BODY MASS INDEX IN YOUNG DUTCH ADULTS:

ITS DEVELOPMENT AND THE ETIOLOGY OF ITS DEVELOPMENT

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PROEFSCHRIFT
ter verkrijging van de graad van
doctor in de landbouwwetenschappen,
op gezag van de rector magnificus,
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in het openbaar te verdedigen
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Het Onderzoek naar Overgewicht waarvan de studies die in dit proefschrift worden beschreven deel uitmaken, werd financieel ondersteund door het Praeventiefonds.
Het verschijnen van dit proefschrift werd mede mogelijk gemaakt door steun van de Nederlandse Hartstichting.

1. De bevinding dat overgewicht op jonge leeftijd een groter risico voor de gezondheid met zich meebrengt dan overgewicht op oude leeftijd, rechtvaardigt niet de conclusie dat de geleidelijke gewichtstoename bij jonge volwassenen het gezondheidsrisico niet verhoogt.

(dit proefschrift)

2. Het is uiterst onbevredigend dat de Gezondheidsraad aanbeveelt tot gewichtsvermindering over te gaan bij een Queteletindex groter dan 30 kg/m^2 terwijl matig overgewicht geaccepteerd dient te worden; enerzijds zullen meer mensen ziek worden binnen de grote groep met matig overgewicht dan binnen de kleine groep met ernstig overgewicht, anderzijds is overgewicht als dit zich eenmaal heeft ontwikkeld voor veel mensen irreversibel.

(Advies inzake adipositas, Gezondheidsraad 1983)

3. De aan leeftijd gerelateerde toename in Queteletindex van jonge volwassenen met overgewicht en van jonge volwassenen zonder overgewicht is vergelijkbaar.

(dit proefschrift)

4. De vetverdeling is naast gewicht en lengte van meer belang dan categorieën voor lichaamsbouw; bij elke weegschaal hoort een centimeter.

5. Naarmate men meer overgewicht heeft kan men van eenzelfde gewichtsvermindering een sterkere risico-afname verwachten.

6. Regelmatige ijking en - bij een niet volstrekt vlakke ondergrond - een eenvoudige plank als ondergrond zijn essentieel voor een weegschaal waarmee individuele gewichtscurves worden gemeten, zoals in de medische praktijk.

7. Het is vreemd dat bij het opzetten van de landelijke kankerregistratie het realiseren van een van de hoofddoelstellingen, het stimuleren van het gebruik van de gegevens voor epidemiologisch onderzoek, niet in de begroting is opgenomen.

(Bestek 1985)

8. De meeste vormen van voedselhulp hebben ondervoeding en gebrek in de derde wereld eerder verergerd dan verholpen.

9. In een tijd waarin bezuinigingen en een 'tolerante' houding elkaar versterken, dreigen psychisch gestoorden in groeiende aantallen te vervallen tot daklozen in de grote steden.

10. Het toenemend aantal tijdelijke arbeidsplaatsen ten koste van vaste aanstellingen bemoeilijkt het vinden van werk voor vrouwen met kinderen en het krijgen van kinderen voor vrouwen met werk.

11. De ergernis bij treinvertragingen is omgekeerd evenredig met de frequentie van treingebruik.

Proefschrift M.A. Rookus
Body mass index in young Dutch adults: its development and the etiology of its development
Wageningen, 19 november 1986.

aan mijn ouders en Jaap

CONTENTS

VOORWOORD

In dit proefschrift worden enkele studies beschreven die deel uitmaken van een uitgebreid Onderzoek naar Overgewicht (het ONNO-projekt). Dit onderzoek dat vanaf 1979 tot 1986 door medewerkers van de vakgroep Humane Voeding van de Landbouwuniversiteit te Wageningen werd uitgevoerd, werd financieel ondersteund door het Praeventiefonds.

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Matti Rookus, augustus 1986

1 GENERAL INTRODUCTION

1. GENERAL

This thesis deals with the development of the body mass index (weight/height^2), as an indicator of overweight (body mass index > 25 kg/m^2, Garrow 1981), and some determinants of this development in young adults. In this chapter the reason why overweight was the subject of a mixed-longitudinal study is explained. The first part deals with the health riks of overweight with special reference to young adults. The second part deals with the questions whether or not the age-related weight gain should be prevented, whether it can be prevented and what its determinants are.

2. RELATION BETWEEN OBESITY AND MORTALITY

2.1. General

It is in the United States that the relationship between obesity and mortality has been most extensively studied. Since about 1930, studies on insured persons have consistently indicated that the lowest relative mortality is found at weights somewhat below the average weight in both sexes. Also, these studies have repeatedly shown that relative mortality is higher below this optimal weight range, and that relative mortality increases steadily with an increase in weight above the optimal weight range. The latest study of the build of insured persons, the Build Study 1979, again shows this U-shaped relationship, with the lowest relative mortality in men and women who are 5-15% under the average weight (Lew 1985).

The relevance of drawing conclusions from these studies has been questioned, because they have been conducted on selective groups; initially healthy subjects, predominantly drawn from the middle and high socio-economic classes. Other studies on the risk of mortality incurred by being overweight have not always found an U-shaped relationship (Larsson et al. 1981). This has resulted in a continuing debate on whether overweight really is a risk factor for increased morbidity and mortality, as the Build Studies had indicated (Andres 1980, Keys 1980, Gezondheidsraad 1984). Recent analyses have shown that the discrepancies among studies may be largely caused by confounders, the most important of which are duration of follow-up, age at start of follow-up, and smoking behavior.

2.2. Confounding by duration of follow-up

During the first years of follow-up, relative mortality is high among underweight subjects and low among overweight subjects (Lew 1985, Garrison and Castelli 1985). With time, relative mortality decreases among the initially underweight and increases among the initially overweight subjects. Thus, the Build Study 1979 (Lew 1985), as well as the Framingham study (Garrison and Castelli 1985) show that the U-shaped relationship between mortality and obesity only appears after long periods (>15 years) of follow-up. This has been supported by other extensive follow-up studies of long duration (Lew and Garfinkel 1979, Waaler 1984).

The effect of duration of follow-up may interact with the dependence on age of the risk associated with overweight; the same degree of overweight produces a greater increased risk during follow-up in younger subjects than in older subjects (Waaler 1984, Van Itallie 1985). This aspect will be considered in more detail below (4.2.).

2.3. Confounding by smoking behavior

Since smokers generally weigh less than non-smokers and smoking is related to mortality, the mortality risk of being underweight may be overestimated and the mortality risk of being overweight may be underestimated, if smoking behavior is not adjusted for. So far, two long-term follow-up studies have been able to take smoking status into account: the Framingham study (Garrison et al. 1983, Garrison and Castelli 1985) and the American Cancer Society study (Lew and Garfinkel 1979, Lew 1985). The relationship between mortality and obesity was found in both smokers and non-smokers, but the curve for smokers ran at a markedly higher level than did the one for non-smokers.

In the American Cancer Society study, optimal weights in men who never smoked were lower than in men who smoked, whereas there was no difference in women (Lew and Garfinkel 1979, Lew 1985). In the Framingham study no U-shaped relationship among the non-smokers could be established, because more than 80% of men under the desirable weight were smokers. This indicates that the weight range with lowest mortality in non-smokers may be the same or lower than the desirable weight range in the total sample.

3. RELATION BETWEEN OBESITY AND MORBIDITY

3.1. Relative weight and morbidity

To examine the health risks associated with obesity, the quality of life seems to be a criterion at least as important as longevity. Relative weight (weight corrected for height) has been shown to be associated with a vast range of risk factors and diseases, such as hypertension, hyperinsulinemia, diabetes mellitus, hypercholesterolemia, hypertriglyceridemia, heart and circulatory diseases, digestive and endocrine disorders, gall and kidney stones, arthritis, gout, various types of cancers, and ventilatory disorders (Keys 1980, Larsson et al. 1981, Royal College of Physicians 1983, Simopoulos and Van Itallie 1984, Waaler 1984, Bray 1985a, Donahue et al. 1985, Kral 1985, Seidell et al. 1986a). Moreover, functional impairment of activities of daily living is associated with obesity (Kral 1985). This might be the most prevalent complication of obesity and since these impairments are feared most (Thomson 1986), they might be as difficult to endure as those imposed by other diseases.

As Waaler (1984) has most clearly shown, mortality at low levels of the body mass index is caused by categories of diseases that differ from those responsible for mortality at high levels of the body mass index. Total morbidity seems to have a U-shaped relationship with the body mass index, as does mortality (Bray 1985a).

In addition to these somatic consequences of overweight, psychosocial consequences of overweight are clearly not to be neglected. In industrialized countries, where a slim body shape is favored, body fatness has its impacts on the individual's self-concept, anxiety and emotional well-being (Rodin 1981, Van Strien 1986).

3.2. Fat distribution and morbidity

Recent studies show that apart from and in addition to indices of relative weight, indices of body fat distribution are strongly associated with somatic morbidity (Kissebah et al. 1982, Larsson et al. 1984, Hartz et al. 1984, Björntorp 1985a, Seidell et al. 1985, 1986b). Fat cells at different locations differ in their metabolism. For instance, fat accumulated in the abdominal region is more sensitive to lipolytic stimuli than fat in the femural/gluteal region. The resulting free fatty acids are released into the portal vein, and high concentrations seem to inhibit uptake of insulin by the liver, providing a possible explanation for the peripheral hyperinsulinemia, insulin resistance, diabetes mellitus, hypertriglyceridemia and possibly hypertension, associated

with abdominal obesity (Björntorp 1985b).

4. DEVELOPMENT OF RELATIVE BODY WEIGHT

4.1. General

According to longitudinal studies conducted in industrialized countries, relative weight increases with age until circa 50 years of age in men and until the menopause in women, then remains fairly constant and decreases after the age of 70 (Forbes and Reina 1970, Hsu et al. 1977, Kannel et al. 1979, Noppa et al. 1980, Lindquist 1982, Borkan et al. 1983a, Khoury et al. 1983, Waaler 1984). Cross-sectional studies show a similar upward trend (Montoye et al. 1965, Forbes and Reina 1970, Khosla and Lowe 1968, Bjelke 1971, Cronk and Roche 1982, Rosenbaum et al. 1985, Kok et al. 1983, Van Sonsbeek 1985). The weight decrement in elderly women is not always found, however (Noppa et al. 1980, Kok et al. 1983). Waaler (1984) found that before middle age the gain was curvilinear in men, with larger gains between 20 and 30 years of age than between 30 and 50 years of age, and was almost linear in women.

With respect to this weight increment three basic questions arise, (1) whether it should be prevented in order to avoid a relative increase in mortality and morbidity, (2) whether it can be prevented, (3) why it occurs; i.e. whether the weight gain with increasing age is genetically determined and/or is influenced by environmental/behavioral factors.

4.2. Evaluation of adult weight gain

4.2.1. Arguments in favor of a health risk that does not increase with adult weight gain

The answer to the question of whether the weight gain should be prevented is clearly affirmative under the assumption of a U-shaped relationship between overweight and risk of mortality/morbidity that does not depend on age. The age-dependency of the U-shaped curve has only recently begun to be understood.

After the same period of follow-up the U-shaped relationship between relative mortality and overweight was steeper for men who had been issued policies at ages 15-39 years than at ages 40-69 years (Seltzer 1966, Simopoulos and Van Itallie 1984). A similar steep trend at lower ages emerged in 13 years follow-up of 750.000 subjects (Lew and Garfinkel 1979) and in 12-years follow-up of 1.8 million Norwegians (Waaler 1984). The NHANES study showed that the relative risk of morbidity revealed by comparing overweight with

non-overweight subjects was higher in 20-44 year old subjects than in 45-74 year old subjects; the risk ratio was 2.9 for hypertension, 1.9 for elevated cholesterol and 1.8 for diabetes (Van Itallie 1985). In several other studies, the association between relative morbidity and obesity was present or most pronounced in young adulthood (<45 years) (Shapiro et al. 1969, Rabkin et al. 1977, Pooling Project 1978, Larsson et al. 1981). So, the same level of overweight seems to carry a greater risk at young ages than at older ages, and thus it is tempting to suggest that with some weight gain during adulthood the risk may remain constant, whether or not one is initially overweight.

More evidence that optimal weight may increase with age has been presented by the Build Study 1979 (Royal College of Physicians 1983, Lew 1985). The minimum of the U-shaped curve, the relative weight with lowest mortality, shifted to higher values with increasing age. The body mass index associated with lowest mortality increased from 21.4 kg/m^2 in men and 19.5 kg/m^2 in women, 20-29 years of age to 26.6 kg/m^2 in men and 27.3 kg/m^2 in women, 60-69 years old (Andres et al. 1985).

The American Cancer Society study based on subjects largely of middle socio-economic class and ostensibly in good health shows the same picture; optimal weight was 80-89% of average weight until the age of 70, and above this age was nearer to average (Lew 1985). Average weights increased with increasing age and thus optimal weight increased also. This was also shown in a review by Andres et al. (1985) who compared the body mass index associated with minimal mortality in 23 populations with the cut-off points of the Metropolitan Height-Weight tables.

These studies suggest that a weight gain during adulthood may not result in an increased risk of mortality insofar as the gain is equivalent to the gain in optimal weight; if some degree of overweight is present in young adulthood, a slight weight gain with increasing age may not always enlarge the risk, that is already present, since overweight in younger ages is more detrimental for health than overweight in older ages. Although this contention already has led to the recommendation of age categories in weight-height tables (Andres et al. 1985), it is based on implicit assumptions that will be explained below.

4.2.2. Arguments against a health risk that does not increase with adult weight gain

4.2.2.1. Optimal weight versus optimal weight range

First, it is assumed that the age-dependency of the optimal weight is

comparable with that of the optimal weight range. Bearing in mind the U-shaped curve between overweight and mortality, the relevance of considering one optimal value only can be questioned. No age trend in optimal weight range was found in men in the Framingham study (Feinleib 1985, Garrison and Castelli 1985). The Norway study (Waaler 1984) showed that in men the range of the body mass index with lowest relative mortality was constant over age, whereas in women the range extended to higher values as age increased: at higher ages women seemed to tolerate overweight better than men. Thus, an age trend in the range of optimal body mass index is not clear, certainly not in men.

Secondly, as a contradiction in terms, the age-related mortality curves are considered to be longitudinally interpretable; distinctive age-related causes of mortality or cohort effects, are assumed not to be present. As is explained below, this assumption may not be right.

4.2.2.2. Age-related causes of mortality

It can be argued that the higher risk accompanying obesity at younger ages is associated with obesity that has a strong genetic component. As they age, a selection takes place within the obese population: the 'stronger' subjects survive and the risk of mortality is increasingly caused by obesity that has weaker genetic components and thus depends more heavily on environmental/behavioral factors.

4.2.2.3. Cohort effect

In addition to cohort effects in both relative weight and mortality/ morbidity, there might also be a cohort effect in the association between the two.

In a study done in the United States a positive cohort effect in relative weight was found to be accompanied by a negative cohort effect in mortality/morbidity in the United States (Feinleib 1985); at the same age, subjects that were born later were more obese than subjects born earlier this century, but displayed a lower risk of morbidity. These observations suggest that there might be a cohort effect in the association between relative weight and mortality/morbidity: this is supported by the finding that the range of weight for height, associated with lowest mortality was lower in the 1957 Metropolitan Desirable Weight Table, than in the 1983 Height-Weight Tables (Metropolitan Life Insurance Company 1959, 1983).

In contrast to the United States, in Sweden a positive cohort effect in relative weight was probably one of the reasons of the positive cohort effect

in ischaemic heart diseases (Welin et al. 1983). Whereas the body mass index was not an independent risk factor in 50 year old men born in 1913, it had become an independent risk factor in 50 year old men born in 1923 (Welin et al. 1983). This cohort effect in relative weight was not present in the median, but in the higher percentiles (Christensen et al. 1981).

In the Netherlands, a positive secular trend in the body mass index of 19-year-old conscripts was present until it peaked in the cohort born in 1945. Men born later showed no or a slightly negative secular trend (Deurenberg et al. 1986). Until 1972, there was a positive secular trend in the mortality caused by ischaemic heart diseases (Central Bureau of Statistics 1983). Since then, the mortality has been declining again. Since most subjects, who died from ischaemic heart diseases around 1972 were part of cohorts that still showed a positive trend in body mass index, a cohort effect in the association between body mass index and ischaemic heart diseases might be present in the Netherlands, as in other countries. It is clear that the presence of a cohort effect in the U-shaped curves of risk of morbidity hampers the interpretation of age trends.

4.2.2.4. Longitudinal studies

In addition to the use of age-related mortality curves, weight gain during adulthood can be evaluated by means of longitudinal observations; a positive relationship has been found between changes in body weight and simultaneous changes in blood pressure (Gillum et al. 1982, Hsu et al. 1977, Ashley and Kannel 1974) as was true for serum cholesterol (Ashley and Kannel 1974, Gordon and Kannel 1973, Kromhout 1983), blood glucose and uric acid (Ashley and Kannel 1974). Weight reduction has been advocated as an effective and safe treatment for hypertension (Hovell 1982). Adult weight gain has been shown to determine the incidence of cardiovascular diseases (Hubert et al. 1983, Shapiro et al. 1969). In the Framingham study, this association was independent of the level of body mass index at which this increment took place (Hubert et al. 1983). So, these longitudinal studies do not support the contention that some weight gain during adulthood might not increase the risk to health.

4.2.2.5. Anthropometric studies

Anthropometric studies also suggest that adult weight gain is undesirable. Even if a subject does not gain weight during adulthood, some changes in body composition nevertheless take place; longitudinal studies show that the fat mass increases and the fat free mass decreases with increasing age (Borkan et

al. 1983b, Forbes and Reina 1970). Moreover, the fat distribution becomes more android with aging in men and postmenopausal women, as was shown by cross-sectional studies based on skinfolds in men aged 13-65 years (Garn and Young 1956), and in subjects aged 6-18, 30-60 years (Mueller 1982), or based on the waist/hip circumference ratio in women aged 38-60 (Lapidus et al. 1984) or based on computed tomography in men aged 46-69 (Borkan et al. 1983b) or based on waist-hip ratio, waist-thigh ratio, or the ratio of intra-abdominal to subcutaneous fat (computed tomography) in men aged 19-85 and women aged 19-72 years (Seidell et al. 1986c). In men, fat distribution was more strongly related to age than in women (Seidell et al. 1986c). Fat that accumulates in women of child-bearing years shows a preference for the gluteal region. This fat seems to have a specific function during lactation (Rebuffé-Scrive et al. 1985). Self-reports of adult weight-history suggest that an unfavorable fat distribution at older ages is strongly associated with adult weight gain in men as well as in women (Seidell et al. 1986a).

4.2.3. Conclusion

It can be concluded that although the desirable weight seems to increase with age, this increment is less clear for the desirable weight range. Since the age-specific risk curves may represent distinct risks and may be biased by a cohort effect, they are difficult to interpret. Longitudinal and anthropometric studies support the contention that adult weight gain may increase health risks, especially in men.

4.3. Reversibility of adult weight gain

Since adult weight gain seems to increase health risk the question arises whether this weight gain can be prevented or reversed. Many treatment studies have shown that obesity, once developed, is difficult if not impossible to cure (Stunkard and McLaren-Hume 1959, Jeffery et al. 1978, Stunkard and Penick 1979, Wing and Jeffery 1979). These studies have generally been conducted on highly selective severely obese subjects. The results of treatment within a less extreme sample may be more encouraging: this is suggested by retrospective studies (Schachter 1982, O'Clarey and Nelson 1985, Jeffery et al. 1984). In these studies a high proportion of normal-weight subjects (50-72%) responded that they had once been overweight as adults. Obviously, their assessment of their former weight status could not be checked.

In the prospective Tecumseh Community Health Survey (Garn et al. 1980, 1984) the initial level of one skinfold was found to be negatively related to

subsequent changes in another skinfold, during 10 years and 20 years of follow-up. Though this association might at least partly be caused by a spurious regression to the mean effect due to biological fluctuation, the stronger association after 20 years than after 10 years of follow-up suggests that overweight subjects might indeed lose weight after many years of follow-up. This is encouraging whether or not the weight loss is caused by conscious attempts to lose weight. However, if this weight loss were also present in the studies on which the U-shaped relationship between obesity and mortality is based, this would imply either that being overweight has long-term detrimental effects on health, independent of future weight development, or that the detrimental effects of being and staying overweight are underestimated in these risk curves. This latter possibility is more likely, because the health and life expectancy of subjects who have lost their overweight have been found to improve (Hovell 1980, Dublin 1953).

4.4. Causes of adult weight gain

4.4.1 General

Like all aspects of growth and development, the growth and development of the body mass index depends on both genetic and environmental factors. The impact of these factors on the development of the body mass index in young adults living in industrialized countries is described in the following sections.

4.4.2. Genetic factors

Many studies have indicated that obese parents are likely to beget children who will become obese adults (Mayer 1965). To study the genetic and environmental determinants of obesity, specific samples such as twin or adoptee groups, are necessary. Whereas most studies concentrate on childhood and adolescence (Hartz et al. 1977, Annest et al. 1983, Fischbein 1977), some studies have been conducted on adults.

A follow-up study on 1028 white male veteran twins showed that although the impact of genetic factors on obesity decreased during follow-up from age 15-28 onward, this impact was still present. The intraclass correlation coefficient of the change in body mass index from 25 to 48 years of age was 0.6 for monozygotic pairs and 0.3 for dizygotic pairs (Fabsitz et al. 1980). This demonstrates an important genetic impact on change in body mass index even after maturation. It might be possible that monozygotic twins share environments more closely than dizygotic twins, but an extensive adoptee study

also showed that genetic factors are important determinants of adult changes in body mass index (Stunkard et al. 1986). Of the 540 adoptees who were studied, 55% had been transferred to the adoptive homes within the first month, and 90% within the first year of life. It was found that the weight class of adoptees when adult (mean age 42.2, SD: 8.1) correlated strongly with the body mass index of their biological parents, whereas there was no relation with the body mass index of their adoptive parents. So, a strong genetic component in the development of adult fatness seems to be clear.

4.4.3. Environmental factors

As indicated above, a strong genetic component does not mean that adult fatness is independent of environment or behavior. In the Framingham study (Heller et al. 1984), the body mass index of the parental generation, aged 30–62 years, was compared with that of the offspring, aged 20–49 years. It was found that the age-adjusted correlation between first-degree relatives was 0.2–0.3, whether it concerned two generations or only one generation. The correlations for second degree relatives were 0.05–0.14 and the correlation for spouses was 0.19 for the parental generation and 0.12 for the offspring generation. So, the correlation for the spouses (who were genetically unrelated) was comparable or higher than the correlation for second degree relatives.

Evidence that living together or general environmental time trends have their impact on the development of body fatness was presented in the Tecumseh Community Health Survey (Garn et al. 1979a, 1979b). During a nine-year period a clear association between parents and children was found in changes in fatness (Garn et al. 1979a). In addition, the correlations of changes between spouse pairs in triceps and subscapular skinfolds between spouse pairs were 0.32 and 0.25 respectively, over a follow-up period of 10 years. So, husbands and wives generally fluctuated in fatness together over the same period of time (Garn et al. 1979b).

Other indications that environmental/behavioral factors have their impact on adult body fatness are:

- Obesity is markedly more prevalent in white inhabitants of the United States than in white inhabitants of Australia or Britain (Bray 1985b), but this does not seem to be attributable to a difference in genotype. If subjects emigrate to a country with a high prevalence of obesity, they become more obese (Eveleth and Tanner 1976).
- The level of fatness is related to the socio-economic status of the subject

(Goldblatt et al. 1965, Silverstone et al. 1969, Garn et al 1977, Baecke et al. 1983). If a subject changes socio-economic status, his/her fatness also changes (Goldblatt et al. 1965).

- Secular trends in obesity (Feinleib 1985, Sonne-Holm and Sørensen 1977, Deurenberg et al. 1986) reflect changes in prosperity and related behavioral changes in diet and activity, rather than genetic effects.

So, although it is difficult to estimate the relative importance of genetic versus environmental/behavioral factors, both factors are pertinent to the development of adult obesity. Environmental/behavioral factors seem to determine the extent to which genetic predispositions are expressed.

To understand the etiology of overweight it is essential to characterize these factors and their working-mechanisms. A model for the etiology of obesity in adults has been developed by Baecke (1982). It is clear that an increase in energy intake and/or a decrease in energy expenditure, of which physical activity (Tuomilehto et al. 1985) is obviously only one aspect, may result in weight gain. Less clear, however, is under what daily circumstances these changes occur.

Many case reports are known in which obesity developed following the experience of life events, such as breakdown of a marriage, bereavements, etc. (Kaplan and Kaplan 1957). These life events may have diverse effects on the energy balance; e.g. a new demanding job may have an impact on the time available for meals and sport. In addition, the job may bring about a state of arousal that may have its own less apparent effect on eating behavior and energy balance in general.

Animal studies have shown that stressors like tail-pinching, noise or electric shock induce animals to eat (Kupfermann 1964, Rowland and Antelman 1976, Robbins et al. 1981). In animals made aphagic by lesions of the lateral hypothalamus, it has been shown that environmental stimuli can temporarily reverse the aphagia (Robbins et al. 1981).

In humans, increased food intake as a response to stressors has also been described (Rodin 1978, Slochower 1983). Slochower (1983) manipulated the recognizability of the stressor and found that obese subjects ate more as a response to diffuse arousal conditions than in response to labeled arousal conditions. In addition to their specific informational properties, the stressors may have non-specific properties that cause a state of arousal. If the informational properties of these stressors are not easy to discriminate, the arousal they cause may be channelled into different forms of behavior,

depending on the external cues that are present (Rodin 1978, Slochower 1983, Robbins et al. 1981). In addition to the presence of stressers, the absence of stressors may also be a diffuse arousal condition (Gersten et al. 1974), which may result in an increased food intake (Abramson and Stinson 1977).

Another mechanism that might be involved in the relation between stressor and energy balance is the breakdown of the cognitive restraint of dieters; as Lipinski (1978) and Wechsler et al. (1978) have shown, in initially successful dieters, weight loss was maintained worst in subjects who experienced more life events during follow-up.

In conclusion, arousal may be one of the intermediate factors between environmental factors and changes in energy balance.

5. CONCLUSION

The risk to health posed by obesity seems to be larger in younger subjects than in older subjects. Though this might suggest that moderate weight gain after the termination of growth may not increase the risk to health, this contention is not supported by other observations. Overweight acquired at adult years may even carry a greater toll than does lifelong overweight of moderate severity. Because studies on the effect of dieting show that overweight is difficult, if not impossible, to cure, it may be better prevented. In this context it is essential to gain more insight into the etiology of overweight.

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2 DESIGN OF THE MIXED-LONGITUDINAL STUDY

A mixed-longitudinal study is based on the following general strategy (Kowalski and Prahl-Anderson 1980): i samples of subjects, a_1, a_2,...., a_i years of age at the start of the study, respectively, are observed for y years. The samples are chosen in such a way that the first sample at the end of the follow-up study is older than the second sample at the beginning of the follow-up study; $a_2 < a_1+y$,...., $a_i < a_{i-1}+y$. Thus, overlapping age intervals arise. The information from all samples may be combined to obtain data over the entire age range a_1, a_i+y. By doing this, both the within-subject changes (longitudinal data) and the between-subject differences (cross-sectional data) are used. Based on these two aspects the study is designated 'mixed-longitudinal'. In this way, a relatively wide age range can be studied in a relatively short time span, which makes the study more efficient.

Descriptive analysis of a mixed-longitudinal study

The magnitude of a parameter in a mixed-longitudinal study depends on three independent effects: differences associated with age (A = age effect), differences associated with time of measurement (P = period effect) and differences associated with time of birth (C = cohort effect). These effects will be illustrated with the weight data from the five-year follow-up Normative Aging Study on 2000 white male veterans born in 1885-1943 (Friedlaender et al. 1977).

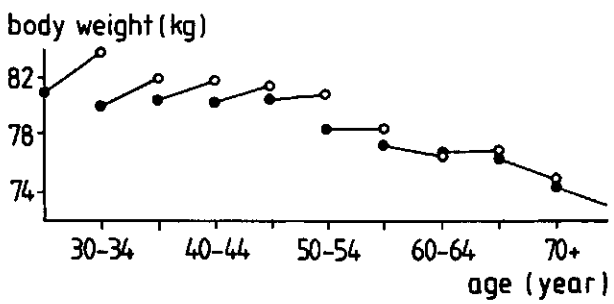

Figure 1: Five year mean longitudinal changes of body weight with age in men (Friedlaender et al. 1977)

The age effect is due to maturation and development. These latter factors, thus, are the prime objects of inference in studies that aim to estimate age-reference curves. The weight change during the five year follow-up period (Figure 1) renders the age effect in the Normative Aging Study, though confounding by a period effect may be present. The decreasing slopes with increasing age represent diminishing age effects, but these slopes may also be confounded by cohort effects (Schaie 1965); over the same age range, different cohorts may have different slopes. This indicates that the 'pure' age effect can only be estimated cohort-specifically in the absence of a period effect.

The cohort effect is caused by long-term environmental conditions, which may even occur before birth. The difference between weights of different cohorts measured at the same age renders the cohort effect (though confounding by a period effect or by random sampling may be present). Figure 1 shows that at the same age American men that are born later have a higher body mass index. Departures due to sampling procedures cannot be distinguished from the cohort effect.

The period effect is caused by temporary environmental conditions. These effects might be caused, for instance, by a calibrating error of the balances, seasonal fluctuations and in a special case of temporary environmental conditions, by a spurious intervention effect caused by the repeated measurements in the study itself (i.e. obese subjects may be motivated to lose weight because of the repeated weight measurements). This last period effect can only be detected by means of a control group.

The weight differences measured in a cross-sectional study are interpreted as age effects under the assumption of no cohort effect, while in a purely longitudinal study the weight changes within a cohort are interpreted as age effects, under the assumption of no period or cohort effect. Based on the data of Figure 1, a cross-sectional study at the first examination (solid points) would have concluded that weight is relatively stable through young adulthood and decreases above 50 years of age, while a longitudinal study on the youngest cohort would have shown a sharp weight gain.

In a mixed-longitudinal study, the age-period-cohort analysis generally aims to estimate the three effects under less stringent assumptions than is true for cross-sectional or purely longitudinal studies. However, though intrinsically the three effects are independent, this is not so within the data, since the year of birth plus the age is the time of measurement (Baltes 1968, Van 't Hof et al. 1977). This necessitates assumptions also being made in a mixed-longitudinal study, to make the effects identifiable. Since no clear-cut criterion

exists to decide which assumption is best, this has been designated 'the identifiability problem' (Kupper et al. 1985). As Kupper et al. have argued, the comparison of the fitted curves with the traditionally graphical representation of the data, may be the best evaluation criterion of the imposed assumption. Even if the same assumptions are made as generally apply to cross-sectional or longitudinal studies, the advantage of the mixed-longitudinal study (in addition to its efficiency) is that it explicitly requires the investigator to recognize the assumptions that are made.

Etiologic analysis of a mixed-longitudinal study

To study the effect of a determinant on the change over time of a dependent variable, the mixed-longitudinal study is generally analyzed as a cross-sectional or a purely longitudinal study, with the extension to more than one cohort.

The advantage of a cross-sectional analysis is clear: the determinants have to be measured at one examination only. A cross-sectional analysis is sufficient for the study of permanent, unvarying, determinants (gender, socio-economic status). In other words, the estimation of the effects of these determinants based on differences within or between subjects will be the same. This is true only if the effect of the determinant is not biased by a cohort effect; but in the study of an association between two variables this is generally ignored. So, with this limitation, the effect of the determinant will be validly estimated cross-sectionally, whether the effect is constant over age (Figure 2a) or modified by age (Figure 2b).

The effect of non-permanent determinants cannot validly be estimated in cross-sectional studies, since at the same age such a determinant may have fully affected one subject, but may just have started to take effect in the other (Figure 2c,2d). So, a valid estimate of the effect can only be derived from a longitudinal study. A cross-sectional analysis of these determinants may be useful, though, in deciding which determinants deserve further consideration in the longitudinal part of the study. This is another advantage of the mixed-longitudinal study.

Present mixed-longitudinal study

In spring 1980, all the inhabitants of the county of Ede who were 19-21, 24-26 and 29-31 years of age in January 1980, were invited by mail to participate in the study. Their addresses were obtained from the Civil Registration Office in Ede. The respondents were split into two subgroups, the

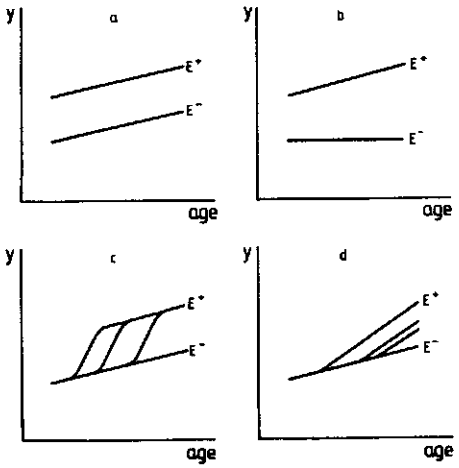

Figure 2: The effect of permanent (a,b) or non-permanent (c,d) determinants

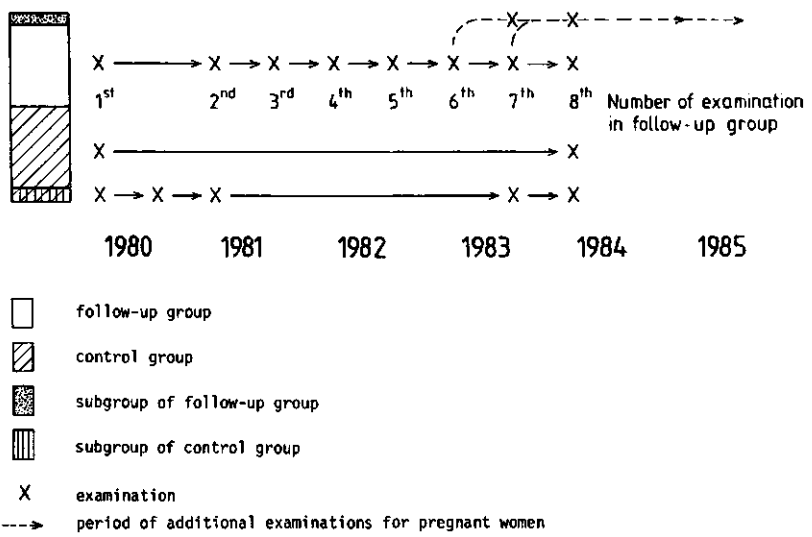

Figure 3: Design of the mixed-longitudinal study

follow-up group and the control group. From spring 1981 through spring 1984, the follow-up group was examined every six months. The control group was examined in spring 1984 only (Figure 3). At each examination, body weight was measured and a questionnaire was completed.

In subsamples of the follow-up group and the control group, additional examinations were conducted. All women who were pregnant at the 6th and 7th examination participated in a study on the effect of pregnancy on the development of the body mass index. A subsample of the control group participated in three additional examinations for a detailed study on anthropometry, physical activity and food consumption.

Scheme 1 gives an overview of the studies conducted within the mixed-longitudinal study.

Outline of the thesis

In Chapter 3 the change in body mass index during the four years of follow-up is compared between the follow-up group and the control group to test whether a spurious intervention effect is present. For the follow-up group the development of the body mass index from 19 to 35 years of age is described, based on an age-period-cohort analysis.

In Chapter 4 several aspects of the development of the body mass index are considered more specifically, exploiting the advantages of a longitudinal design. Since there are indications that subjects initially overweight lose more body mass than subjects who are not initially overweight, body mass changes and fluctuations within subjects are analyzed. The age-specific incidence of overweight is assessed.

With respect to a possible intervention in the development of the body mass index, described in Chapter 3 it is important to estimate the effect of attempts to lose weight by dieting. This is described in Chapter 5. The effect on the body mass index of dieting on medical advice or on one's own initiative is assessed, as well as the effect of repeated attempts to lose weight. Determinants of successful dieting behavior are characterized.

To investigate the impact of environmental factors on the development of the body mass index the effect of life events was estimated. This follow-up study

Scheme 1: Overview of the studies conducted within the mixed-longitudinal study

Subject	Body fatness	Behavior	Personal characteristics	Environment	Examination	Reference
1. Socio-demographic determinants of the BMI[1]	BMI (Dep)[2]		age family of origin birth order	SES[3] parity marital status religion urbanization occupation father	1	Baecke et al. 1983a
2. Life-style determinants of the BMI	BMI (Dep)	slimming CASS[4] smoking eating: sweets/snacks health consciousness physical activity			1	Baecke et al. 1983b
3. Validation of a short questionnaire on physical activity	LBM[5] (Dep) body fatness	physical activity during: work sports leisure time	height		detailed study fall 1980	Baecke et al. 1982a
4. Food consumption and physical activity in relation to body fatness	body fatness (Dep)	food consumption food groups meals energy physical activity during: work sports leisure time	lean body mass		detailed study spring 1981	Baecke et al. 1983c
5. Construction of an index of relative weight, adjusted for height and frame	body fatness (Dep)		frame size		detailed study fall 1980	Baecke et al. 1982b
6. Evaluation of frame categories in weight-height tables	body fatness (Dep)		frame size		detailed study fall 1983	this thesis: chapter 8
7. Relation between BMI and self-reported health	BMI (E)[6]		health status (Dep)		8	Seidell et al. 1986
8. Relation between the BMI and eating behavior in a longitudinal versus cross-sectional study	BMI (Dep)	eating behavior (E)			4 and 7	Van Strien et al. 1985
9. Life events as modifiers of the relation beween change in BMI and emotional eating	DBMI[7](Dep)	emotional eating		life events	4 and 7	Van Strien et al. 1986a
10. Age-reference curves of the BMI in young adults	DBMI (Dep)		age		1 - 8	this thesis: chapter 3
11. Interrelationships of level, change and fluctuation	DBMI (Dep) $BMI_{initial}$				1 - 8	this thesis: chapter 4

Scheme 1 continued

Subject	Body fatness	Behavior	Personal characteristics	Environment	Examination	Reference
12. Effect of dieting on changes of the BMI	DBMI (Dep)	dieting (E) physical activity during: work sports leisure time smoking	age	SES season life events	2 - 8	this thesis: chapter 5
13. Effect of life events on changes of the BMI	DBMI (Dep)	physical activity during: work sports leisure time emotional eating smoking	age	life events (E) SES	2 - 8	this thesis: chapter 6
14. Effect of pregnancy on changes of the BMI	DBMI (Dep)	breast-feeding smoking	pregnancy (E) age	SES season employment	2 - 8 and detailed study 1983-1985	this thesis: chapter 7
15. Construction of the Dutch Eating Behavior Questionnaire (DEBQ)		eating behavior				Van Strien et al. 1986b
16. Relation between eating behavior and sex-role orientation		eating behavior (Dep)	androgyny (E)		7 and 8	Van Strien et al. 1986c

[1] BMI : body mass index
[2] Dep : dependent variable
[3] SES : Socio-economic status
[4] CASS : coffee-drinking - alcohol consumption - smoking - sleeping behavior
[5] LBM : lean body mass
[6] E : exposure
[7] DBMI : change in body mass index

is described in Chapter 6. The change in body mass in subjects experiencing few or many life events was compared with the change in body mass in subjects with a moderate number of life events. As is done for dieting behavior, subgroups with different responses are traced.

Cross-sectional analysis of the first examination (Baecke et al. 1983a) indicated that women with two or more children had a larger body mass index than women with one child or without children. Pregnancy as a potential determinant of overweight in women is examined in a detailed study, described in Chapter 7. The women participated in the follow-up study and were followed one year postpartum. Their change in body mass is compared with that of women who were not pregnant during the four-year follow-up study.

Frame categories in weight-height tables have hardly ever been evaluated, except qualitatively. In Chapter 8 a detailed study on the impact of these frame categories is described. Six frame diameters were examined to improve the estimation of body fatness by the body mass index.

The ninth and last chapter comprises a general discussion of the studies presented in this thesis.

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3 THE DEVELOPMENT OF THE BODY MASS INDEX IN YOUNG ADULTS, I: AGE-REFERENCE CURVES BASED ON A FOUR-YEAR MIXED-LONGITUDINAL STUDY

by Maartje A. Rookus, Jan Burema, Martin A. van 't Hof, Paul Deurenberg, Wilhelmina A.M. van der Wiel-Wetzels, Joseph G.A.J. Hautvast

ABSTRACT

The development of the body mass index (BMI, weight/height^2) during young adulthood was studied in 518 men and 624 women, initially 19-21, 24-26, 29-31 years of age, in a four-year mixed-longitudinal study in the Netherlands. Body height and body weight were measured in spring 1980. Subsequently, body weight was measured every six months from spring 1981 through spring 1984.

The BMI continued to increase after the termination of growth. From 19 to 35 years of age the median BMI increased from 22.1 kg/m^2 to 24.4 kg/m^2 in men and from 21.1 kg/m^2 to 23.0 kg/m^2 in women. All percentiles showed a comparable increment, resulting in an age-independent variation of the BMI.

Since the NHANES I curves diverge with age, it is suggested that American and Dutch samples not only differ in the prevalence of overweight, but also in the incidence of overweight during young adulthood.

INTRODUCTION

It is well known that in industrialized countries relative body weight increases with age until middle age, then remains relatively constant, and decreases in old ages. This relationship has been shown in longitudinal studies (Forbes and Reina 1970; Hsu et al. 1977; Kannel et al. 1979; Noppa et al. 1980; Cronk et al. 1982; Borkan et al. 1983; Khoury et al. 1983; Stark et al. 1981; Waaler 1984; Lindquist 1982), and, less clearly, in cross-sectional studies (Montoye et al. 1965; Khosla and Lowe 1968; Bjelke 1971; Cronk and Roche 1982; Rosenbaum et al. 1985; Van Sonsbeek 1985). The increment in relative body weight is generally due to an increment in body fatness, because the lean body mass tends to decrease with age (Forbes and Reina 1970; Borkan et al. 1983). This increment in body fatness may result in overweight.

The prevalence of overweight differs between the industrialized countries. If the body mass index (weight/height^2) is used as a measure of fatness, the prevalence of overweight (body mass index > 25 kg/m^2) in representative samples

of adult populations is, for men and women respectively 43% and 36% (20–74 years of age) in the USA (Bray 1985), 41% and 31% (25–64 years of age) in Australia (Bray 1985), 39% and 32% (16–64 years of age) in Britain (Rosenbaum et al. 1985) and 38% and 30% (more than 20 years of age) in the Netherlands (Van Sonsbeek 1985).

Of these countries, overweight is most prevalent in the USA. The lower prevalence in the other countries may not only be the result of a difference in growth but also of a difference in the development of weight during adulthood. Young adulthood seems to be a particularly critical period for the development of adult overweight, as is suggested by self-reported weight histories of selective, mainly female, samples (Hartz and Rimm 1980; Bradley 1985) and, at least for men, by the distribution of the body mass index for selected age groups of a random sample of the Norwegian population (Waaler 1984). So far, body weight development in young adults has mainly been studied in the USA.

The aim of the present study was to describe the development of the body mass index in young Dutch adults, living in a county which comprises urban and rural areas.

METHODS

In the county of Ede in the Netherlands, which comprises one town and six villages, a sample of young adults was studied at regular intervals between 1980 and 1984. The mixed-longitudinal design of the study is shown in Figure 1. In spring 1980, all residents of Dutch nationality in Ede, born in 1948–1950, 1953–1955 and 1958–1960 were invited by mail to participate in an extensive study. Though the response was only 33%, the body mass index of a sample from the group that did not cooperate in the first instance was found not to differ from that of the response group. This indicated that a selection bias was unlikely to be present (Baecke et al. 1983).

Participants in spring 1980 were followed for four years (1980–1984) in two groups, each with a similar composition with respect to age, sex and level of education. From 1981 onward one of these groups, the follow-up group, was approached twice a year in seven consecutive examinations. The other, the control group, was re-measured in spring 1984 only. To avoid bias from a seasonal effect on body weight, only the five examinations in the spring have been used in this report.

Subjects were visited at home, except in spring 1980 when they were asked to visit a mobile research unit. Body height without shoes was measured by means of a Microtoise to the nearest 0.1 cm in spring 1980 only. At each examination

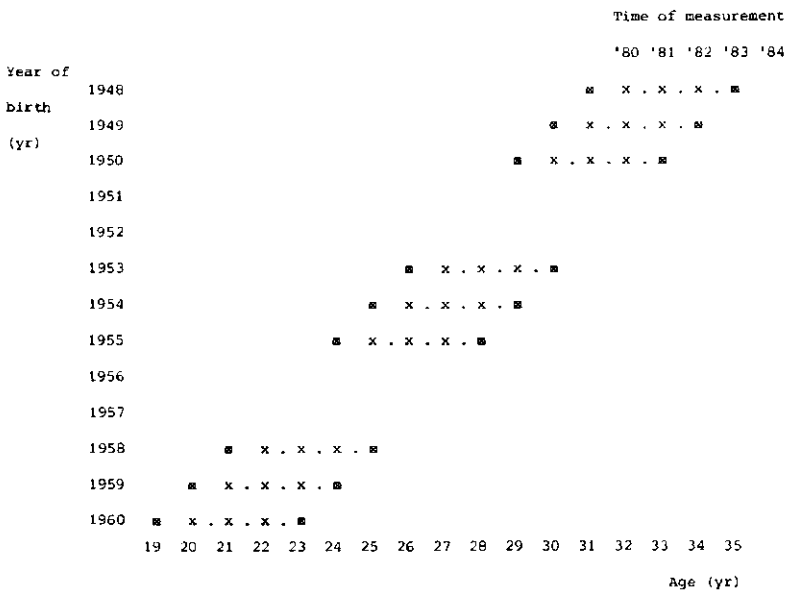

Figure 1. Design of the mixed-longitudinal study

body weight in light indoor clothing and without shoes was measured to the nearest 0.5 kg, between 4 p.m. and 9.30 p.m., on a Seca-scale that was calibrated weekly. From spring 1983 onward, each scale was fixed on a plywood base, because the own base of the scale was not rigid. In this way the random error of the weight measurements was reduced.

Subjects that moved out of the county during the follow-up period reported their body weight on a questionnaire that was sent and returned by mail. These data were excluded from the study, however, because of the findings of an evaluation of the validity of the self-reported body weights. For this evaluation, the body weights of 97 subjects who had moved out of the county were measured on a calibrated scale within one month after they had returned the questionnaire with their reported body weights. The subjects had not been warned that they would be visited and weighted. It was found that the reported body weights were, on average, 1.9 kg (S.D.: 1.6, n=97) lower than the values, we

subsequently measured on our visit. Though 57% of this discrepancy was attributable to a calibrating error of the homescales, the magnitude of the underreporting was positively related to the level of the actual body weight, as other authors have also found (Stewart 1982). Though this bias in reported body weights may be no serious problem in cross-sectional studies, the discrepancy between the two methods of measurement rendered any comparison of data within our subjects spurious.

As a measure of body fatness the body mass index was used since it is widely accepted for epidemiologic studies (Garrow 1981; Bray 1985; NIH-Consensus Conference 1985) and has been shown not to be improved by the inclusion of body diameter measurements (Chapter 8). The calculation of the body mass index was based on the body height measurement of spring 1980 only, since body height is relatively constant during the age-range that was considered (Roche and Davila 1972). This means that the change in the body mass index in the present study was entirely attributable to a change in body weight.

Sample

Of the 1670 subjects who initially comprised the follow-up group, 323 did not respond at all examinations, 187 moved away from the county and 132 women were more than 3 months pregnant at one of the examinations in the spring. These subjects were only included in the sample if their missing weights could be estimated as the mean of two valid measurements from adjacent years; this was possible for 114 subjects. Thus, complete follow-up was available for 518 men and 624 women (= 1142 subjects or 69%). In the control group valid measurements in spring 1980 and spring 1984 were obtained for 811 men and 885 women (=1696 subjects, or 64%).

Analysis

The subjects were classified into nine cohorts, on the basis of their year of birth. The aim of the analysis was to integrate the data collected over four years from the cohorts, so that the development of the body mass index from 19 to 35 years of age, adjusted for possible generational or sampling effects, could be described. Age was recorded as the age at the beginning of the year in which the examination took place. The body mass index (BMI) was transformed into ln(BMI-12) for both sexes. By substracting 12 from the body mass index the range of the index was shifted to an area for which the logarithmic transformation was more powerful. After transformation the symmetry of the normal distribution was approached sufficiently (Table 1).

Table 1. Number of age groups (19, 20, 21, ..., 35 years of age) within the follow-up group, classified by the skewness of the body mass index (BMI) and of two transformations of the body mass index

Range of skewness	Men (n=518)			Women (n=624)		
	BMI	ln(BMI)	ln(BMI-12)	BMI	ln(BMI)	ln(BMI-12)
-0.6 - -0.2	-	-	4	-	-	1
-0.2 - +0.2	1	5	9	-	-	9
+0.2 - +0.6	4	8	4	-	8	7
+0.6 - +1.0	6	4	-	8	8	-
+1.0 - +1.4	-	-	-	4	1	-
+1.4 - +1.8	3	-	-	5	-	-
+1.8 - +2.2	3	-	-	-	-	-

The distribution of the transformed body mass index could be described solely by the mean and the variance, where the mean in the transformed scale corresponded with the median (P_{50}) in the original scale. A model was fitted for each of these two parameters, using data that had been aggregated for each of the 45 combinations of ages at the five examinations in the spring and the nine cohorts (Figure 1). One model was intended to estimate the average curve of development, representing the 50th percentile (Van 't Hof, et al. 1977), the other model was to estimate the variance around the 50th percentile (Van 't Hof and Kowalski 1977). Our application of both models assumed no time of measurement effect since the strong time of measurement effect due to the season was eliminated by using the measurements in the spring only. In each model 25 effect parameters were estimated: a constant representing the level of the reference group, 17-1=16 age effects and 9-1=8 cohort effects. This full number of age and cohort effects was chosen, because the former were the parameters of interest and thus had to be estimated as well as possible, and the latter provided a nuisance parameter to account for the specific sampling dependent level of each cohort. The two models were:

$$\ln(\text{BMI}-12) = a + b_i + c_j + \text{residual}_{ij} \quad (1)$$

$$s^2(\ln(\text{BMI}-12)) = d + e_i + f_j + \text{residual}_{ij} \quad (2)$$

where, $s^2(\ln(\text{BMI}-12))$ is the variance of the transformed body mass index, a and d are constants, b_i and e_i are the age effects for i= 19, 20,.....35, and c_j and f_j are the cohort effects for j= 1, 2, ...9. As a side condition b_{19}, c_1, e_{19}, and f_1 were set to zero, so as to render the model parameters identifiable. The

residuals of each model are mainly due to error of measurement and biological fluctuation.
The residuals correlate, because the measurement of the cohorts (j) were repeated. Thus, the models were fitted by the Generalized Least Squares Method (see Appendix). The pertinent covariances were assessed empirically by estimating them for each of the nine cohorts separately, according to Van 't Hof et al. (1977).

The cohort effects showed no indication of a systematic generational effect and therefore the following procedure was pursued: the weighted means of the estimated cohort effects were calculated (a' for equation 1 and d' for equation 2): each cohort was weighted with the number of subjects in that cohort. Subsequently, the specific percentiles were computed as follows:

$$BMI_p = \exp(a' + \Sigma b_i + Z_p(d' + \Sigma e_i)^{1/2}) + 12 \qquad (3)$$

where Z_p was obtained from the standard normal distribution (e.g. for the 25th and 75th percentiles, the values of Z_p were -0.67 and +0.67, respectively). To evaluate the final fit, the distribution of the crude data was compared with the expected distribution, based on equation 3.

RESULTS

To study whether the body mass index of the follow-up group was biased because of loss of follow-up, the initial body mass indices of three groups: the group for which data were complete, the group with missing data and the control group, were compared per age class. There was no clear indication of any bias in either sex (Table 2). Furthermore, the comparison of the changes in body mass index of the follow-up group versus the control group showed that within age classes a study-generated effect was also very unlikely. In the follow-up group the mean yearly increase in body mass index was 0.16 kg/m^2/year in men and 0.13 kg/m^2/year in women.

To compute the standard curves of the development of the body mass index at age 19 through 35, the mixed-longitudinal design of the study was utilized. No systematic generational trend in the cohort-parameters was found (Table 3), and therefore these effect parameters could be averaged. So, we expressed the body mass index as a function of age alone after adjustment for the cohort effects. In Figure 2 the development of the body mass index is graphically represented by means of the median (Table 3) and six other percentiles.

Table 2: Evaluation of potential selection or study-generated effects on the body mass index (BMI, weight/height^2) within the four-year mixed-longitudinal study

		Year of birth	Follow-up group			Group lost to follow-up			Control group			p^+
			n	mean	SEM	n	mean	SEM	n	mean	SEM	
MEN	Initial BMI* (kg/m^2)	'58–'60	130	22.6	0.2	112	22.2	0.2	460	22.3	0.1	0.92
		'53–'55	167	23.5	0.2	84	23.6	0.3	361	23.9	0.2	
		'48–'50	221	24.2	0.2	41	24.4	0.4	342	24.1	0.1	
	Change in BMI (kg/m^2/4 yr)	'58–'60	130	0.7	0.1				282	0.9	0.1	0.77
		'53–'55	167	0.6	0.1				252	0.5	0.1	
		'48–'50	221	0.5	0.1				277	0.5	0.1	
WOMEN	Initial BMI (kg/m^2)	'58–'60	150	21.8	0.2	119	21.7	0.2	527	21.7	0.1	0.66
		'53–'55	226	22.1	0.2	91	21.7	0.3	403	22.4	0.1	
		'48–'50	248	22.7	0.2	54	23.2	0.4	480	22.7	0.1	
	Change in BMI (kg/m^2/4 yr)	'58–'60	150	0.6	0.1				252	0.4	0.1	0.48
		'53–'55	226	0.4	0.1				259	0.7	0.1	
		'48–'50	248	0.5	0.1				374	0.5	0.1	

+ Test of main effect of group (follow-up, lost to follow-up and control group) in a two-way analysis of variance.
* As measured at the start of the study in spring 1980.

Table 3: Generalized least squares solution of the transformed body mass index (kg/m^2) (ln(BMI-12)) on age, adjusted for cohort effects

Variable		Men (n=518)			Women (n=624)		
		Intercept	b	se(b)	Intercept	b	se(b)
Age(yr)	19	2.361			2.294		
	20		0.029	0.010		0.011	0.016
	21		0.027	0.013		0.019	0.016
	22		0.062	0.014		0.041	0.016
	23		0.065	0.013		0.059	0.017
	24		0.099	0.016		0.084	0.019
	25		0.109	0.017		0.084	0.020
	26		0.117	0.018		0.106	0.021
	27		0.131	0.018		0.107	0.021
	28		0.157	0.019		0.116	0.022
	29		0.163	0.019		0.140	0.022
	30		0.164	0.020		0.146	0.023
	31		0.173	0.020		0.150	0.024
	32		0.188	0.020		0.173	0.024
	33		0.205	0.021		0.174	0.024
	34		0.205	0.021		0.191	0.024
	35		0.205	0.022		0.196	0.025
Year of birth	'59		-0.049	0.049		-0.096	0.044
	'58		-0.123	0.050		-0.034	0.044
	'55		-0.062	0.049		-0.085	0.047
	'54		-0.039	0.050		-0.144	0.045
	'53		-0.029	0.044		-0.089	0.046
	'50		-0.084	0.047		-0.113	0.046
	'49		-0.006	0.048		-0.111	0.046
	'48		-0.070	0.046		-0.081	0.044

Our results clearly show that the body mass index continued to increase after the termination of growth. Between 19 and 35 years of age the median increased from 22.1 kg/m^2 to 24.4 kg/m^2 in men and from 21.1 kg/m^2 to 23.0 kg/m^2 in women. In both sexes the total variation of the body mass index remained relatively constant within the age range that was being considered; this can be seen from the similar slopes of the percentiles. To check the validity of the assumption of no time of measurement effect the fit of the two models was evaluated simultaneously by comparing the crude distribution of the body mass index of all subjects with the distribution expected from equation 3 (Figure 2). From Table 4 can be seen that in general the fit was acceptable, though the fit was poorest in the extremes of the distribution.

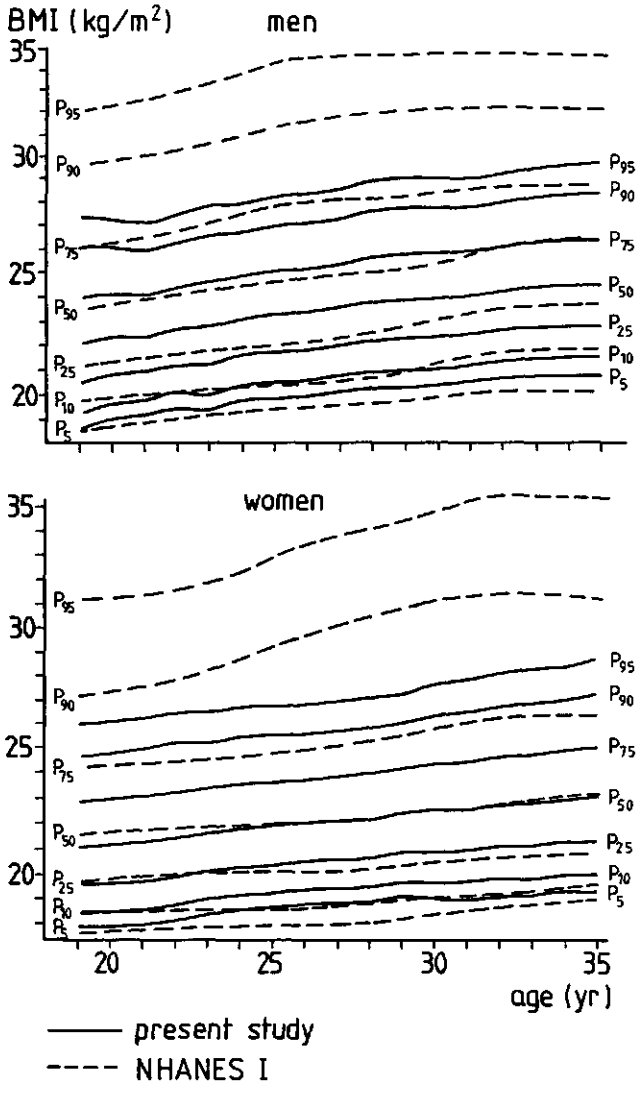

Figure 2: Age-reference curves of the body mass index of young adults based on the present Dutch sample and the National Health and Nutritional Examination Survey I (NHANES I) (Cronk and Roche 1982)

Table 4: Observed and expected distribution of the body mass index

Range of percentiles	Distribution of the body mass index				
		Men (n=518)		Women (n=624)	
	Expected (%)	Observed (%)	Difference (%)	Observed (%)	Difference (%)
$> P_{95}$	5	5.6	+0.6	6.5	+1.5
$P_{90} - P_{95}$	5	4.3	-0.7	3.7	-1.3
$P_{75} - P_{90}$	15	15.4	+0.4	13.7	-1.3
$P_{50} - P_{75}$	25	24.6	-0.4	24.0	-1.0
$P_{25} - P_{50}$	25	24.9	-0.1	26.3	+1.3
$P_{10} - P_{25}$	15	14.4	-0.6	15.8	+0.8
$P_{5} - P_{10}$	5	4.7	-0.3	4.6	-0.4
$< P_{5}$	5	6.0	+1.0	5.3	+0.3

DISCUSSION

It was shown in the present study that the body mass index of the Dutch young adults examined, continued to increase after the termination of growth. Body weight gain during adulthood is a cause of concern, because it is related to atherogenic traits, even more than the level of the body mass index (Shapiro et al. 1969; Ashley and Kannel 1974; Hubert et al. 1983). For various reasons it is especially important to prevent overweight during young adulthood. First, in young ages overweight is associated with a higher risk of morbidity and mortality than in older ages (Hubert et al. 1983; Lew and Garfinkel 1979; Rabkin et al. 1977). This might be related to the redistribution of body fat that seems to occur in young adulthood (Mueller 1982). Secondly, morbidity and mortality from cardiovascular disease increase with age in overweight subjects (Hubert et al. 1983; Rabkin et al. 1977). And thirdly, the increment of body weight in young adults seem to explain a significant proportion of the variance in the body fatness in later adulthood (Hartz and Rimm 1980).

Since weight for height of young Dutch adults is closest to the mean of several European samples (Eveleth and Tanner 1976), the development of the body mass index in young Dutch adults is of special interest. Little usable longitudinal data are available from European samples, so in Table 5 the present study is compared with longitudinal studies on (selective) samples of American young adults. The comparison can only be made, however, under two assumptions. First, in these studies the trend in weight gain or gain in body mass index is assumed to be linear, which enables the annual gain to be computed. The longer follow-up

Table 5: The comparison of the present Dutch mixed-longitudinal study with American longitudinal studies on the development of body weight or the body mass index (BMI) in young adults

Sex	Study	Reference	n	Initial age	Period of follow-up	Estimated yearly gain in Weight mean	Estimated yearly gain in BMI mean
				(yr)	(yr)	(kg/yr)	(kg/m^2/yr)
Men	Normative Aging Study	Friedlaender et al. 1977	86	30	5	0.6	
	Six American Growth Studies	Cronk et al. 1982	146	18	12	0.8	
	Minnesota Student Health Study	Gillum et al. 1982	112	20	20	0.6	
	Princeton School Study+	Khoury et al. 1983	36	18	12-16	0.7	
	Manitoba Follow-up Study	Hsu et al. 1977	218	17-24	27		0.13
	Present Study*		518	19-31	4	0.4	0.16
Women	Six American Growth Studies	Cronk et al. 1982	185	18	12	0.2	
	Princeton School Study+	Khoury et al. 1983	30	18	12-16	0.4	
	Present Study*		624	19-31	4	0.3	0.13

+ Body weight at age 18 reported.
* Estimates based on linear trend in within-subject changes.

periods in the American studies, in combination with the smaller gain in body weight that is to be expected with increasing age (Friedlaender et al. 1977), may spuriously decrease the difference between the results of these studies and our study. The second assumption is that weight changes in these selective samples give an indication of weight changes in American subjects. Under these (severe) assumptions the data show that the weight gain tends to be slightly smaller in the Dutch men than in the American men and about the same in the Dutch women as in the American women.

In addition to the within-subject changes in body mass index, based on the four-year follow-up period, we described the development of the body mass index within the total age range 19 to 35 years by means of an age-period-cohort analysis (Figure 2). As Kupper et al. (1985) have clearly shown, the validity of the regression coefficients estimated in an age-period-cohort analysis, cannot be evaluated statistically. In case an age-period-cohort analysis is conducted, these authors recommend to do this in conjunction with traditional graphical reproductions of the data. From a comparison of Figure 2 with the crude cohort-specific graphs (not shown) and from Table 4 we concluded that the fit was acceptable, and that therefore the assumption that the time of measurement had no effect was acceptable.

A weakness of longitudinal studies is that the samples might be more selective than is generally true for cross-sectional studies. So, a comparison with cross-sectional studies is interesting. However, this is true only if an unequivocal interpretation can be given to any differences found: in addition to a selection bias, differences generally might result from a generation effect. Other Dutch studies (Van Wieringen 1972; Deurenberg 1986) have found no clear secular trend in weight for height or body mass index in the cohorts we studied. We also found no indication of a bias by secular trend or, more generally, by a generational effect. Thus, a comparison with a national Dutch cross-sectional study was felt to be admissable.

In the Netherlands, the Continuous Dutch Health Interview Survey (CDHIS 1981) was conducted on an apparently representative sample of the Dutch non-institutionalized civilian population (Van Sonsbeek 1983; Seidell et al. 1986). A comparison of the present curves (Figure 2) with their counterparts in the CDHIS study (which was based on 2856 subjects) shows that the slopes of all percentiles are comparable for both sexes (data not shown). The same is true for the level of all percentiles in men. In women, however, the values for the percentiles of the CDHIS study are consistently about 1 kg/m^2 lower. The reason for this discrepancy may be the different methods of measurement used (reported;

CDHIS study versus measured; present study). It is known that women tend to underestimate their body weights more than men (Stewart 1982).

We also compared our curves with cross-sectional studied representative samples of other populations under the same assumption of no generational effect. Using the results of a study done in Great Britain (Rosenbaum et al. 1985), we found that the gain in mean body mass index in the British sample was comparable with the increment in the median of our sample for men, but was slightly lower for women. Changes in means and medians may be compared between these two studies, because the standard deviations of the British and Dutch samples were relatively constant.

In Figure 2 our data are compared with those of the white subjects in the National Health and Nutritional Examination Survey I (NHANES I) (Cronk and Roche 1982). Only the 10th percentile in men and the median in women are similar in both studies. In the NHANES I study the smaller percentiles run at a slightly lower level, whereas the larger percentiles run at a markedly higher level. Though this difference in level may partly be caused by differences in methods of measurements (minimal clothed, NHANES versus lightly clothed, in present study), this is not an explanation for the discrepancy in slope, found especially in the more extreme curves. This results in an increment of the skewness of the distribution of the body mass index with age in the American sample, which is virtually absent in the Dutch sample, suggesting a difference in the incidence of severe overweight in young adulthood. This difference in development of overweight in young adults between the Dutch and American samples can be explained in several ways (Eveleth and Tanner 1976). It may be contributed to genetical or secular/generational factors. However, these factors are not likely to have such an impact within this limited age range. Another possibility is a difference in behavior (diet, physical activity) and attitudes (body image) (Kushi et al. 1985; Van Itallie 1986); a difference in the etiology of overweight between the two populations.

It can be concluded, first, that the slope of the present curves gives a good representation of the change in the body mass index in young Dutch adults. Secondly, in addition to a difference in prevalence of overweight between the American and Dutch young adults a difference in incidence of overweight during young adulthood seems to be present.

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APPENDIX

The 45 combinations of the 15 ages at the examinations in the spring and the nine cohorts together with the 25 effect parameters (a, b_i and c_j for model 1 and d, e_i and f_j for model 2) determine the design matrix X. In this 45 x 25 matrix the first column consists of ones and all other cells contain zero, or one if that particular combination of age plus cohort is present.

The vector y of means (or variances, for model 2) of the observations for each of the age - cohort combinations, together with its covariance matrix Σ, constitutes a sufficient statistic for estimating the 16 age and 9 cohort effects on the level of the transformed body mass index, or on its variance, respectively. The Ordinary Least Squares (OLS) solution for the vector b of effect parameters is:

$$\hat{b}_{OLS} = (X' X)^{-1} X' y , \qquad (4)$$

and its covariance matrix is to be estimated as:

$$\mathrm{cov}(\hat{b}_{OLS}) = (X' X)^{-1} X' \Sigma X (X' X)^{-1} \qquad (5)$$

When $\Sigma = s^2 I$, where I is the identity matrix, all observations have the same variance, s^2, and are uncorrelated. In this case, (4) is the best linear unbiased estimator of b, which implies that it has minimum variance. However, the data in our study are from repeated measurements, and thus

$$\Sigma = \begin{bmatrix} \Sigma_1 & \emptyset & \emptyset & . & . & . & \emptyset \\ \emptyset & \Sigma_2 & \emptyset & & & & \emptyset \\ \emptyset & \emptyset & \Sigma_3 & & & & \emptyset \\ . & & & . & & & . \\ . & & & & . & & . \\ . & & & & & . & . \\ \emptyset & \emptyset & \emptyset & . & . & . & \Sigma_9 \end{bmatrix} ,$$

where Σ_i are square submatrices of order 5, representing interdependence of data from the same cohort on each of 5 subsequential examinations,and where $\emptyset$ are 5x5 matrices with all cells zero. The General Least Squares (GLS) solution for the parameters is then:

$$\hat{b}_{GLS} = (X' \Sigma^{-1} X)^{-1} X' \Sigma^{-1} y , \qquad (6)$$

where Σ^{-1} is the inverse matrix of Σ, and its estimated covariance is:

$$\text{cov}(\hat{b}_{GLS}) = (X' \Sigma^{-1} X)^{-1} . \qquad (7)$$

In particular, the standard errors of the estimated effect parameters are the square root of the diagonal elements of expression (7), which are now, on average, smaller than those of expression (5).

The matrix Σ can be estimated from the data. When $\Sigma_j^{(1)}$ is the covariance matrix of the individual measurements (after transformation) for 5 successive years (times of measurement) for a specific cohort j, then the sample means have covariance matrix

$$\Sigma_j^{(m)} = n_j^{-1} \Sigma_j^{(1)} . \qquad (8)$$

where n_j is the number of subjects in cohort j. The covariance matrix of the sample variances is approximately equal to

$$\Sigma_j^{(v)} = 2n_j^{-1} \Sigma_j^{(2)} , \qquad (9)$$

where $\Sigma_j^{(2)}$ is the matrix obtained by squaring each element of $\Sigma_j^{(1)}$. For a proof of this result see e.g. Kendall and Stuart, (1967), vol II, Chapter 18, paragraph 18.30.

4 THE DEVELOPMENT OF THE BODY MASS INDEX IN YOUNG ADULTS, II: INTERRELATIONSHIPS OF LEVEL, CHANGE AND FLUCTUATION, A FOUR-YEAR LONGITUDINAL STUDY

by Maartje A. Rookus, Jan Burema, Martin A. van 't Hof, Paul Deurenberg, Joseph G.A.J. Hautvast

ABSTRACT

To investigate the development of the body mass index (BMI, weight/height^2) in young adults, bpdy height and weight of 518 men and 624 women, initially 19-31 years of age, were measured in spring 1980 and body weight was measured every six months from spring 1981 through spring 1984.

The incidence of moderate overweight (BMI>25 kg/m^2) increased with age from 4.8% per 4 year to 15.5% per 4 year in men and was more stable, 5.3% per 4 year to 5.7% per 4 year, in women. The rate of gain in BMI was not found to differ according to the initial BMI in men, whereas in women a slight negative association existed between the initial BMI and its subsequent change. The within-subject standard deviation of the yearly measured BMI was 0.69 kg/m^2 in men and 0.74 kg/m^2 in women, and was larger both at higher levels of the BMI and at both extremes of the distribution of the rate of change in BMI.

The considerable fluctuation in BMI may mask the gradual increment in BMI and may thus hinder young adults from being aware of their becoming overweight.

INTRODUCTION

Overweight has been studied extensively for many years. In most studies of the development of fatness, however, attention has been restricted to the development in the sample in its entirety, as was done in a former paper (Chapter 3). This provides only a limited insight into the development of fatness in the individual.

Only recently, has this subject received some attention. A 20-year follow-up study has shown that obese subjects tend to lose body fat, whereas the lean tend to gain it (Garn and Roche 1984). The data from a three-year follow-up study show a similar tendency in men, but not in women (Garn and Pilkington 1984).

These studies, however, may be biased by the biological fluctuation and error of measurement for which the measurements were not corrected. In the relation between the initial level of body fat and its subsequent change this

bias may come through as a spurious regression to the mean effect. In the present study the question was investigated whether the obese and the lean still display a differential change in body fatness, taken into account this spurious regression to the mean effect.

So far, day-to-day fluctuations in body weight have received more attention than the long-term fluctuations. Day-to-day fluctuations mainly reflect the fluid-balance, changes in intestinal content and glycogen status, and alterations in metabolic state. In addition to these factors, the long-term fluctuations in body weight generally reflect changes in body fatness (Chien et al. 1975; Sjöström 1980). Two studies have given some indication about the magnitude of the long-term fluctuation in body weight. In a 7-year follow-up study of long-stay prisoners, the difference between the highest and the lowest body weights for individuals was 7.5 kg (Garrow 1974). In a follow-up period of 18 years in the Framingham study, this figure was on average, about 10 kg (Gordon and Kannel 1973). In these studies, however, no correction for linear trend was made.

The aim of the present study was to investigate the individual developmental curve of the body mass index (weight/height^2), as a measure of body fatness. First, the level, the linear trend in change, and the fluctuation around this linear trend in body mass index was assessed, as well as the incidence of overweight, based on the first two variables. Secondly, the interrelationships of these variables were examined.

METHODS

In spring 1980, 1670 men and women in three cohorts, years of birth 1948-1950, 1953-1955, 1958-1960, participated in the first examination of a four-year follow-up study (for details see Chapter 3). From spring 1981 onward measurements took place every six months from March through May and from September through November at seven consecutive examinations.

In spring 1980 body height without shoes was measured to the nearest 0.1 cm with a Microtoise. At each examination body weight in light indoor clothing without shoes was measured to the nearest 0.5 kg, between 4 pm and 9.30 pm on a balance that was calibrated weekly.

As a measure of body fatness the body mass index was used, since it is widely accepted for epidemiologic studies (Garrow 1981; Bray 1985; NIH-Consensus Conference 1985) and has been shown not to be improved by the inclusion of body diameter measurements (Chapter 8). In the present study, changes in body mass are completely caused by changes in body weight,

which are closely related to changes in total adipose tissue (Chien et al. 1975; Sjöström 1980).

Sample

A complete follow-up of the participants over the five examinations in the spring was available for 1142 (69%) subjects. The age in 1980 was 19-21 for 130 men and 150 women, 24-26 for 167 men and 226 women and 29-31 for 221 men and 248 women within the follow-up group. This sample did not differ significantly from a control group with respect to initial body mass index or change in body mass index during the four-year follow-up period (Chapter 3).

Level and rate of change in body mass index

To study the relationship between initial level and subsequent change in body mass index, for each subject the intercept and slope of the least squares linear regression of the body mass index, measured in the spring, on time were computed. The intercept and slope further are designated as the estimated initial level and the rate of change (trend), respectively.

By using the intercept and slope rather than the measured initial level and the difference between two measurements, most of the spurious effect of biological fluctuation and error of measurement in the body mass index was eliminated. However, the adjustment of this bias was not complete because of the error of estimate of the individual regression lines. By means of the average of the individual residual variances around the lines, this error was adjusted for according to Svärsudd and Blomqvist (1978). If a_i, b_i and SEE_i^2 are the intercept, slope and the residual variance, associated with the individual regression lines, respectively, then the regression of b_i on a_i results in a biased estimate, b, of the true regression coefficient, b_{adj}. The true coefficient is obtained as follows:

$$b_{adj} = (b + mc_1) / (1 - mc_2) ,$$

where, $m = \overline{SEE^2} / var(a)$, $c_1 = \bar{t} / \sum_{i=1}^{k} (t_i-\bar{t})^2$ and $c_2 = \sum_{i=1}^{k} t^2 / (k \sum_{i=1}^{k} (t_i-\bar{t})^2)$.

The standard error of b_{adj} is:

$$se(b_{adj}) = [\{ \frac{var(b)}{(c_1+c_2b)^2} + \frac{2m^2}{(1-mc_2)^2} (\frac{1}{f_1} + \frac{1}{f_2}) \} (c_1+c_2b_{adj})^2]^{\frac{1}{2}}$$

where, f_1 and f_2 are the degrees of freedom of the numerator and denominator of m, respectively.

Incidence and recovery rates

Since the risk for morbidity and mortality increases more rapidly with age in overweight subjects than in subjects of normal weight (Rabkin et al. 1977; Hubert et al. 1983), the linear trend in the development of the body mass index was considered more important with respect to a measure for incidence and recovery of overweight than the biological fluctuation. Thus, the incidence and recovery rates of overweight were computed from the intercept and slope of the individual regression lines.

The cut-off points for moderate and severe overweight were 25 kg/m^2 and 30 kg/m^2, respectively (Garrow 1981; Bray 1985). An incident case was defined as having an estimated initial body mass index below the cut-off point and an estimated final body mass index above it, in combination with a rate of gain of at least 0.25 $kg/m^2/yr$. This arbitrary limit corresponds with a weight gain of about 3 kg/4yr, which was considered as a meaningful constraint to avoid subjects to be counted as cases merely because of a random fluctuation around a long-term constant level around a body mass index of 25 kg/m^2 or 30 kg/m^2. The incidence rate was computed as the ratio of the number of incident cases and the number of subjects at risk, the latter being estimated as the number of subjects that had an estimated initial body mass index below the cut-off point, minus half the number of incident cases. The recovery rates were computed accordingly, replacing gain by loss in body mass index. For these binomial distributed rates the exact confidence intervals are given.

Fluctuation in body mass index

The fluctuation of the body mass index was split up into a random yearly fluctuation and a seasonal fluctuation. Two measures for the random yearly fluctuation in body mass index were computed for each subject: (1) the within-subject standard deviation, (2) the within-subject standard deviation after allowing for linear trend, i.e. the standard error of estimate associated with the individual regression line.

The seasonal fluctuation was computed as the difference between the mean of the four last measurements in the spring and the mean of the three measurements in the fall. The fall measurements were used solely to compute the seasonal fluctuation.

RESULTS

The body mass index at the consecutive examinations are given in Table 1. In these young adults the body mass index increased slightly during the four-year follow-up period. The group standard deviation of the body mass index was fairly stable within this period.

Table 1: Body height and body mass index (BMI = weight/height^2) in young adults during a four-year follow-up study

	Men (n = 518)			Women (n = 624)		
	median	mean	SD	median	mean	SD
Body height(cm) in 1980	180.4	180.2	6.7	166.9	166.8	6.3
BMI (kg/m^2)						
in 1980	23.2	23.6	2.9	21.9	22.3	2.8
in 1981	23.4	23.7	2.8	22.0	22.3	2.8
in 1982	23.6	23.9	2.9	21.9	22.4	2.9
in 1983	23.8	24.1	3.0	22.2	22.7	2.9
in 1984	23.9	24.2	3.0	22.1	22.7	2.9

Estimated characteristics of the development of the body mass index are given in Table 2. The fluctuation, expressed as the within-subject standard deviation adjusted for trend, was considerable with respect to the rate of change. The seasonal fluctuation indicated that the body mass index was slightly higher ($p < 0.001$) in the spring than in the fall. The difference was small, but compared with the rate of change, not ignorable.

Table 2: The within-subject rate of change and fluctuation in body mass index (BMI=weight/height^2) in young adults during a four-year follow-up study

Variable	Men (n = 518)			Women (n = 624)		
	median	mean	SD	median	mean	SD
Rate of change in BMI (kg/m^2/yr)	0.16	0.16	0.32	0.10	0.13	0.37
Random yearly fluctuation:						
within-subject SD in BMI (kg/m^2)	0.62	0.69	0.38	0.64	0.74	0.44
within-subject SD in BMI, adjusted for trend (kg/m^2)	0.43	0.48	0.28	0.45	0.52	0.30
Seasonal fluctuation[1] in BMI (kg/m^2)	0.14	0.14	0.40	0.06	0.06	0.43

1 Seasonal fluctuation = difference of the mean of the four last measurements in the spring and the mean of the three measurements in the fall, based on 504 men and 555 women.

The relation between level and rate of change in body mass index is given in Figure 1. The regression of the rate of change on the estimated initial level produced a slope of -0.012 (95% CI: -0.022,-0.002) in men and -0.021 (95% CI: -0.031,-0.011) in women. After adjustment for the remaining bias, the slope was -0.007 (95% CI: -0.017, +0.004) in men and -0.014 (95% CI: -0.024, -0.004) in women. Clearly, the mean gain in body mass index was similar at all levels of the body mass index for men, whereas the significant slight negative association in women reflected an extremely small differential change for the various classes of the initial level of the body mass index.

Since the adjustment for the remaining bias resulted in a minor correction only, the incidence and recovery rates of overweight were estimated from age-specific analogues of Figure 1. The remaining spurious regression to the mean effect may render a slight underestimation of the incidence rates and a slight overestimation of the recovery rates. Table 3 shows the incidence rates of moderate and severe overweight. These figures were high compared with the prevalence of moderate and severe overweight, assessed from the estimated initial level, that was 24.4% and 1.1% in men and 12.8% and 2.5% in women, respectively.

Table 3: Four-year incidence rate (I) of moderate (body mass index > 25 kg/m^2) and severe (body mass index > 30 kg/m^2) overweight in young adults

		Men			Women		
Developing:	Initial age	N at risk	I (%/4yr)	95%CI	N at risk	I (%/4yr)	95%CI
Moderate overweight	19 - 21	105	4.8	1.5, 10.8	133	5.3	2.1, 10.6
	24 - 26	116	15.8	9.5, 23.5	192	5.7	2.9, 10.1
	29 - 31	138	15.2	9.7, 22.4	199	5.5	2.8, 9.7
Severe overweight	19 - 21	129	0.8	0.0, 2.8	146	0.0	0.0, 2.5
	24 - 26	164	1.2	0.0, 3.4	220	1.4	0.3, 4.0
	29 - 31	212	0.9	0.1, 3.4	239	2.5	0.9, 5.5

With respect to this relative comparison as well as to the absolute value, the incidence of moderate overweight was high for the middle and highest age groups in men. The recovery rate of moderate overweight was 7.6% (95% CI: 3.4, 13.6) in men and 18.2% (95% CI: 10.3, 28.6) in women. Because of the small number of subjects that was at risk, no age-specific recovery rates of moderate overweight or recovery rates of severe overweight are presented.

The relationship of the fluctuation in body mass index with the level and rate of change in body mass index was studied. For this purpose, the sample was

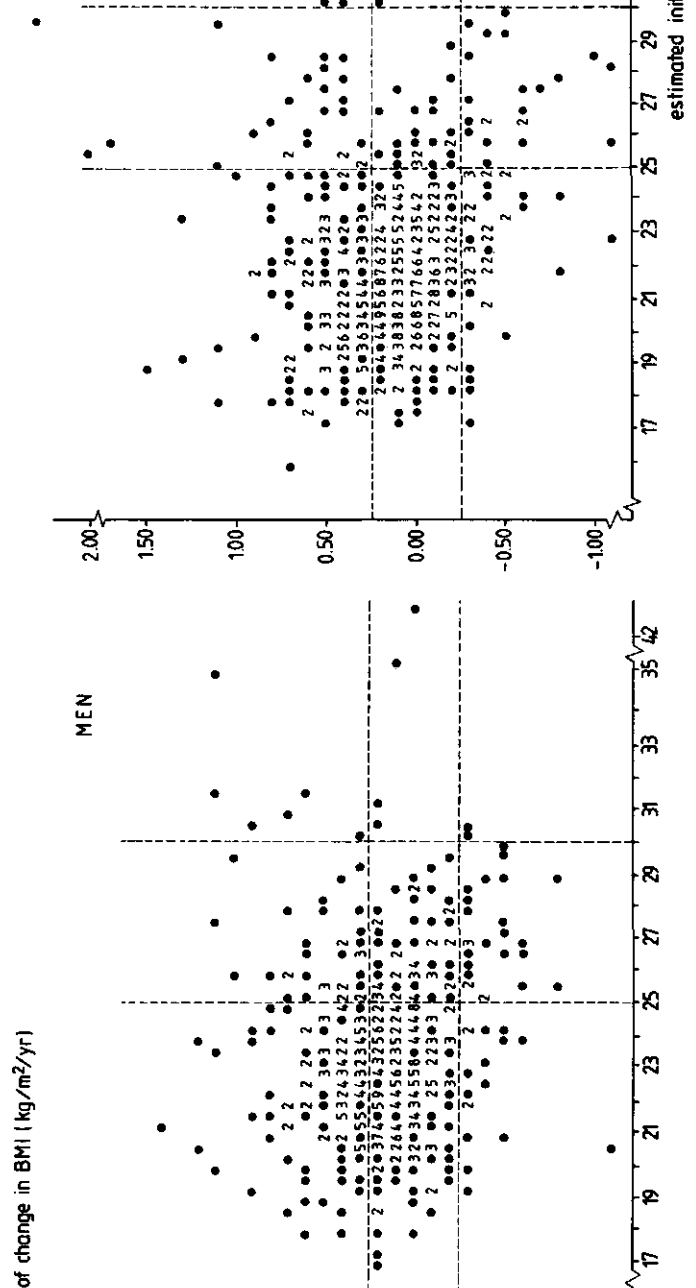

Figure 1: Relationship between the rate of change in body mass index and the estimated initial body mass index, based on a four-year follow-up of 518 men and 624 women, initially 19-31 years of age

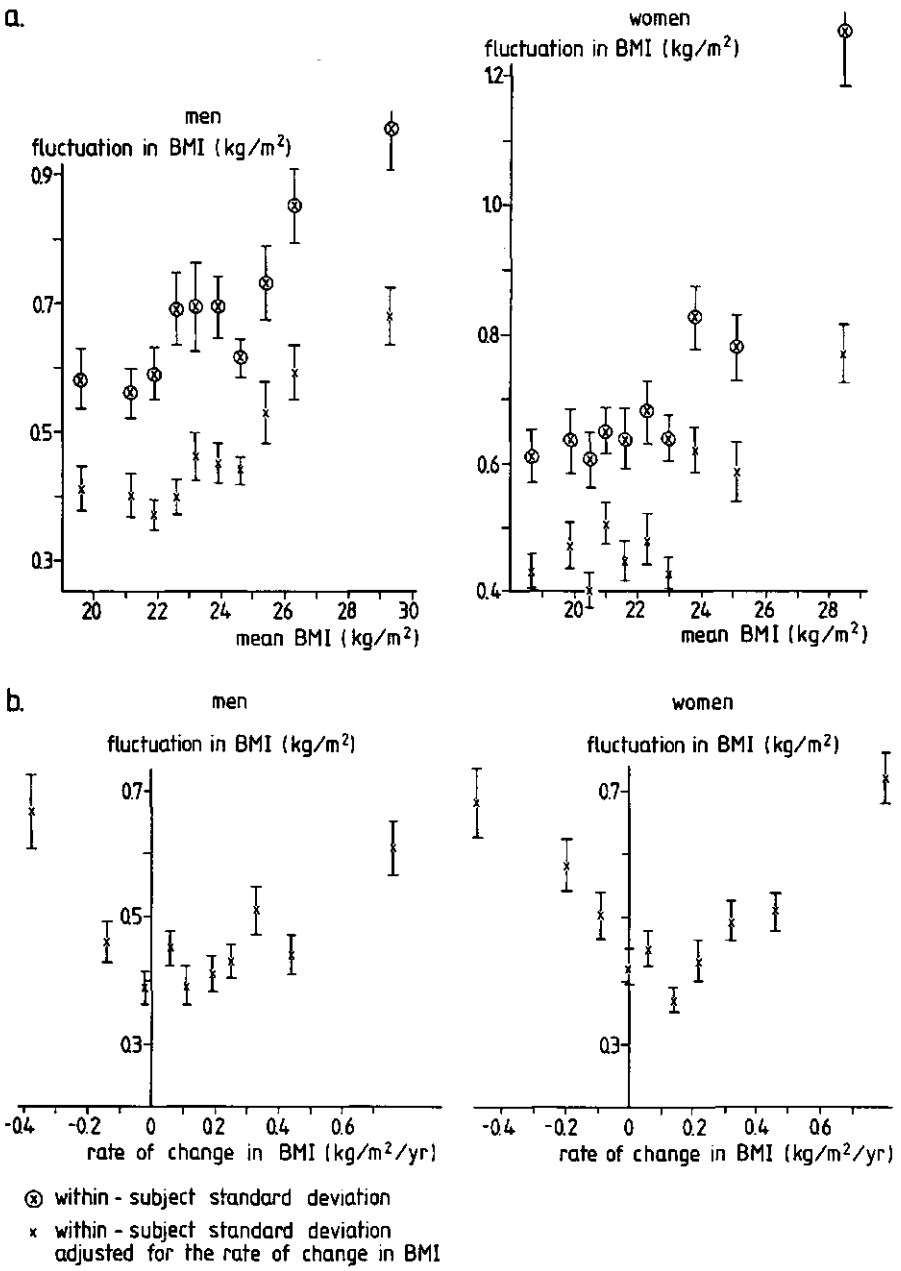

Figure 2: The within-subject standard deviation of the body mass index (BMI) with its SE, adjusted or not adjusted for rate of change, plotted against subgroups based on deciles of the mean body mass index (A) and deciles of rate of change in body mass index (B), in 518 men and 624 women followed for four years

split up into ten groups in two ways, first according to the deciles of the individual means of the body mass index and secondly, according to the deciles of the rate of change in body mass index. In Figure 2a the average within-subject standard deviation of the body mass index has been plotted against the deciles based on the individual means. A J-shaped relationship was found; for the higher levels of the body mass index an increased fluctuation arose.

The plot of the within-subject standard deviation against the rate of change in body mass index shows that both a positive and negative increasing rate of change was associated with an increasing within-subject standard deviation, even after adjustment for linear trend (Figure 2b). In subjects whose body mass index hardly changed during the four-year follow-up period (change between -0.10 and +0.10 $kg/m^2/yr$) the mean within-subject standard deviation was still 0.44 kg/m^2 (SD 0.21, n=124) in men and, incidentally, also 0.44 kg/m^2 (SD 0.22, n=160) in women.

DISCUSSION

In young adults body weight continues to increase after the termination of growth (Chapter 3; Cronk et al. 1982; Rosenbaum et al. 1985; Van Sonsbeek 1985). In the present study we confined ourselves to the linear trend and fluctuation around this trend in the description of the individual development of the body mass index, though the true individual curve may show gains in body mass index that are episodic rather than continuous, as has been suggested by the results of a retrospective study (Bradley 1985). The comparison of the prevalence with the incidence of overweight during the four-year follow-up period clearly illustrated that the slow gain in mean body mass index of the total sample results in a rapid increase in the proportion of subjects that runs an elevated health risk because of overweight, especially in men. In women the recovery of moderate overweight was relatively high, though it may be a slight overestimation, as already discussed.

The definition of the incidence of overweight, however, is not a clear-cut matter. The incidence of overweight, as a risk factor for increased morbidity and mortality can scarcely be determined on the basis of one or two measurements, because of the relatively large fluctuation in body weight. So, to characterize the cases and the at-risk group, we used the intercept and slope of the individual regression lines for the four-year follow-up period, instead of the five observed body mass indices. In consequence of this method, the incidence rate is specific for the four-year follow-up period; a shorter

follow-up period would result in a higher incidence rate, because subjects would be classified as cases that developed overweight but (shortly thereafter) recovered from it again. For the same reason, a longer follow-up period might result in a lower incidence rate. Still, the present method was considered the best choice to minimize the occurrence of false positives arising from the large within-subject fluctuation.

Data on the relationship between level and rate of change in body mass index are sparse. From a 20-year follow-up study, Garn et al. (1984) concluded that the obese become less obese and the lean become less lean during the time of follow-up. For the male obese and the female lean, the analysis of 10-year follow-up of the same study led to analogous results (Garn et al. 1980). Other long-term follow-up studies have reported a negative association between initial level and subsequent change in body mass index, as well (Hsu et al. 1977; Gillum et al. 1982). This can also be deduced from the data of men in a three-year follow-up study, but not from the data of women (Garn et al. 1984).

In these studies, however, in general no adjustment was made for biological fluctuation or error of measurement, that give rise to a spurious effect of regression to the mean. Some of these studies report an increment of the between-subject standard deviation of the body mass index with age, which rather points to a positive association between level and change (Garside 1983). After we had eliminated the biological fluctuation and the error of measurement, no differential change at the various initial levels of the body mass index was found in men, whereas the association was only slightly negative in women. This is in line with the lack of increase in standard deviation of the body mass index during the four-year follow-up period (Table 1). Thus, in men the obese do not gain less body weight than the rest of the sample, whereas this tendency is only weak in women. It would be interesting to examine this same relationship after a follow-up period of more than four years.

In our study, the long-term fluctuation in body mass index was divided into the random yearly fluctuation and the seasonal fluctuation. The random yearly fluctuation in body mass index is for a small part the result of the day-to-day fluctuation; the mean day-to-day changes in body weight, ignoring sign, have been found to be 0.42 kg (SD 0.23) in men and 0.28 kg (SD 0.24) in women (Robinson and Watson 1965; Cronk and Roche 1982). Long-term fluctuation of body weight in other studies confirm our findings that body weight varies within wide ranges within the individual (Garrow 1974; Gordon and Kannel 1973). In the Framingham study, the within-subject standard deviation, based on an examination each two years during a follow-up period of 18 years, was 3.22 kg

in men and 3.19 kg in women (Gordon and Kannel 1973). Since the follow-up period is longer than in our study and the fluctuation is not corrected for trend, these values cannot be compared to our findings quantitatively.

The seasonal fluctuation was small, but considerable with respect to the rate of change in body mass index, and thus should be taken into account in longitudinal studies. Another Dutch study showed that winter-summer differences are even slightly larger than spring-fall differences (Van Staveren et al. 1986). The latter were comparable to the differences we found.

In addition to factors that cause short-term fluctuation, the long-term fluctuation is caused by changes in lean and fat tissue. It has been found in a cross-sectional study that differences in weight between women of similar height are attributable to tissue which is 70-78% fat and 22-30% lean (Webster et al. 1984). This suggests that long-term changes in body weight mainly reflect changes in energy balance. Though thermogenesis and changes in the metabolic efficiency may play a role in the regulation of the energy balance, recent studies suggest that energy intake and physical activity are the most important in this respect (Rand et al. 1985; Forbes 1984). Apart from these factors (Van Staveren et al. 1986). the sinusoid trend of body weight during the year may be caused by slight changes in metabolic activity (Zahorska-Markiewicz and Markiewicz 1984; Ingemans-Hansen and Halkaer-Kristensen 1982).

In the present study the yearly fluctuation was higher at higher levels of the body mass index as found by others (Bray 1976). The larger fluctuation in body mass index in the obese subjects reflects a less accurately working regulatory mechanism than in the lean subjects. Insufficient feed-back may in turn result in a larger variation in the rate of change in the obese, as was especially marked in women in the present study and in both sexes in a three-year follow-up study (Garn and Pilkington 1984). Indeed, Figure 2b shows an association between the yearly fluctuation and the rate of change in body mass index. However, to study the chronological relationship between fluctuation and the rate of change, more examinations would be necessary.

From the present study it can be concluded that the mean gain in body mass index in young adults is similar among groups that differ with respect to the mean body mass index; at all levels there is a positive drift in the body mass index. This drift, however, is masked by seasonal and random yearly fluctuation: one out of ten men with a relatively small change in body mass index (between -0.1 and +0.1 $kg/m^2/yr$) had a within-subject standard deviation based on yearly measurements, of at least 0.71 kg/m^2 (adjusted for linear trend), which was considerable compared with the mean rate of change of

0.16 kg/m^2/yr. This relative large fluctuation may hinder young adults from being aware of the small but steady positive trend in the development of their body weights. For the same reason, health workers may have difficulty in identifying subjects that are developing overweight. Yearly measurements, recommended by health workers, may reveal this trend, so that they become aware of their increasing weight. This might be a first step in the prevention of a further gain.

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5 THE EFFECT OF DIETING IN YOUNG ADULTS

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ABSTRACT

As part of an extensive longitudinal study, the effect of dieting on the development of the body mass index (BMI=weight/height^2) during half a year, one year, or two years of follow-up was examined in a sample of the general population consisting of 216 men and 279 women, 20-35 years of age. Body weight was measured and a questionnaire was completed every six months from spring 1981 through spring 1984. The difference between change in BMI of dieters and non-dieters, matched on initial (for dieters: pre-dieting) BMI was estimated.

The effect of dieting was rather stable during follow-up, but was very small. After two years of follow-up, the average reduction in body mass was -0.51 kg/m^2 (95%CI:-1.04,+0.02) in men and -0.38 kg/m^2 (95%CI:-0.77,+0.01) in women. Determinants of loss in body mass were initial BMI, dieting during the summer and frequent periods of dieting in men, and being older than 30 and dieting on medical advice in women.

It is concluded that the effect of dieting may be enough to prevent the age-related gain in body mass. However, subjects who have already developed overweight are unlikely to decrease their risk to health markedly by dieting, as generally practiced, unless they persist.

INTRODUCTION

Reviews on the effect of the treatment of obesity are clear in their conclusions[15,30,31,38]; whereas the results during treatment may be impressive, relapse during follow-up is common. However, many studies on the treatment of overweight (a) are conducted on small, highly selective samples, consisting of severely obese subjects, (b) have a short follow-up period, (c) have many subjects lost to follow-up, (d) examine only one attempt to lose weight, and (e) do not include a control group. These limitations may restrict the

universality of the disappointing results. As Schachter[26] has suggested, the rates of successful self-cure might be considerably higher in 'non-therapeutic' populations. In two admittingly non-representative samples he found that respectively 55% of the men and 72% of the women that stated to have been once overweight were no longer overweight at the time of the study. In a similar retrospective study of a sample of two English towns it was found that these proportions were 66% in men and just under 50% in women[25]. In a third study, the prospective Tecumseh Community Health Survey[9], 68% of the initially obese men and 25% of the initially obese women were found to be no longer obese after 10 years of follow-up.

In none of the studies mentioned above, is it known whether the weight reduction is the result of dieting or is associated with other factors that may occur during adulthood, such as change in life style. An indication that dieting does play a role is given by a retrospective study in two population-based samples (25-74 years of age) of the Minneapolis-St. Paul metropolitan community[16]. It was found that 38% of the male and 30% of the female subjects that were once overweight and that had once dieted, were no longer overweight at the time of the survey.

The aim of the present follow-up study on young adults living in a county with urban and rural areas in the Netherlands, was to investigate the effect of dieting more explicitly by comparing the change in body mass index (weight/height^2) of dieters and non-dieters during follow-up periods of half a year, one year, and two years. Because there is a U-shaped relationship between mortality and the body mass index[35], the absolute change in body mass was considered a better criterion variable than the proportion of overweight that was lost; the same amount of loss in body mass may give a larger, not a smaller, improvement in risk to health in subjects with severe overweight than in subjects with moderate overweight. In addition, determinants of success during dieting and throughout follow-up were examined.

METHODS

Between 1980 and 1984 an extensive follow-up study was conducted in the county of Ede, the Netherlands (for details see: reference 2 and Chapter 3). Figure 1 shows the design of the study. In spring 1980, all residents, born in 1948-1950, 1953-1955 and 1958-1960 were invited by mail to participate. Of the 3936 respondents in 1980, 1670 subjects were examined at home twice a year from spring 1981 onward to spring 1984. During each examination, body weight without shoes and in light indoor clothing was measured to the nearest 0.5 kg on a

scale that was calibrated weekly, and a questionnaire that had been sent by mail in advance was checked for completeness. Body height without shoes was measured with a Microtoise to the nearest 0.1 cm in spring 1980 only.

The remaining respondents in 1980 were remeasured in spring 1984 only. The change in body mass index during the four years of follow-up, did not differ between the two groups (Chapter 3).

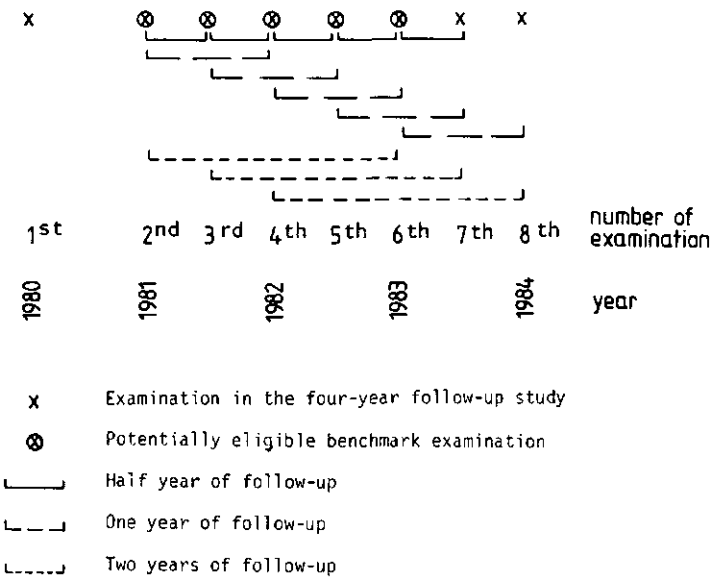

Figure 1: Design of the study

Questionnaire

Attempts to reduce body weight were assessed by means of the question: 'did you diet on your own initiative during the last 6 months' or 'did you diet on medical advice during the last 6 months'. In addition, the subjects were asked to specify the diet so that we could determine whether it was intended to reduce body weight. Those who positively responded to at least one of the two questions on dieting at examinations 3-7 were designated 'dieters'. The subjects who at examinations 2-8 reported they had not been dieting were designated 'non-dieters'.

The highest level of education attained by the end of the study, was used as an indicator of socio-economic status. Three levels were distinguished: low level, comprising primary (grade) school and primary occupational training, middle level, comprising secondary (middle) school and secondary occupational training, and high level, comprising university and tertiary occupational training.

The number of cigarettes that was smoked per day and changes in physical activity during sports or daily acitivities were recorded at each examination.

To study the modifying effects of environmental and behavioral factors on the effect of dieting the following questionnaires were administered: the Dutch Life Events Questionnaire[34] at examinations 3-7, the Dutch Eating Behaviour Questionnaire[33] at examinations 4 and 7, and the Short Questionnaire on Habitual Physical Activity[3] at examinations 5 and 6. From the first questionnaire a score for negative life events was computed, from the second a score for emotional eating and from the third a score for physical activity during work, sports and leisure time.

Analysis

To examine the effect of dieting, the change in the body mass index during a follow-up of half a year, one year, or two years, was compared between dieters and non-dieters so as to take the usual development into account (Figure 1). For the dieters, the first half year of these follow-up periods comprised the period of dieting.

To be able to analyze simultaneously the change in body mass of dieters who started their attempts to lose weight at different times during the follow-up period, one of the examinations 2-6 was used as the benchmark for each dieter and non-dieter. For the dieters, the condition was imposed that they had to have dieted during the six months following the benchmark examination but not during the six months prior to the benchmark examination. The first of the eligible examinations was used as the benchmark for each of the dieters, for non-dieters the benchmark examination was chosen randomly.

The body mass index at the benchmark (the benchmark or initial body mass index) was used for the computation of the three evaluation variables, i.e. the change in body mass index during half a year, one year, and two years following the benchmark examination. The latter variable could be computed only for subjects with examination 2, 3 or 4 as their benchmark.

Matching

The dieters and non-dieters were category-matched on their initial body mass index. This matching procedure ensured that the estimation of the effect of dieting was not highly dependent on the extrapolation of the expected change in body mass for dieters with a low initial body mass index and for non-dieters with a high initial body mass index. The matching was done season-specifically, because body mass fluctuates during the year[32].

Effect modification and confounding

The effect of dieting was assessed in a multiple linear regression model as follows:

$$Y = a + bD + \Sigma c_i C_i + \Sigma d_j M_j + \Sigma e_j DM_j + \text{residual}$$

where, Y=change in body mass index, D=indicator for diet-behavior, C_i=confounders, M_j=effect modifiers.

The initial body mass index, the initial age (20-24, 25-29, 30-34 years), socio-economic status (low, middle, high), and season of the year following the benchmark examination, were considered as potential modifiers of the effect of dieting (i.e. coefficient b) in this additive model. As an adaptation of the method of Kleinbaum et al.[17], an effect modifier was retained in the model for all three follow-up periods, if its F-to-remove in the final model of the backward elimination procedure was significant ($P<0.10$) for at least one of the three follow-up periods. If the continuous initial body mass index modified the effect of dieting, its effect was illustrated by the estimation of the effect of dieting at two levels of the initial body mass index; at 25 kg/m^2 (reference category) and at 30 kg/m^2. If potential effect modifiers were not retained in the model as such (i.e. interaction), they were retained as main effect to control confounding. In addition, a change in smoking behavior (stop or start) and a change in physical activity during sports or daily activities (less or more) were included as confounders in the analysis. The changes were defined for each of the specific follow-up periods.

The score for negative life events during the half year of dieting (used as a linear and as a quadratic term) was added as a potential modifier to the model that resulted from the procedure described above. In two separate analyses this was also done for emotional eating (linear term) and physical activity (linear term), as scored at the examination succeeding the benchmark examination.

RESULTS

The proportion of subjects who dieted during the first year of the survey with half-yearly measurements (examinations 3 and 4) was 7.5% (95%CI:5.6,9.9)in men and 20.8% (95%CI:18.1,23.9) in women. It is clear that more women reported to have been dieting than men.

To compare the change in body mass index between dieters and non-dieters, one of the examinations 2-6 was allocated to subjects as their benchmark examination. At this benchmark examination 61% of the male dieters and 29% of the female dieters were overweight (BMI_{ini} > 25 kg/m^2, reference 10). Matching on initial body mass index with non-dieters was possible for 100% of the male dieters and for 76% of the female dieters, and was sufficiently successful (Figure 2). Of these matched female dieters 16% were initially overweight. So, a large number of female overweight dieters could not be matched, because comparable non-dieters were not available. As a result, the effect of dieting in the overweight women could not be estimated with satisfactory precision.

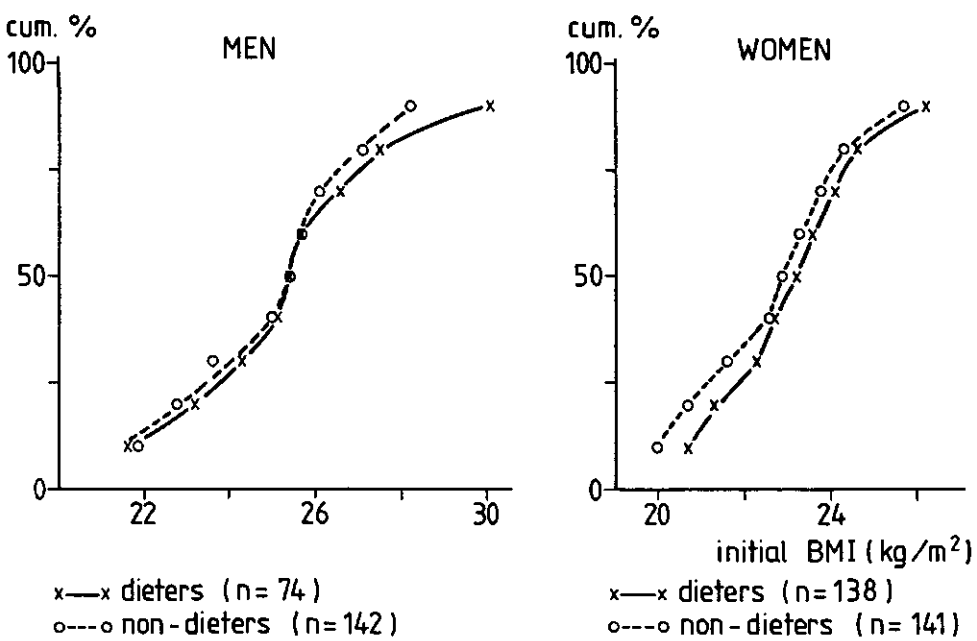

Figure 2: Evaluation of the stratified matching on the initial body mass index (BMI) of dieters with non-dieters

Yet, to obtain an idea of the results of dieting in overweight women, the changes in body mass index of female dieters with an initial body mass index >25 kg/m^2 (N=80) and with an initial body mass index <25 kg/m^2 (N=135) were mutually compared before the matching. Within the total group of female dieters, the overweight women lost 0.37 kg/m^2 (SE:0.16) more body mass during half a year of follow-up than the dieters who were initially not overweight. After one and two years, these figures were 0.46 kg/m^2 (SE:0.20) and 0.48 kg/m^2 (SE:0.28), respectively. Though this crude comparison may be biased, for instance by a spurious regression to the mean effect, it suggests that the higher the body mass index, the more body mass was lost as a result of dieting.

Table 1: The differential change in body mass (kg/m^2) in young adult dieters and non-dieters during a follow-up period of half a year, one year, and two years, respectively

	Duration of follow-up: 1/2 yr			1 yr			2 yr		
	N	change	SE	N	change	SE	N	change	SE
MEN									
Dieters	74	-0.67	0.17	74	-0.38	0.14	34	-0.30	0.25
Non-dieters	142	-0.06	0.06	142	-0.02	0.07	72	+0.21	0.12
Effect of dieting	216	**-0.61**	0.18	216	**-0.36**	0.16	106	**-0.51**	0.27
WOMEN									
Dieters	138	-0.20	0.08	138	-0.09	0.09	81	-0.06	0.15
Non-dieters	141	-0.04	0.06	141	+0.02	0.07	71	+0.32	0.14
Effect of dieting	279	**-0.16**	0.10	279	**-0.11**	0.12	152	**-0.38**	0.20

In Table 1 the change in body mass of matched dieters and non-dieters and the difference between these two (the effect of dieting) is given. Expressed in differential change in body weight, the effect of dieting for the three follow-up periods, respectively, was -1.98 kg (SE:0.57), -1.21 kg (SE:0.51) and -1.50 kg (SE:0.87) in men and -0.46 kg (SE:0.27), -0.29 kg (SE:0.33) and -1.08 kg (SE:0.56) in women. Thus, the effect of dieting was rather stable in men, and in women even tended to increase with follow-up. The magnitude of the effect, however, was very limited.

Table 2: The adjusted[1] difference between change in body mass index (kg/m^2) of dieters and of non-dieters, 20-35 years of age

	Duration of follow-up: 1/2 yr		1 yr		2 yr	
	Differential change	95%CI	Differential change	95%CI	Differential change	95%CI
MEN		N_2=216 R^2=0.28		N_2=216 R^2=0.17		N_2=106 R^2=0.29
Reference group[2]	-0.23	-0.68,+0.23	+0.15	-0.29,+0.60	-0.45	-1.19,+0.29
MODIFIERS:						
BMI_{ini}= 30 kg/m^2	-1.17	-1.74,-0.61	-0.57	-1.11,-0.02	-1.28	-2.22,-0.35
SES:low	-0.91	-1.70,-0.12	-0.86	-1.63,-0.10	-2.32	-3.84,-0.79
high	-0.81	-1.34,-0.28	-0.40	-0.91,+0.12	-0.94	-1.87,-0.01
Winter	-0.05	-0.54,+0.44	-0.08	-0.55,+0.39	+0.63	-0.34,+1.60
WOMEN		N_2=279 R^2=0.11		N_2=279 R^2=0.09		N_2=152 R^2=0.16
Reference group	-0.11	-0.35,+0.12	+0.02	-0.27,+0.32	+0.00	-0.52,+0.53
MODIFIERS:						
Age:30-34	-0.34	-0.65,-0.02	-0.37	-0.76,+0.02	-0.95	-1.58,-0.31

1 Adjusted for: initial age (20-24,25-29,30-34), initial body mass index (BMI_{ini}), socio-economic status (low,middle,high), season when dieting (one dummy), change in smoking behavior (two dummies), and in physical activity during work, sports, and daily activities (each two dummies).

2 The reference category comprised the total group of dieters and non-dieters, with the exception of those categories for which effect modification was present; if the initial body mass index modified the diet effect, the effect was computed at an initial body mass index of 25 kg/m^2.

Table 2 shows the results of the multiple regression analyses of change in body mass index on diet behavior. In the male reference category (middle socio-economic status, dieting during the summer) no effect of dieting, computed at an initial body mass index of 25 kg/m^2, was found for any of the three follow-up periods.

Specific subgroups were slightly more successful, however. Dieting men with a higher initial body mass index lost more body mass. This was true for all follow-up periods. Also, men of low or high socio-economic status achieved more result from dieting than those of middle status. However, a significant ($P<0.05$) countereffect of dieting was found after two years of follow-up in men who had dieted during the winter. In women, only those older than 30 showed an effect of dieting. No effect modification of the initial body mass index was

found in women. This was possibly caused by the scarcity of data on the effect of dieting in the overweight.

Motive for dieting

To study whether the effect of dieting was associated with the motive for dieting, the total group of dieters was split into two categories: those dieting on medical advice (19 men and 15 women) and those dieting on their own initiative (55 men and 123 women). Table 3 shows that in men no difference between the two diet groups was found. In women, however, dieting on medical advice gave better results than dieting on one's own initiative.

Table 3: The adjusted[1] difference between change in body mass index (kg/m^2) of dieters on medical advice or on their own initiative, and non-dieters

	Duration of follow-up: 1/2 yr		Duration of follow-up: 1 yr	
Motive of dieting	Differential change	P[2]	Differential change	P
MEN	N=216		N=216	
Medical advice	-0.85*	ns	-0.50*	ns
Own initiative	-0.39*		-0.24	
WOMEN	N=279		N=279	
Medical advice	-0.58*	<0.05	-0.82*	<0.01
Own initiative	-0.15		-0.03	

1 adjusted for: initial age, initial body mass index, socio-economic status, season when dieting, change in smoking behavior and in physical activity during work, sports, and daily activities.
2 test of difference between the two diet groups.
* P<0.05 (test of difference between dieters and non-dieters).

Frequency of dieting

To study whether follow-up results were better in subjects that dieted more frequently, the total group of dieters was split into two categories: those who reported at only one examination that they had been dieting (50 men and 77 women) and those who reported at two consecutive examinations that they had been dieting (24 men and 61 women).

Table 4 shows that in men more frequent periods of dieting resulted in more loss of body mass, whereas in women no difference according to frequency of dieting was found.

Table 4: The adjusted[1] difference between change in body mass index (kg/m^2) of subjects dieting during the first half year of follow-up only, or subjects dieting more frequently, and non-dieters

Persistence of dieting	One year of follow-up			
	Men N=216		Women N=279	
	Differential change	P[2]	Differential change	P
Once only	-0.01	<0.001	-0.04	ns
More frequently	-0.93*		-0.22	

1 adjusted for: initial age, initial body mass index, socio-economic status, season when dieting, change in smoking behavior and in physical activity during work, sports, and daily activities.
2 test of difference between the two diet groups.
* P<0.05 (test of difference between dieters and non-dieters).

Table 5: The impact of negative life events (NLE), emotional eating (EMOT) and physical activity (PA) on the effect of dieting

Variable	Men Duration of follow-up:			Women Duration of follow-up:		
		1/2 yr	1 yr		1/2 yr	1 yr
	N	P[1]	P	N	P	P
NLE:						
linear	160	0.81	0.27	210	0.87	0.47
quadratic		0.39	0.81		0.97	0.80
EMOT	74	0.37	0.53	84	0.99	0.19
PA:	89			114		
work		0.97	0.39		0.18	0.91
sports		0.58	0.33		0.02	0.19
leisure time		0.43	0.11		0.38	0.73

1 P-value of interaction with the indicator for 'dieting'.

Environmental and behavioral factors

To study whether other environmental or behavioral factors could be identified to characterize successful dieters, the scores of negative life events, emotional eating or physical activity were added as potential effect modifiers to the model shown in Table 2 in three analyses, respectively. Table 5 shows that no effect of negative life events or emotional eating was found. Only in women did more physical activity during sports result in a larger loss of body mass during the first half year of follow-up. However, after another half year of follow-up this effect was no longer significant.

DISCUSSION

The suggestion[16,26] that attempts to lose weight may be more successful in the general population than is usually found in the more selective therapeutic samples was not strongly supported by the present findings. Though specific subgroups according to initial body mass index, socio-economic status, season when dieting and frequency of dieting in men, and age and motive of dieting in women were relatively successful, the effect of dieting during half a year, one year, and two years of follow-up was small. This magnitude deserves further comments.

First, the magnitude of the effect of dieting might have been slightly underestimated in the present study because of (a) the fixed time intervals between the examinations, (b) the lack of female overweight non-dieters and (c) a possible information bias. To start with the fixed time intervals: the examinations were not associated with the timing or duration of the period of dieting. Thus, part of the gain in body mass that led to the decision to diet and/or part of the regained weight following the end of the diet-period might be included in the 'effect of dieting'. If body weight had been measured immediately preceding and following the period of dieting, this underestimation might have been avoided, but this would most probably have been achieved at the cost of a spurious interference in the spontaneous attempts to lose weight by dieting (i.e. an overestimation of the effect of dieting). In the present design, this interference is less likely to occur, which is confirmed by the lack of any difference in the change of the body mass index between the present sample and a control-sample which was measured at the beginning and at the end of the four-year follow-up study (Chapter 3).

The lack of female overweight non-dieters is another factor that might have contributed to an underestimation of the effect of dieting in women. Although severely overweight women seemed to achieve more loss in body mass than

moderately overweight women, the initial body mass index did not prove to be an effect modifier in the multiple regression analysis (Table 2) possibly because of the lack of overweight non-dieters.

The third factor that might have caused an underestimation of the effect of dieting is a possible information bias. An affirmative answer to the question whether one has followed a diet gives no information about the extent to which a subject has actually reduced the usual energy intake. If the social environment encourages subjects to diet, the respondent may be inclined to answer the question in the affirmative even though he/she has not dieted wholeheartedly. So, a higher prevalence of dieting might be associated with a lower proportion of serious dieters. This might be the case for women in general, subjects with a high initial body mass index, younger subjects, and subjects of high socio-economic status[8,14]. So, in these subgroups, the effect of dieting might be slightly underestimated. In the opposite case, subjects within a subgroup that is relatively tolerant to overweight, who nevertheless indicate that they have dieted, may have done this more intensively. This may explain the relatively large effect in men of low socio-economic status. More exact information about the energy deficit is needed to avoid this response bias. However, several studies have illustrated the difficulty of gathering valid data on food consumption from obese and/or dieting subjects[7,19,36].

There is a second aspect concerning the magnitude of the diet effect: whereas the mean effect of dieting is generally small, the variance is often large. This means that dieting may certainly be efficacious for individual dieters, especially in categories defined by the effect modifiers. Thus, it is worth considering these modifiers more specifically.

Men with a high initial body mass lost more than men with a low value. An inverse association between pre-treatment body weight and weight loss during treatment has been reported by Christakis[4] but most studies[1,15,18,24,28,39] have found a positive association as we did for men. In contrast to the findings of Stuart and Guire[29], the follow-up results also were better for men who were initially more overweight. Our findings (Table 4) suggest that this is caused by repeated attempts to lose weight.

A notable effect modifier was the season of the year. In men most weight loss was obtained and retained if the period of dieting took place during the summer. Other studies have supported this seasonal effect[15,22,41] which might be caused by biological and/or social/environmental factors[13,42].

The effect of age was not significant after adjustment for the initial body mass index in two studies[28,37]. Even after this adjustment we found better

results in women older than 30 years. This same effect, though not adjusted, was reported for female members of UK slimming clubs[1].

As discussed above, the effect of socio-economic status might partly be caused by a response bias. A better result by dieting in subjects of high socio-economic status was found also in other studies[5,6,27].

We found that dieting on medical advice gave better results in women but not in men. Dieting for a specific medical reason has been found to predict success in losing weight[6]. The observations by Hoiberg[12] showed that self-reported improvement in health status determined weight loss during treatment and throughout follow-up. In both these studies the data of men and women were combined.

Wooley et al.[40] have suggested that repeated dieting does not give better results, because dieting causes a decrement in metabolic rate which might be permanent in subjects who persist in dieting. Their suggestion was supported by Hoiberg[12] but countered by others[11,15,26]. Our findings in men supported the more optimistic view that subjects who persist in dieting obtain better results.

The environmental and behavioral factors examined in the present study hardly modified the effect of dieting. A worse maintenance of weight loss has been reported during the experience of life events[21], but was not found in the present study. Subjects who are less likely to eat for emotional reasons, may maintain their weight loss better[20,23,27]. Again, this was not found in the present study, in accordance with Hoiberg[12]. A higher level of physical activity might give better follow-up results, at least for a follow-up of seven months[11,27]. For the first six months of follow-up, this finding was supported by our results for women who sported. However, six months later, this effect was no longer significant.

As the foregoing discussion has shown, the magnitude of the effect of dieting might be slightly underestimated in the present study. Even so, however, the effect in several subgroups was comparable with that found in a Community approach treatment study[39]. This emphasizes the importance of the inclusion of a control group in such studies. The present study suggests that the effect may be enough to prevent the age-related gain in body mass of 0.16 $kg/m^2/yr$ in men and 0.13 $kg/m^2/yr$ in women (Chapter 3). However, subjects who have already developed overweight are unlikely to decrease their risk to health markedly unless they persist in dieting. In many treatment studies it has been found that the weight loss that was achieved during treatment was regained completely during follow-up, or even surpassed. In our sample the small weight

loss achieved during half a year was fairly stable during further follow-up. The season of the year, when dieting, seemed to be important with respect to these results. So, though Schachter's contention[26] that dieting is more successful in less selective samples cannot strongly be supported by our findings, they endorse his recommendation that more than one attempt to lose weight should be taken into account. It is clear that a loss in body mass occurs very slowly.

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6 CHANGES IN BODY MASS INDEX IN YOUNG ADULTS IN RELATION TO NUMBER OF LIFE EVENTS EXPERIENCED

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ABSTRACT

As part of an extensive longitudinal study, the effect of experiencing few or many life events on the development of the body mass index (BMI= weight/height^2) over periods of one year and two years was examined in 350 men and 395 women, 20-35 years of age. Body weight was measured and a life event questionnaire referring to the preceding six months was completed every six months from spring 1981 through spring 1984. The change in BMI of subjects who experienced few or many life events was compared with the change in BMI of subjects who experienced an intermediate number of life events (the intermediate group).

During the first year of follow-up, several subgroups of men and women who experienced many life events showed a gain in body mass. In several subgroups of men who experienced few life events a comparable effect occured. After another year of follow-up this gain in body mass had disappeared in almost all subgroups. In the subgroup of men that tried to reduce their body weight by dieting during the follow-up period, did the gain in body mass following few or many life events seem to be permanent.

In conclusion, the gain in body mass that may occur following the experience of many life events in women, seems to be effectively counterbalanced by regulatory mechanisms. In men, however, the experience of few or many life events may play a part in the etiology of overweight.

INTRODUCTION

Since 1950 it has been widely accepted that no aspect of human growth, development or disease can escape the influence of a person's relation to his/her social or interpersonal environment[15]. Several case reports or surveys suggest that environmental factors may have an impact on the development of obesity. For example, obesity was found to follow the experience of stressful life events such as death of a parent, financial reverses[16], hospitalization, situations involving social or intellectual failure[8], marriage and childbirth[9],

breakdown of marriage, or illness of a close relation[6]. The increase in body weight following stressful life events has been designated as 'Kummerspeck'[4] or reactive obesity[7].

But a lack of stimulation by life events may also be followed by the development of overweight[23,25]. Emotional arousal, evoked by the presence or absence of life events may induce hyperphagia in some subjects, who may subsequently become obese.

Any empirical test of the effect of emotional arousal on body weight implied by these case reports and surveys, is beset by practical limitations. First, one may question whether experimentally invoked stress is comparable with stress outside the laboratory[37]. Secondly, in most previous research the response-parameter of interest, e.g. the amount of food eaten, was masked; for instance by presenting the study to the subjects as a taste-test[36] or a study on mood states[1]. Under these circumstances the response behavior may differ from such behavior under natural conditions. Thirdly, in many experiments the response behavior of normal-weight and overweight subjects was compared in order to explain why the latter group had become overweight whereas the first group had not[1,36,37]. However, any differences in response behavior that appeared could have been the cause as well as the effect of the existing differences in body weight.

In fact, most experimental studies do not unequivocally support the case reports or clinical observations on the relationship between arousal and eating. Whereas in normal weight subjects emotional arousal generally led to less food being eaten, the results in overweight subjects were not conclusive[37,40]. Yet experiments have revealed a number of modifiers of the eating response to emotional arousal: e.g. boredom or interest evoked by the experimental condition[1].

Our observational follow-up study differs from the experiments on stress-induced eating behavior in two main aspects. First, in order to assess naturally occurring stress we used the life-event score. While conceding that a life-event score is a crude measure, we consider it to be a more satisfactory representation of the intended concept than experimentally invoked stress. Secondly, the parameter of interest is not the body mass index (weight/height^2) as such, but the change in body mass index. This kind of longitudinal design is a more sound basis for investigating cause-effect relationships than experimental studies comparing normal-weight and overweight subjects. Because other studies have revealed that there is a time-lag between life events and their effects on health[35,38], we measured the change in body mass index over

two follow-up periods, of one year and two years, respectively.

Of the several hypotheses regarding life stress that have been proposed[11], the one that the individual may be vulnerable to the impact of life events because of pre-existing personal disposition and socio-environmental conditions (the "vulnerability hypothesis") was the one we chose to test.

METHODS

Between 1980 and 1984 an extensive follow-up study was conducted in the county of Ede, the Netherlands (see reference 3 and Chapter 3). Figure 1 shows the design of the study. In spring 1980, all residents born in 1948-1950, 1953-1955 and 1958-1960 were invited by mail to participate.

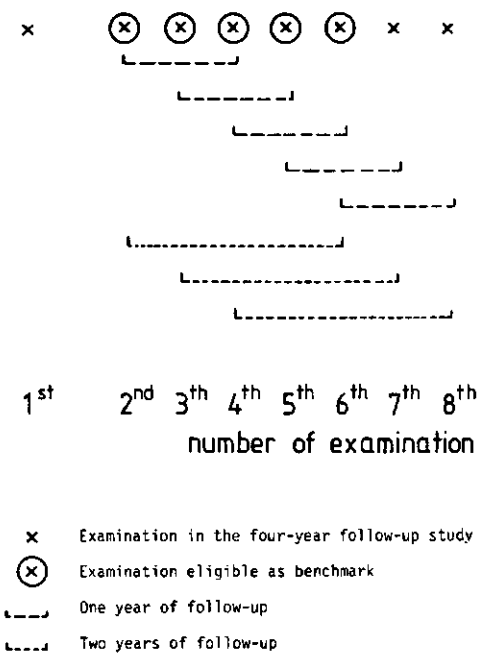

Figure 1: Design of the study

Of the 3936 respondents in 1980, 1670 subjects were examined at home twice a year from spring 1981 to spring 1984. During each examination, body weight without shoes and in light indoor clothing was measured to the nearest 0.5 kg on a scale that was calibrated weekly, and a questionnaire that had been sent

by mail in advance was checked for completeness. Body height without shoes was measured with a Microtoise to the nearest 0.1 cm in spring 1980 only. From weight and height the body mass index was computed as a measure of body fatness[13]. The body mass indices of women who were more than three months pregnant were not used in the present report.

The remaining respondents in 1980 (the control group) were remeasured in spring 1984 only. The change in body mass index during the four year of follow-up did not differ between the two groups (Chapter 3).

Questionnaire

Life events were assessed by means of the Dutch Life Events Questionnaire which contains questions derived from the Social Readjustment Rating Questionnaire[17], the Questionnaire of Paykel and Uhlenhuth[27], the Recent Life Change Questionnaire[28], the Life Experience Survey[34] and the Life Events Inventory[39]. No questions directly related to body weight were included. At each examination, the subjects checked off the events that had occurred in the preceding six months. A cumulative index derived from 85 life events was used. The unity weights were not refined, because it has been shown that it is difficult to improve upon a simple counting[32]. Additionally, the subscore of undesirable life events[41] was studied. We found that the change in body mass correlated more closely with the sumscore of all life events than with the subscore of undesirable life events, and therefore in this report the results of the total sumscore are reported.

The highest level of education that had been attained by the end of the study was used as an indicator of socio-economic status. Three levels were distinguished: low level, comprising primary (grade) school and primary occupational training, middle level, comprising secondary (middle) school and secondary occupational training, and high level, comprising university and tertiary occupational training.

At each examination, dieting behavior during the preceding six months was ascertained. The Short Questionnaire on Habitual Physical Activity[2] was administered at examinations 5 and 6. From this questionnaire, scores for physical activity during work, sports and leisure time were computed.

Analysis

To examine the effect of life events, the change in body mass index during follow-up periods of one year and two years was compared among subjects that experienced few, intermediate or many life events. The respondents were

assigned to these three subgroups as follows. For each subject, five life-event scores were computed; each score indicated the number of life events experienced during a six-month period. The scores concerned the six-month periods preceding each of the examinations 3-7 (Figure 1). The highest of the five life event scores was determined for each subject. The tertiles of this highest life-event score, 6 and 8 for men and 5 and 7 for women, were used as criteria to classify the total sample.

To be able to analyze simultaneously the change in body mass index of all subjects, for each subject the data from one of the examinations 2-6 was used as the benchmark for the body mass index and other variables. For subjects with many life events (more than the 66th percentile of the highest life-event score), the first examination prior to the six-month period with the highest number of life events was used as the bechmark examination. For subjects with few life events (less than the 34th percentile of the highest life-event score) the first examination prior to the six-month period with the fewest life events was used as the benchmark examination. For the remaining subjects in the intermediate range of life events, the benchmark examination was chosen randomly.

The body mass index at the benchmark examination (the benchmark or initial body mass index) was used for the computation of the two evaluation variables, i.e. the change in body mass index in the first year and first two years succeeding the benchmark examination. The latter variable could only be computed for subjects having examination 2, 3 or 4 as their benchmark (Figure 1).

Effect modification and confounding

The effect of life events was assessed by multiple linear regression as follows:

$$Y = a + b_1 LE_{low} + b_2 LE_{high} + \Sigma c_i C_i + \Sigma(d_j M_j + e_{1j} LE_{low} * M_j + e_{2j} LE_{high} * M_j) + R$$

where Y = change in body mass index, LE_{low} and LE_{high} = indicators for the subgroups low and high in life events, respectively, C_i = confounder, M_j = effect modifier and R = residual.

The initial body mass index (lower or higher than median), initial age (20-24,25-29,30-34 yrs), socio-economic status (low, middle, high), season in which the life events were experienced, and reported dieting behavior during the follow-up period (i.e. one year or two years), were considered as potential modifiers of the effect of life events (i.e. the coefficients b_1 and b_2, respectively) in this additive model. As an adaptation of the method described

by Kleinbaum et al.[19], an effect modifier was retained in the model for both follow-up periods and for both life-event groups if its F-to-enter in the final model of the backward elimination procedure was significant ($P<0.10$) for at least one of the follow-up periods, or if it was significant for at least one of the two life-event groups. If potential effect modifiers were not retained in the model as such, they were retained as main effect, to control confounding. In addition, changes in smoking behavior (start or stop) during the two follow-up periods, were included as confounders (two dummy variables) in the analysis.

In a second analysis, indices of physical activity (at the benchmark examination) were studied as modifiers of the effect of life events. The main effect and interactions were added to the model that resulted from the procedure described above; this model included all confounders and a subgroup of modifiers.

RESULTS

Table 1 shows the change in body mass index for the three life-event groups during follow-up periods of one year and two years. The table shows that after two years of follow-up, the gain in body mass in men with many or with few life events was slightly larger than in men with an intermediate number of life events (the intermediate group). In women with few life events the gain was lower than in women with many life events after one year of follow-up, but this was no longer the case after another year of follow-up. Overall, the differences between the three life event groups were small.

Table 1: Change in body mass index (kg/m^2), according to the experience of life events (LE)

		Duration of follow-up:			
		1 year		2 years	
MEN	N	mean	SE	mean	SE
LE:few	180	+0.21	0.06	+0.41	0.07
intermediate	108	+0.30	0.08	+0.23	0.10
many	85	+0.24	0.10	+0.40	0.13
WOMEN					
LE:few	173	+0.07	0.06	+0.29	0.09
intermediate	134	+0.17	0.09	+0.39	0.11
many	116	+0.27	0.10	+0.31	0.14

The result of the multiple regression analysis is given in Table 2. The following factors appeared to modify the effect of life events on the body mass index in men: initial body mass index, high socio-economic status, season in which the life events were experienced and attempts to lose weight during the follow-up period. In women, age and season were influential. The remaining male subgroup (reference category) thus consisted of men whose initial body mass index was below the median, who were of middle or low socio-economic status, who experienced the life events during the winter and who did not diet during the follow-up period. The remaining female subgroup (reference category) consisted of women 25-30 years of age who experienced the life events during the winter. During the first year of follow-up, men with few or with many life events in the reference category had more gain in body mass, than was found in the intermediate group of the reference category. During the summer, the change in body mass of men who experienced few life events was unaffected. In men of high socio-economic status many life events were found to have no effect. After two years of follow-up, no apparent effect of life events was present in any of these subgroups, except that men with many or few life events, who tried to lose weight by dieting during all or part of the follow-up period gained more body mass than men who experienced an intermediate number of life events and had dieted.

During the first year of follow-up women in the reference category with many life events gained more body mass than was found in the intermediate group of the reference category (Table 2). One year later, no effect of life events was present in any of the subgroups, except in women 30-34 years old who experienced few life events during the winter; they lost more body mass than women of the same age who experienced an intermediate number of life events during the winter.

In conclusion, during the first year of follow-up there was a gain in body mass in several subgroups of men and women who experienced many life events. In several subgroups of men the experience of few life events induced a similar effect. After another year of follow-up, this gain in body mass was still present only in the subgroup of dieting men.

For two examinations, the modifying effect of physical activity during work, sport and leisure time was estimated. An indication was found that physical activity during work prevented any effect from few life events in men ($P=0.04$) or from many life events in women ($P=0.03$).

Table 2: The adjusted[1] difference between change in body mass index (kg/m^2) of subjects experiencing few or many life events (LE) compared with subjects experiencing an intermediate number of life events

	Duration of follow-up: 1 yr			Duration of follow-up: 2 yr		
	P[2]	Differential change	95%CI	P	Differential change	95%CI
MEN			N=540			N=350
Reference category						
LE:few		+0.45	+0.07,+0.84		-0.12	-0.75,+0.50
many		+0.43	+0.06,+0.80		+0.26	-0.38,+0.90
Modifiers:						
BMIini						
LE:few		+0.64	+0.25,+1.03		+0.13	-0.50,+0.77
many		+0.29	-0.08,+0.66		+0.39	-0.26,+1.03
SES:high						
LE:few		+0.37	-0.05,+0.80		+0.04	-0.63,+0.71
many	x	-0.06	-0.50,+0.37		+0.06	-0.69,+0.81
Summer						
LE:few	x	+0.06	-0.29,+0.40		-0.11	-0.62,+0.40
many		+0.44	+0.09,+0.80		-0.10	-0.64,+0.45
Dieted						
LE:few	x	+1.70	+0.81,+2.59	x	+1.29	+0.01,+2.57
many	x	+1.23	+0.53,+1.92	x	+1.76	+0.49,+3.04
WOMEN			N=658			N=395
Reference category						
LE:few		+0.15	-0.23,+0.53		-0.48	-1.12,+0.16
many		+0.57	+0.21,+0.94		-0.07	-0.78,+0.63
Modifiers:						
Age:20-24						
LE:few		+0.11	-0.38,+0.59		-0.61	-1.41,+0.20
many	x	+0.13	-0.28,+0.54		+0.03	-0.72,+0.78
Age:30-34						
LE:few	x	-0.27	-0.66,+0.11		-0.83	-1.44,-0.21
many	x	+0.08	-0.31,+0.46		+0.15	-0.53,+0.82
Summer						
LE:few		+0.22	-0.11,+0.54	x	+0.23	-0.27,+0.73
many		+0.61	+0.27,+0.95		+0.08	-0.52,+0.67

1 Adjusted for: initial age (20-24,25-29,30-34), initial body mass index (BMIini),socio-economic status (SES:low,middle,high), season in which the life events were experienced (1 dummy), change in smoking behavior (two dummies).

2 x indicates: regression coefficient of the interaction-term significant (P<0.05).

DISCUSSION

The two most striking observations of the present study were, first, that in addition to the experience of many life events the experience of few life events was associated with a gain in body mass during the first year of follow-up in several subgroups of men, and, secondly, that in the subgroups that showed a stress-induced gain in body mass during the first year of follow-up, this gain was effectively counterbalanced by regulatory mechanisms during further follow-up, except for the subgroup of men who tried to lose weight by dieting. In this subgroup the gain seemed to be permanent.

The situation of an event being desired or anticipated but not occuring (a non-event[14]) might generate a state of emotional arousal comparable to that evoked by the occurrence of the event. In combination with the effect of many life events, an optimal level of stimulation by life events seems to be present with respect to the body mass changes in men. This U-shaped relationship between life events and gain in body mass was not present in women.

The modification of the effect of life events by dieting, as found in men even after two years of follow-up, can be interpreted in two different ways. (1) As a result of the experience of few or many life events, subjects gained so much weight that they subsequently tried to lose it again by dieting; attempts that provisionally failed. (2) Another factor induced the weight gain, and in response the subjects started to diet. The attempts to lose weight were, however, less successful in subjects with many or few life events than in the intermediate group.

The second interpretation is supported by the finding that in initially successful dieters, weight loss was maintained worst in subjects who experienced more life change events during follow-up[21,42]. This finding suggests that experiencing many life events might modify the effect of dieting: many life events break down the cognitive restraint of the dieters. The experience of few life events might have a similar effect. However, in an earlier analysis of the same data in which the effect of dieting was studied, we did not find a modification by few or many negative life events (Chapter 5) and, though we did not study the effect of the total number of life events, this earlier result supports interpretation (1) above: a direct effect of life events on body weight, which at least provisionally cannot be undone by dieting.

From the literature it is not obvious why arousal invoked by few or many life events, should induce a gain in body mass, if only temporarily. On the basis of experiments on animals and humans it has been proposed that stress

induces a state of arousal (e.g. an electrocardial response, or a state of activation) as a behavioral response, which is channelled by salient external cues[29,30,31,37]. In the presence of palatable ready available food cues, eating may be stimulated, though it is not clear whether this eating reduces the state of arousal or is a learned response with its own reinforcing values[29,37].

We found no long-lasting effects of many life events in women, and the experience of few life events seemed to induce a permanent weight loss rather than a weight gain. This suggests that studies on the relationship between emotional arousal and increased eating behavior have unjustly concentrated on women; the effect may be stronger in men. Several studies have shown that compared with men, women rate life events as more stressful, requiring more adjustment[5,18]. Whereas the neuroendocrine response to experimental stressors has been shown to be lower in women than in men, women have been found to be more inclined to verbalize their experience of stress than men[12]. Thus, there may be a sex difference in coping behavior; women being more verbal and men more somatic.

In either sex, subjects with a body mass index above the median showed no differential effect of life events on change in body mass, indicated by the lack of significance of the interaction term. Since some studies suggest that negative emotional arousal is larger in overweight subjects than in subjects of normal weight[20,22], the response to this arousal might have been larger in the subgroup with a body mass index above the median than in the subgroups with a body mass index under the median. Our finding that this was not the case clearly does not exclude the possibility that the long-term weight gain that is found as a result of life events in some male subgroups, may actually have played a role in the weight history of men who now have a high body mass index.

The present study showed that the level of physical activity may prevent a gain in body mass following the experience of life events. This supports the findings by Roth and Holmes[33] who showed that life events were related to poorer subsequent physical health (e.g. self-reported ailments, doctor visits, use of medication) in subjects with a low level of physical fitness (aerobic capacity).

Since its inception the methodology of life events research has been discussed. Several topics in these discussions are relevant to the present study. First; some life events experienced during the preceding six months may be forgotten. Though the optimum period is not yet known[26], even a six-month period may be too long to recall life events[24]. Since underreporting may increase the amount of misclassification in tertiles, it may introduce a bias

towards the null in the estimated effect, and thus decrease the association between exposure to life events and change in body mass.

Secondly; several studies[32,34] have favored a weighting of life events, by using the negative life-event score. Though we do not present the results here, we analyzed our data for both the total sumscore of life events and the subsumscore of undesirable life events, as used in a former report[41]. Contrary to our expectations, the total sumscore of life change was followed more clearly by systematic changes in body mass index than was true for the undesirable life event score. Thus, in confirmation of the first concept of Homes and Rahe[17] the score for the total life change seems to be the most relevant with respect to the development of overweight.

Thirdly; other factors that may modify the effects of life events were not considered in the present study, such as: social support, experience, anticipation, coping behavior, locus of control, ability to affect the occurrence of the event[10,34]. These specifications were not included because the between-examination period of six months might have been too long to obtain a valid response and the item-specific recording might have been too demanding for the participants, who completed the questionnaire seven times during the four years of follow-up.

Without taking these modifiers into account, the present study showed that the effect of life events on body mass is mostly effectively counterbalanced by regulatory mechanisms. However, the finding that in dieting men with few or many life events a long-lasting gain in body mass was present compared to dieting men with an intermediate number of life events reveals an impact of life events on the etiology of overweight.

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7 BODY MASS INDEX IN PAROUS WOMEN

Maartje A. Rookus, Peter Rokebrand, Jan Burema, Paul Deurenberg

ABSTRACT

The effect of pregnancy on the development of overweight was studied. The change in the body mass index from pregestation through 9 months postpartum of 49 pregnant women was compared with the change in the body mass index during the same period of follow-up in 400 non-pregnant women.
All women participated in a four-year follow-up study in which body weight was measured every six months. The body weight of the pregnant women was also measured at 6 and 12 months postpartum.
Nine months postpartum women who breastfed their child for more than two months gained +0.6 kg/m^2 (90%CI:+0.1,+1.0) more body mass than expected from aging. This difference was not present in women who did not breastfeed their child or who breastfed for a period shorter than 2 months. Women who used bromocriptine to stop lactation lost body mass (-0.5 kg/m^2, 90%CI:-1.1,+0.0), unexpectedly. These observations suggest that 'maternal obesity' may especially occur in women who breastfeed their children for long periods.

INTRODUCTION

Sheldon[24] was the first to draw attention to the development of obesity in association with pregnancies. This 'maternal obesity' has often been reported by women requesting medical assistance with weight reduction[7,16,22]. Cross-sectional studies have also shown parity to be an independent determinant of overweight in child-bearing years[2,13,23] as well as in middle age[4,8,13,19,23]. Maternal obesity may be the result of factors directly related to the pregnancy itself, or may be the result of the changed life-style associated with having children. The latter possibility has been suggested by the association between parity and overweight found in women as well as in men[23]. Thus, longitudinal studies are necessary for a better understanding of

maternal obesity.

Compared with the large number of longitudinal studies on weight gain during pregnancy and its relation to birth weight, pregnancy as an etiologic factor of obesity in women has been relatively neglected. The longitudinal studies that have been conducted on this subject generally include an impressive number of women, but suffer from methodological drawbacks.

First, the chief difficulty in assessing the effect of pregnancy on body weight development is that of obtaining a valid measurement of body weight prior to pregnancy[15]. Often without much consideration, a reported/estimated pre-pregnancy body weight[1,18] or measured/estimated body weight at several weeks of gestation[11,15] has been used as the benchmark value.

Studies on the validity of self-reported body weights have shown that women underestimate their weights more than men (mean: 1.4 kg versus 0.7 kg) and that this underestimation increases as actual weight increases[25]. This bias might be compounded if body image is distorted during pregnancy[20]. An estimation of pre-pregnancy body weight or an estimation of body weight at several weeks of gestation are based on the assumption that weight gain early in pregnancy is independent of age and parity. Though this may be true for groups of women, individual changes in body weight computed from this estimated weight may be distorted.

A second methodological drawback of the longitudinal studies that have been done on body weight development associated with pregnancy, is the lack of an adequate control group, necessary to control for the weight gain that usually occurs with increasing age. If aging is adjusted for, this adjustment is based on cross-sectional data of body weights in primiparae early in gestation[5], or it is based on the cross-sectional data of estimated pre-pregnancy weights[18]. To date, only one study has used longitudinal data; the body weight changes of the pregnant women themselves, 12-24 months postpartum[15].

Thirdly, the unit of analysis in studies relating pregnancy to the development of obesity may be the pregnancy, not the women[18]. In consequence, dependent variables are unjustifiably treated as being statistically independent.

As part of an extensive four-year follow-up study of the development of obesity in young adults the changes in the body mass index (weight/height^2) of women who became pregnant and the changes in non-pregnant women were compared.

METHODS

Between 1980 and 1985 an extensive follow-up study was conducted in the county of Ede, the Netherlands (for details see: reference 2 and Chapter 3). In spring 1980, all residents born in 1948-1950, 1953-1955, 1958-1960 were invited by mail to participate. About half of the respondents in 1980 (initially 755 men and 915 women) were examined at home twice a year from spring 1981 through spring 1984 (Figure 1).

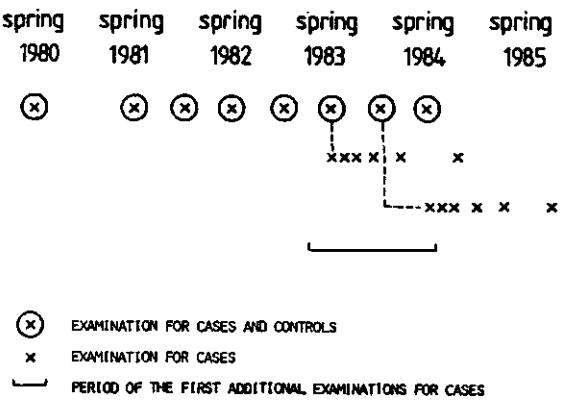

Figure 1: Design of the study

Women participating in this follow-up study and pregnant during at least one of the two examinations in 1983 (the cases), were additionally examined at home (or in hospital) every fortnight from the seventh month of pregnancy until delivery, and subsequently, ten days, two months, six months and twelve months after delivery. Changes in body mass index (weight/height^2) from spring 1980 through spring 1984 of the cases were compared with those of women who did not become pregnant (the controls).

At each examination, body weight without shoes and in light indoor clothing was measured to the nearest 0.5 kg on a scale that was calibrated weekly, and a questionnaire that had been mailed in advance was checked for completeness. Body height without shoes was measured to the nearest 0.1 cm with a Microtoise in spring 1980.

The change in body mass index was computed as the difference of two means (designated the initial and final body mass indices, respectively). By taking

the mean of two measurements, six months apart, the seasonal fluctuation in body weight[27] (Chapter 4) was excluded. For each case, the initial body mass index was the mean of the two last measurements in a non-pregnant state prior to the pregnancy at issue. The initial body mass index applied to a date (T_{ini}) at least nine months after any preceding pregnancy. The final body mass index (applying to T_{fin}) was the mean of the measurements taken six months and one year after delivery. The length of time between T_{ini} and T_{fin} is designated the index-period, which mean was 1.75 year (SE:0.02 yr). In order to obtain a similar index-period for the controls, the initial body mass index of this group was taken as the mean of the measurements in autumn 1981 and spring 1982 and the final body mass index was taken as the mean of the measurements in autumn 1983 and spring 1984. This resulted in an index-period of two years for the controls.

Sample

The follow-up study started with 915 women. Of these, 646 women participated in all half-yearly examinations from spring 1981 through spring 1984 and, of them, 400 women (the controls) did not give birth to a child from spring 1981 through spring 1984. Possible abortions in the control group could not be controlled for.

During the examinations in 1983, 63 women indicated to be pregnant. All agreed to participate in additional examinations. Of these, data were incomplete for four women, six women were pregnant twice in succession and four women had an abortion. For nine women peripartum weight measurements were not available, but these variables were not used in further analyses. So, sufficient information was obtained for 49 women (the cases). This number of cases was small compared with the number of controls, but we found no reason to select a smaller control group; the controls were available all the same and selection would only have decreased precision.

Questionnaire

Table 1 shows the variables in addition to height and weight that were obtained by questionnaire. The highest level of education that was attained in spring 1984 was used as an indicator of socio-economic status. Three levels were distinguished: low level, comprising primary (grade) school and primary occupational training, middle level, comprising secondary (middle) school and secondary occupational training, and high level, comprising university and tertiary occupational training.

Table 1: Distribution of pregnancy-specific variables in 49 women, 22-35 years of age at delivery

Variable	Original categories N	Combined* categories N	Variable	Original categories N	Combined* categories N
Parity prior to pregnancy at issue			Medicines after delivery		
0	17	36	bromocriptine	10	11
1	19		ergometrinmaleate	5	
2	10	13	irontablets	8	
⩾2	3		antibiotics	3	38
			other	4	
			none	27	
Complications during delivery			Duration of breastfeeding**		
vacuum/forceps	6	12	no	13	31
Caesarian	6		1-8 weeks	18	
other	4	37	2-6 months	8	18
no	33		> 6 months	10	
Season at delivery			First use of oral contraceptives after delivery		
winter	24		0-6 months	14	19
summer	25		7-12 months	5	
Sex of child			no use during first year	30	30
boy	29				
girl	20				

* categories similar with respect to change in body mass were combined in the regression analyses.
** either or not supplemented with other food for the child.

If a woman was a smoker (> 1 cigarette/day) on at least one of the two examinations at which the initial or final body mass index was based, she was designated a smoker at T_{ini} or T_{fin}, respectively. Based on the smoking behavior at T_{ini} and T_{fin} the smoking behavior during the index-period (smoker, non-smoker, stopped smoking, started smoking) was determined. The employment status during the index-period was defined similarly.

Analysis

To explain differences in change in body mass index across pregnant women, a stepwise multiple linear regression analysis (forward selection and backward elimination) was done on the pregnancy-specific dummy variables, listed in

elimination) was done on the pregnancy-specific dummy variables, listed in Table 1, and either birth weight or weight loss during delivery and early puerperium (the difference between the last body weight measured prepartum and body weight at ten days postpartum). To avoid multicollinearity the last two variables (each used as a linear and quadratic term) were included in two separate analyses. In both analyses, breastfeeding for longer than two months (henceforth designated 'long-term breastfeeding') was forced into the model, because this variable was considered too important to be excluded if it appeared not to be significant (α=0.05).

Because the index-period varied across pregnant women, it was accounted for in the analysis by using the index-period as a confounder in the model, and simultaneously setting the intercept to zero (regression through the origin). A similar multiple regression analysis was done to compare the change in body mass index of pregnant with non-pregnant women, adjusted for confounders. Based on the results of the analysis in pregnant women, this group was divided into subgroups for each of which a regression coefficient was estimated. Corresponding to a one-sided test, 90%-confidence intervals were computed for these coefficients.

RESULTS

Figure 2 shows the measurements of the body mass index for the pregnant women for whom all weight measurements were available (N=40), and the non-pregnant women. Based on the initial and final body mass index, the cases gained 0.61 kg/m^2 (SE:0.15,N=49) whereas the non-pregnant women gained 0.27 kg/m^2 (SE:0.05,N=400) during the index-period. The difference was significant (P<0.05).

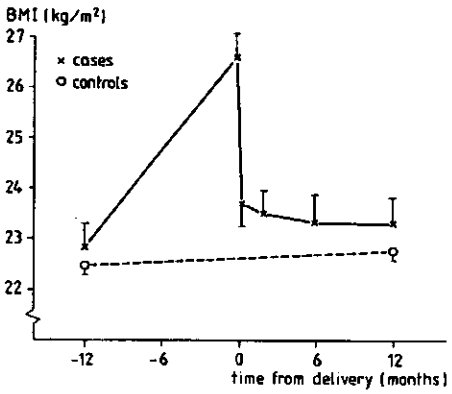

Figure 2: Development of the body mass index of cases (N=40) and controls (N=400)

At 12 months postpartum menstruation had returned in all cases except one. Mean birth weight was 3490 g (SE:84,N=49) and mean weight loss during delivery and early puerperium was 8.0 kg (SE:0.4,N=40).

In Table 1 the distributions of other pregnancy-specific variables are given. Most women (N=36) in our study were pregnant for the first or second time. Categories that appeared to have a similar influence on changes in body mass index, were combined.

Table 2 shows the results of regression analyses on the pregnancy-specific variables. The only coefficient that was significant, was the coefficient of use of bromocriptine and/or ergometrinmaleate (further designated 'medicines'). The former medicine stops lactation by inhibiting the secretion of prolactin. The latter causes contraction of the uterus. Four of the five subjects who used ergometrinmaleate also used bromocriptine, and none of those who used either or both did breastfeed their child for longer than two months. Use of medicines was associated with a marked loss in body mass, compared with the group who did not use these medicines and who breastfed their child for 0-2 months. Compared with the latter group, women who gave breastfeeding for longer periods displayed a similar change in body mass.

Table 2: The adjusted* effect of pregnancy-specific variables on the change in body mass index (kg/m^2) within the group of 49 pregnant women

Breastfeeding	Medicines**	N	Regression coefficient	95%-Confidence interval
> 2 months	no	18	+0.13	-0.50,+0.76
0-2 months	yes	11	-0.73	-1.43,-0.03

* adjusted for index-period (yr) and age at delivery (32-35 versus 22-29 year of age).
** bromocriptine and/or ergometrinmaleate

Subsequently, the three lactation categories (no use of medicines plus breastfeeding for 0-2 months, or more than 2 months, and use of medicines) were compared with the non-pregnant women, controlling for age, level of education, parity, employment, and smoking status (see Appendix). Table 3 shows the differential mean gain in body mass in the three groups, as compared with the mean gain in the controls. After correction for confounders, women who breastfed their child for 0-2 months and did not use medicines tended to gain

as much body mass as was expected from aging. Women who breastfed their child for longer than two months gained more body mass than expected from aging, whereas women who used medicines tended to lose body mass, unexpectedly.

Table 3: The adjusted* effect of pregnancy versus non-pregnancy on the change in body mass index (kg/m^2), based on 49 pregnant and 400 non-pregnant women

Breastfeeding	Medicines**	N	Regression coefficient	90%-Confidence interval
> 2 months	no	18	+0.56	+0.09,+1.04
0–2 months	no	20	+0.20	-0.26,+0.66
0–2 months	yes	11	-0.54	-1.10,+0.03

* adjusted for index-period (yr), age at T_{fin} (23-25,28-30,33-36 years), socio-economic status (low,middle,high), parity (initially 0, ≥1 children), whether the woman had given up work (yes/no), smoking status (started, stopped, continued, no smoking): for details see Appendix.

** bromocriptine and/or ergometrinmaleate.

DISCUSSION

Several longitudinal studies of the impact of pregnancy on body weight have been conducted (Table 4). These studies show that pregnancy is generally associated with an additional weight-gain. Even after correction for aging, the net weight gain ranged from +0.5 to +2.5 kg[5,15,18]. In addition to differences in estimation of pre-gestational weight and differences in age-correction, part of this range in weight gain may be attributed to differences in smoking behavior[10] and differences in breastfeeding practices and associated use of medicines, as found in the present study; after correction for age, smoking behavior and other confounders, the only women who gained more body mass than expected from aging, gave breastfeeding for more than 2 months.

The effect of breastfeeding on maternal body weight has been investigated in several studies, but the findings are inconclusive. It has been reported that women who breastfed their child for longer periods lost more weight postpartum[9,15], but this is not a uniform finding[17]. Although Sheldon[24] found no differences in frequency of former breastfeeding between women with maternal obesity and women without maternal obesity, Richardson[22] showed that women who developed maternal obesity had breastfed their child more frequently. The findings of Richardson are in accordance with those of Newcombe[18] who reported

Table 4: Longitudinal studies of the effect of pregnancy on body weight

References	Cases	Age correction	Pre-pregnancy weight	Time of weight postpartum	Pregnancy associated weight change mean (kg)
McKeown & Record[15]	1110 women; population-based sample:	yes	estimated: measures/estimated at 124 days gestation -		
	N=383		2.3 kg	12 months	2.4
	N=289			24 months	2.3
Abitbol[1]	700 clinic patients	no	self-reported	six weeks	1.8
Billewicz & Thomson[5]	5830 women; population-based sample	yes	measured at 20th week of gestation	*	0.5–1.0
Garn et al.[10]	223 smoking women	no	not indicated	*	0.5–1.3
	254 non-smoking women				1.4–1.5
Beazley & Swinhoe[3]	50 clinic patients	no	20 weeks gestation	*	1.0
Gormican et al.[11]	600 clinic records	no	measured when first seen at clinic	4–8 weeks	2.1
Newcombe[18]	35556 deliveries; population-based sample	yes	estimated: (weight measured at 20 weeks gestation)-4 kg	*	0.7

* pregnancy-associated weight change based on changes in body weight between consecutive pregnancies.

a gain of 1.1 kg/pregnancy in breastfeeding women as opposed to 0.8 kg/pregnancy in women who bottle-fed their children. In the present study, the same tendency, at least for long-term breastfeeding, was found.

There are several explanations for these observations. To provide enough energy for breastfeeding, women may increase their usual energy intake[17] and may do this to such an extent that they do not completely use the fat depots they accumulated during pregnancy for lactation. This would imply that the extra weight is predominantly located in the femural region[21]. Although the observations made by Sheldon[24] do not strongly support the hypothesis that maternal obese women have excess femural fat-depots, since his study this subject has been neglected. Yet, with respect to the association between fat distribution and morbidity/mortality risks it is of much interest[6,14]; if the excess weight is located in the femural region, the risk to health of maternal obesity might be lower than general indices of obesity such as the body mass index would lead one to expect.

In contrast to breastfeeding women, bottle-feeding women may not increase but may consciously or unconsciously decrease their usual food intake postpartum. In the present study, attempts to measure patterns in restrained eating behavior during pregnancy and follow-up were unsuccessful since the answers to the Dutch Eating Behavior Questionnaire[28] under these circumstances proved to be invalid (data not presented here).

In addition, an own effect of the medicine to stop lactation, bromocriptine, might be present, though its effect on body weight is not clear. In a study of 21 patients with galactorrhoea, five subjects lost 4-18 kg in weight over 3-18 months in spite of no apparent change in food intake[26]. They had all gained this weight during the time that they had been galactorrhoeic. Harrower[12] found that the use of bromocriptine in eight women with anorexia nervosa and nine women with obesity did not result in consistent weight changes.

In the present study the 'long-term' effect of pregnancy was estimated by following pregnant women 12 months postpartum. From 6 through 12 months postpartum a small loss in body mass (-0.05 kg/m^2) still occurred. Thus, it might be possible that after some years of follow-up the effect of pregnancy is minimized. However, this seems to be unlikely, because McKeown and Record[15] found a weight gain of 0.3 kg from 12 to 24 months postpartum. Another indication that the effect of pregnancy may indeed be permanent is given by a cross-sectional study of 378 monozygotic twin-pairs. Of each pair, one twin had had at least one child and the co-twin was childless. The intra-pair difference

in body weight was 1.90 kg (SE:0.35) at 37-77 years of age[8].

Though the mean amount of body mass gained as a result of one pregnancy is small, it is found that at higher parities the effect might increase[3,5]. The present study suggest that 'maternal obesity' may especially occur in women who breastfeed their children for long periods.

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APPENDIX

Parametrization of variables in analysis of Table 3:

Variable	Code	Number of women	
		pregnant	non-pregnant
Breastfeeding 0-2 months	1	31	0
	0	18	400
Breastfeeding > 2 months	1	18	0
	0	31	400
Use of medicines	1	11	0
	0	38	400
Index-period (yr)	continuous value		
Age at T_{fin}: 23-25 years	1	14	98
	0	35	302
33-36 years	1	8	186
	0	41	214
Socio-economic status: low	1	4	76
	0	45	324
high	1	10	56
	0	39	344
Parity: ≥ 1	1	32	19
0	0	17	381
Gave up work	1	14	20
	0	35	380
Smoking status: started	1	3	0
	0	46	400
stopped	1	5	96
	0	44	304
continued	1	19	118
	0	30	282

8 THE IMPACT OF ADJUSTMENT OF A WEIGHT-HEIGHT INDEX (W/H^2) FOR FRAME SIZE ON THE PREDICTION OF BODY FATNESS

Maartje A. Rookus, Jan Burema, Paul Deurenberg, Wilhelmina A.M. van der Wiel-Wetzels

ABSTRACT

The impact of frame size categories in weight-height tables was studied by comparing the efficiency of the body mass index (W/H^2) and weight adjusted for body height and a body diameter, $W/(H^2 D^p)$, in predicting body fatness.

Body weight, body height, six body diameters and four skinfold thicknesses were measured in 95 men and 70 women, aged between 23 and 35 years. Percentage of body fat was calculated from skinfold thicknesses using regression equations according to Durnin & Womersley (1974).

The inclusion of a body diameter increased the explained variation of body fatness from 57% to 62% (knee) and 63% (shoulder) in men and from 63% to 69% (knee) in women.

It can be concluded that in the present population the efficiency of the prediction of percentage of body fat was not improved markedly by the inclusion of a body diameter in the body mass index, thus giving no support for the inclusion of frame size categories in weight-height tables.

INTRODUCTION

The inclusion of frame size categories in weight-height tables seems to be of disputable value. A criticism of the Metropolitan Life Insurance Company weight standards (1959) in respect of frame size categories was that no description of the method, used to determine these categories, was given (Brožek 1956, Seltzer & Mayer 1965). In the new Metropolitan height and weight tables (1983), elbow diameter was introduced as a measure of frame size. Recent reviews have emphasized the lack of any measurement of body diameter in the insurance examination information, resulting in frame size categories not based on frame size measurements (Knapp 1983, Garn & Hawthorne 1984). Apparently, weight differences at a given height are assumed to be attributable to frame size differences as assessed from anthropometric measurements.

It is clear that body weight should be corrected for frame size to obtain a

better indication of body fatness. The percentage of body fatness will be lower of a tall than of a short person of the same body weight and skeleton width, and similarly, lower of a person of a wide skeleton than of a narrow skeleton of the same weight and height. However, it is yet to be established whether adjustment for frame size should take into account body diameter(s) in addition to body height. Arguments for the inclusion of both body height and body diameter(s) have been put forward by several investigators. Seltzer et al. (1970) found differences in elbow and chest diameters in individuals of two relative weight classes of comparable age and height, and Frisancho and Flegel (1983) found larger differences in body weight among frame size categories based on elbow diameter than among body height categories. Several equations have been devised to estimate fat-free mass or body weight from body height and body diameters. These diameters include shoulder, chest, wrist, pelvis, hip, knee and ankle (Brožek 1956, Behnke 1959, von Döbeln 1959, Hechter 1959, Wilmore & Behnke 1968, Wijn & Zaat 1968, Forsyth & Sinning 1973).

If an estimation of frame size can be improved by using body diameter(s) in addition to body height, then the next question is, to what extent can body diameter(s) improve a weight-height table to make the recommended weight range more appropriate. In other words, as a weight-height table can be considered as a categorized weight-height index, to what extent can the prediction of body fatness from a weight-height index be improved by the inclusion of a body diameter.

Baecke et al. (1982) estimated body weight from height, knee, and wrist diameters and used this weight estimate in an index of relative weight, giving $W/\hat{W}$, where W is body weight and $\hat{W}$ is estimated weight. The improvement of the frame size correction by the addition of body diameters was evaluated by comparing $W/\hat{W}$ with weight-height indices in predicting body fatness. No indication was found that frame size categories based on knee and wrist diameters improved this prediction.

In the present study, additional body diameters were taken into account, some of which have been suggested recently to be frame size indicators (Katch and Freedson 1982, Metropolian Life Insurance Company 1983, Frisancho & Flegel 1983). A further reason is that Behnke (1959) indicated that limb and trunk diameters together give the best estimation of fat-free mass. For evaluation of the diameters used as frame size indicators in weight-height tables, an analysis procedure somewhat different to that of Baecke et al. (1982) was developed. The body mass index ($BMI=W/H^2$) was selected as reference index because of its high correlation with measures of body fatness found in several populations (Keys et

al. 1972, Womersley & Durnin 1977, Frisancho & Flegel 1982). Percentage of body fat was estimated by the log sum of four skinfold thicknesses (Durnin & Womersley 1974). To assess whether an index consisting of weight, height, and one body diameter is more efficient than the body mass index in predicting percentage of body fat, an alternative index was constructed of the form $W/\hat{W}$, where $\hat{W}$ is an estimation of body weight from both body height squared (H^2) and body diameter to exponent p (D^p);

$$\hat{W}=c\ D^p\ H^2, \qquad (1)$$

where c is a constant. Then the efficiency of the new index and the body mass index to predict body fatness was compared. Some anthropometric body diameters may be biased by the subcutaneous fat layer and give a biased estimate of body weight, which may well result in overcorrection in the index $W/\hat{W}$, and thus not improve the estimation of body fatness. In other words, an expected improvement in prediction of body fatness by the inclusion of body diameters in addition to body height as frame size indicators in $\hat{W}$, may well be nullified by the fact that some body diameters and body fatness are correlated. Since the contribution of body height to the body mass index (W/H^2) and to $W/\hat{W}$ ($\hat{W}= cD^p\ H^2$) is identical, a difference in the estimation of body fatness is due entirely to the corresponding body diameter only.

METHODS

Population and procedure

In November 1983, participants in a project on overweight being conducted in the municipality of Ede, the Netherlands (Baecke et al. 1982) were invited to participate in the present study. Complete information was obtained from 95 men and 70 women, aged between 23 and 35 years and of various levels of education. Pregnant women were excluded from the study population.

All subjects were invited by mail to visit a mobile research unit, which was stationed in their respective section of the municipality for seven days. The anthropometric parameters were measured at the mobile research unit. For various reasons, 18 of the men were unable to attend the unit and were thus measured at home.

Measurements

Body height and body weight without shoes and in scant clothing only were measured to the nearest 0.1 cm and 0.1 kg, respectively. Body weight of those visited at home was measured to the nearest 0.5 kg. All measurements were made between 16.00 and 20.00 hours.

The diameter of knee, wrist and elbow was measured on both sides of the body according to Weiner and Lourie (1969). If the difference between the left and right side measurement was greater than 5%, then the measurement was repeated. The sum of left and right measurement was used in analyses. The shoulder and pelvic diameters were measured according to Keys (1967), and the hip diameter according to Wilmore and Behnke (1969). The chest diameter was not measured because it is subject to large degree of measurement error (Katch & Freedson 1982, von Döbeln 1959). All diameters were measured to the nearest 0.1 cm using a GPM spreading caliper.

Duplicate measurements of the biceps, triceps, suprailiac, and subscapular skinfold thicknesses were made according to Durnin and Rahaman (1967), on the left side of the body to the nearest 0.2 mm using a Holtain skinfold caliper. Body fat was calculated as a percentage of the total body weight from the average log sum of the four skinfold thicknesses, using linear regression equations for men and women aged from 20 to 29 years available from Durnin and Womersley (1974). All measurements were made by one examiner for each sex. As the data were analysed for men and women separately, it was not necessary to make adjustment for possible observer bias.

Analysis

An alternative index for body fatness, $W/\hat{W}$, was constructed where W is body weight and $\hat{W}$ is body weight estimated from body height squared (H^2) and a body diameter to the exponent p (D^p). To estimate body weight, regression coefficients b_k (with corresponding intercept a_k) were estimated from a linear regression of ln body mass index on ln body diameter D_k (k=1,..,6)

$$\ln(W/H^2) = a_k + b_k \ln D_k + \text{error} \qquad (2)$$

$$\hat{W}_k = \exp(a_k)\, D_k^{b_k}\, H^2. \qquad (3)$$

Subsequently, the efficiency of each index $W/\hat{W}$ in predicting body fatness was compared with the efficiency of the body mass index to make the same prediction. The relationships were examined visually. The evaluation criterion was the

residual variance of body fatness after adjustment for the body mass index and the respective $W/\hat{W}$ indices. Since these indices were evaluated against the same observations of the external criterion, body fatness, comparison of unexplained variances is equivalent to comparison of correlation coefficients (proportion of variance explained). As these correlation coefficients were not pairwise independent, the difference of the two correlation coefficients was tested according to Olkin and Siotani (1976) with the asymptotically standard normal test statistic z*:

$$z = (r_{13} - r_{12})\,[\mathrm{var}(r_{13} - r_{12})]^{-1/2}, \qquad (4)$$

where,

$$\mathrm{var}(r_{13}-r_{12}) = N^{-1}[(1-r_{12}^2)^2+(1-r_{13}^2)^2-2r_{23}-(2r_{23}^3-r_{12}r_{13})(1-r_{12}^2-r_{13}^2-r_{23}^2)] \qquad (5)$$

and N=number of subjects, r = correlation coefficient between two X variables, X_1=percentage of body fat, X_2 = the body mass index, $X_3 = W/\hat{W}$.

Table 1. Anthropometric variables in men and women aged between 23 and 35 years (Mean values with their standard errors)

	♂ (n=95)		♀ (n=70)	
Variable	Mean	SE	Mean	SE
Body-weight (kg)	76.0	1.0	61.4	0.9
Body-height (m)	1.792	0.007	1.675	0.007
BMI (kg/m^2)	23.6	0.3	21.9	0.3
Body fat (%)**	19.5	0.5	26.1	0.6
Diameter (mm)				
Knee (left and right)	195	1	172	1
Wrist (left and right)	121	1	100	1
Elbow (left and right)	139	1	117	1
Shoulder	394	2	347	2
Pelvis	282	2	281	2
Hip	325	2	314	2

BMI, body mass index (weight-$height^2$).
** Estimated from skinfold measurements.

* In the present study ($r_{12} \approx r_{13} \approx 0.8$ and $r_{23} \approx 0.97$), the statistic defined by Equations 4 and 5 gave similar results as a statistic proposed by Hotelling (1940). This statistic is also defined by Equation 4, but here

$$\mathrm{var}(r_{13}-r_{12}) = (N-3)^{-1}[1-r_{12}^2-r_{13}^2-r_{23}^2+2r_{12}r_{13}r_{23}].$$

RESULTS

The mean with its standard error of each anthropometric variable is given in Table 1, and Table 2 gives the regressions of the ln BMI on ln body diameter (for each body diameter). The correlation coefficients show that shoulder and pelvic diameters in men and pelvic and hip diameters in women gave the best estimate of the body mass index. Thus, these body diameters together with body height, gave an estimation of body weight, $\hat{W}$, that was most closely related to measured body weight.

Table 2. Regression models with the ln body mass index (weight/height^2; kg/m^2) as the dependent variable and the respective ln body diameters (k=1,...6) (cm) as the explanatory variable

Sex	Body diameter	Intercept (a_k)	Regression coefficient (b_k)	Correlation coefficient r
♂ (n=95)	Knee (left and right)	1.96	0.40	0.19
	Wrist (left and right)	3.14	0.01	0.00
	Elbow (left and right)	2.32	0.32	0.17
	Shoulder	0.82	0.64	0.33
	Pelvis	0.85	0.69	0.34
	Hip	1.60	0.45	0.21
♀ (n=70)	Knee (left and right)	0.95	0.75	0.28
	Wrist (left and right)	3.43	-0.15	0.08
	Elbow (left and right)	2.47	0.25	0.11
	Shoulder	2.53	0.15	0.06
	Pelvis	-0.33	1.02	0.57
	Hip	-0.84	1.14	0.55

The difference of the correlation coefficient between $W/\hat{W}$ and body fatness and the correlation between the body mass index and body fatness, show whether $\hat{W}$ is more efficient than body height squared in adjusting body weight for frame size (Table 3). The limits of the 90% confidence interval of this difference are given. Some of the body diameters, which gave the best estimates of body weight, were shown to be poor in the new index. Indices based on pelvic and hip diameters did not improve estimation of body fatness from the body mass index. Knee and shoulder diameters in men and the knee diameter in women improved the estimation of body fatness from the body mass index significantly (lower limit of the 90% confidence interval > 0).

Table 3. Product-moment correlation coefficients (r) of the the body mass index (BMI, weight/height^2; kg/m^2) and weight/estimated weight ($W/\hat{W}$)* with percentage of body fat

Sex index	r	z +	90% Confidence interval‡ lower limit	 upper limit
♂ (n=95)				
BMI	0.758	-	-	-
$W/\hat{W}$:				
knee (left and right)	0.785	2.158	0.006	0.048
wrist (left and right)	0.759	0.334	-0.004	0.006
elbow (left and right)	0.765	0.630	-0.011	0.025
shoulder	0.794	1.648	-0.000	0.072
pelvis	0.726	-1.327	-0.072	0.008
hip	0.746	-0.866	-0.035	0.011
♀ (n=70)				
BMI	0.795	-	-	-
$W/\hat{W}$:				
knee (left and right)	0.828	1.677	0.000	0.066
wrist (left and right)	0.787	-1.201	-0.019	0.003
elbow (left and right)	0.802	0.817	-0.007	0.021
shoulder	0.794	-0.218	-0.009	0.007
pelvis	0.729	-1.507	-0.137	0.007
hip	0.704	-2.030	-0.165	0.017

* weight is predicted from $\hat{W} = \exp(a_k)(\text{diameter}_k)^{b_k}\ \text{height}^2$.
\+ Standard normally distributed, see analysis.
‡ Limits of two sided 90% confidence interval of the difference between the correlation coefficient of body fatness and $W/\hat{W}$ (r_{13}) and the correlation coefficient of body fatness and the body mass index (r_{12}) are $(r_{13}-r_{12}) \pm 1.66\ s$, where $s = [\text{var}\ (r_{13}-r_{12})]^{1/2}$.

To illustrate what this improvement means in practice, the regression was carried out of body fatness on $W/\hat{W}$ based on body diameters that gave the best estimates, i.e. knee and shoulder. By means of the resulting regression coefficients [b_{knee} : 35.58 (men) and 36.02 (women), $b_{shoulder}$: 37.34 (men)] the effect of a difference in diameter on the prediction of body fatness can be examined. Consider, for example, two men of equal body weight and height (group means) but of different knee diameter, [15th percentile (9.2 cm) and 85th percentile (10.3 cm), respectively]. On the basis of height and weight only, the percentage of body fat is estimated to be 19.5%, and taking knee diameter into account, it is estimated to be 20.4% for the man of the smaller frame size and 18.8% for the other. Thus, even with this large difference in knee diameter, height and weight being equal, the measurement of an additional parameter results in a estimated difference of only 1.6% in percentage of body fat. In

other words, for 70% of the population, frame size adjustment by including the knee diameter, accounts for no more than approximately 0.8% body fatness, where the standard deviation of body fatness is about 5%. It can be shown that the same difference in predicted body fatness will result from a relative error in the measurement of body height of 1%, that is, about 1.8 cm for a subject of median height.

To assess the potential improvement to prediction of body fatness by taking into account a diameter and also its interaction with body height (D,D*H), or an additional diameter (D_1, D_2), corresponding analyses were carried out for knee and shoulder diameters in both sexes. The improvement was found not to be greater than that obtained by using the best diameter (knee) alone, in either sex.

As suggested by Katch and Freedson (1982), in subjects of extreme body height, frame size may be determined by height alone, but this may not be the case for those in normal height ranges. To obtain support for this hypothesis, the analysis was repeated excluding those subjects of body heights less or greater than the first or ninth decile, respectively. In the total group, W/W based on the most promising diameters improved the explained variance by 5% (knee:r^2_{13} = 0.62, r^2_{12} = 0.57) and 6% (shoulder: r^2_{13} = 0.63) in men and by 6% (knee: r^2_{13} = 0.69, r^2_{12} = 0.63) in women, but in the subgroups with reduced range in body height, this was no more than 6% (knee: r^2_{13} = 0.63, r^2_{12} = 0.57) in men. The improvement when using shoulder diameter in men or knee diameter in women was no better in the subgroups than in the total group. Thus, by restricting the study population to people with more common values of body height, the increment of the small beneficial effect of using body diameters was not striking.

The improvement gained by the inclusion of a body diameter may be greater for subjects of extreme body diameters than for the total population. To study this, the analysis was repeated for subjects of a body diameter less or greater than the first or second tertile, respectively, thus excluding subjects with more common values of the body diameter. This was done for the knee and shoulder diameter, separately. The improvement in explained variation was 6% (r^2_{13} = 0.65, r^2_{12} = 0.59) for the knee diameter in men, but was not significant in the other cases, giving no support for the statement that the prediction of body fatness is better in subjects having relative small or large body diameters compared with the total population.

DISCUSSION

In 1980, the 95 men and 70 women in the present study did not differ significantly with respect to body weight, height and the body mass index from the 1667 men and 1996 women participating in a project on overweight (Baecke et al. 1983). Thus, it may be concluded that the present study population was not highly selected.

From the results, it can be concluded that indices ($W/\hat{W}$) based on knee and shoulder diameters in men and knee diameter in women, improved the estimation of body fatness from the body mass index slightly. However, while the body mass index alone explained 57% of variation in body fatness in men and 63% in women, the inclusion of an additional variable increased the proportion of explained variation to no more than 62% (knee) and 63% (shoulder) in men and 69% (knee) in women. The numerical example shows that a moderate improvement in the measurement of body height may result in an increment in precision in estimation of body fatness of the same order of magnitude as obtained by the measurement of an additional parameter.

The question can be asked whether this result depends on the way in which information on body diameters was used in the analysis. Body height and various body diameters were used to estimate body weight, $\hat{W}$, which was then used to adjust for frame size in the index $W/\hat{W}$. An alternative measure for frame size, for example, fat-free mass (FFM), which is more specific than body weight, may have been more appropriate. However, to estimate FFM ($\widehat{FFM}$), another measure for body fatness in addition to the skinfold measurements is necessary to ensure that the index $W/\widehat{FFM}$ is not dependent on the criterion, body fatness. This would introduce new errors in measurement which may weaken the possible effect. Thus, as body weight is less specific but more accurate to measure, it was decided to estimate it by means of body height and diameters, giving $\hat{W}$ (equation 3) as reference value for body weight.

To check whether exponents of the body diameter in $W/\hat{W}$, other than regression coefficient b based on weight estimation, yielded higher correlation coefficients with body fatness than found for $W/\hat{W}$, several other exponents were examined. The correlation coefficients, rounded to the second decimal, were equal to or lower than the correlation coefficient between body fatness and $W/\hat{W}$. Thus, a measure for frame size more specific than W would not yield results strikingly different from those found in the present study.

Pelvic and hip diameters were biased by body fatness, as in other studies (Behnke 1959, Pollock et al. 1975, Pollock et al. 1976). For other body diameters, also, a fat association may be a reason that inclusion does not

really improve the prediction of body fatness from the body mass index. This fat association may be an artefact of measurement, resulting from the subcutaneous fat layer, but may also be true to some extent. Keys (1967) found an association between body fatness and the sum of shoulder and hip diameters divided by body height. Katch and Freedson (1982) suggested that a causal relationship between fat storage and frame size is present in women. Fat children have been shown to be of greater height (Garn et al. 1974, Forbes 1977) and to have larger body diameters (Beunen et al. 1983) than lean children. Data from longitudinal studies have suggested a cause-effect relationship (Forbes 1977). These anthropometric studies have been affirmed by a study using radiography (Beunen et al. 1982), which suggests that fatness may be a factor accelerating skeletal growth. It is likely that this effect is weakened in adults. Only radiography can show the extent to which an association between body diameters and body fatness is a true association or whether it is an artefact of measurement. A true association will inevitably produce a bias in the estimation of body weight from frame size, but an artefact of measurement can be avoided by the use of radiography. Thus measured, body diameters may be shown to improve a weight-height index more than has been the case in the present study. However, since weight-height tables are used in situations where the measurements of skinfold thicknesses is problematic, difficulties are also likely to be encountered with radiography. Thus, for the use of frame size categories in weight-height tables, anthropometry seems to be of more practical value.

For epidemiological studies, investigators should decide whether the increment in precision warrants the measurement of an additional parameter. If possible, measurement of skinfold thicknesses is preferable. As the present study shows that anthropometric diameters used on an interval scale provide only a small improvement in the estimation of body fatness from the body mass index alone, it is not likely that weight-height tables will be improved by the inclusion of (three) frame size categories, as indicated by the elbow diameter suggested recently (Metropolitan Life Insurance Company 1983, Frisancho and Flegel 1983), or by any of the other five diameters, including indices based on them (Katch & Freedson 1982). This conclusion is in agreement with other investigators (Baecke et al. 1982, McKay et al. 1983) and supports the reconstruction of the Metropolitan height and weight tables by Andres (1983) and the construction of the weight-height tables of the Fogarty Center Conference on Obesity (1973), which was adopted by the Royal College of Physicians (1983).

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9 GENERAL DISCUSSION

1. GENERAL

In this chapter, the findings of the study are discussed. In order to evaluate the findings properly, aspects relating to the validity of the mixed-longitudinal study (i.e. selection bias, information bias, confounding bias and the body mass index as a measure of body fatness) are considered.

2. VALIDITY

2.1. Selection bias

A selection bias is a distortion in the estimate of the effect, resulting from procedures used to select subjects for the study population (Kleinbaum et al. 1982).

2.1.1. Descriptive studies

Chapters 3 and 4 report on aspects of the development of the body mass index in young adults. In these studies selection bias will occur if the subjects have been selected with respect to the body mass index. This selection may result from the initial non-response, or the loss to follow-up; severely overweight subjects may not be as willing to participate in a study on overweight as subjects of normal weight. Equally, subjects who display a marked weight gain may refuse to participate further. In consequence, the level of the age-reference curve or the estimates of incidence rates may be too low.

The presence of a possible selection bias has been evaluated in the present study in three ways. First, participants and a sample of the subjects who did not respond initially (non-participants) were compared (Baecke et al. 1983). Since the response rate of the non-participants for this evaluation study was high (92%), it is unlikely that a selection bias was present in the sample of non-participants. Although the participants had a higher level of education than the non-participants, the two groups did not differ with respect to the body mass index. Secondly, the initial body mass index of the follow-up group was compared with that of the control group (the latter group was measured at the first and last examinations only; Chapter 1, Figure 3). The two groups did not differ significantly nor were the differences between age categories of the two groups consistently in the same direction (Chapter 3). Thirdly, the change in body mass index between the two groups was compared, and again no difference was apparent (Chapter 3).

It may be argued that the loss to follow-up in the control group (36%) may also be selective with respect to the body mass index. For example, in the subgroups within the follow-up and control groups that moved out of the county, young subjects of high socio-economic status might be overrepresented, because they move on for further education: if they display a differential gain in body mass index, a differential selection bias will result. Since measured and reported body weights were shown not to be comparable (which is why the group that moved out of the county was excluded from further analysis; Chapter 3) a potential differential loss to follow-up could not be estimated within the present study.

For further evaluation of the loss to follow-up, the change in body mass index was compared with that of young adults observed in the Continuous Dutch Health Interview Survey (Van Sonsbeek 1981), in which all body weights had been reported. The slopes of the age-reference curves of the body mass index of both studies were not markedly different (Chapter 3) and, thus, no indication of a strong selection bias with respect to the body mass index was found.

2.1.2. Etiologic studies

Chapters 5 to 7 report on determinants (dieting behavior, life events and pregnancy) of the development of the body mass index. If there is no selection bias in the dependent variable, a selection bias may still occur if subjects are included in the study as dieters/non-dieters, or experiencing few, an intermediate number or many life events, or as pregnant/non-pregnant women, because of their change in body mass index (Criqui 1979). For life events and pregnancy this is very unlikely, but for dieting behavior it might be possible for successful dieters to be overrepresented and unsuccessful dieters to be underrepresented within the sample that remains in the study. This same differential loss to follow-up may be present in the control group, but this is less likely because participation meant only one additional examination. Though this is only a crude evaluation, the lack of a difference in the change in body mass index between the follow-up group and the control group suggests that if this selection bias is present, it may not be very large.

2.2. Information bias

Information bias refers to a distortion in the estimate of the effect resulting from subjects being misclassified on one or more variables (Kleinbaum et al. 1982).

2.2.1. Descriptive studies

In the studies reported in Chapters 3 and 4, a spurious intervention effect might give rise to an information bias; subjects may watch their weight more carefully because they are measured every six months and thus they might display a smaller gain than would have occurred if they had not participated in the present study. The main reason for including the control group in the present study was to detect this bias. The lack of a difference in change in body mass index between follow-up and control groups (Chapter 3) clearly shows that no intervention effect was present.

From spring 1984 onward each scale used in an examination was fixed on a plywood base, because the own base of the scales was not rigid. This intervention in the method of measurement was applied to both the follow-up group and the control group, and thus could not be controlled for. Since the base was fixed in order to remove random error from the measurements, it is unlikely to have introduced a systematic intervention effect.

2.2.2. Etiologic studies

Information bias in etiologic studies may be nondifferential or differential; if subjects are misclassified according to the independent variable this misclassification might or might not be the same within categories of the dependent variable. Whereas nondifferential misclassification results in a bias towards the null, the differential misclassification may give rise to a bias in either direction (Kleinbaum et al. 1982). In follow-up studies misclassification bias may be generated and/or enlarged by the growing relationship between the respondent and the observer, resulting in response biased by acquiescence and social desirability (Van Strien 1985).

A nondifferential misclassification might result from questionnaires being completed less carefully the seventh time than for the first time, e.g. resulting in the life events score decreasing over time. This was, however, not supported by the data (data not shown).

A differential misclassification might result from subjects who indicated they had been dieting although they had not dieted wholeheartedly (Chapter 5). In this case the differential misclassification would result in the effect of dieting being underestimated. In order to prevent differential information bias in the study of life events, questions relating to changes in body weight were not included in the questionnaire on life events.

In addition, a differential misclassification bias may arise from a study-generated intervention in the change in body mass. For instance, this

might occur in the study on dieting, if the ongoing follow-up study stimulated subjects to make a better attempt to lose some weight. As discussed in Chapter 5, the design of the present study is less likely to invoke this differential intervention bias than a design that depends on the period of dieting. A similar misclassification might be present in the pregnancy study, reported in Chapter 7. Since this misclassification results in an underestimation of the effect, it is unlikely to bias the conclusions of this study much.

2.3. Confounding bias

For a factor to be a confounder it must be related to the exposure as well as to the dependent variable (Kleinbaum et al. 1982).

The assumption of no time of measurement effect, that was made in the age-period-cohort analysis (Chapter 3) may have introduced a confounding bias in the estimated age effects. As discussed above, the comparison of the change in body mass index between the follow-up group and the control group suggested that no intervention effect was present. By restricting the data used for the analysis, to the examinations in the spring, a systematic time of measurement effect, i.e. the seasonal effect, was excluded. Even so, however, a time of measurement effect may distort the data of one or more examinations. Because the assumption of no time of measurement effect cannot be evaluated statistically, it was concluded from a traditional graphical representation of the crude cohort-specific data that this bias did not seem to be present.

The evaluation of all the other confounders included in the various analyses is beyond the scope of this discussion, but two variables that were included as a confounder in most analyses deserve special attention; i.e. age and socio-economic status. Since these variables were the strongest determinants of the level of the body mass index (Baecke et al. 1983), they were suspected beforehand as determinants of the change of the body mass index. A relation between age and socio-economic status and the determinants that were studied has been suggested in several reports; for dieting behavior: Jalkanen (in press), for life events: Dohrenwend and Dohrenwend (1969), Markush and Favero (1974) and for pregnancy: McKeown and Record (1957) and Newcombe (1982).

2.4. The body mass index as a measure of level and change in body fatness

In the present mixed-longitudinal study the body mass index was used to measure body fatness as well as changes in body fatness. Of all weight/height indices studied, the body mass index had a comparable or higher association with more direct measurements of body fatness (Baecke et al. 1982, Keys et al.

1972). Yet, the validity of the body mass index has often been challenged: the excess weight or the weight change might be both muscle and fat (Durnin 1986). In young adults, the bias by lean tissue in the body mass index as such, seems to be present, especially in men (Baecke et al. 1983). The bias in the gain in body mass index is likely to be much smaller, because large increments in physical activity during work or sports very rarely occur (data not presented here).

Skinfold thicknesses might yield a more valid estimate of body fatness than the body mass index, provided that they are measured at several sites (Wilmore and Durnin 1977). However, it is unlikely that this gain in validity counterbalances the loss in precision, especially with respect to the measurement of change in body fatness. Moreover, the precision of the measurement of skinfold thicknesses is smaller at higher levels of body fatness. For the measurement of body weight this loss in precision is less marked. In addition to the lower precision, the measurement of skinfold thicknesses has another important drawback. Since the measurement of skinfold thicknesses requires subjects to undress, this might have been too much of a burden for the subjects, with its possible impact on the response rate.

Thus, the body mass index was prefered as a measure for body fatness. Body height was measured only once, and thus any change in body mass index was completely caused by a change in body weight. This might have been an argument for using the body weight changes themselves. We preferred the body mass index also as a measure of changes in fatness, for three reasons.(1) By expressing level and change in the same unity, both variables may be related to each other more easily (Chapter 4). (2) The body mass index permits comparison with other cross-sectional and longitudinal studies that have used the body mass index or comparable measurements of relative weight (Cronk and Roche 1982, Hsu et al. 1977, Kannel et al. 1979, Borkan et al. 1983, Waaler 1984). (3) From longitudinal studies the relation between health risk and changes in relative weight (e.g. in the body mass index) is known (Hsu et al. 1977, Hubert et al. 1983). In addition, the Build Study 1979 suggests that the same change in body weight might have fewer repercussions on health in taller subjects than in shorter subjects (Van Itallie 1985).

With respect to specific questions, other indicators of change in body fatness may be more informative. In studies on the effect of dieting behavior, measures of change in amount of initial overweight have been used as the dependent variable (Gormally et al. 1980). However, this relative measure is more demanding for overweight subjects than for subjects of normal weight and

thus, this relative measure does not indicate the decrement in health risk caused by the weight reduction. The U-shaped relationship between relative mortality and the body mass index suggests that the same loss in body mass yields a larger benefit for subjects who were severely overweight than for subjects who were only moderately overweight. So, with respect to the evaluation of attempts to lose weight we also preferred the change in body mass index as the criterion variable (Chapter 5).

3. DESIRABLE WEIGHT

Though studies on the association between health risks to relative weight show a U-shaped continuous relationship, an indicator of a "desirable weight range" has been much in demand for therapeutic and educational purposes. With respect to the body mass index this has led to the following categorization (Garrow 1979, Garrow 1981): 20-25: desirable range of body mass index; 25-30: moderate overweight; 30-40: overweight/obesity; >40: severe overweight/obesity. The range 20-25 is close to the equivalent of the Metropolitan Life Insurance desirable weight-for-height over the range from the minimum for small frame to the maximum for large frame: 19.7-24.9 for men and 19.1-24.6 for women (James 1976). The use of frame categories based on waist or chest diameter (White 1956) or on elbow diameter (Metropolitan Life Insurance Company 1983) is supported by common sense rather than by mortality data.

The study done by Baecke et al. (1982) showed that the estimation of body fatness by weight and height was not improved by measuring knee or wrist diameter. Himes and Bouchard (1985) concluded that wrist and ankle diameters satisfied the assumptions inherent in a frame-weight-height table best. However, although they showed that wrist and ankle diameters can improve the estimation of fat free mass, they did not evaluate whether these diameters can improve the estimation of body fatness by weight and height.

The study reported in Chapter 8 shows that the impact of adjustment of the body mass index for frame size on the prediction of body fatness is limited. Six body diameters were studied: the diameters of knee, wrist, elbow, shoulder, waist and hip. The explained variation of body fatness increased from 57% to 62% by inclusion of the knee diameter and to 63% by the shoulder diameter in men and from 63% to 69% by the knee diameter in women. We considered this improvement of the explained variation by a diameter (used as a continuous variable) too small to justify the inclusion of frame categories in weight-height tables.

In some recent weight-height tables frame categories have indeed been omitted (Andres et al. 1983, Royal College of Physicians 1983). Rather than frame categories, subcategories according to age have been proposed (Andres et al. 1985). However, this implicitly assumes that the age-related weight gain is not associated with an increase in risk. As discussed in Chapter 1, the data in support of this assumption are not convincing. Indeed morbidity data support new subcategories in weight-height tables or new nomograms in which circumference measures are incorporated (Björntorp 1985, Deurenberg et al. 1986).

4. AGE-RELATED GAIN IN BODY MASS INDEX IN YOUNG DUTCH ADULTS

Longitudinal and anthropometric studies support the contention that it is best to avoid the age-related weight gain if one wishes to reduce health risk (Chapter 1). Cross-sectional Dutch studies suggested that the age range 20-40 years is a critical period for the development of overweight (Kok et al. 1979, Kromhout 1979). This led to the study of the relationship between the body mass index and age in young Dutch adults. This relation in the Dutch population is of special interest, because weight for height is closest to the mean of several European samples (Eveleth and Tanner 1979). In the present study the age curves based on the cross-sectional data of the first examination for the age range 19-31 years (Baecke et al. 1982) were extended to 35 years of age with correction for sampling effects (Chapter 3). This resulted in a slight correction of the curvature of the curves, whereas the steady increasing trend was maintained. Chapter 4 shows that the steady increment in body mass index within the present sample gave rise to an incidence of moderate overweight that increased with age from 5% per 4 years to 16% per 4 years in men and was more stable (5-6% per 4 years) in women (Chapter 4). This clearly indicated that young adulthood is indeed a critical period for the development of overweight. From the age-reference curves it could be estimated that by the age of 35 almost 50% of the men and 25% of the women were at least moderately overweight.

The health risk of moderate overweight itself is not very alarming for the individual (Garrow 1981). But the risk that the age-related weight gain will give rise to cases of overweight, is larger among the moderately overweight subjects than among the subjects of normal weight. This is suggested by the lack of a clear association between the level and change of the body mass index (Chapter 4); during the follow-up period of four years the gain in body mass was similar at all levels of the body mass index.

Whereas for the individual, moderate overweight may be a health problem mainly in relation to future weight gain, for public health services it is a problem in its own right. The 25-50% moderately overweight subjects at the age of 35 may make large demands on health facilities in the future. As Rose (1985) put it: "a large number of people at a small risk may give rise to more cases of disease than the small number who are at a high risk". Thus, from the point of view of public health services it is of much interest (1) to know whether attempts to lose weight, as they are practiced in the general population, are successful and (2) to characterize determinants of the age-related weight gain.

5. DIETING IN THE GENERAL POPULATION

The suggestion, based on several retrospective studies, that attempts to lose weight may be more successful in the general population than is usually found in the more selective therapeutic samples (Schachter 1982, O'Clarey and Nelson 1985, Jeffery et al. 1984), was not strongly supported by the follow-up study presented in Chapter 5. Several subgroups lost some body mass as a result of their efforts at dieting, but the amount was generally small. Although the effect might be slightly underestimated (see above), the findings were generally not in accordance with the optimism about the efficacy of dieting, expressed in the popular press (Parham 1986). Rather, the findings supported studies on more selective samples (Stunkard and McLaren-Hume 1959, Stunkard and Penick 1979, Wing and Jeffery 1979, Jeffery et al. 1976); subjects with a higher health risk due to overweight are unlikely to reduce that risk markedly by dieting. Only if they persist, especially during summer periods, may they gradually lose their overweight, but this seems to take some years. It is clear that this is difficult, if not impossible, and has detrimental psychological side-effects (Van Strien 1986). Psychologists have therefore frequently questioned whether the vicious circle of losing and gaining weight is to be preferred over being quietly overweight (Wooley et al. 1979).

The report described in Chapter 5 shows that dieting may be efficacious in preventing the age-related weight gain from occurring. In this respect, it may be useful for subjects who are not overweight to diet, although again the psychological consequences must not be ignored, especially in the large group of women who diet but gain as much weight as women who do not diet. Clearly, the prevention of weight gain remains to be preferred, and in this respect more needs to be known about the etiology of overweight in the general population.

6. DETERMINANTS OF GAIN IN BODY MASS INDEX

In Chapters 6 and 7, the experience of life events in general and the experience of pregnancy in particular are studied as potential determinants of change in body mass index. Initially, the experience of few (for men) or many (for both men and women) life events resulted in a larger gain in body mass than occurred following the experience of a moderate number of life events. This gain was effectively counterbalanced by regulatory mechanisms during further follow-up in all women and most men. However, in dieting men the gain remained, even after two years of follow-up. This finding suggested that even deliberate attempts to lose the additional weight were not efficacious, although the other interpretation, i.e. less efficacious attempts to lose weight when experiencing few or many life events, may not be completely ruled out (Chapter 6). Thus, few or many life events may directly or indirectly determine a gain in body mass index in some men. This supports experimental studies in which an increased food intake was observed in situations of boredom (Abraham and Stinson 1977) or stress (Slochower 1983). The effect of life events on long-term change in body mass may be modified by coping behavior or social support systems (Sarason et al. 1978).

The life event that was suggested by several retrospective studies (Richardson 1952, Sheldon 1949, Bradley 1985) to be of special relevance for the development of overweight in women is pregnancy. The study reported in Chapter 7 shows that the effect of pregnancy may be associated with subsequent breastfeeding practices. The findings suggested that 'maternal obesity' may especially occur in women who breastfeed their children for long periods (> 2 months). If this excess body mass consists of fat depots left unused after the period of breastfeeding, it is likely that they are located in the femural region (Rébuffé-Scrive 1985). This suggests that the health risks associated with maternal obesity are less severe than might be expected on the basis of the body mass index.

As described in Chapter 1, environmental/behavioral factors seem to determine the extent to which genetic predispositions are expressed, even in young adulthood. The studies described in Chapters 6 and 7 have supported this contention. The observations showed that adult body weight development is not completely genetically determined but can be manipulated. However, the situations in which these manipulations occur in real life appear to be complex. Clearly, developing overweight is not just a matter of being too weak to resist the temptation of eating. This is an important issue for the

self-esteem of overweight subjects (Putchik 1976) and for the attitudes of others towards the overweight (De Jong 1980). The mixed-longitudinal study of which this thesis has described only a part (see Chapter 2), has contributed to the characterization of several determinants of overweight. Knowledge of these environmental conditions, personal characteristics and behavioral factors is the first step in the prevention of overweight.

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SUMMARY

Follow-up studies of long duration have shown a U-shaped relationship between mortality/morbidity and the body mass index (BMI, weight/height^2). The risk to health posed by obesity appears to be larger in younger subjects than in older subjects. Though this might suggest that a moderate weight gain after the termination of growth may not increase the risk to health, this contention is not supported by other observations (Chapter 1). These are: (1) the range of relative weight associated with minimum mortality does not seem to shift to higher values with increasing age, at least not in men. (2) Diseases associated with overweight at younger ages need not be the same as those associated with overweight at older ages. (3) Cohort effects may bias the age trend in the risk associated with overweight. (4) Longitudinal studies on adults show that changes in body weight are associated with changes in risk factors of diseases. (5) Anthropometric studies show that even at the same weight, the fat mass increases with increasing age. In addition, several studies suggest that weight gained during adulthood mainly accumulates in the abdomen, which means a shift to a fat distribution more strongly associated with risk to health. The risk to health therefore does not seem to remain constant if body weight increases during adulthood.

As part of an extensive mixed-longitudinal study (Chapter 2), this thesis deals with the development of the BMI and the etiology of this development in young adults. In addition, one methodological study is described. These studies build on cross-sectional observations that were made in the first examination of the study.

In spring 1980, all residents of Dutch nationality in the county of Ede, born in 1948-1950, 1953-1955, 1958-1960 were invited by mail to participate in the study. Participants in spring 1980 (N=3936) were followed for four years (1980-1984) in two groups, the follow-up group (N=1670) and the control group (N=2266). From spring 1981 onward the follow-up group was approached twice a year in seven consecutive examinations. The control group was re-measured in spring 1984 only. At each examination a questionnaire was completed and body weight in light indoor clothing and without shoes was measured to the nearest 0.5 kg. Body height was measured to the nearest 0.1 cm in spring 1980. Subgroups of the follow-up and control groups were additionally examined for the studies described in Chapter 7 and 8.

The development of the BMI is dealt with in Chapter 3, in which the

mixed-longitudinal design of the study was utilized, and Chapter 4, in which the data were analyzed longitudinally. Over the four years, the change in BMI of the follow-up group was the same as that of the control group. From 19 to 35 years of age the median BMI increased from 22.1 kg/m^2 to 24.4 kg/m^2 in men and from 21.1 kg/m^2 to 23.0 kg/m^2 in women (Chapter 3). This increment in BMI gave rise to an incidence of moderate overweight (BMI>25 kg/m^2) which increased from 4.8% per 4 years to 15.5% per 4 years in men, and was more stable (5.3-5.7% per 4 years) in women (Chapter 4). Thus, especially in men, young adulthood appears to be a critical period for the development of overweight.

The age-reference curves (Chapter 3) suggested that the variation of the BMI over subjects was independent of age. In accordance, the longitudinal analyses showed that the initial BMI and the rate of gain in BMI were unrelated in men and only slightly negatively related in women. Thus, overweight subjects did not appear to gain more or less body mass than non-overweight subjects.

The within-subjects standard deviation of the yearly measured BMI was 0.69 kg/m^2 in men and 0.74 kg/m^2 in women (Chaper 4). This fluctuation was larger in overweight subjects than in normal-weight subjects and was larger in subjects with a larger long-term change (gain or loss) in BMI than in subjects who had a more stable BMI. The considerable fluctuation in BMI may mask the gradual long-term increment in BMI and may thus hinder young adults from being aware that they are becoming overweight.

The effect of dieting is dealt with in Chapter 5. After two years of follow-up, the average decrease in body mass as a result of dieting was -0.5 kg/m^2 (95% confidence interval (CI):-1.0,+0.0) in men and -0.4 kg/m^2 (95%CI:-0.8,+0.0) in women. This approximately amounts to an average weight reduction of 1.5 kg in men and 1.0 kg in women. The effect of dieting was most pronounced in men whose initial BMI was high, who dieted during the summer, or who dieted more frequently, and in women who were older than 30 or who dieted on medical advice. Though subjects who have already developed severe overweight may achieve a larger reduction in the risk to health by losing the same amount of body mass as subjects who are only moderately overweight, it is unlikely that the benefit is large unless they persist. However, the effect of dieting may be enough to prevent the age-related gain in body mass and thus dieting may especially play a part in the prevention of overweight.

Chapter 6 deals with the changes in BMI in relation to number of life events experienced. During the first year of follow-up several subgroups of men and women who experienced many life events and several subgroups of men who experienced few life events showed a gain in body mass. After another year of

follow-up this gain in body mass had disappeared in almost all subgroups; compared with dieting men with an intermediate number of life events, dieting men with few life events gained +1.3 kg/m^2 (95%CI:+0.0,+2.6) more body mass, whereas dieting men with many life events gained +1.8 kg/m^2 (95%CI: +0.5,+3.0) more body mass. Thus, life events seem to have an impact on the etiology of overweight in men.

One life event in women is considered more specifically (Chapter 7); i.e. pregnancy. Women who breastfed their child for more than two months gained more body mass than was expected from aging. Nine months postpartum they were +0.6 kg/m^2 (90%CI: +0.1,+1.0) heavier than expected. This difference was slightly smaller (not significantly) in women who breastfed their child for 0-2 months. Women who used bromocriptine to stop lactation lost body mass unexpectedly.

In a methodological study (Chapter 8) the impact of adjustment of the BMI for body diameters (i.e. knee, wrist, elbow, shoulder, pelvis and hip) on the prediction of body fatness was examined. In addition to what was explained by the BMI, 6% of the variation in body fatness was explained by the shoulder diameter in men, and also 6% by the knee diameter in women. This improvement in estimating body fatness by the diameters, used as continuous variables, was considered too small to justify frame categories being included in weight-height tables. Instead of frame size, fat distribution might be a more useful attribute in these tables.

In the last chapter (Chapter 9) several aspects relating to the validity of the mixed-longitudinal study are discussed. The main conclusions of this thesis are:

- Young adulthood seems to be a critical period in the development of moderate overweight, expecially in men.
- The age-related gain in BMI of overweight young adults and of young adults of normal weight is similar.
- Overweight subjects are unlikely to decrease their risk to health markedly by dieting as generally practiced, unless they persist. Dieting may be more effective in preventing overweight.
- The experience of many life events may play a part in the etiology of overweight in men, as does the experience of few life events.
- Women who breastfeed their child for more than two months may gain more body mass than expected from aging.
- In weight-height tables the fat distribution might be a more useful attribute than frame size categories.

SAMENVATTING

Langdurende vervolg-onderzoeken hebben aangetoond dat tussen mortaliteit/morbiditeit en de Queteletindex (QI=gewicht/lengte^2) een U-vormige relatie bestaat. Het risico ten gevolge van overgewicht lijkt groter te zijn voor jongeren dan voor ouderen. Hoewel dit zou kunnen betekenen dat na het beëindigen van de groei, een matige gewichtstoename niet hoeft te leiden tot een verhoging van het risico voor de gezondheid, wijzen andere bevindingen niet in deze richting (Hoofdstuk 1). Deze zijn: (1) bij mannen lijkt het gebied van het relatieve gewicht, dat is geassocieerd met de laagste mortaliteit, niet te verschuiven naar hogere waarden met toenemende leeftijd. (2) Ziekten die op jonge leeftijd met overgewicht zijn geassocieerd hoeven niet dezelfde te zijn als ziekten die op oude leeftijd met overgewicht samenhangen. (3) Cohort-effecten kunnen de leeftijds-trend in het risico ten gevolge van overgewicht verstoren. (4) Longitudinale onderzoeken bij jonge volwassenen wijzen op een verband tussen veranderingen in lichaamsgewicht en veranderingen in risicofactoren voor de gezondheid. (5) Uit anthropometrische onderzoeken blijkt dat de vetmassa toeneemt bij het ouder worden, zelfs als het gewicht constant blijft. Hiernaast suggereren verschillende onderzoeken dat bij een gewichtstoename van een volwassene de vetverdeling verandert; er hoopt zich meer vet op in het abdomen. Dit betekent een verschuiving naar een vetverdeling die sterker met gezondheidsrisico's samenhangt. Een gewichtstoename van een volwassene lijkt daarom wel degelijk gevolgen te hebben voor het risico voor de gezondheid.

Als onderdeel van een uitgebreide mixed-longitudinale studie (Hoofdstuk 2), beschrijft dit proefschrift de ontwikkeling van de QI en enkele determinanten van deze ontwikkeling bij jonge volwassenen. Hiernaast wordt een methodologische studie beschreven. Deze studies bouwen voort op cross-sectionele waarnemingen die tijdens het eerste meetpunt werden gedaan.

In het voorjaar van 1980 werden alle inwoners met de Nederlandse nationaliteit, woonachtig in de gemeente Ede en geboren in 1948-1950, 1953-1955, 1958-1960, per post uitgenodigd deel te nemen aan het onderzoek. De deelnemers in het voorjaar van 1980 (N=3936) werden vier jaar lang gevolgd in twee groepen, de follow-up groep (N=1670) en de controle groep (N=2266). Vanaf het voorjaar in 1981 werd de follow-up groep tweemaal per jaar, in totaal zeven keer, benaderd. De controle groep werd alleen in het voorjaar van 1984 opnieuw benaderd. Tijdens elk meetpunt werd een vragenlijst afgenomen en het

lichaamsgewicht in lichte huiskleding en zonder schoenen tot op 0,5 kg nauwkeurig gemeten. De lengte werd tot op 0,1 cm nauwkeurig gemeten in het voorjaar van 1980. Bij subgroepen van de follow-up groep en de controle groep werden extra metingen verricht ten behoeve van de studies beschreven in Hoofdstuk 7 en 8.

De ontwikkeling van de QI wordt beschreven in Hoofstuk 3 en 4. In Hoofdstuk 3 wordt de mixed-longitudinale opzet van de studie uitgewerkt. In Hoofdstuk 4 worden de gegevens longitudinaal geanalyseerd. Tussen de 19 en 35 jaar nam de mediaan van de QI toe van 22,1 kg/m^2 tot 24,4 kg/m^2 bij mannen en van 21,1 kg/m^2 tot 23,0 kg/m^2 bij vrouwen (Hoofdstuk 3). Deze toename van de QI had een incidentie van matig overgewicht (QI>25 kg/m^2) tot gevolg, welke toenam van 4,8% per 4 jaar tot 15,5% per 4 jaar bij mannen en meer stabiel was (5,3-5.7% per 4 jaar) bij vrouwen (Hoofdstuk 4). De jong-volwassen leeftijdsfase blijkt daarom met name bij mannen een kritische periode voor de ontwikkeling van matig overgewicht te zijn.

De leeftijd-referentiecurven (Hoofdstuk 3) wezen erop dat de populatie-variatie van de QI niet van de leeftijd afhankelijk was. Hiermee in overeenstemming, toonden de longitudinale analyses (Hoofdstuk 4) aan dat de initiële QI en de toenamesnelheid van de QI niet met elkaar waren gerelateerd bij mannen en slechts zwak negatief gerelateerd waren bij vrouwen; jonge volwassenen met overgewicht bleken niet meer of minder in QI toe te nemen dan jonge volwassenen zonder overgewicht.

De binnen-persoons standaardafwijking van de jaarlijkse QI-metingen was 0,69 kg/m^2 bij mannen en 0,74 kg/m^2 bij vrouwen (Hoofdstuk 4). Deze fluctuatie was groter bij personen met overgewicht dan bij personen zonder overgewicht en was groter bij personen met een grote 4-jaars verandering (toename of afname) van de QI dan bij personen bij wie de QI meer stabiel was. De aanmerkelijke fluctuatie in QI kan de geleidelijke toename in QI maskeren en kan op deze manier verhinderen dat jonge volwassenen zich bewust zijn van het feit dat ze overgewicht ontwikkelen.

Hoofdstuk 5 heeft betrekking op het effect van lijngedrag. Na twee jaar follow-up was de gemiddelde afname van de QI tengevolge van een lijnpoging -0,5 kg/m^2 (95%CI:-1,0;+0,0) bij mannen en -0,4 kg/m^2 (95%CI:-0,8;+0,0) bij vrouwen. Dit komt ongeveer overeen met een gemiddelde gewichtsafname van 1,5 kg bij mannen en 1,0 kg bij vrouwen. Het effect van lijnen was het meest uitgesproken bij mannen met een hoge initiële QI, die in de zomer lijnden of die het lijnen volhielden, en bij vrouwen ouder dan 30 jaar of lijnend op doktersadvies. Een afname in QI bij personen die reeds ernstig overgewicht

hebben ontwikkeld lijkt een grotere afname van het risico voor de gezondheid tot gevolg te kunnen hebben dan eenzelfde afname in QI bij personen met matig overgewicht. Het is evenwel niet waarschijnlijk dat de verbetering ten gevolge van het lijnen, zoals dat in het algemeen wordt beoefend, groot zal zijn, tenzij gewichtsvermindering voortdurende aandacht krijgt. Het effect van lijnen kan echter wel voldoende zijn om de toename van de QI met de leeftijd te voorkomen. Lijnen kan op deze manier een rol spelen in de preventie van overgewicht.

Hoofdstuk 6 handelt over de relatie tussen veranderingen in de QI en het aantal belangrijke levensgebeurtenissen die werden meegemaakt. Tijdens het eerste jaar van de follow-up periode namen verschillende subgroepen van mannen en vrouwen die veel levensgebeurtenissen meemaakten en verschillende subgroepen van mannen die opvallend weinig levensgebeurtenissen meemaakten toe in QI. Na het tweede jaar van follow-up was deze toename in QI verdwenen in praktisch alle subgroepen; alleen mannen die aan de lijn deden en weinig of veel levensgebeurtenissen meemaakten namen respectievelijk +1,3 kg/m^2 (95%CI:+0,0;+2,6) en +1,8 kg/m^2 (95%CI:+0,5;+3,0) meer in QI toe dan mannen die aan de lijn deden en een tussenliggend aantal levensgebeurtenissen meemaakten. Belangrijke levensgebeurtenissen blijken dus een rol te spelen in de etiologie van overgewicht van mannen.

Op één belangrijke levensgebeurtenis bij vrouwen wordt apart ingegaan in Hoofdstuk 7, en dat is zwangerschap. Vrouwen die hun kind langer dan twee maanden borstvoeding gaven namen meer toe in gewicht dan op grond van de leeftijdstrend werd verwacht; negen maanden postpartum waren ze +0,6 kg/m^2 (90%CI:+0,1;+1,0) zwaarder dan verwacht. Dit verschil was (niet significant) kleiner bij vrouwen die hun kind niet of korter dan 2 maanden borstvoeding gaven. Vrouwen die bromocriptine gebruikten om de borstvoeding te remmen, namen, tegen de verwachting in, zelfs in gewicht af.

In een methodologische studie (Hoofdstuk 8) werd nagegaan in hoeverre de voorspelling van de vetmassa door de QI kon worden verbeterd door één van de volgende breedtematen: knie, pols, elleboog, schouder, bekken en heup. De door de QI verklaarde variantie werd met 6% verbeterd door de schouderbreedte bij mannen, en eveneens 6% door de kniebreedte bij vrouwen. Deze verbetering door breedtematen, gebruikt als continue variabelen, werd te klein bevonden om de categorieën voor lichaamsbouw in gewicht-lengte tabellen te rechtvaardigen. In plaats van de lichaamsbouw zou beter de vetverdeling bij gewicht-lengte tabellen kunnen worden betrokken.

In het laatste hoofdstuk (Hoofdstuk 9) worden verschillende aspecten

besproken die met de validiteit van de mixed-longitudinale studie samenhangen. De belangrijkste conclusies van dit proefschrift zijn:

- De jong-volwassenen leeftijdsfase lijkt een kritische periode te zijn voor de ontwikkeling van matig overgewicht, vooral bij mannen.
- De toename in QI met de leeftijd van jonge volwassenen met overgewicht en van jonge volwassenen zonder overgewicht is vergelijkbaar.
- Het is niet waarschijnlijk dat personen met overgewicht een grote verbetering van hun gezondheidsrisico bereiken door te lijnen zoals dat in een steekproef van de populatie wordt beoefend, tenzij ze het volhouden. Lijnen kan echter wel succesvol zijn voor de preventie van overgewicht.
- Het meemaken van veel, maar ook het meemaken van opvallend weinig belangrijke levensgebeurtenissen kan een rol spelen in de etiologie van overgewicht bij mannen.
- Vrouwen die hun kind langer dan twee maanden borstvoeding geven lijken meer in gewicht toe te nemen dan verwacht op grond van de leeftijd.
- In plaats van de lichaamsbouw zou de vetverdeling beter bij gewicht-lengte tabellen kunnen worden betrokken.

ABSTRACT

As part of an extensive four-year mixed-longitudinal study in young adults (initially 19-31 years of age), the development of the body mass index (BMI, weight/height^2) and some determinants of this development were examined.

The following conclusions were drawn from the study. (1) Young adulthood seems to be a critical period in the development of moderate overweight, expecially in men. (2) The age-related gain in BMI of overweight young adults and of young adults of normal weight is similar. (3) Overweight subjects are unlikely to decrease their risk to health markedly by dieting as generally practiced, unless they persist. Dieting may be more effective in preventing overweight. (4) The experience of many life events may play a part in the etiology of overweight in men, as does the experience of few life events. (5) Women who breastfeed their child for more than two months may gain more body mass than expected from aging. (6) In weight-height tables the fat distribution might be a more useful attribute than frame size categories.

CURRICULUM VITAE

De auteur werd op 30 november 1955 geboren te Amsterdam. Na het behalen van het gymnasium beta diploma op het Stedelijk Gymnasium te Breda, studeerde zij een jaar psychologie. Na voor de tweede maal uitgeloot te zijn voor medicijnen, begon ze in 1975 met haar studie biologie aan de rijksuniversiteit van Utrecht. In 1982 slaagde zij voor het doktoraal examen met Voedingsleer als hoofdvak en Experimentele ontwikkelingsbiologie en Beleidsgerichte biologie als bijvakken. Vanaf oktober 1982 tot april 1986 verrichtte zij met financiële steun van het Praeventiefonds het in dit proefschrift beschreven onderzoek bij de vakgroep Humane Voeding van de Landbouwuniversiteit te Wageningen. In deze periode werd zij ook in staat gesteld de Epidemiologie-cursus van het New England Institute of Epidemiology te Amherst te volgen. Vanaf mei 1986 is zij werkzaam bij het Antoni van Leeuwenhoekhuis te Amsterdam.