cohort had a 2.1 units higher mean BMI at a given age in comparison to the cohort who was born 30 years earlier.

When modelling BMI as a function of age, independent of period (a 'cross-sectional' approach), the highest BMI was attained at age 58 years for men (Figure 3.5a). In women, highest BMI was not attained before age 70 years (Figure 3.5b). Since there was an interaction between age and period in relation to BMI in women, the effects of age on BMI are displayed for each period

separately. Based on cross-sectional data, the increase in BMI with ageing in women was lower with later periods (Figure 3.5b). When modelling BMI as a function of age, independent of cohort (a 'longitudinal' approach), BMI did not decline before age 70 years in both men and women (Figure 3.5, black dots).

TABLE 3.2 Mean body mass index with 95% confidence limits of the mean for age groups across periods.

Age group during measurement	**1987-1991**		**1993-1997**		**1998-2002** [1]	
	Mean (kg/m^2), (n)	**95% CL**	**Mean (kg/m^2), (n)**	**95% CL**	**Mean (kg/m^2), (n)**	**95% CL**
Men						
30-39 years	24.43 (667)	24.22; 24.64	25.02 (488)	24.76; 25.27	25.50 (303)	25.15; 25.86
40-49 years	25.37 (619)	25.16; 25.59	25.61 (725)	25.40; 25.83	26.08 (656)	25.84; 26.32
50-59 years	25.74 (370)	25.46; 26.02	26.23 (492)	25.98; 26.49	26.58 (642)	26.34; 26.82
Women						
30-39 years	23.29 (706)	23.04; 23.54	23.96 (549)	23.64; 24.27	24.79 (336)	24.35; 25.24
40-49 years	24.49 (630)	24.23; 24.75	24.84 (760)	24.57; 25.11	25.19 (692)	24.88; 25.50
50-59 years	25.64 (381)	25.31; 25.98	26.04 (458)	25.69; 26.38	26.16 (645)	25.84; 26.48

CL, confidence limits. [1] Note that in 1998-2002 the youngest participants were aged 31 years.

TABLE 3.3 Effects of age and period (cross-sectional) and effects of age and cohort (longitudinal) on body mass index in men and women.

	Men				Women			
	Cross-sectional		Longitudinal		Cross-sectional		Longitudinal	
	Beta (SE)	*p*-value	Beta (SE)	*p*-value	Beta (SE)	*p*-value	Beta (SE)	*p*-value
Age (years)	0.230 (0.015)	<0.001	0.297 (0.014)	<0.001	0.035 (0.060)	0.559	0.287 (0.017)	<0.001
Age*age (years^2)	-0.002 (0.000)	<0.001	-0.002 (0.000)	<0.001	0.001 (0.001)	0.173	-0.001 (0.000)	<0.001
Period1[1]	Reference	-			Reference	-		
Period2	0.386 (0.051)	<0.001			1.519 (0.427)	<0.001		
Period3	0.801 (0.078)	<0.001			3.158 (0.773)	<0.001		
Age*Period1	-	-			Reference	-		
Age*Period2	-	-			-0.027 (0.010)	0.006		
Age*Period3	-	-			-0.053 (0.017)	0.002		
Cohort1[2]			2.012 (0.229)	<0.001			2.111 (0.285)	<0.001
Cohort2			1.254 (0.193)	<0.001			0.973 (0.243)	<0.001
Cohort3			0.754 (0.190)	<0.001			0.538 (0.240)	0.025
Cohort4			Reference	-			Reference	-

SE, standard error. [1] Period1 = baseline; Period2 = 6 years follow-up; Period3 = 11 years follow-up. [2] Cohort1 = 20-29 years at baseline, Cohort2 = 30-39 years at baseline, Cohort3 = 40-49 years at baseline, Cohort4 = 50-59 years at baseline.

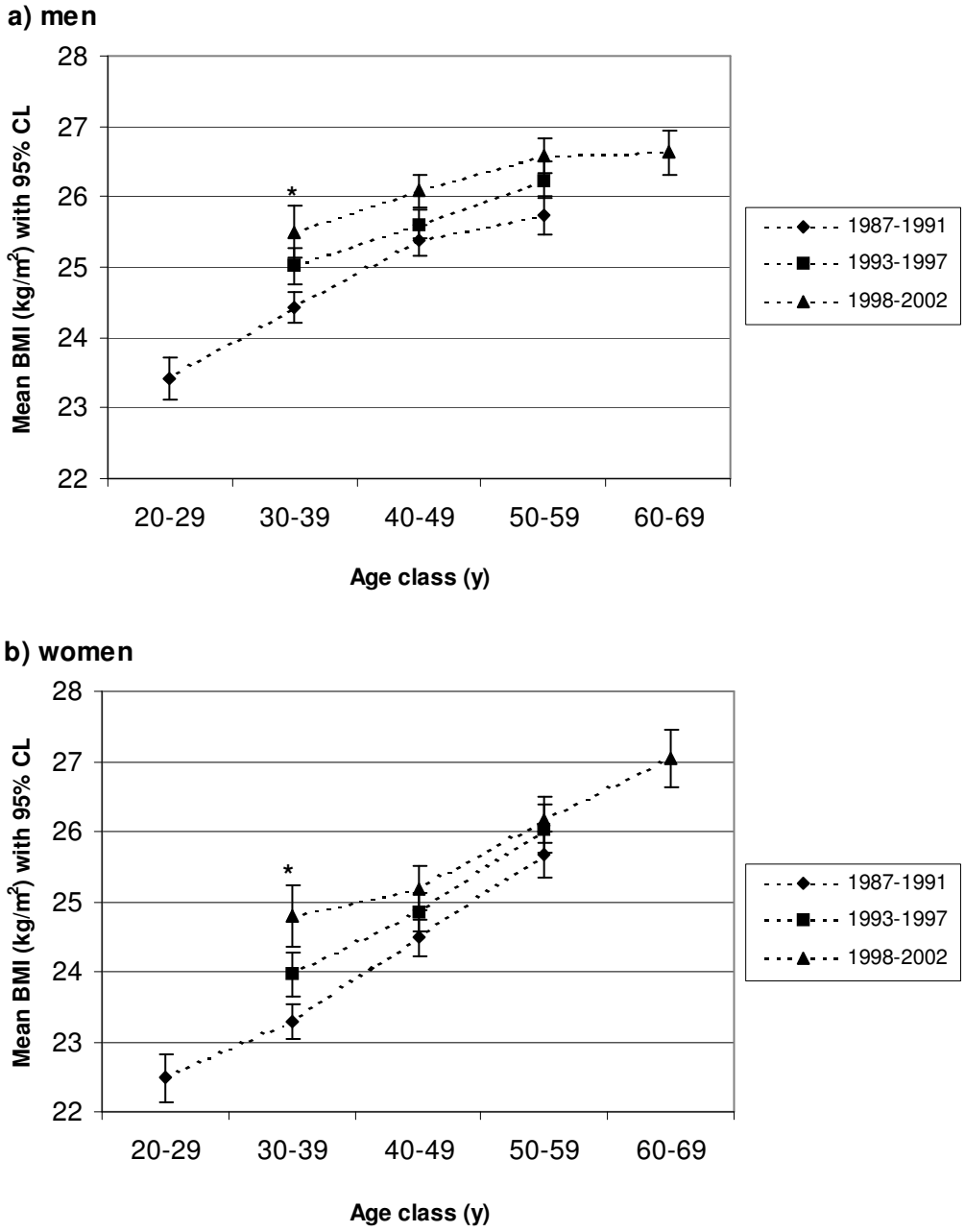

FIGURE 3.1 Cross-sectional data of body mass index (BMI) in men (a) and women (b) in the three successive measurement rounds (1987-1991, 1993-1997, 1998-2002): Age and Cohort effects (with a constant Period). CL, confidence limits. Each line represents data from one measurement round. * Note that the youngest subjects in 1998-2002 were aged 31 years.

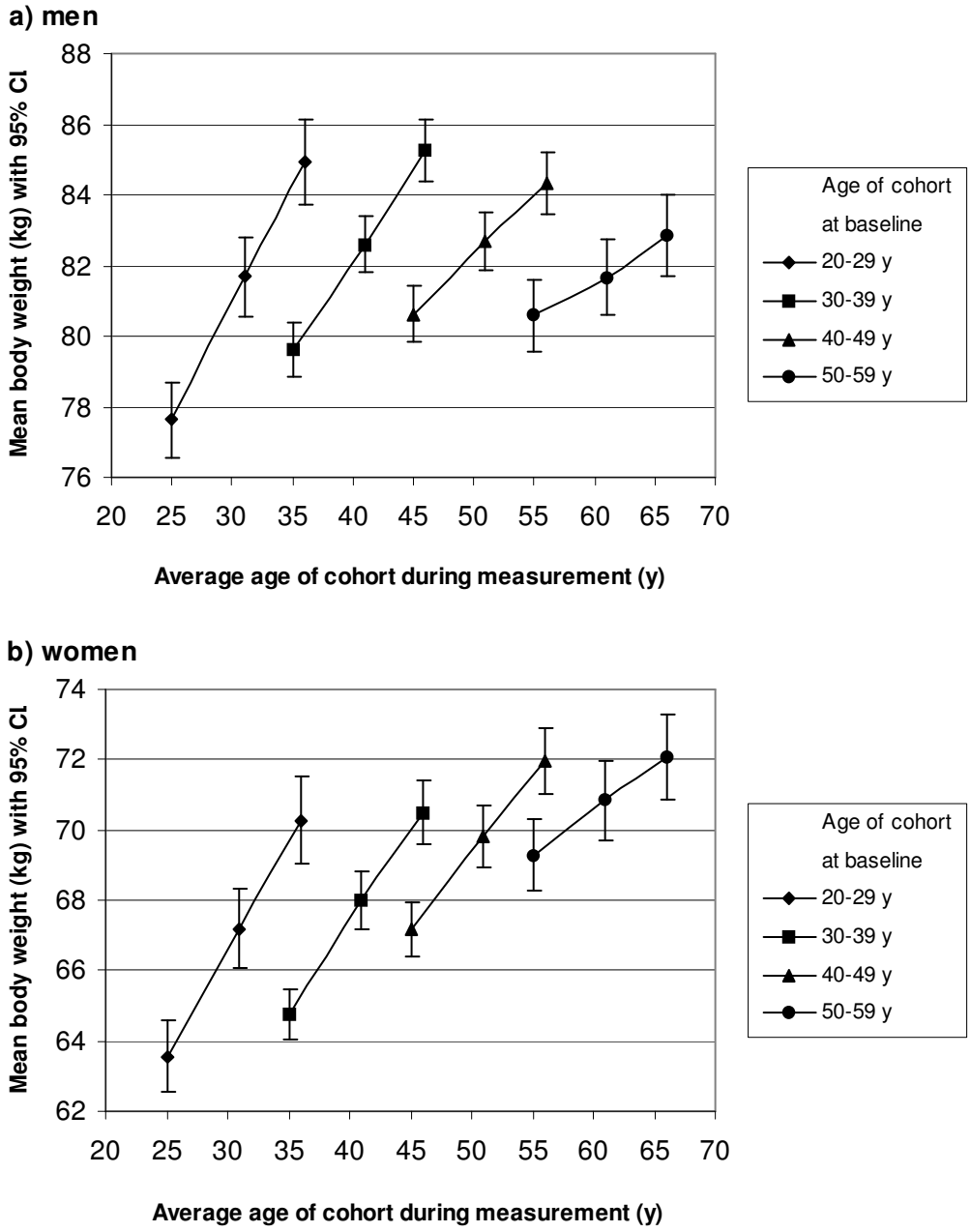

FIGURE 3.2 Longitudinal data of (change in) body weight in men (a) and women (b) by age group at baseline: Age and Period effects (with fixed cohort). CL, confidence limits. Each line represents data from one cohort.

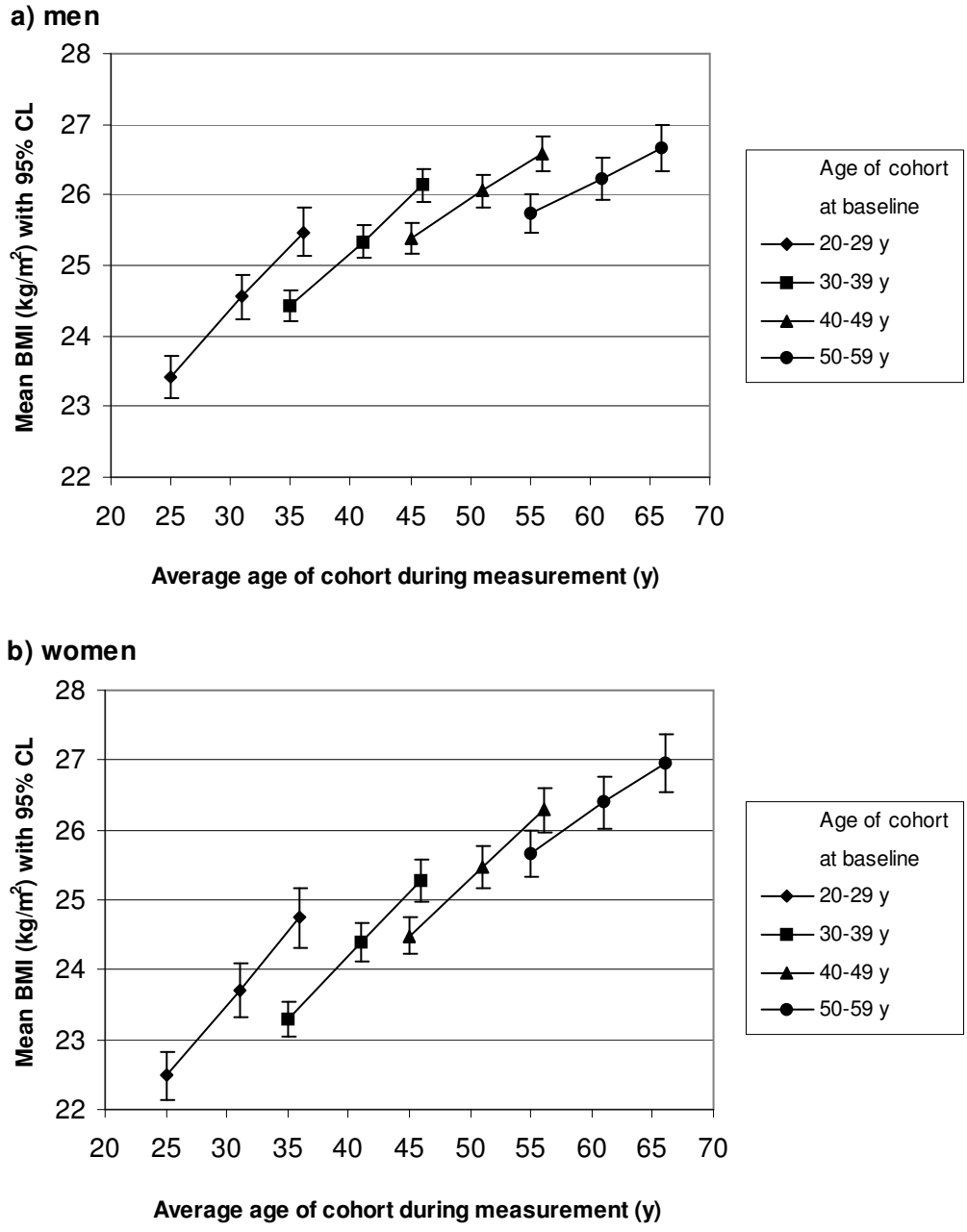

FIGURE 3.3 Longitudinal data of (change in) body mass index (BMI) in men (a) and women (b) by age groups at baseline: Age and Period effects (with fixed cohort). CL, confidence limits. Each line represents data from one cohort.

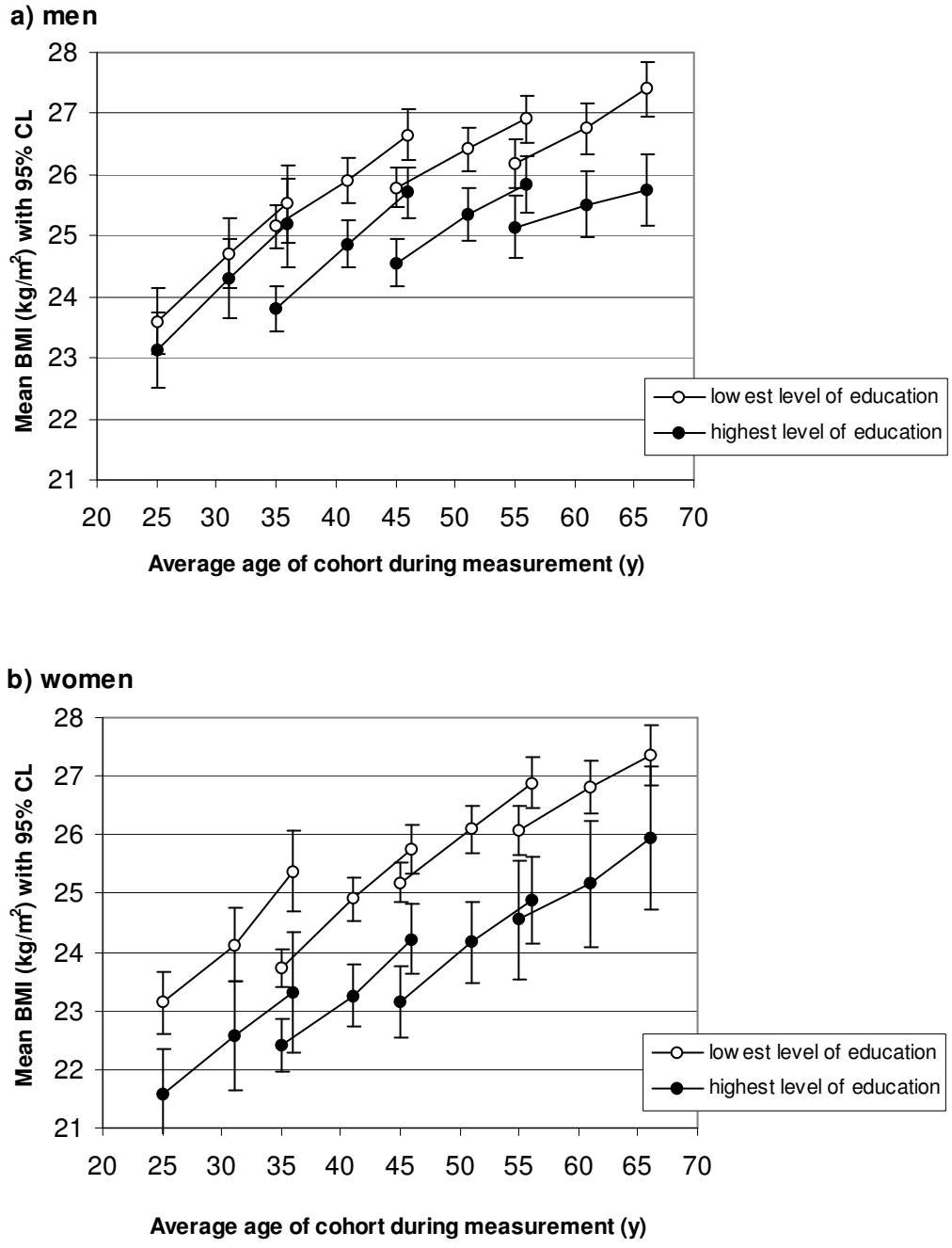

FIGURE 3.4 Longitudinal data of (change in) body mass index (BMI) with age in men (a) and women (b) by age group at baseline, stratified by educational level. CL, confidence limits. Each line represents data from one cohort. Note that moderate level of education is omitted in the graphs, for clarity reasons. BMI of the groups with moderate level of education was always in between the BMI's of the groups with low and high level of education.

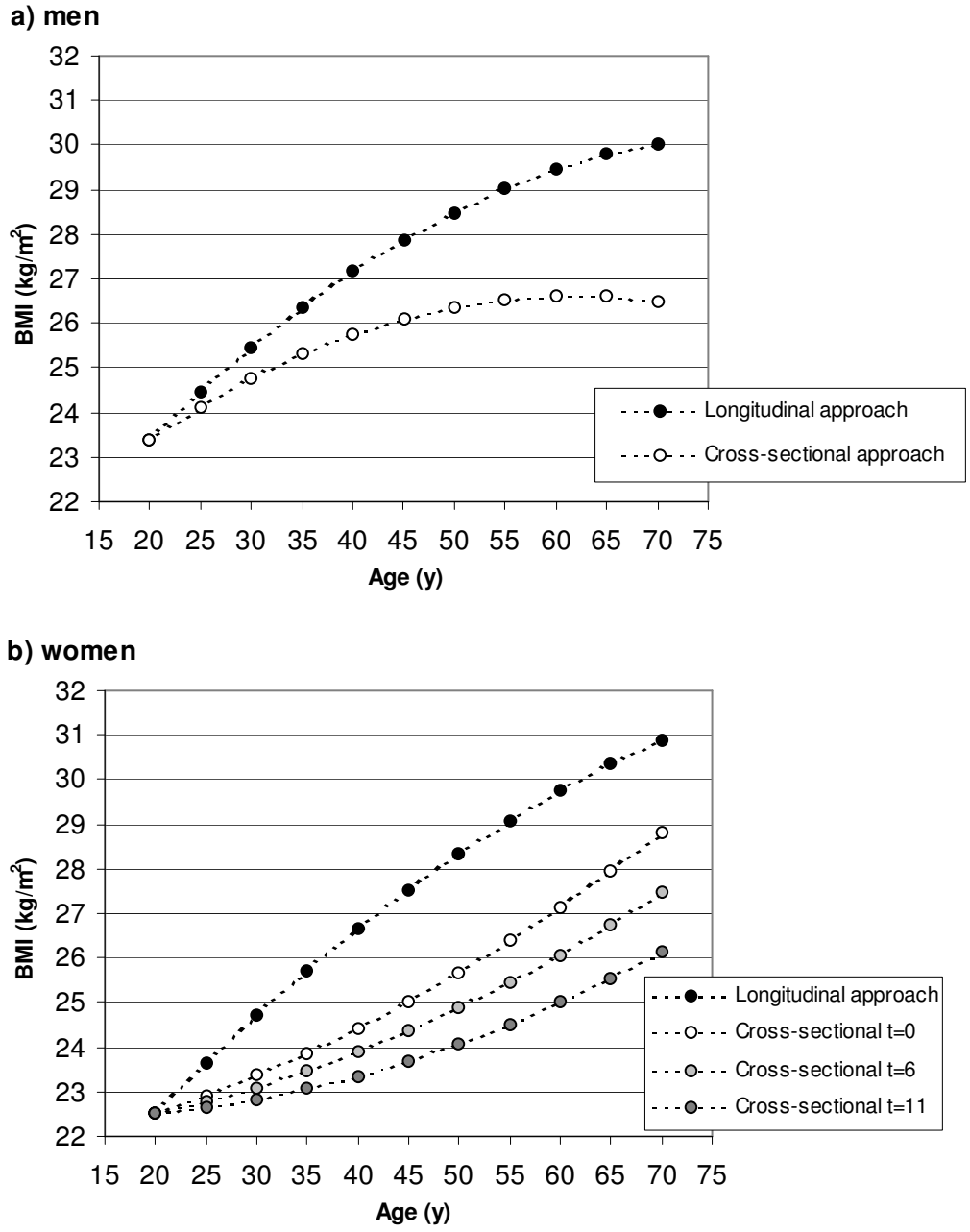

FIGURE 3.5 Mean body mass index (BMI) by age for men (a) and women (b), based on random intercept models, taking into account period (cross-sectional approach) and cohort effects (longitudinal approach).

DISCUSSION

In the present study, age, period and cohort effects on BMI were studied. One of the main findings of the present study was that the apparent decline in BMI with ageing in the oldest group of men seen in the cross-sectional analysis was shown to be an artefact due to a cohort effect. When evaluating longitudinal data, BMI still increased in the oldest age group, in both men and women. Furthermore, increase in BMI was greatest in the youngest age group, and the BMI of younger cohorts was higher than that of older birth cohorts at any given age between 30 and 60 years. Although the mean BMI of higher educated persons was lower than the mean BMI of lower educated persons, the increase in BMI with ageing was in general similar for different levels of education.

The tendency for younger adults to have large increases in BMI is confirmed by other studies.[3,21,25] Given the already higher BMI levels in the younger cohort, this steep increase in weight will definitely result in an increased prevalence of overweight and obesity over time in young adults. Prevention of weight gain in young adulthood, and also earlier in life, is therefore important for public health.

Contrary to our study, some previous longitudinal studies have observed an apparent decrease in body weight or BMI after age 55-65 years.[3,21,26,27] A possible explanation for differences in results between these studies and the current study could be that the Doetinchem cohort was born at least two decades later than those analysed in the previous studies. It may be that cohorts born earlier than the cohort analysed in the present paper actually did lose weight after age 55-65 years, in contrast to more recently born cohorts, where (positive) cohort and/or period effects were possibly larger than the (negative) effects of ageing per se. This hypothesis is strengthened by results of two recent studies, in which no decline in BMI was observed in both men[18,19] and women,[18] although decline after age 70 was observed in African-American men.[18]

No differences in secular trends of BMI were observed between groups of different educational level or between never-smokers and the overall group. Similar findings were observed among young adults in the CARDIA cohort.[10] Although BMI was higher in lower educated subjects, changes in BMI over follow-up were in general similar to those of higher educated subjects. These findings suggest that differences in BMI due to differences in educational level particularly occur before adulthood and that prevention of weight gain in adults should also include people with relatively high levels of education. Moreover, results of the present study suggest that also differences in absolute levels of BMI among young adult men may have disappeared over the past decades. The observed higher increase in BMI over follow-up among lower educated men aged 50-59 years at baseline can be explained by effects of retirement: men who retire from

active jobs (in general lower educated) gain more weight after retirement than men who retire from sedentary jobs (with in general a higher education).[28]

Results of our cross-sectional analyses in men (but not in women) confirmed results of previous cross-sectional studies, which showed that mean BMI declines after the age of 60 years.[1-3] At least three explanations can be given for observing decreasing BMI with ageing in cross-sectional studies: selective survival, cohort-effect, or weight loss after the age of 60 years.[4] Because subjects were only included in the present study if they had participated in all three surveys, selective survival did not affect our results. Our longitudinal analyses showed that mean body weight and BMI still increased in subjects over 60 years of age. Results of the present study therefore suggest that the relationship between age and BMI based on cross-sectional data is underestimated because of cohort effects. With a difference of more than one BMI-unit over an 11 years period, this underestimation was largest in young adults.

Probably the most important drawback of this study is the dropout of participants during follow-up, which is inherent to longitudinal studies. No information on level of education, BMI or lifestyle behaviours is available from non-participants at baseline. During the second and the third measurement of the Doetinchem Cohort Study, responders have been compared to the non-responders with respect to several variables.[29,30] Compared to responders, non-responders were younger, more often lower educated, obese and smokers. Although the increase in BMI was highest in younger persons, the present study has shown that the age effect on BMI was, in general, similar for different levels of education. *Ad hoc* analyses showed that overweight and obese women at baseline increased more in BMI over follow-up than normal weight female participants at baseline. For men, no effect of overweight was observed on future weight gain. Also smoking status at baseline did not predict future weight gain. Taken together, this implies that if the results of the present study would be biased by selective dropout, this bias would have led to an underestimation of the age effects on BMI.

Due to the relatively short follow-up period with only three measurements in the present study, we were not able to draw firm conclusions about period effects on BMI. However, we were predominantly interested in age effects on BMI. Effects of period and cohort were used to make the difference between the cross-sectional and the longitudinal approach.

One of the most important advantages of the present study was that body weight was not self-reported, but measured. Next, subjects over a large adult age range (20-59 at baseline) were included in the study and followed over an 11-year period, including three measurement periods.

Some have argued that prevention of weight gain is not important in older adults, but should be directed to young adults.[31] We have argued before that weight gain prevention at older age is still important in preventing adverse health outcomes,[32] since weight gain in this age group is progressively related to higher absolute risk for disease and mortality.[4,33-39] Because of the combination of higher prevalence of overweight and the observed weight gain at older age, prevention of weight gain at older age results in health gain in the short term. In addition, it has been shown that also percentage body fat increases with ageing until age 80 y, again, with higher increases in later birth cohorts.[40] As a result, increased prevalence of adverse health outcomes can be expected for the future.

In conclusion, findings of the present study using longitudinal data suggest that findings based on cross-sectional surveys underestimate the actual increase in BMI with ageing. This is due to cohort effects. Highest weight gain was observed in young adults, but weight gain was still present after the age of 60 years. Furthermore, weight gain was observed in all age groups and was independent of the level of education. This implies that prevention of weight gain should follow a population-wide, life-course approach.

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The development of lifestyle from adolescence into adulthood in relation to adult body composition – the Amsterdam Growth and Health Longitudinal Study

Limitations of observational studies to assess determinants of weight gain

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ABSTRACT

Background – An increase in energy intake (EI) and/or a decrease in physical activity (PA) results in body weight gain, until a new balance is reached.
Objective – To evaluate whether longitudinal observational data fit this theory of energy balance, by studying the developments of EI and PA from adolescence into adulthood in relation to body composition at adult age.
Methods – Dietary intake and physical activities were assessed in structured interviews, seven times from age 13 to 32 years in the Amsterdam Growth and Health Longitudinal Study. At age 36, percentage body fat was measured with dual-energy X-ray absorptiometry, and fat mass and lean body mass was calculated. The developments (i.e. baseline level, slope, and fluctuation) of EI and PA from adolescence to adulthood were calculated in 149 men and women and associated with percentage body fat, fat mass index and lean body mass index at age 36 years in linear regression analyses, adjusting for age, body weight and body fatness (based on sum of skinfolds) at baseline, and growth in height.
Results – In men and women, low EI at adolescence was associated with high percentage body fat, and tended to be associated with higher fat mass index at adult age. High PA at adolescence was associated with high lean body mass index at adult age in women, and with high fat mass index at adult age in men.
Conclusion – Also in longitudinal studies, associations between energy balance related behaviours and weight gain are not always straightforward. Therefore, serious caution is required at interpretation of such associations that might seem cause-effect relationships.

INTRODUCTION

The prevalence of overweight and obesity has increased substantially over the past decades.[1] Prevention of weight gain is crucial for the prevention of overweight and obesity and their related chronic diseases. In order to prevent weight gain, determinants of weight gain need to be ascertained.

In a previous study[2] we have shown that anthropometrical measures differ between adolescents who do and do not become adults with objectively assessed high body fatness. In addition, the increase in body mass index (BMI) and the sum of four skinfolds (S4SF) between age 13 and age 36 years was higher in subjects who became adults with high body fatness. These differences in weight gain should theoretically be accompanied by differences in (the development of) energy balance related behaviours.

In theory, an increase in energy intake (EI) or a decrease in physical activity level (PA) causes weight gain. Based on observational epidemiological data, it seems hard to verify this theory. In several observational studies[3-8] only few associations between (changes in) energy balance related behaviours (e.g., dietary intake, physical (in-)activity) and weight gain were observed, while in theory many more associations were expected. In these studies, follow-up time was often rather short and/or behaviour was measured only once or twice.

When results based on observational data do not fit the theory of energy balance, results on relations between energy balance related behaviours and weight gain can not be interpreted straightforward. This may have implications for the implementation of such results in interventions on weight gain prevention.

Lifestyle behaviours between adolescence and adulthood are subject to change.[9-12] Changes in dietary intake and PA are likely to result in changes in body weight and body fatness. In the present study, we will evaluate the associations between the developments of EI and PA from adolescence into adulthood, based on multiple measurements, in relation to body composition at age 36 years.

METHODS

Population

The Amsterdam Growth and Health Longitudinal Study (AGAHLS) is an observational, longitudinal study with a total inclusion of 698 subjects. The initial goal of the AGAHLS was to describe the natural development of growth, health and lifestyle of adolescents, and to investigate longitudinal relationships between biological and lifestyle variables.[13-15] The study started in 1976 with boys and girls (mean age 13 years) from the first and second form of two secondary schools in the Netherlands – one in Amsterdam and one in Purmerend. Informed consent was obtained from the children and their parents, and all subjects agreed to participate in the study. The AGAHLS was approved by the medical ethics committee of the VU University Medical Center in Amsterdam, the Netherlands. Measurements took place at mean ages 13, 14, 15, 16, 21, 27, 32 and 36 years. Pupils from the school in Amsterdam were invited for all eight measurements. Pupils from the school in Purmerend have not been invited for the measurements at mean ages 21 and 27 years.

Inclusion

For the present study, only the subjects who underwent a DXA scan at age 36 years and who participated in at least 5 (out of 7) measurements from age 13 to 32 years were included, since fewer measurements would yield less precise parameters to describe the development of the lifestyles. If women had reported to be pregnant during any of the measurements, the data from that particular measurement were excluded from the analyses. Subjects who reported to suffer from diabetes, cardiovascular disease or other chronic diseases that are related to overweight or have impact on weight gain were excluded from the analyses also (n=6). In total, 71 men and 78 women participated in 5 or more measurements and were included in the present study.

Anthropometrical measures

At each measurement, body height [with a wall-mounted stadiometer (Holtain, Crymych, United Kingdom), to the nearest 0.1 cm], body weight [with a spring balance (van Vucht, Amsterdam, the Netherlands), to the nearest 0.1 kg] and S4SF [the sum of the thickness of the biceps, triceps, subscapula, and suprailiac skinfolds to the nearest 0.1 mm with a Harpenden capiler (Holtain)] were assessed according to standard procedures.[16] At the mean calendar age of 36 years, a whole-body DXA scanner [Hologic QDR-2000, software version V5.67A; Hologic Inc, Waltham, MA) was used to assess percentage body fat (PBF) for all participants. Fat mass (in kg) was calculated as percentage body fat times body weight (kg); lean body mass (in kg) as body weight minus fat mass (kg). Body mass index (BMI) was calculated as body weight (kg) divided by body height squared (m^2). Fat mass index was calculated as body fat mass (kg) divided by body height squared (m^2). Lean body mass index was calculated as lean body mass (kg) divided by body

height squared (m^2). Percentage body fat at baseline was calculated according to Durnin and Rahaman[17] from the sum of skinfolds. Overweight at adolescence was assessed based on age and gender specific cut-off points for BMI.[18] At adult age, overweight was defined as a BMI ≥25 kg/m^2. High percentage body fat (PBF) was defined as ≥25% for men and ≥35% for women.[2]

Energy intake (EI)

At all time points, habitual dietary intake was measured with a modification of the crosscheck dietary history interview that referred to the last month.[19] All participants were interviewed by a dietician to recall their usual intake of foods and drinks by reporting the frequency, the amounts and methods of preparation of the foods consumed. All consumed food items were transformed into nutrients in accordance with the Dutch Food and Nutrient Table[20] and the total EI (MJ/day) was calculated. In the year 2000 (at mean age 36 years), the method to determine dietary intake has been changed.[21] Therefore, dietary intake data obtained at age 36 years were not used in the analyses of the present study.

Physical activity (PA)

Habitual PA was assessed at all time points in a structured interview.[14] In this interview, the intensity, frequency and duration of PA during the past three months were recorded. Activities were translated into metabolic-equivalent (MET) scores (indicating the intensity of an activity relative to the energy equivalent of the Basal Metabolic Rate) and classified in three intensity levels of at least moderate intensity: 4-7, 7-10, and >10 METs.[22] A weighted activity score was calculated from all types of activities demanding more than 4 METs, by multiplying the total amount of time spent per week doing an activity with the respective level of intensity (respectively 5.5, 8.5 and 11.5). This score (MET*hour/week) was used as the indicator of total activity level. In the year 2000, the method to assess PA level has been changed.[21] Therefore, PA data obtained at age 36 years were not used in the analyses of the present study.

Analyses

The development of EI and PA was operationally defined as a function of three parameters. These development-parameters were obtained by individual linear regression equations of continuous levels of EI and PA as a function of time (year of measurement) over up to 7 measurements with 19 years of follow-up. Intercept, slope and residual mean square were calculated for each individual participant in order to obtain estimates of baseline (year 1977) level, the degree of increase over time and the degree of fluctuation, respectively. Calculated baseline levels (intercepts) of EI and PA were statistically significantly correlated with assessed baseline EI and PA, r's ranging from 0.73-0.86.

The three development parameters (graphically shown in Figure 4.1.) were compared between the groups of participants who did and who did not become 36-years old adults with high body

fatness, using t-tests. The development parameters were associated with percentage body fat, fat mass index and lean body mass index at age 36 years with linear regression analyses, adjusting for baseline age, body weight and body fatness, growth in height (by including height at age 13 years and at age 36 years), and other development parameters of energy intake and PA. These latter analyses were also performed on data from adults (age 21-32 years, n=121) only, in order to evaluate whether growth in height from adolescence into adulthood interfered with associations between the developments of energy balance related behaviours and body composition. All factors in these analyses were entered as continuous ones. Since a significant interaction effect was observed between development parameters of lifestyle and gender on the associations with body fatness, all analyses were performed for men and women separately. SAS version 9.1 was used [SAS Institute Inc., North Carolina].

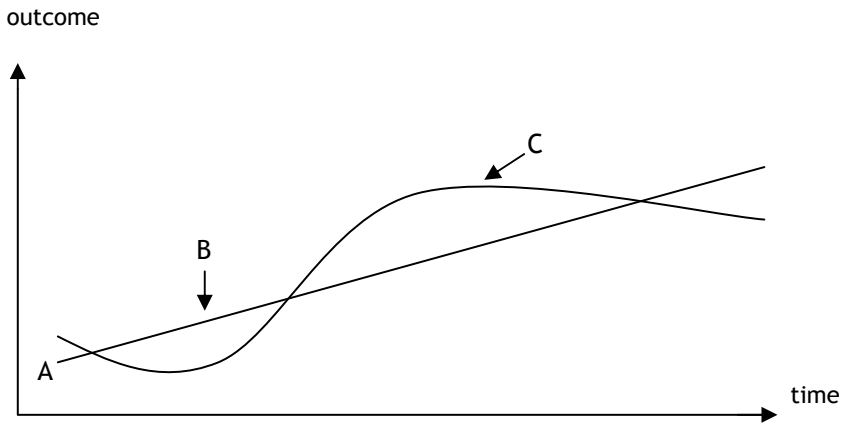

FIGURE 4.1 Graphical representation of the development-parameters: A refers to the intercept (the calculated baseline level), B refers to the linear slope over time, and C refers to the fluctuation around the slope.

RESULTS

In Table 4.1, the general characteristics of the study population are presented. Average calendar age at baseline was 13 years for both boys and girls. None of the included participants were overweight at baseline, based on their BMI. Average body fatness was higher in girls compared to boys. After 23 years of follow-up, 37% of men and 24% of women were overweight, and 30% and 37% had a high percentage body fat, respectively. Total EI at baseline was higher in men than in women. On average, total EI did hardly increase over time, but a fluctuation of about 13% of the baseline level was present around the slope. PA at baseline was higher in men than in women and decreased more in men than in women from age 13 years to 32 years. A considerable fluctuation around the slope of PA was present: on average 33% of the baseline levels.

No statistically significant differences in developmental parameters of EI and PA were observed between boys who did and did not become adults with high body fatness. Among girls, those who became adults with high body fatness had lower EI and lower PA at baseline compared to those who had lower body fatness at adult age (Table 4.2).

Table 4.3 shows the associations between the developments of EI and PA from age 13 to 32 years and aspects of body fatness at age 36 years. In men, associations between baseline levels of EI and PA and percentage body fat and fat mass index became stronger after additional adjustment for the development of the other behaviour. In both men and women, higher EI at age 13 years was associated with lower percentage body fat and fat mass index at age 36 years. Higher baseline PA was associated with higher percentage body fat and fat mass index at age 36 years in men. In women, high baseline PA and increase in PA were associated with higher lean body mass index at adult age. P-values of all these associations were around 0.05. Increases and fluctuations in EI and PA between adolescence and adulthood were never statistically significantly associated with percentage body fat or fat mass index at age 36 years. In men, developments of EI en PA were not associated with lean mass index at adult age.

Additional analyses on associations between the development of EI and PA from age 21-32 years and aspects of body fatness at age 36, revealed no statistical significant associations (data not shown). Associations that were found to be statistical significant over the period 13-32 years in men, were far from statistically significant (p's>0.84) in the analyses on adult data, only. In fact, some of the associations in adult men were oppositely directed to the effects found over the period 13-32 years. In women, associations observed over the period 21-32 years were mostly in line with associations observed over the period 13-32 years.

TABLE 4.1 General characteristics of the study population – the Amsterdam Growth and Health Longitudinal Study.

	Men (n=71)	Women (n=78)	p-diff*
Baseline (1977)			
Age (years), mean (SD)	13.5 (0.7)	13.5 (0.6)	0.73
Height (cm), mean (SD)	161.6 (7.7)	162.3 (6.9)	0.53
Weight (kg), mean (SD)	45.5 (6.6)	47.7 (7.4)	0.07
BMI (kg/m^2), mean (SD)	17.4 (1.4)	18.0 (2.1)	0.03
Overweight (IOTF-cut-offs BMI), %	0	0	-
Percentage body fat**, %	16.0 (3.8)	23.7 (4.4)	<0.01
Follow-up (2000)			
Age (years), mean (SD)	36.5 (0.6)	36.6 (0.6)	0.71
Height (cm), mean (SD)	183.1 (6.5)	170.3 (6.5)	<0.01
Weight (kg), mean (SD)	82.7 (10.5)	67.6 (9.3)	<0.01
BMI (kg/m^2), mean (SD)	24.6 (2.6)	23.3 (3.0)	<0.01
Overweight (BMI ≥25 kg/m^2), %	36.6	24.4	0.10
Percentage body fat**, mean (SD)	22.1 (5.8)	32.2 (6.6)	<0.01
High percentage body fat***, %	29.6	37.2	0.33
Lean body mass (kg), mean (SD)	64.1 (6.8)	45.5 (5.9)	<0.01
Energy intake (MJ/day)			
Calculated baseline, mean (SD)	12.0 (2.0)	9.3 (1.7)	<0.01
Average linear change per year, mean (SD)	0.02 (0.14)	0.01 (0.12)	0.75
Fluctuation, mean (SD)	1.7 (0.7)	1.1 (0.4)	<0.01
Physical activity level (METh/week)			
Calculated baseline, mean (SD)	71.3 (25.9)	58.2 (17.6)	<0.01
Average linear change per year, mean (SD)	-1.51 (2.18)	-0.37 (1.62)	<0.01
Fluctuation, mean (SD)	23.5 (11.3)	18.9 (9.0)	<0.01

* p-diff was assessed with t-test for continuous variables and with Chi-square for categorical variables.

** Percentage body fat at baseline was calculated from the sum of four skinfolds; at follow-up it was assessed using a whole-body DXA scanner. *** High percentage body fat was defined as ≥25% for men and ≥35% for women.

TABLE 4.2 Average values of developmental parameters of EI and PA level from age 13 to 32 years for participants who did and did not become adults with high body fatness* - the Amsterdam Growth and Health Longitudinal Study.

	Men			Women		
MEN	Low fatness	High fatness	*p*-diff	Low fatness	High fatness	*p*-diff
Energy intake (MJ/day)						
Baseline level	12.3 (1.8)	11.4 (2.3)	0.07	9.6 (1.6)	8.7 (1.6)	0.02
Increase	0.01 (0.14)	0.03 (0.15)	0.71	0.01 (0.13)	0.02 (0.10)	0.85
Fluctuation	1.6 (0.7)	1.7 (0.8)	0.71	1.1 (0.5)	1.1 (0.4)	0.72
Physical activity (METh/week)						
Baseline level	68.9 (21.8)	77.0 (33.6)	0.31	61.5 (18.8)	52.7 (14.2)	0.03
Increase	-1.32 (1.98)	-1.97 (2.61)	0.26	-0.63 (1.64)	0.08 (1.49)	0.06
Fluctuation	23.1 (11.2)	24.4 (11.9)	0.66	17.9 (8.5)	20.6 (9.8)	0.21

*High body fatness was defined as ≥25% for men and ≥35% for women. *p*-diff involves the statistical significance of the difference between the groups with high and low body fatness at adult age and was assessed with t-test.

TABLE 4.3 Associations of the developments of EI and PA from age 13 to 32 years in relation to percentage body fat, fat mass index, and lean body mass index at age 36 years - the Amsterdam Growth and Health Longitudinal Study.

	Percentage body fat				Fat mass index				Lean body mass index			
	Model 1*		Model 2**		Model 1*		Model 2**		Model 1*		Model 2**	
MEN	stb	p	stb	p	stb	p	stb	p	stb	p	stb	p
Energy intake (MJ/day)												
Baseline level	-0.19	0.20	-0.31	0.04	-0.16	0.28	-0.30	0.05	0.19	0.13	0.10	0.45
Increase	-0.01	0.96	-0.00	0.99	-0.02	0.88	-0.02	0.90	0.01	0.90	-0.03	0.81
Fluctuation	0.13	0.32	0.10	0.42	0.11	0.39	0.08	0.50	-0.08	0.45	-0.10	0.33
Physical activity (METh/week)												
Baseline level	0.19	0.32	0.34	0.09	0.25	0.18	0.40	0.04	0.10	0.54	0.06	0.71
Increase	-0.11	0.49	-0.04	0.79	-0.08	0.61	-0.01	0.96	-0.01	0.93	-0.02	0.91
Fluctuation	-0.04	0.79	-0.03	0.82	-0.03	0.82	-0.02	0.88	0.16	0.18	0.18	0.15
WOMEN												
Energy intake (MJ/day)												
Baseline level	-0.32	0.04	-0.34	0.05	-0.25	0.12	-0.29	0.09	0.18	0.23	0.10	0.54
Increase	-0.22	0.10	-0.22	0.10	-0.17	0.21	-0.17	0.21	0.09	0.49	0.08	0.52
Fluctuation	0.22	0.07	0.21	0.10	0.20	0.10	0.21	0.11	-0.00	0.98	0.06	0.63
Physical activity (METh/week)												
Baseline level	-0.19	0.20	-0.12	0.44	-0.10	0.49	-0.03	0.84	0.29	0.03	0.30	0.04
Increase	-0.03	0.85	0.01	0.95	0.05	0.75	0.08	0.57	0.26	0.04	0.25	0.06
Fluctuation	0.04	0.75	-0.05	0.69	-0.02	0.86	-0.11	0.42	0.18	0.11	-0.17	0.17

Stb = standardized beta. All associations are adjusted for age, weight, and body fatness at baseline, growth in height, and all other parameters of EI and PA.

*In model 1, associations are additionally adjusted for other developmental parameters of the same behaviour (e.g. baseline EI for increase and fluctuation in EI).

**In model 2, associations are additionally adjusted for other developmental parameters of both EI and PA.

DISCUSSION

In the present study, the developments of EI and PA from adolescence into adulthood were studied in relation to body fatness at adult age. Based on the concept of energy balance, it was assumed that especially a high baseline or increase in total EI and/or a low baseline or decrease in PA from adolescence into adulthood would be associated with a higher percentage body fat at adult age. Indeed, we observed that girls who became adults with high body fatness had a lower PA (but also a lower EI) at baseline compared to the girls who became adults with lower body fatness. Results of our multiple adjusted linear regression analyses did not support the stated hypothesis. In fact, we observed associations of low EI and high PA at baseline with more increase in (percentage) body fat between adolescence and adult age. The only associations that were plausibly directed were those between developmental parameters of PA and the lean body mass index among women. At adult age, no statistically significant associations between the development of EI and PA and aspects of body fatness were observed.

Associations between energy expenditure and percentage body fat have been evaluated by Paul et al in a cross-sectional study, taking gender and food intake into account.[23] Opposite associations between (PA) energy expenditure and percentage body fat were observed in that study: higher PA in men was associated with lower body fatness, while higher total energy expenditure (but not PA) was associated with higher body fatness in women.[23] In the present study, associations of PA and EI with body composition were also different in men compared to women. However, we (unexpectedly) observed higher initial PA to be associated with higher body fatness at adult age in men. The development of PA in women was not associated with body fatness at adult age, but higher initial PA and an increase in PA over time were associated with higher lean body mass index at adult age. Since dietary intake was taken into account in both studies, resulting in different results for men compared to women, the concept of energy balance was not met in both studies.

In our study, higher total EI at adolescence tended to be associated with lower body fatness at adult age in both men and women. In the study by Paul et al[23] dietary intake was correlated with body fatness in women only: percentage energy intake as carbohydrates was associated with lower percentage body fat and percentage energy intake as fat was associated with higher percentage body fat. However, no associations between total energy intake and percentage body fat were reported.[23] Kimm et al[24] found in a longitudinal study among girls aged 9-10 at baseline with 9 years follow-up, that EI during follow-up was not associated with changes in BMI and S4SF, while PA was inversely associated as expected. They blamed under-reporting of EI for the non-finding for EI.[24] In a cross-sectional study among 17 year olds, self-reported PA was

independently inversely associated with fat mass in men, but not in women. In women, fat mass was associated with fat mass of the mother.[25]

In another longitudinal study among adolescents, higher baseline PA was associated with stronger gains in adiposity. Decreases in PA level appeared to explain this paradoxical finding.[26] In a cross-sectional study in Australian boys and girls, lower fat intake, lower consumption of energy-dense snacks and higher consumption of vegetables were associated with higher risk of overweight/obesity. These unexpected associations were explained by dieting or knowledge of favourable dietary choices in overweight/obese children.[27]

Women who developed high body fatness at age 36 years were less active and had a lower EI at age 13 years compared to those who developed lower body fatness at age 36 years. This observation is plausible since more active persons need higher EI. Since EI and PA should always be viewed together in relation to weight gain, we adjusted for other developmental parameters of EI and PA in our linear regression analyses. In these analyses, associations between developmental parameters and body composition should be interpreted independently from other developmental parameters. In these analyses, the observed association between low baseline EI and high body fatness does not fit the concept of energy balance. The same is true for the association between high baseline PA and high body fatness.

The observed association between high total energy intake and low fat mass may be explained by three reasons: 1) the population was not obese and a high total energy intake per se does not necessarily lead to the accumulation of body fat, 2) under-reporting of dietary intake by the interview method, and 3) repeated measurements can introduce a negative testing effect.[28] This observation was also made in an earlier study in our data, but is now confirmed based on a longer follow-up period and a more valid measure for body composition (DXA instead of S4SF).

Although our calculated developmental parameters of EI and PA fit our data, the parameters may not describe the true developments, due to bias in self-reported EI and PA. It is often difficult for participants to report the exact diet and PA over the last few months. Therefore we assume that reporting bias has occurred in our studies. This bias may have been larger than the (small) changes in lifestyle behaviour resulting in weight gain as observed.[19] In addition, in the interviews, habitual dietary intake and habitual PA were recorded. However, also incident food consumption and incident PA count up for the energy balance, resulting in weight change. In addition, only PA of at least moderate intensities was recorded. However, the time engaged in low-intensity PA is mostly larger than in high intensity PA and therefore counts up for a significant part of the total energy expenditure by PA.[29]

Next to the difficulties of estimating PA, energy expenditure is not determined by PA, only. Especially from adolescence into adulthood, energy expenditure is determined also by growth. Therefore we adjusted in our analyses for growth by adding height to the analyses. But also body composition, gender, the kind of food consumed and even the environmental temperature are determinants of total energy expenditure. Therefore, PA, as assessed in this study, may not be a sufficiently good marker for total energy expenditure, or the percentage of the total energy expenditure by PA may change during aging. When we performed our analyses on data from adults (age 21-32) only, excluding effects of growth, we did not observe any significant association between EI or PA and percentage body fat at age 36 years. In adult men, associations were sometimes oppositely directed, but in adult women associations were more or less in line with results over the total study period. Probably, in men associations between EI and PA observed over the total study period (13-32 years) are determined during adolescence only, while in women associations remain constant over the total study period. This could reflect effects of growth in height on the associations between EI and PA on body fatness: growth in men was more than twice the growth in women over the entire study period. However, since we did not observe any significant associations at adult age, we cannot fit the concept of energy balance with our observational data free from effects of growth in height.

In our analyses, we adjusted for baseline body composition. Therefore, associations between the developmental parameters of EI and PA with body composition at adult age can be interpreted as determinants of changes in body composition. We mostly expected effects of changes and/or fluctuations in energy balance related behaviours in relation to changes in body composition. However, in the present study we mostly observed effects of baseline behaviours on later body composition. This may be due to reporting bias, of which the error is larger than the observed changes in behaviour over time.

Between age 32 and 36 years, lifestyles and body composition may have changed. Unfortunately, at age 36 years, the method to assess dietary intake and PA had been changed. Therefore we could not include lifestyle data obtained at age 36 years into our calculation of the development of lifestyles from adolescence into adulthood. In contrast, we did use body composition at age 36 years and not at age 32 years, since DXA measurement (at age 36 years) is more valid for body composition than the anthropometric indices BMI and S4SF (at age 32 years). Associations between the developments of EI and PA from age 13-32 years and body composition at age 36 years may have weakened due to (unmeasured) changes between age 32 and 36 years. However, gender-specific correlations between percentage body fat at age 32 years and age 36 years, based on S4SF was high, about 0.8. Therefore we do not believe that associations between the developmental parameters and body composition would have been

differently directed when the lifestyle parameters at age 36 years would have been included in our analyses.

Strengths of our study are that participants were followed over a long time span, from age 13 to age 36 with up to eight repeated measurements of anthropometric parameters and interview data on lifestyles. In this way, we were able to calculate robust developmental parameters for EI and PA level from adolescence into adulthood. Next, body composition at age 36 years was measured with DXA.

Our study including multiple measurements regarding EI and PA over a long time period resulted in sparse associations of EI and PA with obesity, as were found in earlier studies with only one of two measurements of EI and PA. Taking into account the strengths and weaknesses of our analyses, our study offers strong presumptions that longitudinal observational data based on self-report do not fit the theory of energy balance. Probably, a mixture of measurement error, reporting error, time between measurements and reporting habitual lifestyles makes it very difficult to associate self-reported lifestyle behaviours to weight gain (and resulting body fatness). Therefore, we believe that findings of relations between energy balance related behaviours and weight gain based on self-reported data, that might seem cause-effect relationships, should be interpreted with serious caution.

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Impact of life transitions during young adulthood on body weight and body fat mass – the Amsterdam Growth and Health Longitudinal Study

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Submitted for publication

ABSTRACT

Objective: To investigate whether life transitions in the period between late adolescence and young adulthood affect body weight and/or body fatness.
Methods: Leaving the parental home, starting to work, living alone or with a partner, and parenting children were studied in relation to changes in anthropometric measures (body weight, body mass index, sum of skinfolds, and waist circumference) in men (n=69-78) and women (n=74-87) in the Amsterdam Growth and Health Longitudinal Study, using linear regression analyses.
Results: Between age 16 and 27 years, men who started to work at young age while still living with their parents, showed largest increases in body weight (4 kg additional increase). Between age 27 and 36 years, men and women who became parents showed a larger increase in body weight (3.5 and 3.9 kg larger increase, respectively) and body fat than those who did not parent children. Women living alone without children gained more body weight than cohabiting women.
Conclusion: Men who start to work while living with their parents, women living alone and new parents seem high-risk groups for targeted weight gain prevention.

INTRODUCTION

During young adulthood, relatively large increases in body weight are observed.[1,2] In the same period, several life transitions can take place: e.g. leaving the parental home and starting to live alone or cohabiting, starting to work, and having children to take care for. Obesity research regarding this age-period has a strong focus on marriage and women delivering children. Married women are in general less physically active than unmarried women.[3] Furthermore, women who get married or have children have a higher risk to become physically inactive, than women who do not experience these life events.[4] Entering marriage/start living together has been associated with body weight gain in both men and women,[5-7] with changes in diet among women, and with a decrease in physical activity among men.[7]

It is likely that several other life transitions (leaving the parental home, starting to live together, starting to work, and having children) during young adulthood are accompanied by changes in physical activity and / or diet, which may affect body weight and / or body fatness. For instance, women who start working have a higher risk to become inactive than women who do not start working.[4] Apparently some life transitions are accompanied by changes in physical activity, dietary intake and weight gain and may therefore ask for preventive measures. Data on these life transitions in relation to body weight gain is scarce and mainly focused on women.

The purpose of the present study is to investigate the impact of leaving the parental home, starting to work, living together, and parenting children on body weight, body mass index, skinfold thickness and waist circumference. If differences were observed, we investigated the underlying changes in lifestyles (i.e. total energy intake and physical activity level). Data were derived from the Amsterdam Growth and Health Longitudinal Study.[8]

METHODS

The Amsterdam Growth and Health Longitudinal Study (AGAHLS)

The AGAHLS is an observational, longitudinal study with a total inclusion of 698 subjects. The initial goal of the AGAHLS was to describe the natural development of growth, lifestyle and health of adolescents, and to investigate longitudinal relationships between lifestyle and biological variables.[8-10] The study started in 1976 with boys and girls (mean age 13 years) from the first and second form of two secondary schools in the Netherlands – one in Amsterdam and one in Purmerend. Informed consent was obtained from the children and their parents, and all subjects agreed to participate in the study. The AGAHLS was approved by the medical ethics committee of the VU University Medical Center in Amsterdam, the Netherlands. Measurements took place at mean ages 13, 14, 15, 16, 21, 27, 32, 36 and 42 years. Pupils from the school in Amsterdam were invited for all nine consecutive measurements. Pupils from the school in Purmerend were not invited for the measurements at mean ages 21 and 27 years.

Inclusion

For the present study, the participants who were measured at mean ages 16, 21, 27, 32 and 36 years were eligible for inclusion, since it is assumed that most important life transitions take place in that age period. If women reported to be pregnant during any of these included measurements, the data from that particular measurement were excluded from the analyses. Subjects who reported to suffer from diabetes, cardiovascular disease or other chronic diseases that may be related to overweight or weight gain were excluded from the analyses (n=6). Over the age period 16 – 21 – 27 years, 165 participants (78 men) were included. Over the age period 27 – 32 – 36 years, 143 participants (69 men) were included.

Life transitions

In the present study, four life transitions or living situations were studied: 1) leaving the parental home, 2) starting to work, 3) living together and 4) parenting children.
Leaving the parental home and starting to work mainly took place between ages 16 and 27 years and were therefore studied over this 11-year period. Almost each young individual leaves the parental home and starts to work some moment in time. Therefore we investigated the timing of these events (before/after age 21 y) in relation to changes in body weight or body fat mass. No information on living situation was obtained at age 16 years, but we assumed that all participants were living with their parents.

Living situation was assessed with the following question: *"In which situation do you live most days of the week?"* Answering categories were: living with parents, living alone, living together with a partner, living together with friends, living in military service, living intern for school, or

living in a commune. During the measurement in 1985 (mean age 21 years), groups were small. Therefore only two main groups on living situation could be composed: 1) living with parents and 2) living on one's own (living alone or living together with a partner or with friends). Subjects living in military service, living intern for school or living in a commune (n=10 at mean age 21 y) were excluded from the analyses investigating the effects of leaving the parental home, due to small numbers. The effect of living situation (living alone or with a partner) on body weight and body fat mass was studied between mean ages 27 and 36 years. For one subject, no information on living situation at age 36 years was available.

Starting to work was assessed based on the question: *"Do you have a paid job?"* Three answers could be given: "no", "yes, part time", or "yes, full time". Two subgroups were composed: no job or a job, regardless of the number of hours per week.

The impact of parenting children on body weight and body fat mass was only studied between age 27 and 36 years, since before 1991 (mean age 27 y) no information about parenting children was available. Parenting children was assessed with the question *"What is the number of children you take care for?"*

Anthropometrical measures

At each measurement, body height (with a Harpenden wall-mounted stadiometer [Holtain, UK], to the nearest 0.1 cm), body weight (with a spring balance [van Vucht, Amsterdam the Netherlands], to the nearest 0.1 kg), and the thickness of four skinfolds (biceps, triceps, subscapula and suprailiac, with a Harpenden calliper [Holtain, UK], to the nearest 0.1 mm) were assessed in accordance with guidelines of the International Biological Program.[11] Body mass index (BMI) was calculated as body weight (kg) divided by body height squared (m^2). The sum of four skinfolds (S4SF) was calculated and expressed in mm. From 1991 (mean age 27 y) onwards, also data on waist circumference (with a flexible steel tape [Martin circumeter, Franken & Itallie, Amsterdam, the Netherlands] to the nearest 0.1 cm) are available.

Dietary intake

Dietary intake was measured with a modification of the crosscheck dietary history interview that referred to the last month.[12] All participants were interviewed by a dietician to recall their usual intake of foods and drinks by reporting the frequency, amounts and methods of preparation of the foods consumed. All consumed food items were transformed into nutrients in accordance with the Dutch Food and Nutrient Table[13] and the total energy intake (MJ/day) was calculated. At the age of 36 years, dietary intake was measured with a computer-assisted version of the interview method.[14]

Physical activity

Daily physical activities were assessed in a structured interview.[10] In this interview, the intensity, frequency and duration of physical activities during the past three months were recorded. Activities were translated into metabolic-equivalent (MET) scores (indicating the intensity of an activity relative to the energy equivalent of the Basal Metabolic Rate) and grouped in three levels of at least moderate intensity: 4-7, 7-10, and >10 METs.[15] A weighted activity score was calculated from all types of activities demanding more than 4 METs, by multiplying the total amount of time spent per week doing an activity with the respective level of intensity (respectively 5.5, 8.5 and 11.5 MET). This score (kMET*hr/week) was used as an indicator of total physical activity level. At the age of 36 years, physical activity was measured with a computer-assisted version of the interview method.[14]

Since the methods to assess dietary intake and physical activity level were slightly different (computer-assisted) at age 36 years, we transformed dietary intake and physical activity level data obtained at age 27 and 36 y to measurement specific standardized z-scores (=(individual score – mean score) / standard deviation of the mean score). In this way, dietary intakes and physical activity levels became comparable over these measurement years, and relative differences in changes could be compared between subgroups over the age period from age 27 to 36 years.

Other measures

Further demographic measures, the level of education and history of chronic diseases were assessed with a self-administered questionnaire. Level of education was dichotomized into low and high (=higher vocational education and university).

Analyses

Between adolescence and young adulthood, we compared four groups with regard to living and working situation: 1) subjects who left the parental home and started to work after age 21 years, 2) subjects who left the parental home after age 21 years and started to work before age 21 years, 3) subjects who left the parental home before age 21 years and started to work after age 21 years, and 4) subjects who left the parental home and started to work before age 21 years.

Between age 27 and 36 years, we compared three groups with regard to living situation and parenting children: 1) subjects who lived alone and were not parenting children at age 36 years, 2) subjects who cohabited with a partner and were not parenting children at age 36 years, and 3) subjects who cohabited with a partner and were parenting children at age 36 years. The group of subjects who lived alone while parenting children at age 36 years was small (one man and three women) and was therefore excluded from the analyses.

Differences in changes in body weight, BMI, sum of four skinfolds, and waist circumference over follow-up (all as continuous variables) between groups (as dummies) were analyzed with linear regression analyses, adjusting for age (continuous) and the baseline level of the anthropometric measure (continuous). Level of education was considered a potential confounder in the analyses on timing of leaving the parental home and starting to work. However, additional adjustment for level of education did not essentially change the results, and the results are therefore presented without this additional adjustment.

If significant differences in weight gain between groups with and without the life transition were observed, the change in total energy intake and change in energy spent on at least moderate intensity physical activities were studied in order to find the underlying cause of these differences.

All analyses were performed for men and women separately, using SAS version 9.1 [SAS Institute Inc., North Carolina].

RESULTS

Between age 16 and 27 years, men gained weight with an average of 12.9 kg (BMI: 3.0 kg/m^2), women with 6.4 kg (BMI: 1.9 kg/m^2). The sum of four skinfolds (S4SF) increased with an average of 7.5 mm in men and 1.0 mm in women (Table 5.1). Total energy intake and physical activity level decreased over follow-up in men and women. The percentage smokers increased in men, and decreased in women between age 16 and 27.

Between age 27 and 36 years, average weight gain was 6.4 kg among men and 4.3 kg among women. The S4SF and waist circumference also increased among both men and women (Table 5.1). The total energy intake in women and the level of physical activity in both men and women increased. The prevalence of smoking decreased in men and increased in women between age 27 and 36 years.

Age period 16 – 27 years: impact of timing of leaving the parental home and starting to work.
Men who had left the parental home late and started to work early gained more body weight and S4SF than the ones who had not yet started to work at age 21 years (Table 5.2, compare groups 1 and 2). This difference was significant for BMI and was accompanied by a difference in change in total energy intake ($p<0.01$) and a difference in change in at least moderate physical activity ($p=0.10$): the men who had started to work early lowered their energy intake whereas the ones who had started to work later increased their total energy intake. In addition, the men who had started to work early, tended to keep up their physical activity level compared to a drop in physical activity among the ones who had started to work later.

Men and women who had left the parental home before age 21 years and started to work after age 21 gained more body weight and S4SF than the ones who worked at age 21 years (Table 5.2, compare groups 3 and 4). This difference was significant for body weight in women, but no differences in changes in total energy intake or at least moderate physical activity were observed between these groups of women.

TABLE 5.1 Characteristics of the participants – The Amsterdam Growth and Health Longitudinal Study.

	Men (n=78)		Women (n=87)	
Age period 16 – 27 years	Baseline	Change	Baseline	Change
Age (years, mean (SD))	16.4 ± 0.7[1]	11.1 ± 0.1	16.5 ± 0.6	11.1 ± 0.1
Body weight (kg)	62.8 ± 7.6	12.9 ± 7.9	57.2 ± 7.7	6.4 ± 5.4
BMI (kg/m^2)	19.6 ± 1.7	3.0 ± 2.0	20.0 ± 2.3	1.9 ± 1.9
Sum four skinfolds (mm)	29.2 ± 9.8	7.5 ± 13.0	46.1 ± 14.7	1.0 ± 15.3
Energy intake (MJ/d)	12.4 ± 2.8	-0.3 ± 3.1	9.2 ± 2.1	-0.2 ± 2.5
Physical activity (kMET*h/wk)	3.7 ± 2.0	-0.8 ± 2.8	3.4 ± 1.7	-0.3 ± 2.3
Smokers, %	14.1	+20.5	28.2	-4.1
Highly educated[2], %	51.3		41.4	
	Men (n=69)		Women (n=74)	
Age period 27 – 36 years	Baseline	Change	Baseline	Change
Age (years)	27.6 ± 0.7	9.0 ± 0.2	27.6 ± 0.6	9.0 ± 0.1
Body weight (kg)	75.8 ± 8.5	6.4 ± 5.3	63.4 ± 7.9	4.3 ± 5.3
BMI (kg/m^2)	22.6 ± 2.1	1.9 ± 1.5	21.9 ± 2.5	1.5 ± 1.9
Sum four skinfolds (mm)	36.8 ± 13.6	11.6 ± 9.3	46.4 ± 17.1	8.3 ± 14.0
Waist circumference (cm)	78.5 ± 5.6	5.8 ± 5.7	68.0 ± 5.5	6.1 ± 6.7
Energy intake (MJ/d)	11.9 ± 2.6	-0.0 ± 2.8	9.2 ± 2.0	0.4 ± 1.9
Physical activity (kMET*h/wk)	2.8 ± 2.1	0.9 ± 2.4	3.3 ± 1.9	2.0 ± 3.4
Smokers, %	33.3	-7.2	18.9	+6.8
Highly educated[2], %	59.4		46.0	

BMI, body mass index; MJ, megajoules; MET, metabolic equivalent.
1 Mean ± standard deviation (all such values)
2 Educational level was assessed at age 37 years.
Only those who participated both at baseline and at follow-up were included.

Among the men and women who started to work after age 21 years, no differences in changes in body weight and S4SF were observed between the groups who left the parental home early or late (Table 5.2, compare groups 1 and 3).

Men and women who had started to work before age 21 years and stayed with their parents beyond age 21 years had gained more body weight and S4SF than the ones who had left the parental home before age 21 years (Table 5.2, compare groups 2 and 4). These differences were significant for S4SF in men and for body weight in women. No corresponding differences in changes in total energy intake or at least moderate physical activity were observed between these groups.

TABLE 5.2 Average changes in anthropometric measures between age 16 – 27 years, by subgroups based on timing (before/after age 21 years) of leaving the parental home and start to work – The Amsterdam Growth and Health Longitudinal Study.

				Body weight (kg)	Body mass index (kg/m^2)	Sum of four skinfolds (mm)
			n	Mean [95% CL]	Mean [95% CL]	Mean [95% CL]
MEN	Leaving home	Starting to work				
Group 1	Late[#]	Late[#]	25	11.4 [8.6;14.2]	2.4 [1.7;3.2][2]	5.9 [1.1;10.8]
Group 2	Late	Early	19	15.6 [12.2;18.9]	3.7 [2.9;4.6][1]	11.5 [5.7;17.2][4]
Group 3	Early	Late	12	11.6 [7.6;15.5]	2.8 [1.8;3.9]	6.6 [-0.3;13.6]
Group 4	Early	Early	12	10.5 [6.3;14.7]	2.4 [1.3;3.5]	0.3 [-6.9;7.5][2]
WOMEN	Leaving home	Starting to work				
Group 1	Late	Late	10	7.1 [3.8;10.4]	2.3 [1.2;3.4]	1.6 [-7.6;10.8]
Group 2	Late	Early	35	7.1 [5.4;8.8][4]	2.1 [1.5;2.7]	2.1 [-2.7;7.0]
Group 3	Early	Late	14	7.8 [5.1;10.6][4]	2.4 [1.4;3.3]	4.0 [-3.6;11.7]
Group 4	Early	Early	28	4.5 [2.6;6.4][2,3]	1.3 [0.7;2.0]	-2.1 [-7.6;3.3]

All changes are adjusted for age and the baseline level of the anthropometric measure. Only participants who lived either with their parents or on their own (alone or together with partner or friends) were included.

[#] Late denotes after age 21 years, early denotes before age 21 years.

[1,2,3,4] The number after the 95% CL represents a significant difference at p=0.05 to the corresponding group.

Age period 27 – 36 years: impact of living situation and parenting children.

No significant differences in changes in anthropometric measures were observed between the non-parenting men who lived alone or together with a partner (Table 5.3, compare groups 1 and 2). Women who did not parent children at age 36 years and lived alone had gained significantly more body weight, and (borderline significantly) more BMI and waist circumference, compared to women who lived together with a partner (Table 5.3, compare groups 1 and 2). This difference was not accompanied by significant differences in the changes in lifestyles.

Men and women who lived together with a partner at age 36 years and were parenting children at age 36 years gained more body weight, BMI, S4SF and waist circumference than those who did not parent children (Table 5.3, compare groups 2 and 3). For men, these differences were statistically significant for body weight and BMI, for women for body weight and waist circumference. The decrease in physical activity level between age 27 and 36 years was relatively smaller in the men who parented children than in the men who did not parent children ($p<0.05$). Women who parented children at age 36 years had increased their total energy intake over the

preceding period, while women who did not parent children had decreased their total energy intake (p=0.01).

TABLE 5.3 Average changes in anthropometric measures from age 27 – 36 years, by subgroups of living situation (alone/together with partner) and parenting children (yes/no) at age 36 years – The Amsterdam Growth and Health Longitudinal Study.

		Body weight (kg)		Body mass index (kg/m^2)		Sum of four skinfolds (mm)		Waist circumference (cm)	
MEN	n	Mean	[95% CL]	Mean	[95% CL]	Mean	[95% CL]	Mean	[95% CL]
Group 1[#]	17	4.0	[1.4;6.6][3]	1.1	[0.4;1.9][3]	7.5	[3.1;11.9][3]	2.1	[-0.5;4.7][3]
Group 2	16	4.7	[2.1;7.3][3]	1.5	[0.8;2.3][3]	10.5	[6.0;15.0]	5.0	[2.3;7.7]
Group 3	35	8.3	[6.5;10.1][1,2]	2.5	[2.0;3.0][1,2]	13.9	[10.9;17.0][1]	8.0	[6.2;9.8][1]
WOMEN									
Group 1	14	5.8	[3.0;8.5][2]	2.0	[1.0;2.9]	11.4	[4.1;18.7]	7.2	[3.7;10.8]
Group 2	9	1.0	[-2.5;4.4][1,3]	0.4	[-0.8;1.6]	3.2	[-5.8;12.3]	1.6	[-2.7;6.0][3]
Group 3	47	4.8	[3.3;6.3][2]	1.6	[1.1;2.2]	8.5	[4.6;12.3]	6.7	[4.8;8.6][2]

All changes are adjusted for age, level of education and the baseline level of the anthropometric measure.

[#] group 1: living alone and not parenting children at age 36 years; group 2: living together with partner and not parenting children at age 36 years; group 3: living together with partner and parenting children at age 36 years. [1,2,3] The number after the 95% CL represents a significant difference at p=0.05 to the corresponding group.

DISCUSSION

Largest increases in body weight and S4SF between age 16 to 27 years were observed in men who had started to work before age 21 years, while they were still living with their parents. Smallest increases were observed among men and women who left the parental home and started to work before age 21 years.
Between age 27 and 36 years, women who lived alone had a larger increase in body weight and body fat than women who lived with a partner. Parenting children was associated with larger increases in body weight and waist circumference in men and women. Women who were parenting children at age 36 years showed a significant increase in total energy intake between age 27 and 36 years. Unexpectedly, some other significant changes in behaviours observed were contradictory to observed changes in anthropometry: a decrease in energy intake was associated with an increase in BMI and maintaining the physical activity level was associated with an increase in body weight.
The most important drawback of the present study is the small number of participants that could be included in the analyses. This was inherent to the study design, in which half of the sample was not invited for measurements at mean ages 21 and 27 years, but also to the difficulty to follow young people over the long follow-up period of this study. Despite the small numbers in each group, we observed several plausible effects of life transitions on anthropometric indices. This suggests that the life transitions under study may affect body weight or fat mass. However, to detect relations between anthropometry and changes in underlying lifestyle factors a larger sample size is needed, because of the large error in measuring lifestyle behaviours.

Another drawback of the present study is that we do not have information about the kind of job (active / sedentary) the adults had. In an earlier paper, based on other data, we have shown that the activity during the last job is an important predictor for weight and waist change after retirement.[16] Consequently, one could hypothesize that young adults who start to work in sedentary jobs will gain more weight than the ones who start to work in more active jobs. Since the level of education in our sample was rather high (57% completed a higher vocational or university education), we expect that most of them had started in a sedentary job, resulting in relatively large increases in body weight.

Between ages 16 and 21 years, men and women still gained body height. This gain in body height will not have affected the relations between life transitions and weight gain, because in our analyses comparisons were made within groups of the same age.

No information on parenting children was available before age 27 years. In the early nineties, the mean age of women at first childbirth was 27-28 years in the Netherlands.[17] Since the level of

education in our population was relatively high and higher educated women start parenting children at a higher age than lower educated women, we do not think that the omission of data about parenting children before age 27 years has inversely affected the results and conclusions of our study. In our sample, 22% of the women and 6% of the men were parenting children at age 27 years. Further, we did not study the effects of marital change, but we studied the impact of living situation: living alone or cohabiting with a partner. This was done because in the Netherlands people often start cohabiting before, or instead of, marrying.[18]

To our knowledge, reports about the effects of leaving home and starting to work are scarce, probably due to the lack of appropriate longitudinal data. In the study by Burke et al (2004), the prevalence of overweight increased more in men and women who had left the parental home before age 25 years, in comparison to those still living with their parents. In our study, we observed that men who stayed with their parents, but already started to work before age 21 years showed the largest increases in body weight and fat mass. This can be explained by the fact that the working men living with their parents had more money to spend than non-working men or men who live on their own.[19] In men, the difference in weight gain between the group who stayed with their parents and already started to work before age 21 years in contrast to the other three groups was larger than in women. This may be explained by the fact that young men and women have different interests to spend their money on: expenses of young men are more associated with energy balance related behaviours (e.g. snacks or motorized transport) than expenses of young women (e.g. clothes or gifts).[20]

Until now, most studies that investigated life transitions in young adults have evaluated the effects of changes in marital status on anthropometry.[5,7,21-23] These studies showed that entering marriage or cohabitation is associated with additional weight gain among both men and women. One study found that marital status was associated with more fatness in men, but not in women.[22] To our knowledge, our study is the first study making a distinction between cohabiting and parenting children. Results of our study suggest that not the cohabiting, but raising children seems to have caused the effects of increasing body weight, body fatness and waist circumference in both men and women. The finding that these increases are also high in men who start parenting is relatively new, since other studies mostly focused on women only. In the study by Burke, women with at least one child increased more in body weight and waist circumference between age 18 and 25 years than women without children. Weight gain was not different for men with and without children.[7] Our finding that parenting children was associated with weight gain in both men and women suggests parenting children being a lifestyle cause rather than a biological cause (due to pregnancy) of weight gain. Lifestyle interventions may be suitable for both women and men who are about to become parents.

In addition, in most studies, the men and women who entered marriage were compared to those who were already married. In our study, the ones who cohabited with a partner were compared to those who lived alone. The effects of cohabiting seem different for men and women: no effect was observed among men, while a protective effect was seen among women.

In most cases, the impact of changes in living situation could not be explained by (changes in) reported physical activity level, energy intake or smoking. Maybe temporary changes in behaviour have taken place in the period between two measurements that were not administered with the interview. Another explanation could be that persons were not aware of the small changes in behaviour and therefore did not report differently in successive interviews. Only small changes in behaviour are needed to make the differences in weight gain as found in our study. For example, the difference in weight gain over a 9-years period was about 4 kg between parenting and non-parenting young adults. This difference of less than 400 grams change per year corresponds to eating one additional sweet per day only or to walking for a few minutes per day. Differences of this magnitude are easily missed in an interview.

Our study population was rather small and therefore our findings merely give an indication of the impact of the life transitions on body weight and fat mass. Especially the result that parenting children is associated with additional weight gains in both men and women warrants further attention.

In conclusion, life transitions seem to affect body weight and body fatness. Especially men starting to work while living with their parents and young men and women planning a family seem important target groups for intervention programs to prevent (excessive) weight gain.

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Changes in diet and physical activity in relation to weight gain in Dutch young adults

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Submitted for publication

ABSTRACT

In adults, major weight gain often occurs before the age of 35 years. In the present study, we aimed at identifying modifiable determinants of weight gain in this age group. For this purpose we used data from 508 healthy Dutch men and women aged 26-36 years of the Doetinchem Cohort Study. Body weight and height were measured and diet, physical activity and demographic data were assessed using self-administered questionnaires at baseline (1994-1997) and after 5 years of follow-up (1999-2002). Sex specific regression analyses were performed to test for associations between lifestyle (both baseline and change) and change in body weight. The average weight gain in our sample was 0.8 kg/year. This weight gain was accompanied by several changes in behaviour. Baseline behaviour and changes in behaviour were differently, and sometimes oppositely, associated with weight gain. Weight gain in men was significantly associated with a decrease in hours of biking, a decrease in frequency of eating breakfast, and an increase in juice consumption. Among women, weight gain was associated with an increase in sweet snack consumption. We conclude that during young adulthood, several changes in diet and physical activity seem responsible for the elevated gain in body weight. Our study indicates that these determinants are different for men and women. Furthermore, our study shows that it seems pertinent to study changes in behaviour, rather than only baseline behaviour, when studying determinants of weight gain.

INTRODUCTION

The global prevalence of overweight and obesity is increasing.[1-3] In adults, highest weight gain is observed in those younger than 35 years.[4,5] This is probably the result of changes in diet and physical activity associated with life transitions during young adulthood.

The relation between lifestyle and weight gain in a general adult population was investigated in several previous studies,[6-19] but only a few focused on young adults.[20-24] In most studies, baseline energy intake and physical activity was studied in relation to subsequent, usually self-reported, weight gain. Some investigators studied only men[10] or women[6,8,18,20] and usually diet[6,7,13,19] and physical activity[8,10,16] were studied separately.

It is likely that the change in behaviour, rather than the baseline behaviour is associated with weight gain, since effects of changes in energy balance are related directly with changes in weight. This is in contrast to other disease-related associations where lifestyle has an impact many years later (e.g. diet and colon cancer). Further, baseline behaviour is only likely to be associated with future weight gain when behaviour is stable over time, or when baseline behaviour is positively associated with changes in behaviour. Finally, a determinant of weight gain is weight loss in the preceding period.[6,12] Baseline behaviour may therefore actually reflect the end of a period of negative energy balance and an association with subsequent weight gain is therefore conceptually difficult to interpret. These aspects emphasize the need to study changes in behavioural factors in relation to changes in body weight.

The aim of the present study was to identify modifiable determinants of weight gain among young adults. Within these analyses, we studied the development of lifestyle behaviours in young adults over time, and we studied the association between both baseline and changes in diet and physical activity with weight gain.

METHODS

Study population

For the purpose of the present study, we have included subjects who participated in the first three examination rounds of the prospective Doetinchem Cohort Study (1987-1991,[25] 1993-1997,[26] and 1998-2002)[27] and were aged 20-29 years at the first examination. For this group of young adults, the participation rates at the first examination were 32% and 41% for males and females, respectively. Participation rates for the second and third examination were between 71% and 76%. All participants gave written informed consent. The study was approved according to the guidelines of the Helsinki Declaration by the external Medical Ethics Committee of the Dutch TNO Research Institute.

Since 1994 identical questionnaires have been used, allowing us to study changes in dietary intake and physical activity in relation to changes in weight from that year onwards. Therefore, the measurements that were performed before 1994 were excluded for the present analyses. Measures from the first examination round (1987-1991) are only used to determine weight change before baseline. In the present paper, baseline data refer to the second examination (1994-1997), when subjects were aged 26-36 years, and follow-up refers to the third examination (1999-2002).

In total, 639 young adults completed the measurements in 1988-1991, 1994-1997 and 1999-2002. Exclusion criteria for the present study were pregnancy (n=48), suffering from cancer (n=10), cardiovascular diseases (n=2), or diabetes (n=4) during any of the examinations, being underweight (body mass index <18.5 kg/m^2)[1] at baseline (n=10), weight loss during follow-up (≥0.4 kg loss/year, n=73, since the focus of this paper was on determinants of weight gain and to take into account possible underlying diseases causing weight loss and affecting patterns of energy balance related behaviours), or having missing data for confounding variables (n=1). After these exclusions, 254 men and 254 women remained for the present analyses.

Weight, height, and body mass index

Body weight and height were measured at the municipal health centre, with participants wearing light indoor clothing with emptied pockets and without shoes. Height was measured to the nearest 0.5 cm and weight to the nearest 0.5 kg. To adjust for light indoor clothing, one kilogram was subtracted from the measured weight. Body mass index (BMI) was calculated as weight divided by height squared (kg/m^2).

Subjects were assigned to three groups of weight change between baseline and follow-up using cut-off points of 0.4 and 1 kg per year: stable weight (weight change <±0.4 kg/year), moderate

weight gain (0.4-1.0 kg/year) and high weight gain (≥1.0 kg/year). These cut-off points are primarily based on classifications used in previous studies on determinants of weight gain.[11,12,17]

Diet

Habitual diet was assessed using the Dutch version of the EPIC (European Prospective Investigation into Cancer and Nutrition) Food Frequency Questionnaire, a validated semi-quantitative food frequency questionnaire, that was designed to assess the habitual consumption of 178 food items during the previous year.[28,29] Nutrient and energy intake was quantified for each individual using an extended version of the 1996 computerised Dutch food composition table.[30] For potatoes, pasta, rice, meat and vegetables, both portion size and consumption frequencies were available. For fish, meat substitutes, fruits (as times/week), dairy drinks, sugared soft drinks, juices and alcoholic beverages (as drinks/day) only consumption frequencies were available. Sweet and savoury snacks were quantified in grams consumed per day. To determine the average portion size for several food groups (potatoes, rice and vegetables), portion sizes of specific products within the food group were weighted for their frequency of consumption. Average portion sizes were expressed in numbers of standard amounts: 70 grams per serving for potatoes, 45 grams for pasta, 55 for rice, 50 for meat and 60 for vegetables.

Physical activity

Data on physical activity was collected by use of a validated EPIC questionnaire on physical activity.[31] The questionnaire included questions on hours spent per week on leisure time activities, household activities, sports, and activities at the place of work, referring to the previous 12 months. An added question on sports and other strenuous physical activities enabled the respondents to report on type, duration and number of weeks per year for three different activities. The number of stairs climbed on a usual day was also recorded. Average duration per week was weighted by the number of weeks per year that the activity was performed. To be able to take into account intensity of physical activity, all activities were converted into Ainsworth scores[32] expressed in metabolic equivalent values (MET-values). Leisure time physical activity included walking (3.5 MET), bicycling (5 MET), doing odd jobs / do it yourself (3 MET), and gardening (5 MET). Overall leisure time intensity was calculated by weighing intensities of the individual activities by their duration. Occupational physical activity was classified as 'sitting' (1.5 MET), 'sitting and standing' (2 MET), 'walking regularly while carrying light objects' (3 MET), and 'walking regularly while carrying heavy objects' (4 MET). Overall work related intensity was calculated by weighing the intensities of both work and household activities (4 MET) by their duration. An overall level of physical activity was assessed, based on the combination of physical activity levels at work and during leisure time, according to

Wareham's classification, by which subjects are classified as inactive, moderately inactive, moderately active, or active.[33]

Data on demographic factors, medical history of chronic diseases, reproductive status, and smoking, were collected by use of a self-administered questionnaire. Educational level was assessed as highest level reached between 1999 and 2002, and classified into three categories: low (intermediate secondary education or less), moderate (intermediate vocational or higher secondary education) and high (higher vocational education or university). Smoking status was defined as persistent non-smokers (smoking less than one cigarette per month), persistent smokers, quitters and starters, based on a question about current cigarette smoking at baseline and at follow-up.

Data analyses

Average changes in components of diet and physical activity between baseline and follow-up were tested for significance in paired t-tests. Separate baseline behaviours were related to weight gain, adjusting for age and BMI at baseline, prior weight gain, smoking status, and, for women, new deliveries during follow-up, by use of linear regression analyses. When studying the associations between dietary intake and weight gain, an additional adjustment was made for the overall level of physical activity at baseline. When studying associations between aspects of physical activity and weight gain, adjustments were made for total energy intake at baseline. Except for smoking status (four categories, as dummies), overall level of physical activity (four incremental categories) and new deliveries (discrete variable), all other variables were entered in the model as continuous variables. To study whether changes in behaviour were related to weight gain, similar regression models were used. However, in these models, changes in diet were adjusted for *change* in overall level of physical activity, and changes in physical activities were adjusted for *change* in total energy intake. To determine the independent effects of changes in behaviour on weight gain, changes in diet and physical activities that were associated with weight gain ($p<0.20$), were combined into a multivariate linear regression model, adjusting for age, BMI, prior weight gain, smoking status, and, for women, new deliveries. No derived measures of diet (total energy intake, energy percentages from macro-nutrients and fibre-density) were entered in the multivariate models, in order to prevent collinearity with the separate aspects of diet. All analyses were carried out separately for men and women and were conducted in SAS version 8.2.

RESULTS

Weight gainers tended to be younger and were more likely to have quitted smoking during follow-up than subjects who kept their body weight stable (Table 6.1). Men who gained weight had a higher BMI at baseline than men with a stable weight. Women who gained weight were less likely to be cigarette smokers than women with a stable weight (Table 6.1).

Table 6.2 shows that, among men, total energy intake, the average portion size of rice and the frequencies of eating potatoes, meat and vegetables decreased, while the frequencies of eating pasta and fish increased over follow-up. The average time spent on household activities and the average intensity of leisure time physical activities increased, while the average time spent on walking and the average intensity of work related physical activities decreased over follow-up (Table 6.2). Among women, the average portion size of rice, the frequencies of eating potatoes and meat and the consumption of dairy drinks decreased, while the frequency of eating pasta, the average consumption of sweet snacks and alcoholic beverages increased. Average time spent on working and walking decreased among women (Table 6.2).

Table 6.3 shows that in men, some changes in diet and physical activity behaviour were associated with weight gain, but baseline behaviour was not. An increase in portion size of meat, frequency of juice consumption, and a decrease in frequency of eating breakfast and in hours spent biking over follow-up were related to weight gain in men (Table 6.3). Table 6.4 shows that in women, several baseline behaviours were associated with weight gain: a low frequency of eating meat, a low sweet snack consumption, a low total energy intake and consumption of a diet high in fibre density were associated with weight gain during follow-up. In women, also changes in behaviours were associated with weight gain, but sometimes oppositely directed to the associations between baseline behaviours and weight gain: an increase in sweet snack consumption and an increase in total energy intake over follow-up were related to weight gain (Table 6.4).

Entering the variables that were associated with weight gain into a multivariate model lowered the magnitude and significance level of the associations, but did not affect the direction of the associations (Tables 6.3 and 6.4). Weight gain in men remained statistically significantly associated with a decrease in frequency of eating breakfast, an increase in juice consumption and a decrease in time spent on biking (Table 6.3). Weight gain in women remained associated with an increase in sweet snack consumption (Table 6.4).

TABLE 6.1 Demographic and anthropometric characteristics of the study population over a five-year follow-up period in men and women.

	Total population		Stable weight ± <0.4 kg/year		Weight gain 0.4-1 kg/year		Weight gain ≥1 kg/year	
	Men (n=254)	Women (n=254)	Men (n=79)	Women (n=82)	Men (n=89)	Women (n=88)	Men (n=86)	Women (n=84)
Baseline								
Age (years)	31.6 ± 2.9[1]	31.3 ± 2.9	32.0 ± 2.8	31.8 ± 2.7	31.7 ± 2.7	31.1 ± 3.0	31.0 ± 3.1	31.1 ± 3.0
Weight (kg)	81.5 ±10.7	66.8 ± 10.2	78.7 ± 10.7	65.9 ± 9.9	83.0 ± 10.2	66.3 ± 10.7	82.5 ± 10.8	68.3 ± 10.0
Height cm)	182.6 ± 6.4	168.2 ± 6.4	182.1 ± 7.1	167.5 ± 5.5	182.9 ± 6.0	169.4 ± 6.4	182.7 ± 6.0	167.8 ± 7.1
BMI (kg/m^2)	24.5 ± 2.7	23.7 ± 3.5	23.8 ± 2.5	23.6 ± 3.5	24.9 ± 2.8	23.2 ± 3.3	24.8 ± 2.7	24.4 ± 3.7
Normal weight (BMI 18.5 – 24.9), %	63.0	74.4	70.9	79.3	58.4	77.3	60.5	66.7
Moderate overweight (BMI 25.0 – 29.9), %	32.3	18.9	26.6	15.9	34.8	17.1	34.9	23.8
Obese (BMI over 30.0), %	4.7	6.7	2.5	4.9	6.7	5.7	4.7	9.5
Low education level, %	29.5	35.4	29.1	30.5	33.7	34.1	25.6	41.7
Moderate education level, %	49.6	50.4	49.4	53.7	46.1	52.3	53.5	45.2
High education level, %	20.9	14.2	21.5	15.9	20.2	13.6	20.9	13.1
Average prior weight change (kg/year)	0.67 ± 0.82	0.53 ± 0.82	0.70 ± 0.72	0.49 ± 0.70	0.79 ± 0.85	0.59 ± 0.78	0.52 ± 0.87	0.50 ± 0.97
Energy intake (MJ/day)	12.0 ± 3.0	9.0 ± 2.1	11.9 ± 3.0	9.1 ± 2.2	11.7 ± 2.9	9.2 ± 2.1	12.3 ± 3.0	8.5 ± 2.1
During follow-up								
Duration of follow-up in years	5.0 ± 0.2	5.0 ± 0.2	5.0 ± 0.1	5.0 ± 0.2	5.0 ± 0.2	5.0 ± 0.2	5.0 ± 0.2	5.0 ± 0.2
Average weight change (kg/year)	0.82 ± 0.75	0.87 ± 0.87	0.05 ± 0.24	0.05 ± 0.22	0.69 ± 0.18	0.67 ± 0.18	1.66 ± 0.56	1.88 ± 0.71
Persistent non-smoker, %	63.0	65.4	60.8	58.5	65.2	72.7	62.8	64.3
Persistent smoker, %	26.0	26.0	30.4	34.2	24.7	21.6	23.3	22.6
Quitter, %	6.7	5.1	3.8	1.2	4.5	3.4	11.6	10.7
Starter, %	4.3	3.5	5.1	6.1	5.6	2.3	2.3	2.4
New deliveries, %	-	31.5	-	28.1	-	31.8	-	34.5

1 mean ± SD (all such values).

TABLE 6.2 Mean changes in diet and physical activity over the five year follow-up period in men and women.

	Men (n=254)		Women (n=254)	
	Mean [95%-CL]	p	Mean [95%-CL]	p
DIET				
Portion size warm meal (# of servings)				
Potatoes	0.02 [-0.09; 0.13]	0.71	0.06 [-0.02; 0.15]	0.16
Pasta	-0.08 [-0.30; 0.14]	0.47	0.15 [-0.10; 0.40]	0.23
Rice	-0.18 [-0.33; -0.04]	0.01	-0.13 [-0.26; -0.01]	0.04
Meat	-0.06 [-0.12; 0.01]	0.08	0.05 [-0.02; 0.12]	0.18
Vegetables	-0.03 [-0.09; 0.02]	0.25	0.03 [-0.02; 0.09]	0.21
Frequencies (times/week)				
Potatoes	-0.51 [-0.70; -0.32]	<0.01	-0.47 [-0.69; -0.25]	<0.01
Pasta	0.12 [0.01; 0.24]	0.03	0.15 [0.06; 0.24]	<0.01
Rice	0.04 [-0.03; 0.12]	0.27	0.03 [-0.06; 0.12]	0.51
Meat	-0.21 [-0.39; -0.04]	0.02	-0.21 [-0.38; -0.05]	<0.01
Fish	0.08 [0.02; 0.14]	<0.01	0.06 [-0.01; 0.12]	0.08
Meat substitutes	-0.02 [-0.10; 0.06]	0.65	-0.01 [-0.13; 0.11]	0.86
Vegetables	-0.24 [-0.46; -0.01]	0.04	-0.00 [-0.22; 0.21]	0.97
Fruit	-0.60 [-1.52; 0.32]	0.20	-0.09 [-0.82; 0.64]	0.81
Breakfast	0.01 [-0.21; 0.23]	0.93	0.01 [-0.22; 0.24]	0.93
Eating out for dinner	-0.04 [-0.13; 0.06]	0.42	-0.02 [-0.11; 0.06]	0.63
Snacks (100 grams/day)				
Sweet snacks	-0.02 [-0.06; 0.02]	0.31	0.04 [0.01; 0.08]	0.02
Savoury snacks	-0.00 [-0.04; 0.04]	0.89	0.03 [-0.02; 0.07]	0.21
Drinks (glasses/day)				
Dairy drinks	-0.03 [-0.22; 0.15]	0.73	-0.23 [-0.39; 0.06]	<0.01
Sugared soft drinks	-0.01 [-0.17; 0.15]	0.89	-0.04 [-0.14; 0.06]	0.46
Juice	0.03 [-0.05; 0.11]	0.50	0.10 [-0.02; 0.22]	0.10
Alcoholic beverages	-0.11 [-0.29; 0.08]	0.25	0.10 [0.04; 0.16]	<0.01
Energy intake (MJ/day)	-0.55 [-0.83; -0.27]	<0.01	-0.00 [-0.24; 0.23]	0.97
En % Protein	0.23 [-0.00; 0.45]	0.05	-0.23 [-0.44; -0.01]	0.04
En % Carbohydrate	0.39 [-0.11; 0.88]	0.12	0.03 [-0.58; 0.64]	0.93
En % Fat	-0.58 [-1.03; -0.13]	0.01	-0.14 [-0.67; 0.39]	0.60
En % Alcohol	-0.03 [-0.36; 0.30]	0.86	0.34 [0.14; 0.54]	<0.01
Fibre density (grams/MJ)	0.01 [-0.04; 0.06]	0.61	-0.02 [-0.08; 0.04]	0.56
PHYSICAL ACTIVITY				
Mean duration (hours/week)				
Work	-0.52 [-2.19; 1.16]	0.54	-2.91 [-4.58; -1.25]	<0.01
Household	1.14 [0.38; 1.90]	<0.01	-0.23 [-2.20; 1.75]	0.82
Sports	-0.39 [-0.78; 0.00]	0.05	-0.07 [-0.33; 0.20]	0.62
Bicycling	-0.44 [-0.93; 0.06]	0.09	-0.28 [-0.79; 0.24]	0.29
Walking	-4.57 [-6.36; -2.78]	<0.01	-2.10 [-3.67; -0.53]	<0.01
Gardening	0.03 [-0.24; 0.30]	0.81	0.19 [-0.10; 0.49]	0.20
Doing odd jobs	-0.46 [-1.14; 0.23]	0.19	0.10 [-0.27; 0.47]	0.61
Climbing stairs (number per day)	-0.09 [-1.05; 0.88]	0.86	0.66 [-0.54; 1.85]	0.28
Intensity (MET-values)				
Work related physical activity	-0.12 [-0.21; -0.03]	<0.01	0.04 [-0.03; 0.11]	0.22
Leisure time physical activity	0.12 [0.02; 0.23]	0.02	0.05 [-0.02; 0.12]	0.17

CL = Confidence limits, MJ = Megajoule, En % = Energy percentage, MET-values = Metabolic equivalent values

TABLE 6.3 Baseline and changes in diet and physical activity in relation to weight gain (kg/year) over a 5-year period in young adult men (n=254).

	Baseline behaviour		Changes in behaviour			
	Model 1*		Model 1**		Model 2***	
	Beta	p	Beta	p	Beta	p
DIET †						
Portion size warm meal (# of servings)						
Meat			0.21	0.02	0.16	0.07
Frequencies (times/week)						
Potatoes	-0.05	0.08				
Pasta			0.07	0.18	0.06	0.25
Fish			0.18	0.06	0.13	0.15
Breakfast			-0.06	0.03	-0.05	0.03
Drinks (glasses/day)						
Dairy drinks	0.05	0.11				
Sugared soft drinks			0.05	0.14	0.05	0.17
Juices			0.15	0.03	0.15	0.03
En% Protein			0.04	0.12	-	-
PHYSICAL ACTIVITY ‡						
Mean duration (hours/week)						
Bicycling			-0.03	<0.01	-0.03	<0.01
Intensity (METs)						
Work related physical activity			-0.08	0.19	-0.10	0.09
Leisure time physical activity			-0.10	0.06	-0.09	0.08

Associations that reached significance levels of p<0.20 in the analyses of model 1 for the baseline behaviours and for the changes in behaviours are presented only, for clarity reasons. * Beta denotes change in weight over follow-up (in kg/year) when the baseline behaviour is one unit higher. Associations are adjusted for age, BMI, prior weight change and smoking status. ** Beta denotes change in weight over follow-up (in kg/year) when the behaviour is elevated with one unit over the follow-up period. Associations are adjusted for age, BMI, prior weight change and smoking status. † Associations between baseline/change in diet and weight gain were additionally adjusted for baseline/change in level of physical activity. ‡ Associations between baseline/change in physical activity and weight gain were additionally adjusted for baseline/change in total energy intake. *** In model 2, all changes in behaviour that were associated with weight gain in model 1 at p<0.20 are studied together in a multivariate model, adjusting for age, BMI, prior weight change and smoking status. NB Negative effects reflect less weight gain with a relative increase in the behavioural factor.

TABLE 6.4 Baseline and changes in diet and physical activity in relation to weight gain (kg/year) over a 5-year period in young adult women (n=254).

	Baseline behaviour		Changes in behaviour			
	Model 1*		Model 1**		Model 2***	
	Beta	p	Beta	p	Beta	p
DIET †						
Portion size warm meal (# of servings)						
Pasta	-0.04	0.12				
Meat	-0.12	0.19				
Frequencies (times/week)						
Pasta	-0.14	0.07				
Meat	-0.09	<0.01				
Fruit			-0.01	0.18	-0.01	0.21
Breakfast	-0.05	0.09				
Eating out for dinner	-1.13	0.07				
Sweet snacks (100 grams/day)	-0.47	0.02	0.43	0.01	0.37	0.03
Energy intake (MJ/day)	-0.05	0.03	0.06	0.04	-	-
Fiber density (grams/MJ)	0.31	<0.01	-0.19	0.08	-	-
PHYSICAL ACTIVITY ‡						
Mean duration (hours/week)						
Work	0.01	0.07				
Household	-0.01	0.15				
Sports			-0.04	0.09	-0.03	0.25
Walking			0.01	0.18	0.00	0.60
Doing odd jobs			0.03	0.10	0.02	0.30
Intensity (METs)						
Work related physical activity	-0.13	0.12				
Leisure time physical activity	0.17	0.16	-0.18	0.07	-0.12	0.31

Associations that reached significance levels of $p<0.20$ in the analyses of model 1 for the baseline behaviours and for the changes in behaviours are presented only, for clarity reasons. * Beta denotes change in weight over follow-up (in kg/year) when the baseline behaviour is one unit higher. Associations are adjusted for age, BMI, prior weight change, smoking status and new deliveries. ** Beta denotes change in weight over follow-up (in kg/year) when the behaviour is elevated with one unit over the follow-up period. Associations are adjusted for age, BMI, prior weight change, smoking status and new deliveries. † Associations between baseline/change in diet and weight gain were additionally adjusted for baseline/change in level of physical activity. ‡ Associations between baseline/change in physical activity and weight gain were additionally adjusted for baseline/change in total energy intake. *** In model 2, all changes in behaviour that were associated with weight gain in model 1 at $p<0.20$ are studied together in a multivariate model, adjusting for age, BMI, prior weight change, smoking status and new deliveries.

DISCUSSION

In the present study, it was observed that several energy balance related behaviours changed over time in young adults. In men, changes in diet and physical activity over follow-up were associated with weight gain, whereas baseline diet and physical activity were not. In women, both baseline levels and changes of diet and physical activity were associated with weight gain. However, some baseline levels of behaviour and their changes were associated with weight gain in opposite directions. Men and women had different determinants of weight gain. Among men, a decrease in using breakfast, an increase in juice consumption and a decrease in time spent on biking were associated with weight gain. Among women, an increase in sweet snack consumption was associated with weight gain.

Since both the diet and physical activity levels were not stable over time in our study, we have put emphasis on changes in behaviour in relation to weight gain.
There are several reasons why changes in behaviour may give more valid information than baseline behaviour in relation to weight gain, as summed in the introduction: 1) there is a direct link between changes in energy balance and changes in weight, 2) baseline behaviour is only likely to be related to future weight gain when behaviour is relatively stable, and 3) weight gain is predicted by relative weight loss in the preceding period. Furthermore, as well known, the use of questionnaires to assess diet and physical activity is susceptible to misreporting,[34-36] that may influence the relations between behaviour and weight gain. By studying changes in behaviour, it can be argued that a possible distorting effect of misreporting on studied associations is eliminated if the extent of misreporting is similar at both examinations. In our study, systematic underreporting by individuals may partly explain the oppositely directed associations between baseline behaviours and weight gain among women: Especially women with a higher BMI tend to underreport energy intake and over-report physical activities.[35,37] Relations between the changes in behaviours and weight gain were generally in line with biological expectations.

One of the methodological drawbacks in the present study is the possibility of inverse causation. Subjects may change their behaviour in response to weight gain as well as the other way around. Within the present analyses, it was impossible to disentangle this problem. Secondly, the participation rates among the young adults in the present study were low. However, this has probably not affected the association between behaviour and weight gain.[38] It is important to note that relatively small sustained or large temporary changes in behaviour can result in weight gain, which could be missed easily by questionnaire research. Still, we were able to identify associations between components of diet and physical activity and weight gain.

The finding in the present study that behaviour was not stable over time in young adults was also found by Parsons et al, who showed that most young adults in their study changed their physical activity and dietary habits over a period of nine years.[39] Until now, only a few studies were conducted in young adults investigating lifestyle determinants of weight gain.[20-24] Most of these studies related weight gain to lifestyle measured at a single point in time.[20,22,23] Determinants of weight gain in these studies were a sedentary lifestyle at baseline[22] or at follow up,[20] a high consumption of savoury snacks at baseline,[22] high level of eating take-away food at baseline[20] and a low fibre consumption just before follow-up.[23] None of these findings were confirmed in the present study.

Studies in which changes in behaviour were related to weight gain in young adults found that a decrease in physical activity,[21,24] and an increase in relative fat intake[21] were determinants of weight gain. Lewis et al studied both baseline and changes in lifestyles in relation to weight gain among young adults and found that baseline and changes in lifestyle were comparably related to weight gain.[21] Similar to our findings among young adults, Klesges et al found a different pattern of predictors of weight change for adult men than for adult women.[14] Determinants of weight gain in general adult populations were an increase in total energy intake, [9,14] an increase in fat intake,[9,14] an increase in frequency of fast food restaurant use,[18] a decrease in fruit consumption,[19] a decrease in the consumption of (partly) skimmed milk,[19] a decrease in work activity level,[14] and a decrease in frequency of physical activity.[9] However, taking into account the methodological drawbacks, (changes in) lifestyles that are associated with weight gain in the present and previous studies should be interpreted with caution, since they are not by definition determinants of weight gain.

In conclusion, several aspects of energy balance related behaviour are not stable over time in young adults. Furthermore it seems pertinent to study changes in behaviour, rather than only baseline behaviour, in order to study determinants of weight gain. Lifestyles that were associated with weight gain among men were a decrease in using breakfast, an increase in juice consumption and a decrease in time spent on bicycling. Among women, an increase in sweet snack consumption was associated with weight gain. This study can contribute to the development of interventions that aim to prevent weight gain in young adults.

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Effects of retirement on lifestyle in relation to changes in weight and waist circumference in Dutch men – a prospective study

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Published in: *Public Health Nutrition* 2005: **8**(8), 1266–1274

ABSTRACT

Objective: To study changes in lifestyle in relation to changes in body weight and waist circumference associated with occupational retirement in men.
Design: A prospective cohort study with 5 years follow-up. At baseline and at follow-up, questionnaires were completed and body weight and waist circumference were measured.
Setting: The Doetinchem Cohort Study, consisting of inhabitants of Doetinchem, a town in a rural area of the Netherlands.
Subjects: In total 288 healthy men aged 50-65 years at baseline, who either remained employed or retired over follow-up.
Results: The effect of retirement on changes in weight and waist circumference was dependent on type of former occupation. Increase in body weight and waist circumference was higher among men who retired from active jobs (0.42 kg/year and 0.77 cm/year respectively) than among men who retired from sedentary jobs (0.08 kg/year and 0.23 cm/year respectively). Weight gain and increase in waist circumference was associated with a decrease in fruit consumption and fibre-density of the diet, with an increase in frequency of eating breakfast, and with a decrease in several physical activities, such as household activities, bicycling, walking, and doing odd jobs.
Conclusion: Retirement was associated with an increase in weight and waist circumference among those with former active jobs, but not among those with former sedentary jobs. Retirement may bring opportunities for healthy changes in diet and physical activity, which could be used in health promotion programs.

INTRODUCTION

Occupational retirement is usually accompanied by substantial changes in lifestyle. If retiring persons do not replace their former work-related physical activity with other physical activities or do not diminish energy intake to a level that matches the new energy requirements, they are likely to gain weight and/or increase waist circumference. Alternatively, people who had sedentary occupations may, after retirement, have an increased opportunity to engage in physical activities.

People beyond the age of 50 are likely to lose muscle mass,[1] partly due to decreased physical activity, which is accompanied by a decrease in basal energy requirement.[2] They also, on average, accumulate more fat mass in the abdominal region.[3,4] Middle-aged people can experience a gain in abdominal fat mass, and thus increased waist circumference,[4] without a change in body weight.[5]

Prevalence of obesity is highest among subjects aged 50-65 years, then levels off and subsequently declines.[6] In addition, overweight and (particularly abdominal) obesity is associated with higher risks of several chronic diseases, such as type II diabetes and cardiovascular diseases.[7,8]

Surprisingly little is known about the changes in lifestyle after retirement, and their effect on weight and waist circumference. Some studies evaluated changes in lifestyle across retirement, but did not link them to changes in weight or waist circumference.[9-12] In another study, changes in body fat and muscle were evaluated in retiring men, but changes in lifestyle were not included.[13] Others[14] studied the relation between changes in lifestyle and increase in waist circumference, but did not analyse the impact of retirement.

In the present study we evaluate the impact of retirement on diet, physical activity, body mass index and waist circumference, over a five-year follow-up period in a population-based cohort. We hypothesise that a sudden reduction in work-related physical activity of moderate intensity may lead to unfavourable changes in energy balance and thereby lead to an increase in body weight and waist circumference.

SUBJECTS AND METHODS

The Doetinchem Cohort Study is a prospective study carried out among inhabitants aged 20 to 59 years of Doetinchem, a town in a rural area of the Netherlands, who visited the municipal health centre between 1987 and 1991 (n=12 404).[15] A total of 7768 of these participants were re-invited between 1993 and 1997[16] and 6582 participants were again invited between 1998 and 2002. Not all participants in the first examination were invited for the second examination due to logistic reasons. Participants in the first examination who died or emigrated during follow-up or who actively refused to participate in the second examination were not invited for the third examination. The participation rate was 62% for the first examination. Participation rates for the second and third examination were 79% and 75% respectively, among subjects who participated in the first examination and were invited for the second and third examination respectively.

Demographic characteristics and medical history of chronic diseases were collected using standardised questionnaires at baseline and at follow-up, including items regarding educational level, smoking status, and working status. Educational level was assessed as the highest level reached and classified into three categories: low (intermediate secondary education, or less), medium (intermediate vocational, or higher secondary education) and high (higher vocational education, or university). Smoking status was defined as being a persistent non-smoker (i.e. smoking less than one cigarette per month), persistent smoker, quitter or starter, based on a question about current cigarette smoking at baseline and at follow-up.

From 1994 onwards, identical questionnaires were used, allowing to study changes in dietary intake and physical activity in relation to changes in weight and waist circumference. From 632 men aged 50-65 years at the time of the examination between 1994 and 1997 (=baseline), who visited the health centre again in 1999-2002 (=follow-up), complete data on body weight and waist circumference was available. For the purpose of the present study we excluded men who suffered from cancer (n=37), cardiovascular diseases (n=58), diabetes (n=36) and/or men who had a waist circumference below 79 cm at baseline (n=5). From the remaining men (n=516) we selected 288 men, who reported to be employed at baseline, and employed or retired at follow-up. These 288 men were divided into two groups of different employment status: 176 who were still working and 112 who had retired during the follow-up period.

Measurement of body weight and waist circumference
Body weight, height and waist circumference were measured at the municipal health centre at baseline and at the end of follow-up, wearing light indoor clothing with emptied pockets and without shoes. Height was measured to the nearest 0.5 cm, weight to the nearest 0.5 kg. To adjust for light indoor clothing, one kg was subtracted from the measured weight. Body mass

index (BMI) was calculated as weight divided by height squared (kg/m^2). Waist circumference was measured to the nearest 0.5 cm, at the middle of the lowest rib and the iliac crest, with subjects in standing position, after breathing out gently.

Assessment of food intake

Food intake was assessed using the Dutch version of the EPIC (European Prospective Investigation into Cancer and Nutrition) Food Frequency questionnaire, a validated semi-quantitative food frequency questionnaire. In short, the questionnaire addressed habitual consumption of 178 food items during the previous year.[17,18] Energy intake was quantified by use of the 1996 computerised Dutch food consumption table.[19] For potatoes, pasta, rice, meat, and vegetables, portion sizes were assessed by use of photographs. Portion sizes were expressed in numbers of standard amounts: 70 grams per serving for potatoes, 45 grams for pasta, 55 for rice, 50 for meat and 60 for vegetables. Also, consumption frequency of these foods, completed with fish, meat substitutes, fruits, snacks, and drinks were assessed. Further, issues as eating dinner outside the home and eating breakfast were addressed.

Assessment of physical activity

Data on physical activity was collected by use of a validated questionnaire on physical activity,[20] extended with questions on sports and other strenuous leisure time activities. The questionnaire included questions on hours spent per week on leisure time activities (walking, bicycling, doing odd jobs / do it yourself, and gardening), household activities, sports and physical activities at the place of work, referring to the previous 12 months. The question on sports and other strenuous physical activities enabled the respondents to report on type, duration and number of weeks per year for three different activities. Average duration of leisure time and household activities per week were calculated as the average of the summer and the winter season data. For sports and other strenuous activities, duration per week was weighted by the number of weeks per year that physical activities were performed. The number of stairs walked on a usual day was recorded also. Except for stair walking, all activities were converted into Ainsworth-scores[21] expressed in metabolic equivalent values (MET-values). Leisure time physical activities included walking (3.5 MET), bicycling (5 MET), doing odd jobs (3 MET), and gardening (5 MET). Average intensity of leisure time activities including sports, was calculated by weighing intensities of the individual activities by their duration. For the purpose of the present study, occupational activity was dichotomised into sedentary ('sedentary occupation') or active ('standing occupation', 'manual work', or 'heavy manual work') jobs.

Statistical analyses

Demographic and anthropometric factors, and mean changes in weight, waist circumference, diet and physical activity between baseline and follow-up were compared between the groups of

men with sedentary jobs and active jobs who remained working and those who retired during follow-up, based on analyses of co-variance (SAS version 8.2, PROC GLM), adjusting for age.
Associations between changes in behaviour and changes in weight and waist circumference were studied by use of linear regression analyses (PROC REG), adjusting for retirement (yes/no), type of occupational activity (sedentary or active), interaction between retirement and type of occupational activity, age, smoking and the base level (the average of baseline and follow-up) of the behavioural component.
Associations between retirement and changes in weight and waist circumference were studied, adjusting for age, and the changes in behaviour that were associated with changes in weight or waist circumference at $p<0.10$, by analyses of co-variance (PROC GLM).
Since change in working hours was highly correlated with retirement ($r=0.73$), adjustments were not made for change in working hours.

RESULTS

Working men were on average younger in comparison to those who retired (Table 7.1). A total of 10.4% of the men were obese, and 33.7% had abdominal obesity at baseline.

Time spent on work decreased, by definition, significantly more among the men who retired compared to the men who remained working (Table 7.2). Men who retired increased the time spent on household activities and on doing odd jobs, they increased the portion size of rice and the frequency of vegetable consumption, and they decreased the frequency of potato consumption in comparison to the men who remained working (Table 7.2). Men who retired from sedentary jobs, also increased their alcohol consumption and decreased their energy intake from proteins, while men who retired from active jobs decreased their total energy intake and the fibre-density of the diet in comparison to their peers who remained working (Table 7.2).

Men who retired from sedentary jobs tended to gain less weight and to increase less in waist circumference in comparison to their peers remaining at work. Men who retired from active jobs tended to gain more weight and to increase more in waist circumference compared to the men who maintained working in active jobs (Table 7.2). Men who retired from active jobs gained significantly more weight and increased more in waist circumference than the men who retired from sedentary jobs (Table 7.2). Working men with an active job gained more weight and increased more in waist circumference in comparison with working men with a sedentary job, but these differences were not statistically significant.

TABLE 7.1 Demographic and anthropometric characteristics* of the study population, at baseline and during follow-up, stratified for (change) in job status.

	Working Sedentary (n=90)	Retired Sedentary (n=66)	Working Active (n=86)	Retired Active (n=46)
Baseline				
Age (years), mean ± SD	53.3 ± 2.6 [a]	57.5 ± 2.7 [a]	53.1 ± 2.2 [b]	57.4 ± 2.3 [b]
Weight (kg)	84.3	82.1	83.7	82.8
Height (cm)	178.5	177.9	177.5	176.8
BMI (kg/m^2)	26.4	26.0	26.6	26.5
Waist circumference (cm)	98.5	96.5	98.3	97.5
Normal weight (BMI 18.5-24.9), %	33.0 [a]	42.2	21.6 [a]	28.1
Moderate overweight (BMI 25-29.9), %	54.9 [a]	48.9	69.2 [a]	60.5
Obese (BMI ≥30), %	12.1	8.9	9.3	11.4
WC 79 – 93.9 cm, %	28.3	41.5	29.3	30.4
WC 94 – 101.9 cm, %	37.4	30.5	33.4	35.7
WC ≥102 cm, %	34.3	28.0	37.3	33.9
Low education level, %	24.9 [ab]	41.2 [a]	54.9 [b]	54.8
Medium education level, %	31.1 [a]	13.2 [ab]	26.9	28.6 [b]
High education level, %	44.0	45.6[a]	18.3	16.7[a]
During follow-up				
Duration of follow-up (years), mean ±	5.0 ± 0.2	5.0 ± 0.2	5.0 ± 0.1	5.0 ± 0.2
Persistent non-smoker, %	74.7	81.4	70.0	82.2
Persistent smoker, %	20.0 [a]	7.3 [a]	23.3	11.3
Quitter, %	2.4	9.2	5.9	5.9
Starter, %	3.0	2.1	0.8	0.6

SD, standard deviation; BMI, body mass index; WC, waist circumference.
* Except for age and duration of follow-up period, all variables are adjusted for age.
Means or percentages with the same superscript letter within the same row are statistically different from each other at $p<0.10$. (Only relevant differences were tested: within workers, within retired men, within active jobs, and within sedentary jobs.)

Weight gain was statistically significantly associated with a decrease in fruit consumption, an increase in frequency of using breakfast, an increase in consumption of sugar sweetened soft drinks, a decrease in fibre-density of the diet, and with a decrease in time spent on bicycling (Table 7.3). Independent of other changes in behaviour, a decrease in fruit consumption remained associated with weight gain. The total multivariate model explained 22% of variance in changes in body weight in the study population.

TABLE 7.2 Changes in anthropometric characteristics, physical activity and diet* over the five year follow-up period in working and retired men with sedentary and active jobs.

	Sedentary Job				Active Job			
	Working (n=90)		Retired (n=66)		Working (n=86)		Retired (n=46)	
	Mean change	[95%-CL]	Mean change	[95%-CL]	Mean change	[95%-CL]	Mean change	[95%-CL]
Anthropometric characteristics								
Body weight (kg/year)	0.24	[0.06; 0.41]	0.08	[-0.13; 0.30] [a]	0.37	[0.19; 0.55]	0.42	[0.17; 0.67] [a]
Waist circumference (cm/year)	0.31	[0.09; 0.53]	0.23	[-0.04; 0.50] [a]	0.54	[0.31; 0.77]	0.77	[0.46; 1.08] [a]
Physical activity								
Mean duration (hours/week)								
Work	-5.87	[-8.98; -2.76] [a]	-32.66	[-36.51; -28.82] [a]	-4.24	[-7.45; -1.02] [b]	-31.10	[-35.54; -26.65] [b]
Household	0.67	[-0.20; 1.54] [a]	2.16	[1.09; 3.24] [a]	0.23	[-0.67; 1.13]	1.50	[0.26; 2.74]
Sports	0.55	[-0.22; 1.32]	0.68	[-0.28; 1.63]	-0.02	[-0.82; 0.78]	0.15	[-0.96; 1.25]
Bicycling	0.37	[-0.58; 1.32]	0.86	[-0.31; 2.04]	-0.14	[-1.12; 0.85]	1.24	[-0.12; 2.60]
Walking	1.08	[-1.33; 3.49]	-0.62	[-3.60; 2.36]	-4.18	[-6.67; -1.68]	-5.14	[-8.58; -1.70]
Gardening	0.28	[-0.44; 1.01]	0.32	[-0.58; 1.21]	-0.19	[-0.94; 0.56]	0.47	[-0.56; 1.51]
Doing odd jobs	0.07	[-0.92; 1.07]	1.45	[0.21; 2.68]	-0.90	[-1.94; 0.13] [1]	3.01	[1.58; 4.43] [1]
Climbing stairs (number per day)	-0.47	[-2.54; 1.60]	-3.09	[-5.63; -0.55]	-0.17	[-2.31; 1.97]	-2.13	[-5.10; 0.85]
Intensity (MET-values)								
Leisure time physical activity	0.04	[-0.07; 0.15]	0.00	[-0.13; 0.13]	0.08	[-0.03; 0.19]	0.09	[-0.07; 0.24]
Diet								
Portion size warm meal (# of servings)								
Potatoes	-0.12	[-0.28; 0.04]	-0.04	[-0.15; 0.24]	-0.13	[-0.29; 0.03]	-0.24	[-0.46; -0.02]
Pasta	-0.54	[-1.02; -0.06]	-0.37	[-0.97; 0.22]	-0.20	[-0.70; 0.29]	-0.75	[-1.44; -0.07]
Rice	-0.39	[-0.67; -0.11] [a]	0.12	[-0.22; 0.47] [a]	-0.51	[-0.81; -0.22] [b]	0.04	[-0.39; 0.48] [b]
Meat	-0.02	[-0.14; 0.09]	-0.01	[-0.15; 0.13]	0.01	[-0.11; 0.12]	-0.14	[-0.30; 0.02]
Vegetables	-0.05	[-0.14; 0.05]	0.04	[-0.08; 0.16]	0.05	[-0.05; 0.15]	-0.01	[-0.15; 0.13]
Frequencies (times/week)								
Potatoes	-0.08	[-0.76; 0.60] [a]	-1.13	[-1.98; -0.29] [a]	-0.03	[-0.74; 0.68] [b]	-1.41	[-2.39; -0.43] [b]
Pasta	-0.02	[-0.13; 0.08]	0.05	[-0.08; 0.18]	0.01	[-0.10; 0.12]	0.02	[-0.13; 0.17]
Rice	-0.09	[-0.21; 0.03]	0.06	[-0.09; 0.21]	-0.07	[-0.19; 0.06]	-0.07	[-0.24; 0.11]

TABLE 7.2 (continued)

	Sedentary Job				Active Job			
	Working (n=90)		Retired (n=66)		Working (n=86)		Retired (n=46)	
	Mean change	[95%-CL]	Mean change	[95%-CL]	Mean change	[95%-CL]	Mean change	[95%-CL]
Frequencies (times/week)								
Meat	-0.20	[-0.53; 0.13]	-0.46	[-0.87; -0.05]	-0.16	[-0.50; 0.18]	-0.25	[-0.72; 0.23]
Fish	0.34	[0.19; 0.50]	0.18	[-0.01; 0.37]	0.19	[0.03; 0.35]	0.23	[0.01; 0.45]
Meat substitutes	0.02	[-0.12; 0.16]	0.06	[-0.11; 0.23]	-0.01	[-0.16; 0.14]	-0.01	[-0.21; 0.19]
Vegetables	-0.73	[-1.41; -0.05] [a]	0.58	[-0.26; 1.41] [a]	-0.13	[-0.83; 0.57]	0.01	[-0.95; 0.98]
Fruit	0.32	[-1.20; 1.84]	0.09	[-1.81; 1.99]	0.23	[-1.35; 1.80]	-2.16	[-4.33; 0.02]
Breakfast	-0.16	[-0.47; 0.15]	0.02	[-0.36; 0.41]	0.25	[-0.07; 0.57]	0.44	[-0.00; 0.89]
Eating out for dinner	0.14	[0.02; 0.27]	0.10	[-0.06; 0.25]	0.05	[-0.08; 0.18]	-0.02	[-0.20; 0.16]
Snacks (100 grams/day)								
Sweet snacks	-0.04	[-0.10; 0.02]	0.03	[-0.04; 0.10]	0.01	[-0.05; 0.07]	-0.02	[-0.10; 0.07]
Savoury snacks	-0.08	[-0.14; -0.02]	-0.02	[-0.10; 0.05]	-0.04	[-0.11; 0.02]	-0.08	[-0.17; 0.01]
Drinks (glasses/day)								
Dairy drinks	-0.11	[-0.40; 0.18]	-0.40	[-0.76; -0.04]	0.06	[-0.24; 0.36]	-0.01	[-0.43; 0.40]
Sugared soft drinks	-0.08	[-0.24; 0.08]	0.02	[-0.18; 0.22]	0.07	[-0.09; 0.23]	0.09	[-0.14; 0.32]
Juice	0.02	[-0.10; 0.14]	0.09	[-0.05; 0.23]	0.07	[-0.05; 0.19]	0.23	[0.06; 0.39]
Alcoholic beverages	-0.11	[-0.36; 0.14] [a]	0.27	[-0.04; 0.57] [a]	-0.15	[-0.40; 0.11]	-0.04	[-0.39; 0.32]
Energy intake (MJ/day)	-0.80	[-1.22; -0.37]	-0.50	[-1.02; 0.02]	-0.35	[-0.78; 0.09] [a]	-1.13	[-1.73; -0.52] [a]
En % Protein	0.19	[-0.21; 0.59] [a]	-0.60	[-1.09; -0.10] [a]	0.46	[0.05; 0.87]	0.29	[-0.28; 0.85]
En % Carbohydrate	-0.31	[-1.26; 0.63]	0.69	[-0.49; 1.86]	0.11	[-0.87; 1.09]	-0.85	[-2.21; 0.50]
En % Fat	0.10	[-0.83; 1.02]	-1.09	[-2.24; 0.05]	-0.32	[-1.28; 0.64]	0.19	[-1.13; 1.51]
En % Alcohol	0.03	[-0.66; 0.71]	1.00	[0.15; 1.85]	-0.25	[-0.96; 0.46]	0.37	[-0.60; 1.35]
Fibre density (grams/MJ)	0.05	[-0.06; 0.16]	0.02	[-0.12; 0.15]	0.06	[-0.05; 0.18] [a]	-0.18	[-0.34; -0.02] [a]

CI, confidence interval; WC, waist circumference; MET, metabolic equivalent.

* Adjusted for age.

Means with the same superscript letter within the same row are statistically different from each other at P<0.10. (For changes in behaviour, only relevant differences are shown: between men with (former) sedentary jobs, and between men with (former) active jobs.)

TABLE 7.3 Changes in behaviour over follow-up in relation to change in body weight.

	Total group of men (n=288)			
	Univariate models		Multivariate model	
	β*	p	β**	p
Control variables				
Age (years)			-0.00	0.82
Retirement (versus working)			0.08	0.64
Type of job (sedentary versus			-0.02	0.88
Interaction retirement*sedentary			-0.25	0.17
Smoker (versus non-smoker)			0.11	0.39
Ex-smoker (versus non-smoker)			1.00	<0.01
Starter (versus non-smoker)			-0.19	0.58
Physical activity				
Mean duration (hours/week)				
Bicycling	-0.03	<0.01	-0.02	0.13
Doing odd jobs	-0.02	0.06	-0.01	0.21
Diet				
Frequencies (times/week)				
Potatoes	0.04	0.05	0.02	0.17
Fruit	-0.02	<0.01	-0.02	0.03
Breakfast	0.07	0.03	0.04	0.21
Sugared soft drinks (glasses/day)	0.20	<0.01	0.12	0.05
Fibre density (grams/MJ)	-0.31	<0.01	-0.17	0.10

* β refers to the change in body weight (kg/year) per unit change in the behaviour, adjusted for retirement (yes/no), type of job (sedentary or active), interaction between retirement and type of job, age, smoking and the base level of the behaviour. ** β refers to the change in body weight (kg/year) per unit change in the behaviour, adjusted for retirement, type of job, interaction between retirement and type of job, age, smoking, the base level of the behaviour, and all other behaviours in the table. (Only associations with p-value below 0.10 in the univariate models are shown.)

Increase in waist circumference was associated with an increase in frequency of pasta consumption, a decrease in fruit consumption, an increase in frequency of using breakfast, an increase in the consumption of sugar sweetened soft drinks, a decrease in fibre-density of the diet and with a decrease in time spent on walking or on doing odd jobs (Table 7.4). Independent of other changes in behaviour, an increase in frequency of pasta consumption, a decrease in fruit consumption and an increase in frequency of eating breakfast remained associated with increase in waist circumference. The total multivariate model explained 27% of variance in changes in waist circumference among the study population.

TABLE 7.4 Changes in behaviour over follow-up in relation to change in waist circumference.

	Total group of men (n=288)			
	Univariate models		Multivariate models	
	β *	p	β**	p
Control variables				
Age (years)			-0.03	0.13
Retirement (versus working)			0.31	0.13
Type of job (sedentary versus active)			-0.02	0.91
Interaction retirement*sedentary job			-0.47	0.04
Smoker (versus non-smoker)			0.16	0.30
Ex-smoker (versus non-smoker)			1.05	<0.01
Starter (versus non-smoker)			0.38	0.37
Physical activity				
Mean duration (hours/week)				
Household	-0.02	0.09	-0.03	0.07
Bicycling	-0.02	0.07	-0.01	0.59
Walking	-0.01	0.02	-0.01	0.12
Doing odd jobs	-0.03	0.04	-0.02	0.08
Diet				
Frequencies (times/week)				
Pasta	0.29	0.02	0.26	0.03
Fruit	-0.03	<0.01	-0.03	<0.01
Breakfast	0.13	<0.01	0.10	0.01
Sugared soft drinks (glasses/day)	0.16	0.04	0.06	0.47
Alcoholic beverages (glasses/day)	0.09	0.09	0.09	0.09
Energy intake (MJ/day)	0.05	0.08	x	x
En % Carbohydrate	-0.02	0.07	x	x
Fibre density (grams/MJ)	-0.32	<0.01	-0.08	0.55

* β refers to the change in waist circumference (cm/year) per unit change in the behaviour, adjusted for retirement (yes/no), type of job (sedentary or active), interaction between retirement and type of job, age, smoking and the base level of the behaviour.
** β refers to the change in waist circumference (cm/year) per unit change in the behaviour, adjusted for retirement, type of job, interaction between retirement and type of job, age, smoking, the base level of the behaviour, and all other behaviours in the table.
(Only associations with p-value below 0.10 in the univariate models are shown)

When the associations between retirement and changes in weight and waist circumference were adjusted for changes in behaviours that were associated (at p<0.10) with changes in weight or waist circumference, the average gains in body weight and waist circumference for the groups of men who remained working in sedentary or active jobs became equal. The differences in weight gain and in increase in waist circumference between the men who retired from sedentary versus active jobs were maintained (Figures 7.1 and 7.2).

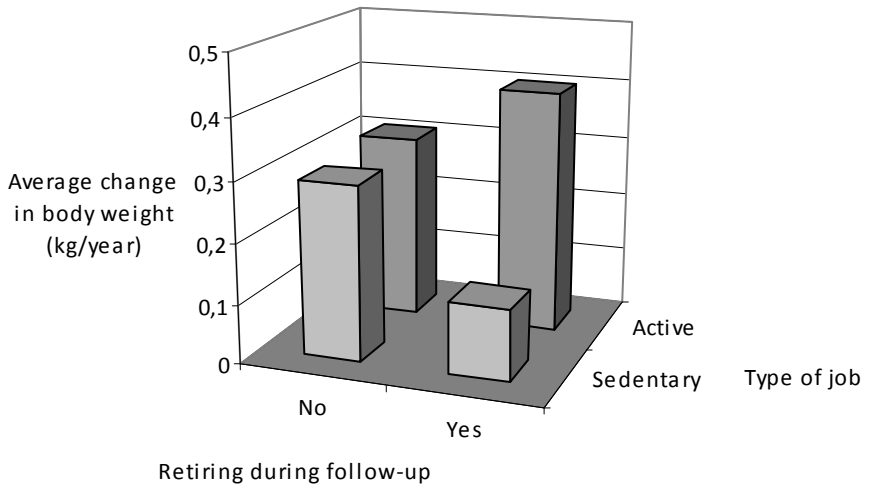

FIGURE 7.1 Changes in body weight among working and retiring men with sedentary or active jobs, adjusted for age, smoking, and changes in behaviour as stated in table 7.3.

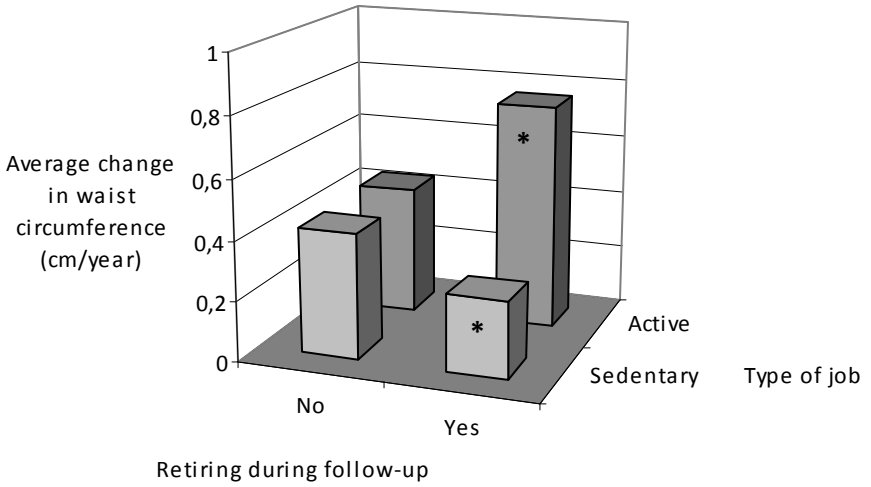

FIGURE 7.2 Changes in waist circumference among working and retiring men with sedentary or active jobs, adjusted for age, smoking and changes in behaviour as stated in table 7.4. Bars with * are statistically different from each other at $p<0.05$.

DISCUSSION

To our knowledge, this is the first study that has analysed the association between retirement and changes in dietary behaviour and physical activities in relation to changes in body weight and waist circumference. Retirement was associated with an increase in weight and waist circumference among those with former active jobs, but not among those with former sedentary jobs. Weight gain and increase in waist circumference were associated with a decrease in fruit consumption and fibre-density of the diet, with an increase in eating breakfast and the consumption of sugar sweetened soft drinks, and with a decrease in several leisure time physical activities.

There are several methodological issues that warrant discussion. First, men who retired were on average four years older than those who did not. Both changes in behaviour and changes in weight may be related to age (e.g. older men gaining less weight than younger men). In the Netherlands, voluntary or mandatory early retirement is relatively common and, in principal, unrelated to health status. People who have to stop working before age 65 due to health status, receive disablement insurance benefits and were not included in the present study. Most people retire between the ages 55 and 65 years with a pension or some other kind of early retirement payment. Most of them retire around 60 years of age.[22] Therefore, there was sufficient overlap in the age-ranges between the groups of men who remained working and who retired during follow up, and we were able to study effects of retirement on body weight and waist circumference, independent of age. Second, the use of questionnaires to assess food-intake and physical activity is susceptible to misreporting.[23-25] However, by studying changes in behaviour, it could be argued that a possible distorting effect of misreporting on studied associations was eliminated when the extent of misreporting was similar within participants in both examinations. Third, one may argue that educational level should have been included in the multivariate analyses, since body weight and waist circumference are associated with level of education. However, these associations are mediated by lifestyle. Therefore we chose to present results of the multivariate analyses on changes in lifestyle in relation to changes in body weight or waist circumference, unadjusted for educational level. Conclusions did not change when educational level was included in the analyses. Another point is that we do not know the exact date of retirement, only that men did, or did not retire during follow-up. Under the assumption that changes in body weight and waist circumference do not differ between the group of men remaining at work and the last years of employment for the retiring men, the effects of retirement on change in body weight and waist circumference may have been underestimated in this study. Finally, the results in the present study are based on a subpopulation of all men in the study. However, we included all healthy men in the cohort who fulfilled the inclusion criteria to be 50-65 years of age and working at baseline and working or retired at follow-up. We have no

reason to believe that the associations between retirement and weight gain are different in the total Dutch population. Since findings in the present study are to a large extent based on the reduction of work related physical activity coupled with occupational retirement, we assume that the findings of the present study are applicable to all men who are about to retire, also in other countries.

Evenson (2002) reported that retirement was associated with increases in sport and exercise participation.[9] In a prospective study among men aged 40-75 years, a decreased fibre-consumption and a decrease in total physical activity were associated with an increase in waist circumference over a 9-year follow-up.[14] Another study-group reported heavy alcohol intake to contribute directly to weight gain in middle-aged men.[26] In a study among middle-aged and old men, increased physical activity was associated with weight loss.[27] These results are largely in line with the findings in the present study. Two studies evaluated the relation between retirement and weight gain. Morris et al reported that retiring men were more likely to gain over 10% body weight in comparison with men who remained employed.[28] Patrick et al reported an increase in fat mass in the first year of retirement, but a decline in body mass and its components between the first and fifth year after retirement.[13] Unfortunately we were not able to make such a distinction.

To prevent an increase in body weight and waist circumference after retirement, energy intake should be changed to a level that matches the new energy requirements or physical activities should replace the former work-related physical activities. In our study population, men who retired from active jobs lowered their total energy intake and they increased doing odd jobs over time, but not enough to prevent weight gain and an increase in waist circumference. However, without these changes in behaviour, even larger increases in weight and waist circumference would have been expected.

Most changes in diet and physical activity were related to both changes in body weight and changes in waist circumference in a similar way. However, some components (e.g. frequency of pasta consumption, drinking alcoholic beverages, walking) were clearly related to change in waist circumference, and not to change in body weight. Apparently, changes in these behaviours have more effect on fat distribution than they have on total fat storage. Since waist circumference has shown to be a better predictor of all cause mortality among the elderly,[29] it seems better to focus on determinants of increases in waist circumference than on determinants of weight gain only, in order to prevent negative effects of overweight and obesity among middle-aged men.

The differences in weight gain and increase in waist circumference between groups of working men in sedentary and active jobs could be explained by differences in changes in lifestyle over follow-up. The differences in weight gain and increase in waist circumference between the groups of men that retired from active versus sedentary jobs that remained after adjusting for changes in behaviours, can be attributed to the cessation of work related physical activity. Apparently, men with former active jobs, become overall less active after retirement than they were before retirement, while men with former sedentary jobs, become overall more active after retirement in comparison with their working years. This is not surprising, since men with former sedentary jobs can almost not become less active after retirement in comparison with their activities during former working hours. For men with former active jobs, however, greater adaptations are required to create a new energy balance.

In conclusion, men who retired from physically active jobs were more prone to gain weight and increase in waist circumference compared to men who retired from sedentary jobs. Since retirement may bring opportunities for healthy changes in diet and physical activity, it seems warranted to develop health promotion programs aimed at prevention of overweight in men who are eligible for retirement, particularly in those who have physically demanding jobs.

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General discussion

The aim of the present thesis was to identify important age specific lifestyle determinants of weight gain. In spite of several methodological limitations, we have been able to identify factors associated with weight gain based on longitudinal data. Observational data do not seem appropriate to assess lifestyle determinants of long term weight gain, due to several methodological issues. However, they seem appropriate to identify high-risk groups for weight gain. Based on our results we can underscore that weight gain prevention should follow a population-wide, life course approach, since weight gain is of all ages, both genders, and all educational levels. In addition, we identified several groups at elevated risk of weight gain within the specific age groups, at which interventions can be targeted.

Main findings

High-risk groups

Among adolescents, we observed that those with a relatively large sum of skinfolds had an increased risk of becoming adults with a high body fatness, even more than adolescents with a relatively high BMI [**chapter 2**]. Therefore, weight gain prevention programmes targeting at adolescents should focus on those with a large sum of skinfolds, rather than those with a high BMI. From adolescence into young adulthood, we observed that men who started to work at a young age, while still living with their parents, showed the largest increase in body weight [**chapter 5**], compared to young men who lived on their own or did not work. Among adults, weight gain was observed at all ages between 20 and 70 years [**chapter 3**]. Highest average weight gain was observed in young adults, but weight gain was still observed after the age of 60 years. In general, persons with a lower educational level had a higher body weight than persons with a higher educational level, but age related weight gain was similar among all educational levels. Based on these results, prevention of weight gain in adults should follow a population-wide, life-course approach [**chapter 3**]. Adults with an increased risk of weight gain are men and women who raise children. In addition, women living alone without children gained more weight than cohabiting women without children [**chapter 5**]. Therefore, especially women who live alone, and young men and women planning a family are important groups for targeted weight gain prevention programs. Finally, among recently retired men, we observed that retirement was associated with an increase in body weight and waist circumference among those with former active jobs, but not among those with former sedentary jobs [**chapter 7**]. Therefore, weight gain prevention programs should target men who have physically demanding jobs and who are eligible for retirement.

Lifestyle behaviour and weight gain

Among young adults (aged 20-40 years), we observed that associations between diet and physical activity with weight gain were different between men and women [**chapter 6**]. Weight gain in young adult men was associated with a decrease in hours of biking, a decrease in frequency of eating meat substitutes and having breakfast, and with an increase in juice consumption, portion size of meat, and frequency of eating fish. Among young adult women, weight gain was associated with an increase in sweet snacks consumption. Among middle-aged men (aged 50-70 years), weight gain and increase in waist circumference were associated with a decrease in fruit consumption and fibre density of the diet, with an increase in having breakfast and in the consumption of sugar-sweetened soft drinks, and a decrease in leisure-time physical activities [**chapter 7**].

Methodology

Both baseline level and changes in diet and physical activity were studied in relation to weight gain in young adults [**chapter 6**]. We observed that the associations between changes in diet and physical activity with weight gain were more plausible compared to the associations between baseline diet and baseline physical activity with weight gain. We concluded that, when studying lifestyle determinants of weight gain, changes in energy balance related lifestyles may be more informative than a single measure of diet and physical activity. In young adulthood, when many changes in social life occur, it seems even more pertinent to look at changes in lifestyles when studying determinants of weight gain. The need for longitudinal data became also apparent in **chapter 3**, where we observed cohort effects on weight status. We showed that younger generations have higher body weight than older generations at the same calendar age. Therefore, comparisons of BMI in different age-categories based on cross-sectional surveys underestimate the actual increase in BMI with aging [**chapter 3**].

Another aspect of methodology was studied in chapter 4, where we studied the baseline level, slope and fluctuation of total energy intake and physical activity from age 13 to age 36 years, in relation to body composition at age 36 years. We showed that, unexpectedly, a higher baseline energy intake was associated with a lower percentage of adult body fatness in both men and women, and a higher baseline physical activity was associated with higher body fat mass index at adult age. Associations between the development of physical activity and fat-free body mass in women were more plausible. Based on these findings, we concluded that observational data are limited in determining which lifestyles are related to weight gain, probably due to measurement error, reporting bias and other methodological factors [**chapter 4**].

Methodological considerations in weight gain epidemiology

In order to identify important age specific determinants of weight gain, we focussed on lifestyle changes related to life transitions (e.g. leaving parental home, starting work, raising children, retirement), living situations (composition of the household), and demographic characteristics (age, level of education). The challenge of this thesis was to evaluate which main lifestyles are associated with weight gain in the three selected age groups, based on data from prospective observational studies. The assumption was that people in these particular age groups share common changes in lifestyle. By studying determinants of weight gain as described above, it was assumed possible to identify some high-risk (changes in) lifestyles. However, this proved to be more complex than anticipated. In the present section of this thesis, the most important methodological strengths and drawbacks encountered in the epidemiological studies presented in this thesis are described and discussed.

Internal validity

Selection bias

Subjects in cohort studies participate on a voluntary basis. In the Doetinchem Cohort Study, only 62% of recruited subjects from a general population participated in the first measurement. Unfortunately, no information is available from the non-participants. In the Amsterdam Growth and Health Longitudinal Study (AGAHLS), all initially invited adolescents participated in the study.[1]

Another issue, evident for all longitudinal studies, is that it is difficult to keep subjects in the study over a long period of time. Therefore, drop-out during follow-up is a common phenomenon in cohort studies. As long as the loss to follow-up is non-selective, this is not a serious problem, except for the loss of statistical power. However, the loss to follow-up can be selective. In the AGAHLS, subjects who dropped out had, on average, a higher BMI and sum of skinfolds at adolescent age than subjects who participated at adult age [**chapter 2**]. In the Doetinchem Cohort Study, especially the younger and the heavier adults were lost to follow-up.[2] Drop-outs in the third measurement had a lower education, consumed less alcohol, spent less time on sports activities, and were more often smokers compared to participants.[3]

Selective participation and drop-out in both cohorts has probably resulted in a lower (increase in the) prevalence of overweight and obesity than in the source cohorts. However, associations between changes in lifestyles and changes in body weight are not necessarily affected by this phenomenon.[4,5]

Reporting bias /Information bias
In the cohort studies described in this thesis, energy intake (diet) and physical activity were assessed using questionnaires or interviews.

It is often difficult for participants to report the exact diet and physical activities over the last few months. Therefore we assume that measurement error has occurred in our studies. This measurement error may have been larger than the (small) changes in lifestyle behaviour resulting in weight change as observed in the two cohorts.[6] As a result, associations between reported lifestyle behaviours and weight gain may have been underestimated, or associations observed may represent lifestyles with lowest measurement error.

Secondly, differential misclassification can be introduced by the phenomenon that overweight persons tend to under-report their energy intake more than persons with a normal weight,[7-10] while over-reporting their physical activity.[11,12] To take this phenomenon into account in our studies, we adjusted for previous weight change or initial weight status when analysing associations between changes in lifestyle behaviour and weight gain, but the effect of this adjustment was rather small. This indicates that the extent of reporting bias may have been similar in the successive measurements, as was mentioned in **chapter 6**.

Thirdly, the questionnaires on dietary intake and physical activity have been validated to *rank* (relative validity) subjects based on their energy intake and physical activity, not to determine absolute levels.[1,6,13,14] Therefore, only directions of the associations between (changes in) lifestyles and weight gain were studied.
However, energy balance is not about ranking energy intake and energy expenditure, but about a delicate balance between absolute levels of energy intake and energy expenditure. For example: a discrepancy of 1% between energy intake and energy expenditure would add up to about 730 MJ over a period of 20 years, equivalent to about 24 kg of fat mass.[15] Therefore, judgement about how much physical activity is needed, or how much decline in energy intake is needed to prevent weight gain based on data from studies using self reported behaviours should always be interpreted with caution.

Even if the total dose of physical activity is assessed correctly, it is impossible to assess total energy expenditure accurately by questionnaires or interviews. This is because total energy expenditure is not a linear function of physical activity level (see Figure 8.1): The largest part (up to 75-80%) of total energy expenditure is used to maintain the body and to keep it on temperature (the basic metabolic rate). About 10 percent of total energy expenditure is determined by food-induced thermogenesis. The remainder of total energy expenditure (less than 10% in sedentary subjects[16] up to 50% in very active subjects[17]) is determined by physical

activity. In addition, basic metabolic rates differ between individuals even when corrected for height and weight. Therefore it is difficult to determine the personal energy needs for energy balance based on questionnaires only: body composition, the kind of food, the amount of total physical activity and even the environmental temperature are determinants.

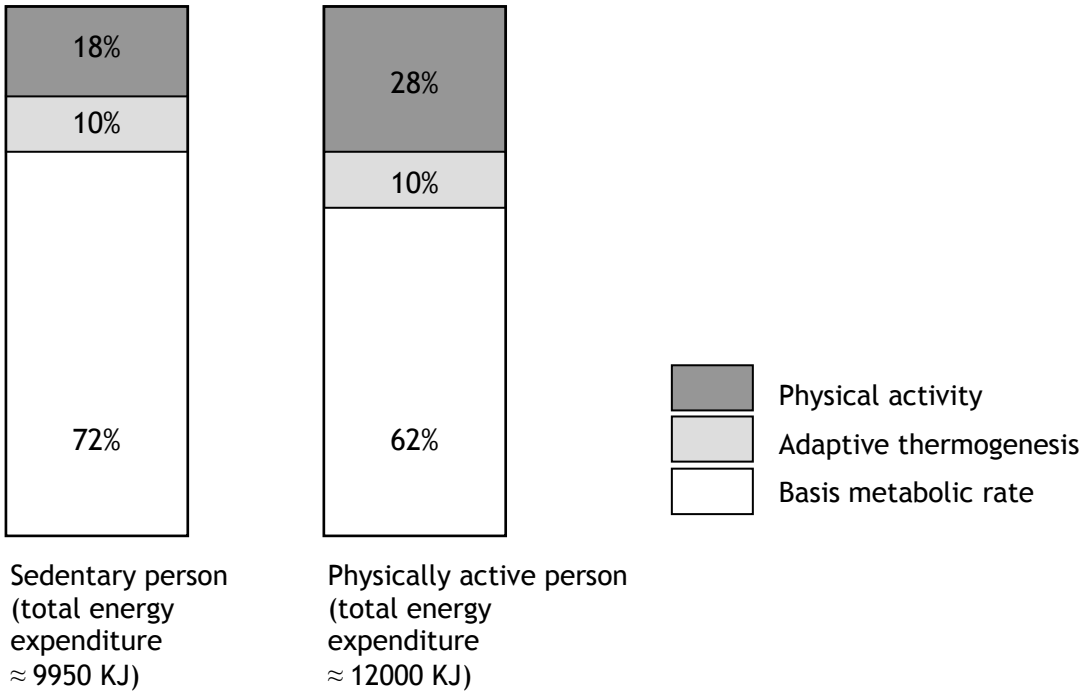

FIGURE 8.1 Example of the difference in composition of the total energy expenditure between a sedentary and a physically active person. *Adapted from Rising et al, 1994.*[18]

Finally, the questions about *habitual* diet and physical activities referred only to the past few months (AGHALS) or year (DCS) before the weight measurements. Consequently, we do not have information on lifestyle between two measurements (see Figure 8.2), and participants may not have reported occasional changes, while these small changes are important in relation to (unintentional) changes in body weight. As stated before, weight gain is a dynamic process: the difference in body weight between measurements is the accumulation of all, also occasional, daily results in energy balance.

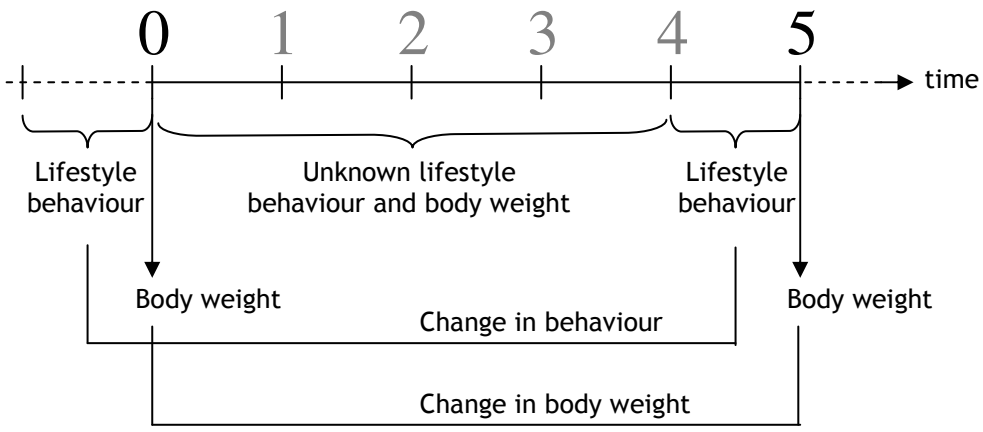

FIGURE 8.2 Schematic representation of collected data in the Doetinchem Cohort Study.

Taking all preceding issues regarding questionnaires and interviews into account, it turns out to be very difficult to detect the small differences in energy balance related behaviours (diet and physical activity) using questionnaires or interviews.[6] A more precise assessment of energy balance related behaviours may be possible in experimental settings, using more direct measures to determine energy intake and energy expenditure (e.g. motion sensors, provide and keep count of all foods using an experimental supermarket), resulting in less information bias. However, apart from high costs, in such experimental settings a long follow-up is not possible and people may behave differently from their free-living routines.

Cross-sectional versus longitudinal studies

Because of its dynamic process, multiple measurements are required to assess weight gain. In the past, correlates of body weight have been identified in cross-sectional studies: differences in body weight between persons were associated with differences in lifestyle behaviour between persons. Correlates of high body weight were often interpreted as determinants of weight gain. However, weight gain reflects the balance between energy intake and energy expenditure. A person who eats a lot, but also has a high physical activity level, may be in energy balance, as well as a person who eats very little in combination with a sedentary lifestyle, regardless of their body weight. So, both fat and thin people may be in energy balance. As a result, correlates of body weight in cross-sectional studies may not be valid determinants of body weight gain.
But also when weight gain, instead of body weight, is evaluated in relation to lifestyle behaviour assessed at baseline only[19-21] similar difficulties appear with the interpretation of the results.

Causality

Also longitudinal studies may suffer from several methodological problems that should be considered before interpreting the results, especially regarding a cause-effect relationship.

First of all, the energy requirement to maintain the body is higher with higher body weight, growth (in children and adolescents) and smoking. The energy requirement decreases with aging. Other features that should be regarded are deliveries and menopause among women, and illnesses that affect body weight, e.g. by altering resting energy expenditure. Another issue that should be considered is whether participants were in energy balance during the measurements. When, for instance, participants were not in energy balance at baseline, changes in body weight assessed over follow-up may be the result of changes in lifestyle before baseline, rather than changes in lifestyle between baseline and follow-up. Next, people can change their lifestyle behaviour in response to weight changes (reverse causation). Finally, as mentioned before, overweight subjects tend to underreport their dietary intake[7-10] and to over-report their physical activity.[11,12] Probably all these, and other, individual scenarios are present in epidemiological observational studies. Therefore, results from longitudinal analyses are still difficult to interpret with respect to temporal relationships. Changes in lifestyle that are associations with weight gain should therefore be interpreted as indicators rather than as determinants.

External validity

The AGAHLS included only adolescents from two higher educational level secondary schools. Therefore the initially recruited population was already more highly educated compared to the general Dutch adolescent population. The initial population for Doetinchem Cohort Study made up a representative sample of the general population in Doetinchem in the same age range (20-59 years at baseline).[22] However, Doetinchem may not be a representative town for the Netherlands.[23]

As stated before, selection has occurred in both cohort studies used in this thesis. However, selection does not necessarily produce different associations between lifestyle behaviours and weight gain[4,5] and we therefore believe that age-specific associations between lifestyle behaviour and weight gain as observed in our studies will also hold in a general population of the same age.

Nevertheless, extrapolation of results in this thesis to the present general Dutch population warrants caution. One of the most positive aspects of the cohort studies (i.e. the long follow-up time) is at the same time a drawback: results based on longitudinal data from the cohorts can

already be outdated. The social environment changes very fast and the behaviour or living situations that were associated with weight gain 10 years ago may already have changed, as is indicated by higher body weight for younger cohorts in **chapter 3**. For instance, in **chapter 5** we found that especially boys who lived with their parents beyond age 21, while already working, gained most body weight from age 16 to 27 years. These results are based on data collected about 20 years ago, and it is therefore not certain whether these living situations are similar nowadays. Similar time-effects may also hold for results of our study on the effect of weight gain associated with retirement, that are based on data collected only 5 to 10 years ago: behaviours related to retiring may have changed over the last decade.

Public health implications / Recommendations for practice

Guidelines for weight gain prevention

Prevention of weight gain is needed, since weight gain and overweight or obesity increase the risk of several chronic diseases.[24] It is important to note that weight gain prevention should fit in programmes and guidelines for a healthy diet and sufficient daily physical activity. The overall goal in public health is the promotion of the number of healthy life years and a reduction of unhealthy life years. For instance, starting to smoke is associated with lower weight gain, but should never be recommended, since smoking has serious adverse effects on health.
In addition, besides their combined effect on body weight, a healthy diet and physical activity are directly associated with a lower risk of cardiovascular disease, diabetes and cancer. An unbalanced diet (e.g., no fat) can be harmful for health in the long term and should therefore not be part of an intervention to prevent weight gain. And although replacement of saturated fatty acids by unsaturated fatty acids has no effect on energy balance (since energy content of saturated and unsaturated fatty acids is the same), this replacement fits well in a health promotion programme, since it contributes to the reduction of cardiovascular risk profile. And, as high intensity physical activity improves cardiovascular risk profile, it should be promoted, although performing high intensity physical activities is less likely to contribute to weight gain prevention than performing lower intensity physical activities.[25] In the Netherlands, several guidelines have been published for a healthy diet and physical activity pattern and more specific to prevent weight gain, e.g. the “Guidelines for a healthy diet”,[26] and the “Nederlandse Norm Gezond Bewegen”.[27] In addition to these general health recommendations, results of the present thesis could be used to design more targeted interventions towards high risk groups for weight gain.

General weight gain prevention programmes should be incorporated in daily life, since weight gain affects the entire population, regardless of age or level of education [**chapter 3**]. Three

specific age groups at elevated risk for weight gain were proposed for this thesis: adolescents, young adults and recently retired subjects.

Adolescents

In order to identify adolescents at high risk for obesity, the Youth Health Services could include anthropmetric measures in their screening for obesity. Special attention could be paid to inclusion of skinfold measurement [**chapter 2**] and possibly waist circumference measurement. In addition to more general attention for healthy lifestyles at school (e.g. more physical education lessons and lower availability of energy dense, nutrient poor foods and drinks[28]), targeted weight gain prevention programmes should focus on adolescents with a large sum of skinfolds.

Young adults

Based on results as observed in **chapter 6**, young men should have breakfast every day, eat smaller portion sizes of meat and use active transport more often. Young women should consume less sweet snacks. Incentives to change behaviours of young adults into more healthy behaviours may be offered by their employers, by offering healthier choices in the canteen or facilitate active transport.[29] A specific group of young adults at elevated risk of weight gain are men and women planning a family [**chapter 5**]. During the visit at the family doctor or midwife, new parents to be may be informed about this elevated risk and about a healthy lifestyle for both mother and father.

Recently retired subjects

To prevent weight gain, also middle-aged men should live according to the general guidelines stated above. Special attention could be given to fruit consumption, fibre density of the diet, and leisure time physical activities. Especially men retiring from physically active jobs are at an elevated risk of weight gain and an increase in waist circumference [**chapter 7**]. These men may be offered an information meeting through their employers, prior to their retirement. This information should enhance awareness on energy balance and include tools to beneficially change the diet and physical activity.[30]

Suggestions for future research

In our study on weight gain with aging [**chapter 3**], we found no substantial differences in weight gain between adult subjects with a lower and higher education. Nevertheless, at the same age, subjects with a lower education weigh more than subjects with a higher education. Therefore, we concluded that the education-associated difference in body weight occurs somewhere before adulthood, although we could not determine when and how. Several other studies have looked

at the association between socioeconomic status and weight status.[31-33] Adolescents of higher socioeconomic status reported lower habitual energy intake and higher levels of habitual physical activity,[32] but underlying mechanisms require further investigation. Such mechanisms can be studied by following a birth cohort.

Further, we observed that young parents experience more weight gain than young men and women without children, but we could not determine which changes or differences in lifestyle behaviours exactly caused this difference in weight gain [**chapter 5**]. Therefore, future research should focus on changes in lifestyle behaviour that accompany parenting. Since questionnaires do not seem to be the best tools for such research questions, more qualitative research designs, such as focus groups, may offer some clues.

In addition, further research is needed to study effects of professional retirement on weight gain and fat mass in middle-aged women. Most research on weight gain in middle aged women has been focusing on effects of the menopause, while retirement may also be an important life transition in middle aged women. In our population, only a small group of women retired from work, and therefore could not be studied properly. In new generations, more women have a job and will retire in the near future. It would be informative to know how retirement affects their lifestyle behaviours and body composition.

Furthermore, cohort data such as data from the AGAHLS should be kept up-to-date. It would be very informative to know the behaviours and weight status from adolescents nowadays and to follow these adolescents into their young adulthood and beyond. Several aspects in the physical and social environment have changed over the past decades, but only limited information about their effects on behaviour, health or weight status is available. In addition, such studies are needed to evaluate associations between different weight status indices (body weight, BMI) or fat-indices (waist circumference, skinfolds, impedance measurements) in adolescents and (future) health status. Based on such research, proper cut-off values for increased health risk may be established for children and adolescents based on indices other than BMI.

In addition, prevention of weight gain in middle-aged and elderly populations deserves further research. We showed that weight gain is at least apparent up to age 70 years [**chapter 3**], but also after the age of 70 years, an increase in percentage body fat has been observed.[34] High body fatness (or obesity and sometimes also overweight) in the elderly has been shown to predict disability in the elderly,[35,36] but also morbidity[37] and mortality.[38-41] Furthermore, it has been proposed that prevention of weight gain in middle-aged and elderly is more cost-effective than in younger populations.[42] Middle-aged and elderly adults have a higher body weight, have a higher absolute risk for cardiovascular diseases, and possible adverse health outcomes resulting

from weight gain and overweight are near. Therefore, weight gain prevention almost directly lowers the incidence of overweight-related disease in the middle-aged and elderly population. The baby boomers, born in the late fourties and early fifties of the last century are now reaching the age at which effects of overweight and obesity manifest themselves: diabetes mellitus, heart diseases and certain forms of cancer. There is need for more information on determinants of increased waist circumference in this age group, and prevention of waist gain in the middle aged and elderly may have important public health benefits.[39,43]

Finally, more research is needed to translate findings of epidemiological studies into interventions or prevention programmes. New challenges in weight gain prevention will be how to make people change their unhealthy lifestyle behaviours into more healthy ones. Three intervention trials within the NHF-NRG project have shown modest effects on this challenge.[28-30] Based on results of these three intervention studies, it seems effective to include environmental changes in the intervention trials.

Conclusions

Based on results as shown in the present thesis, it can be concluded that there is a need for weight gain prevention. Weight gain is of all ages, both genders and all levels of education. Therefore a population wide, life-course approach of weight gain prevention should be implemented.
In addition, we could identify several subgroups that gained even more weight than their peers. Targeted weight gain prevention could be implemented, specifically directed to these subgroups. When studying associations between (changes in) lifestyle and weight gain, several methodological limitations came around. Taking into account these limitations, it seems hard to draw firm conclusions on lifestyle determinants of weight gain. However, when associations between changes in lifestyle factors and weight gain are biologically plausible (in line with the concept of energy balance), there is no problem to address such lifestyle factors in weight gain prevention programs.

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Summary

The present thesis is part of the Netherlands Heart Foundation "Netherlands Research programme weight Gain prevention" (NHF-NRG). The focus of this programme was to study determinants of weight gain, and to develop, carry out and evaluate comprehensive weight gain prevention programmes for three different target groups: adolescents, young adults and recently retired people. In all studies within this programme, both food intake and physical activity behaviours were integrated, rather than focusing on a single behaviour. The present thesis focuses on life-style predictors of weight gain. An introduction and outline of this thesis is described in *chapter 1*.

The main objectives of this thesis were 1) to identify and describe groups at high-risk of weight gain and 2) to describe modifiable determinants of weight gain within the selected age groups (adolescents, young adults and recently retired persons). High-risk groups were identified based on personal circumstances (such as household composition/work etc.), life events or life transition, and on anthropometric indices. Modifiable determinants of weight gain that we studied were (changes in) the main energy balance related behaviours, i.e. total dietary intake and physical activity level, or components of these two main behaviours, e.g. energy density of the food, portion size and eating frequency, and intensity, duration and frequency of physical activities.

In the Netherlands, only a few studies exist in which persons' body weight, body height, and waist circumference are measured over a longer period of time, combined with information on their energy intake and physical activity. Such cohorts are crucial to study lifestyle determinants of weight gain in a real life setting. In this thesis we have used data of two Dutch cohort studies containing both objective measures of body weight, body height and waist circumference as well as self-reported lifestyle measures: i.e. the Amsterdam Growth and Health Longitudinal Study (AGAHLS, adolescents and young adults; 13 years old at baseline followed for 24 years) and the Doetinchem Cohort Study (DCS, adults in general, including young adults and recently retired subjects; 20-59 years old at baseline followed for 11 years).

Adolescence appears to be a critical period for the development of obesity. Until now, little is known about the identification of adolescents who are at increased risk of becoming overweight or obese at an adult age, and it is uncertain which measures should be used to identify adolescents at high risk of adult obesity. In a longitudinal study (*chapter 2*) using data from the AGAHLS, we observed that adults with high body fatness (measured with dual-energy X-ray absorptiometry) had a significant higher body mass index (BMI) and sum of four skinfolds (S4SF) values during adolescence than adults with lower body fatness. In addition, adults with high body fatness had increased more in BMI and S4SF from adolescence into adulthood, than adults with lower body fatness. We showed that high adult body fatness is better predicted by adolescent skinfold thickness than by adolescent BMI. Skinfold thickness should therefore be used as the preferred screening tool to determine which adolescents are at increased risk of

becoming adults with high body fatness. Weight-gain prevention programs should focus on adolescents with large skinfold thicknesses.

In a longitudinal study using data from the DCS, we evaluated age, period and cohort effects on BMI (*chapter 3*). One of the main findings of this study was that the apparent decline in BMI with ageing in the oldest group of men as seen in cross-sectional analyses appeared to be an artefact due to a cohort effect. Our longitudinal data showed that BMI also increased in the oldest age group, in both men and women. Furthermore, increase in BMI was largest in the youngest age group, and the BMI of younger cohorts was higher than that of older birth cohorts at any given age between 30 and 60 years. Although the mean BMI of higher educated persons was lower than the mean BMI of lower educated persons, their increase in BMI with ageing was (in general) similar. This implies that prevention of weight gain should follow a population-wide, life-course approach.

Lifestyle behaviours are subject to change between adolescence and adulthood. Changes in dietary intake and physical activity (PA) are likely to result in changes in body weight and body fatness. In *chapter 4* we evaluated the associations between the developments of energy intake (EI) and PA from adolescence into adulthood, based on multiple measurements of the AGAHLS, in relation to body composition at age 36 years. Based on the concept of energy balance, it was assumed that especially a high baseline or increase in total EI and/or a low baseline or decrease in PA from adolescence into adulthood would be associated with a higher percentage of body fat at adult age. Results, however, showed only sparse associations of EI and PA with obesity and offer strong presumptions that longitudinal observational data based on self-report do not fit the theory of energy balance. Probably, a mixture of measurement error, reporting error, time between measurements and difficulties related to reporting habitual lifestyles makes it very difficult to associate self-reported lifestyle behaviours to weight gain (and resulting body fatness). Therefore, we believe that findings of relations between energy balance related behaviours and weight gain based on self-reported data (that might seem cause-effect relationships) should be interpreted with serious caution.

In *chapter 5*, we investigated the impact of four live events (leaving the parental home, starting to work, living together, and parenting children) on body weight, body mass index, skinfold thickness and waist circumference, using data from the AGAHLS. If effects were observed, we investigated the underlying changes in lifestyles (i.e. total energy intake and physical activity level).
Largest increases in body weight and S4SF between age 16 to 27 years were observed in men who had started to work before age 21 years, while they were still living with their parents. Smallest increases were observed among men and women who left the parental home and

started to work before age 21 years. Between age 27 and 36 years, women who lived alone had a larger increase in body weight and body fat than women who lived with a partner. This difference was not accompanied by significant differences in the changes in lifestyles. Parenting children was associated with larger increases in body weight and waist circumference in both men and women. Women who were parenting children at age 36 years showed a significant increase in total energy intake between age 27 and 36 years. Unexpectedly, some other significant changes in behaviours observed were contradictory to observed changes in anthropometry: a decrease in energy intake was associated with an increase in BMI and maintaining the physical activity level was associated with an increase in body weight. In conclusion, life transitions seem to affect body weight and body fatness. Especially men starting to work while living with their parents and young men and women planning a family seem important target groups for intervention programs to prevent (excessive) weight gain.

The aim of the study presented in *chapter 6* was to identify modifiable determinants of weight gain among young adults. Within the analyses, we studied the development of lifestyle behaviours in young adults over time, and the association between weight gain and both baseline and changes over time in diet and physical activity. We observed that several energy balance related behaviours changed over time in young adults. In men, weight gain was associated with changes in diet and physical activity over follow-up, but not with baseline diet and physical activity. In women, weight gain was associated with both baseline levels and changes in diet and physical activity. However, some baseline levels of behaviour and their changes were associated with weight gain in opposite directions. Different lifestyles were associated with weight gain in men compared to women. Among men, a decrease in using breakfast, an increase in juice consumption and a decrease in time spent on biking were associated with weight gain. Among women, an increase in sweet snack consumption was associated with weight gain. In conclusion, several aspects of energy balance related behaviour are not stable over time in young adults. Furthermore it seems pertinent to study changes in behaviour, rather than only baseline behaviour, in order to study determinants of weight gain.

In *chapter 7* we evaluated the impact of retirement on diet, physical activity, body mass index and waist circumference, over a five-year follow-up period in a population-based cohort. We hypothesised that a sudden reduction in work-related physical activity of moderate intensity may lead to unfavourable changes in energy balance and thereby lead to an increase in body weight and waist circumference. Results showed that retirement was associated with an increase in weight and waist circumference among those with former active jobs, but not among those with former sedentary jobs. Weight gain and increase in waist circumference were associated with a decrease in fruit consumption and fibre-density of the diet, with an increase in eating breakfast and in the consumption of sugar sweetened soft drinks, and with a decrease in

several leisure time physical activities. In conclusion, men who retired from physically active jobs were more prone to gain weight and increase in waist circumference compared to men who retired from sedentary jobs. Since retirement may bring opportunities for healthy changes in diet and physical activity, it seems warranted to develop health promotion programs aimed at prevention of overweight in men who are eligible for retirement, particularly in those who have physically demanding jobs.

In the general discussion (*chapter 8*), we discussed the methodology used in the presented studies and results are integrated into overall conclusions, implications for (clinical) practice and suggestions for future research. Based on results of the present thesis, it can be concluded that there is a need for weight gain prevention. Weight gain is of all ages, both genders and all levels of education. Therefore a population wide, life-course approach of weight gain prevention should be implemented. In addition, we could identify several subgroups that gained even more weight than their peers. Targeted weight gain prevention could be implemented, specifically directed to these subgroups. When studying associations between (changes in) lifestyle and weight gain, several methodological limitations came around. Taking into account these limitations, it seems hard to draw firm conclusions on lifestyle determinants of weight gain. However, when associations between changes in lifestyle factors and weight gain are biologically plausible (in line with the concept of energy balance), there is no problem to address such lifestyle factors in weight gain prevention programs.

Samenvatting

Overgewicht en obesitas komen steeds vaker voor, ook in Nederland. Een te hoog lichaamsgewicht verhoogt het risico op het ontwikkelen van chronische aandoeningen, zoals type 2 diabetes (suikerziekte), hart- en vaatziekten, sommige vormen van kanker, en galstenen. Mensen met overgewicht lukt het vaak wel om gewicht te verliezen op korte termijn, maar het is lastig om het verminderde gewicht vast te houden voor langere tijd. Door preventie van gewichtsstijging lijkt de toename in het vóórkomen van overgewicht beter te stoppen dan door behandeling van overgewicht alleen.

In dit proefschrift zijn verschillende studies opgenomen welke betrekking hebben op het vóórkomen en leefstijl oorzaken van gewichtsstijging binnen de Nederlandse bevolking. We hebben specifiek gekeken naar gewichtsstijging bij jongeren, jong volwassenen en pas gepensioneerden. Doel van deze studies was het aanreiken van handvaten voor interventieprogramma's ter preventie van gewichtsstijging.

We vonden dat adolescenten met relatief dikke huidplooien, maar niet diegenen met een hogere 'body mass index' (BMI, =gewicht (kg)/lengte $(m)^2$) een grotere kans hadden om als volwassene een hoog percentage lichaamsvet te ontwikkelen. Daarom zouden preventieprogramma's zich moeten richten op adolescenten met dikke huidplooien.

Van adolescentie naar volwassenheid kwamen de jonge mannen die vroeg gingen werken en pas later het ouderlijke huis verlieten, meer aan dan jonge mannen die al eerder op zichzelf gingen wonen of pas later gingen werken. Jongvolwassen mannen en vrouwen die kinderen verzorgden kwamen gemiddeld meer aan dan mannen en vrouwen die dat niet deden. Daarnaast kwamen alleenstaande vrouwen meer aan dan samenwonende vrouwen, maar konden deze verschillen niet toegeschreven worden aan verschillen in voeding en lichamelijke activiteit.

Bij volwassenen zagen we dat gewichtsstijging vóórkomt in alle leeftijdsklassen tussen 20 en 70 jaar. De grootste toename zagen we bij de jongvolwassenen, maar ook de groep mannen en vrouwen van 60 jaar en ouder kwam gemiddeld nog steeds aan. Over het algemeen hadden personen die een lagere opleiding hadden genoten een hogere BMI dan personen met een hoger opleidingsniveau, maar de gewichtsstijging met het ouder worden was voor alle opleidingsniveaus gelijk. Het bleek dat personen die later geboren zijn, een hogere BMI hebben op een bepaalde leeftijd dan personen die vroeger geboren zijn.

Effecten van pensionering op lichaamsgewicht en middelomvang hebben we bestudeerd in een groep mannen van 50-65 jaar die we 5 jaar volgden. We zagen dat mannen die met pensioen gingen vanuit een actieve baan meer in gewicht en middelomvang toenamen dan mannen die met pensioen gingen vanuit een zittende baan.

Wanneer we keken naar leefstijl in relatie tot gewichtsstijging, zagen we dat voor jongvolwassen mannen en vrouwen (20-40 jaar) verschillende aspecten van leefstijl gepaard gingen met gewichtsstijging. Bij mannen ging gewichtsstijging gepaard met een afname in het aantal uren fietsen en van de consumptie van vleesvervangers en ontbijt, een toename van de consumptie van fruitdrank en vis en een toename van de portiegrootte vlees. Bij vrouwen ging gewichtsstijging gepaard met een toename van de hoeveelheid geconsumeerde zoete snacks.

Bij mannen van middelbare leeftijd (50-70 jaar), ging gewichtsstijging en een toename in middelomvang gepaard met een afname in fruitconsumptie en de vezeldichtheid van de voeding, vaker gaan ontbijten, een stijging in de consumptie van gesuikerde frisdranken, en met een daling in het aantal uren besteed aan lichamelijke activiteit in de vrije tijd.

Over het algemeen bleek het lastig om relaties tussen leefstijl en gewichtsstijging goed te interpreteren. Om verschillende methodologische redenen leek het beter om, in plaats van baseline gedrag, veranderingen in gedrag te relateren aan veranderingen in gewicht. Dit heeft vooral te maken met rapportage fouten. Maar ook om andere methodologische redenen, lijken observationele data beperkt geschikt om leefstijl oorzaken van gewichtsstijging te bestuderen. Echter, wanneer gevonden relaties tussen leefstijl en gewichtsstijging biologisch te verklaren zijn, is het geen probleem om deze leefstijlfactoren mee te nemen in interventieprogramma's ter preventie van gewichtsstijging.

Dank aan...

De aio-periode tot de uiteindelijke afronding van mijn proefschrift was lang. In die tijd heb ik een hoop geleerd van de vele mensen om me heen. Hierbij wil ik iedereen danken die op wat voor manier dan ook heeft bijgedragen aan de vorming en afronding van dit proefschrift. Maar zeker ook iedereen die er voor heeft gezorgd dat deze periode een tijd is geweest waar ik met plezier op terug kan kijken. Het is onmogelijk om iedereen bij naam te noemen, dus dat ga ik niet eens proberen... allemaal dank!

Toch wil ik enkelen in het bijzonder bedanken.

In de eerste plaats een woord van dank aan mijn promotoren. Jaap, dank voor de inspirerende overleggen. Nieuwe resultaten leidden meestal weer tot nieuwe vragen en ideeën. Je bent kritisch, maar altijd goedlachs en vriendelijk, waardoor ik toch weer vrolijk de data indook om nieuwe tabellen te kunnen vullen. We hebben er door de jaren heen heel wat voorbij zien komen... Willem, dank voor de kritische en vaak hoopgevende commentaren (welke meestal op grote hoogte geschreven werden).

Mijn copromotoren, Jantine en Tommy, wil ik in de eerste plaats danken voor alle gezellige momenten. Maar natuurlijk ook voor de begeleiding tijdens het hele traject. Wat moeten jullie soms een engelengeduld hebben gehad! Dank voor alles!

Dan het NRG-genootschap. Andrea, Amika, Lydia, Gert-Jan en Stef, het was prettig om de interventies er soms bij te hebben om het ‘droge’ werk soms wat beter te kunnen verteren en interpreteren. Soms leek het een lotgenoten-contactgroep, maar meestal waren het vooral functionele en erg gezellige bijeenkomsten!

Collega’s van PZO, dank voor de gezellige werksfeer. Mijn kamergenoten, Lilian, Ester, Annemien, Nienke en Carolien, maar ook alle anderen van zowel voor als achter de klapdeuren: dank voor alle leuke momenten, spelletjesavonden en meer. Wanda, Carolien, erg fijn dat jullie altijd met raad en daad klaar staan. Ook bijzonder dank voor jullie spontane actie om me dit proefschrift nu eens af te laten schrijven. Zie hier het resultaat!

Collega’s van het EMGO en van IGW maakten het de moeite waard om af te reizen naar Amsterdam. Marieke, Judith, Maiza, Amber, Dieuwke en France. Er is blijkbaar meer te doen in Amsterdam en Amersfoort dan alleen werken. Leuk om het mee te maken. Marieke ook dank voor het prettige gezelschap tijdens de reizen naar verschillende congressen, maar vooral de reizen ná de congressen!

Ardine en Monique, dank voor het vertrouwen waardoor ik de afgelopen jaren weer met veel plezier bij PZO heb kunnen werken.

Carolien en Marieke, ik vind het een eer dat jullie mijn paranimfen willen zijn! Dank!

Pa en ma, ook al was het voor jullie soms een raadsel waar ik allemaal mee bezig was daar boven de rivieren: dank voor jullie vertrouwen en onvoorwaardelijke steun. Het is lang zaaien, verzorgen en onkruid wieden geweest, maar de oogsttijd is aangebroken!

Laurens, je hebt veel geduld met me moeten hebben de afgelopen jaren. Dank voor al je geduld, motivering, medeleven, afleiding en liefde! Ik hoop dat we nu samen nóg meer van het leven kunnen gaan genieten...

About the author

Astrid Nooyens was born on October 22nd 1976 in Baarle-Nassau, the Netherlands (no… not in Belgium!). In 1995 she completed secondary school (VWO) at the "Odulphus Lyceum" in Tilburg. In 1995 she started the study "Health Sciences" at the Maastricht University, where she received her MSc degree in 2000.
In June 2002 she started her PhD-project of which the results are described in this thesis. This project was conducted at Centre for Prevention and Health Services Research of the National Institute for Public Health and the Environment (RIVM) in Bilthoven in collaboration with the EMGO Institute of the VU University Medical Center in Amsterdam and the Institute of Health Sciences of the VU University in Amsterdam.
During her PhD period she attended the Postgraduate Epidemiology Programme of the EMGO Institute, and in 2006 she received her MSc degree in epidemiology at the VU University in Amsterdam.
In November 2006 she started her job as epidemiological researcher on lifestyle determinants of cognitive decline at the Centre for Prevention and Health Services Research of the RIVM.

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