Dietary intake, physical activity, and eating behaviour in obese patients with high cardiovascular risk

Assessments and implications for treatment

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Oslo 2008

Series of dissertations submitted to the
Faculty of Medicine, University of Oslo
No. 771

ISBN 978-82-8072-926-2

Cover: Inger Sandved Anfinsen.
Printed in Norway: AiT e-dit AS, Oslo, 2009.

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ACKNOWLEDGEMENTS

The work presented in this thesis was carried out at the Department of Preventive Cardiology, at the Ullevål University Hospital in Oslo from December 1998 to December 2006. Some of the studies were supported by study grants from Hoffman La Roche, Oslo, Norway.

When the first study in this dissertation was initiated, I was married and the mother of small children. Now, I am divorced and the mother of adolescents. Completing this thesis has in many ways been analogous to the divorce…long lasting and frustrating at times, but also giving a sense of pride that I have managed. Several times during this work I wanted to give up, but there have always been some "lights in the end of the tunnel" that inspired me to go on.

Many lights have shown the way. I thank my advisor Professor Serena Tonstad, with whom I have worked between 1991 and 1997 at the Lipid Clinic, Rikshospitalet and from 1998 to present at the Department of Preventive Cardiology, Ullevål University Hospital, for her belief in me, and her care, patience, optimism and support. Thanks also for fruitful discussions, and for instruction, advice and helpful suggestions to improve the articles. I thank all my co-authors for their contribution to this thesis, in particular Bo-Egil Hustvedt for help with the ActiReg® calculations, Lars Ellegård for the doubly labelled water analyses and for teaching me about the method, Rune Blomhoff and the members of his group for the antioxidant analyses, Merete Helgeland for contributions to the SMOMS study and Ingar Holme for statistical advice. I also thank Hanne Dyrendahl for help with some of the dietary calculations.

I am very grateful to all my co-workers at the Department of Preventive Cardiology for your support, intellectual discussions and practical help with this work (Tor Ole Klemsdal, Ingrid Ellingsen, Ragnhild Kleve, Eli Heggen, Lise Bergengen, Lisa Flakk, Tonje Berg, Hilde Iversen, Jorunn Magnæs and Terje R. Pedersen). I thank Leiv Ose for introducing me for nutrition research when I worked at the Lipid Clinic. I thank all the participants for sharing their experiences of weight loss. I also thank Karen Kollien Nygaard, who, though not directly involved in this work, supplemented my knowledge of the difficulties of behavioural change.

I am lucky to have supportive parents, brothers, extended family and friends. Thanks for your patient listening to all my frustrations. I thank Rune for loving care of our children when they are not with me. At last but not at least, my brightest shining lights are Maiken and Marius. Thank you for being my darling children!

Mette Svendsen
August 2008

ABSTRACT

The prevalences of obesity (body mass index ≥ 30 kg/m^2) and its concomitant comorbidities, metabolic syndrome, sleep related breathing disorders and other diseases are increasing worldwide and in Norway. A moderate weight reduction through dietary change and increased physical activity is recommended to decrease risk. Pharmacological therapy is an adjunct to behaviour change. To guide clinical treatment, a better understanding of measurement errors in the reported diet and physical activity of patients with obesity-associated comorbidities and the effects of dietary interventions in this group including anti-obesity drug is needed.

This thesis is based on three cross-sectional studies and two randomized, controlled trials. In a population of obese subjects with metabolic risk factors, energy intake was underreported by 500 kcal and 1000 kcal according to a dietary interview and dietary records, respectively. Thus, in a majority of obese subjects dietary advice to achieve moderate energy restriction cannot be based on dietary assessments. ActiReg® is a monitor that can be used to estimate total energy expenditure and physical activity. Despite an underestimated of 100 kcal, the ActiReg® method gave a valid estimate of total energy expenditure compared to the doubly labelled water method, which is the gold standard. These results may indicate that energy restriction 500 kcal to1000 kcal below measured total energy expenditure is an approach that can be used in counselling for weight reduction. With regard to physical activity, obese subjects seeking treatment spent a mean of 26 minutes per day in moderate physical activity before advice for weight loss was given. This is close to the recommended levels but for weight loss and maintenance purposes the level of physical activity has to be increased.

A simple intervention to increase intake of vegetables and fruit was tested in subjects with sleep related breathing disorders. A behavioural group program that emphasized a 500 g increase in the daily intake of vegetables and fruit resulted in better weight loss, decreased systolic and diastolic blood pressure and increased plasma levels of lipid soluble antioxidants compared to usual care controls after three months. The influence of orlistat on eating behaviour and dietary intake was studied in a long-term weight maintenance trial. After weight loss achieved with the use of a very low energy diet for eight weeks, treatment with orlistat did not influence eating behaviour or fat intake other ways than placebo. Subjects that chose to take orlistat after the end of the study when the use was optional had higher than recommended intake of fat and this may hamper the effect of the drug.

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4. Svendsen M, Blomhoff R, Holme I and Tonstad S. The effect of an increased intake of vegetables and fruit on weight loss, blood pressure and antioxidant defence in subjects with sleep related breathing disorders. Eur J Clin Nutr 2007;61:1301-1311.
5. Svendsen M, Rissanen A, Richelsen B, Rössner S, Hansson F and Tonstad S. Effect of orlistat on eating behavior among participants in a three-year weight maintenance trial. Obesity 2008;16:327–333.
6. Svendsen M, Helgeland M and Tonstad S. The long-term influence of orlistat on dietary intake in obese subjects with components of metabolic syndrome. Accepted, J Hum Nutr Diet.

ABBREVIATIONS

AHI	Apnea-hypo apnea index
BMI	Body mass index
BES	Binge eating scale
CI	Confidence interval
CV	Coefficient of variation
CPAP	Continuous positive airways pressure
CVD	Coronary vascular disease
EI	Energy intake
DEXA	Dual energy x-ray absorptiometry
DLW	Doubly labelled water
DR	Dietary record
FFQ	Food frequency questionnaire
HDL	High density lipoprotein
LDL	Low density lipoprotein
PAL	Physical activity level
PAR	Physical activity ratio
RMR	Resting metabolic rate
SMOMS	Scandinavian Multicenter study of obese subjects with the Metabolic Syndrome
SRBD	Sleep related breathing disorders
TFEQ	Three factor eating questionnaire
TEE	Total energy expenditure
VLED	Very low energy diet

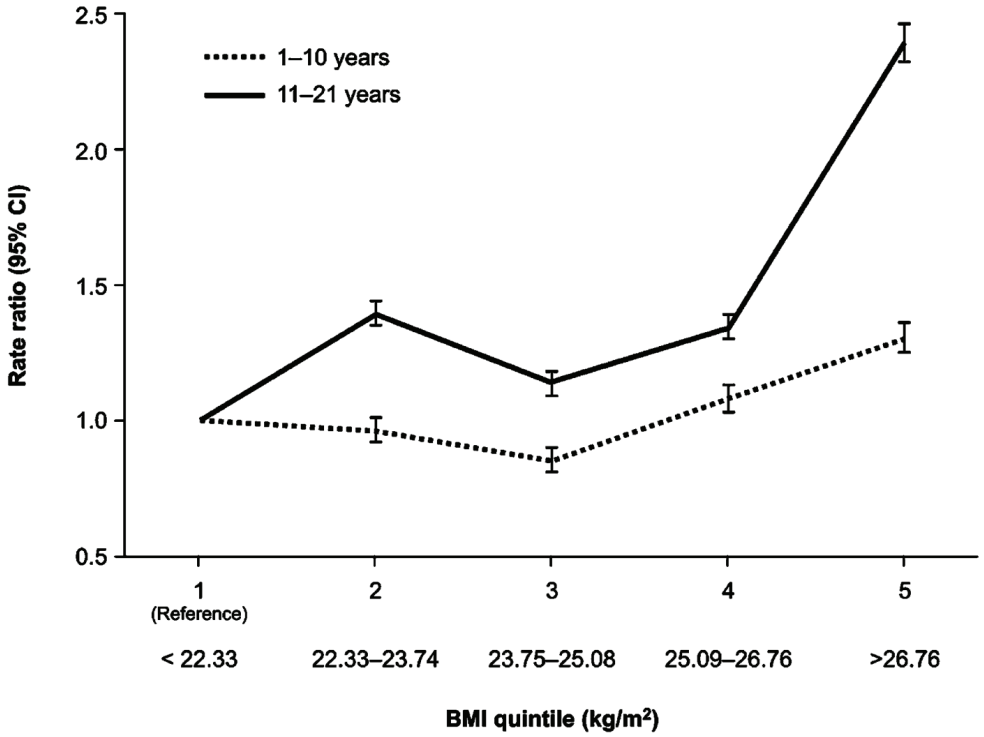
1 INTRODUCTION

1.1 The health consequences of obesity

The prevalence of overweight (body mass index [BMI] of 25-29.9 kg/m^2) and obesity (BMI ≥ 30 kg/m^2) is increasing (Soowon and Hopkin, 2006) and it is estimated that about 315 million people worldwide are obese (James, 2004). Scandinavian countries are no exception (Ulset et al., 2007). The prevalence of obesity has been doubled in Norway during the last 20 years and one of five adults is obese. In both men and women the highest prevalence of obesity is seen in age above 60 years (Ulset et al., 2007).

Obesity has been associated with increased risk of cardiovascular disease, type 2 diabetes, sleep related breathing disorders (SRBD), fatty liver, dyslipidaemia (elevated serum triglycerides and apolipoprotein B, decreased high density lipoprotein [HDL], increased small dense low density lipoprotein [LDL]), hypertension and thromboembolic disease as well as mortality due to certain forms of cancer (colonic, uterine, ovarian, gall-bladder). In addition obesity is associated with increased morbidity due to conditions such as osteoarthritis, gallstones, bladder dysfunction, polycystic ovary syndrome, asthma, lymphoedema, psychological problems, low social status and low levels of physical activity (Coppack et al., 2005). In Norway, it has been shown that an elevation in BMI of one unit increased the relative risk of fatal coronary heart disease by 6% (Håheim et al., 2007). This is shown in figure 1.

Figure 1. Crude rate ratios of cardiovascular heart disease mortality according to BMI quintiles during long-term follow up. From Håheim LL et al. Predictiveness of body mass index for fatal coronary heart disease in men according to length of follow-up: a 21-year prospective cohort study. Scand J Publ Health 2007;35:4-10.



This dissertation focuses specifically on obese individuals at high risk of cardiovascular disease associated with SRBD and metabolic syndrome.

1.1.1 Sleep related breathing disorders

SRBD are an increasingly common cause of morbidity and mortality among overweight and obese individuals. SRBD are characterized by repetitive collapse (apnea) or partial collapse (hypoapnea) of the upper airway during sleep (Barvaux et al., 2000). This results in pauses in breathing while sleeping and intermittent transient hypoxia. SRBD include habitual snoring, increased upper airway resistance syndrome and obstructive sleep apnea. It is estimated that

one of five adults in Western countries has at least mild disease and one of 15 has at least moderate disease (Caples et al., 2005). The apnea-hypo apnea index (AHI) is a commonly used measure of SRBD (Young, 2005) and an AHI ≥ 5 indicates the presence of obstructive sleep apnea. There is a graded increase in the prevalence of SRBD with increasing body weight (Young, 2005). SRBD have been associated with hypertension and an increased risk of cardiovascular disease (CVD) (Lavie, 2004; Stradling, 2004). Sympathetic over-activity, increased oxidative stress, insulin resistance and a pro-thrombotic state are some of the mechanisms thought to mediate the relation between SRBD and CVD (Shamsuzzaman et al., 2002; Fletcher, 2003; Lavie, 2003; Punjabi et al., 2004). The exact independent relation of SRBD to CVD is still not clear. However, there is widespread acceptance for the idea that concomitant factors such as pervasiveness of male gender, age, central obesity, hypertension, diabetes and dyslipidemia contribute to the increased risk of CVD among patients with SRBD (Newman et al., 2001).

Studies have consistently shown that a 3% reduction in weight improves symptoms of AHI by about 1% (Young et al., 2002). Such moderate weight loss is important and can facilitate treatment of SRBD and lower associated risks, but additional treatment with nasal continuous positive airways pressure (CPAP) is necessary for most individuals to improve sleep quality (Barvaux et al., 2000).

1.1.2 Metabolic syndrome

Metabolic syndrome is a constellation of interrelated risk factors that promote the development of atherosclerotic CVD and diabetes type 2 (Grundy et al., 2005). The definition of metabolic syndrome varies (Alberti and Zimmer, 1998; National Cholesterol Education Program, 2002; International Diabetes Federation, 2005). Almost all attempts to characterise metabolic syndrome include abdominal obesity, atherogenic dyslipidaemia, elevated blood

pressure and disturbance of carbohydrate metabolism. The definition of metabolic syndrome given by the International Diabetes Federation emphasises the importance and essentiality of abdominal obesity, which is measured by waist circumference in clinical practice.

In 2005 the American Heart Association/National Heart, Lung, and Blood Institute published a Scientific Statement regarding the diagnosis and management of metabolic syndrome. The goal of treatment is to reduce the overall risk for clinical atherosclerotic disease by modification of the underlying risk factors (obesity, physical inactivity and atherogenic diet) through life style changes. For treatment of obesity a goal of 7-10% weight reduction is recommended (Grundy et al., 2005). Though the concept of metabolic syndrome has been debated in the literature (Grundy, 2006; Reaven, 2006), there is broad agreement that obese subjects with established cardiovascular risk factors are a high-risk group for CVD and type 2 diabetes, and that weight reduction addresses one of the most important causes of increased risk.

1.1.2.1 Weight reduction and metabolic syndrome

A weight reduction of 7-10% gives clinically relevant benefits for subjects with metabolic syndrome or its components. Weight loss of this size has been shown to reduce the incidence of type 2 diabetes (Tuomiletho et al., 2001; Knowler et al., 2002) and ameliorate elevated blood pressure (Apple et al., 2003). In addition reductions in serum triglycerides and increases in HDL-cholesterol have been shown (Dattilo and Kris-Etherton, 1992). It has to be noted that during weight loss HDL-cholesterol is reduced, while the increase in seen when weight is stabilized after weight loss (Dattilo and Kris-Etherton, 1992).

1.1.2.2 Anthropometric risk factors for metabolic syndrome

Accumulation of adipose tissue in the abdominal region has been shown to predict increased risk for CVD independent of BMI (Rexrode et al., 1998; Rexrode et al., 2001) and waist circumference may predict increased risk for CVD better than waist-to-hip ratio (Ketel et al., 2007). The World Health Organization BMI classification of obesity (WHO, 1998) as shown in table 1 is limited by not taken take distribution of body fat into consideration. An additional measurement of waist circumference and waist-to-hip ratio is recommended to assess the distribution of body fat in the abdominal region (Grundy et al., 2005). Assessment of body composition by dual energy x-ray absorptiometry (DEXA) is a more sophisticated method to measure body composition and distribution. When compared to computer tomography scanning this method gives valid estimates of total and abdominal fat mass in obese subjects (Glicman et al., 2004).

Table 1. Body mass index classification of overweight and obesity (WHO, 1998)

BMI	*CLASSIFICATION*
25.0-29.9	Overweight
30.0-34.9	Obesity grade 1
35.0-39.9	Obesity grade 2
>40.0	Obesity grade 3

1.2 Body weight regulation

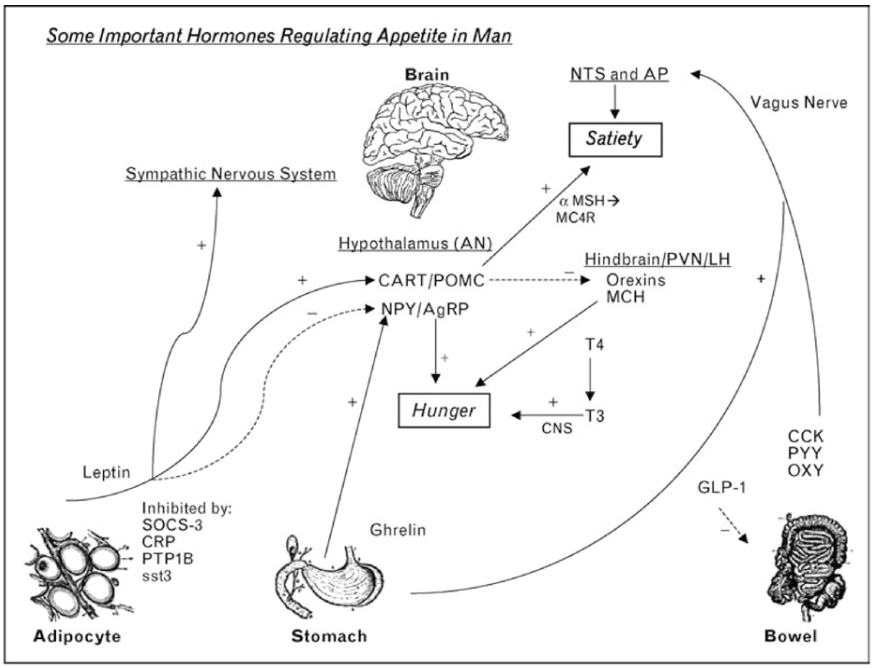
1.2.1 The homeostatic regulation of body weight

To reduce obesity-associated morbidities sustained weight reduction is paramount. One major obstacle is that maintenance of weight loss is difficult to achieve due to redundant mechanisms of body weight regulation that counteract negative energy balance (Knecht et al.,

2008). Within a narrow individually defined range appetite and energy balance are tightly regulated to maintain body weight or adiposity by homeostatic mechanisms (Speakman, 2004).

The homeostatic mechanisms involve hormones secreted from adipose tissue, endocrine glands and enteroendocrine cells which all converge at the vagus nerve, brainstem and hypothalamus to modulate complex interactions of neurotransmitters and central appetite-regulating peptides (Lenz and Diamond, 2008). The hypothalamic arcuate nucleus contains orexigenic (appetite increasing) neuropeptid Y and agouti-related peptid neurons as well as anorexigenic pro-opiomelanocortin and cocaine-stimulated and amphetamine-stimulated transcript peptide producing neurons. These peptides interact with receptors in other areas of the hypothalamus, nucleus tractus solitarius and area postrema to regulate meal size and energy balance (Valassi et al., 2008). Figure 2 illustrates the homeostatic regulation of appetite.

Figure 2. The homeostatic regulation of appetite. From Lenz A, Diamond FB Jr. Obesity: the hormonal milleu. Curr Opin Endocrinol Diabetes Obes 2008;15:9-20.



AN, arcuate nucleus; POMC, proopiomelanocortin; CART, cocaine and amphetamine receptor transcript; NPY, neuropeptide Y; AgRP, agouti-related peptide; PVN, paraventricular nucleus; LH, lateral hypothalamus; MCH, melanocyte concentrating hormone; NTS, nucleus tractus solitarius; AP, area postrema; T4, thyroxine; T3, triiodothyronine; SOC3, suppressor of cytokine; CRP, C-reactive protein; PTP1B, protein tyrosine phosphatase; sst3, somatostatin receptor; PYY, protein YY; OXY, oxyntomodulin; GLP-1, glucagon-like peptide 1; CNS, central nervous system.

Moreover, cognitive, hedonic, and emotional neural processes are also involved in energy intake (EI) and energy balance (Zheng and Berthoud, 2007). As an example, increased levels of leptin that was primarily thought to selectively modulate activity of neuropeptid Y and pro-opiomelanocortin neurons, also acts on neurons in the gut involved in meal termination (Lenz and Diamond, 2008) and on neurons involved in the taste, smell and visual perceptions on food (Zheng and Berthoud, 2007). In obesity, leptin resistance and decreased levels of gut peptide involved in meal termination and suppression of appetite have been demonstrated (Lenz and Diamond, 2008).

The complex regulation of body fat stores is not a major focus of this dissertation. However, understanding that any attempt to lower established fat stores leads to

compensatory mechanisms, both in the short term and in the long term, to restore body fat stores is essential to any clinical approach to overweight and obesity.

1.2.2 The roles of physical activity and diet

1.2.2.1 Physical activity

The cornerstone of treatment for obesity is to help individuals make changes in dietary and physical activity habits. Behaviour change requires long-term support and the acquisition of behavioural skills. Numerous studies have been performed using a variety of approaches to initiate and maintain weight loss in individuals by modifying their behaviour. Medication (see below) has been used as an adjunct to some approaches. In general these studies show that weight loss programs achieve only modest success. For example in a recent meta-analysis Franz et al. found that regardless of the type of intervention (i.e. diet, physical activity, medication) weight loss achieved within the first few months of treatment plateaus at approximately six months (Franz et al., 2007). However, achieving changes in people's patterns of physical activity and diet remains the central goal of all treatment programs, due to salutary effects of moderate activity and a varied, balanced diet on health and longevity (Heilbronn et al., 2006).

In meta-analysis, increasing the amount of exercise of about 30 minutes per day contributed to approximately 2 kg weight loss after one year (Franz et al., 2007). Despite a small effect on body weight loss per se physical activity has other well-established health benefits. Physically active women and men of all ages have a lower mortality rate compared to inactive individuals (Pedersen, 2007). Moreover, exercise alone or in combination with weight loss and dietary interventions has been shown to reduce the incidence of diabetes type 2 (Tuomiletho et al., 2001; Knowler et al., 2002) and improve metabolic risk factors associated with metabolic syndrome (Couillard et al., 2001; Kraus et al., 2002; Boulé et al.,

2005; Shaw et al., 2006). In addition, studies consistently have shown that subjects participating in high levels of physical activity have improved weight loss maintenance compared to subjects with lower levels of physical activity (Johannsen et al., 2007; Franz et al., 2007; Jakicic et al., 2008).

In 2007 the American College of Sports Medicine and American Heart Association updated their guidelines for physical activity and public health. It was recommended that all healthy adults should engage in a minimum of 30 minutes of moderate endurance physical activity on five days each week as well as activities that maintain and increase muscular strength and endurance a minimum of two days each week (American College of Sports Medicine and American Heart Association, 2007). Moreover, for individuals that achieve this level of activity but remain overweight, a further increase in physical activity was stated to be a reasonable component of strategies to lose weight (American College of Sports Medicine and American Heart Association, 2007). This is an area where adherence is a challenge. To be able to design appropriate exercise prescriptions in a clinical setting, a quantified assessment of the activity pattern of obese individuals is needed.

1.2.2.2 Diet

Dietary change seems to play a larger role in the achievement of initial weight loss than physical activity. Recent meta-analyses showed that randomized controlled trials comparing dietary counselling-based weight loss programs with usual care interventions produced a mean net weight loss of approximately 5 kg after one year, of which about half was maintained after three years (Dansinger et al., 2007; Franz et al., 2007). The optimal dietary macronutrient composition for weight loss purposes has not been clearly demonstrated (Franz et al., 2007). Studies comparing various dietary compositions for the purpose of weight loss have given divergent results (Dansinger et al., 2005; Gardner et al., 2007). Some authors

found that dietary composition did not seem to influence weight loss (Dansinger et al., 2005). Others have reported superior effects of a low carbohydrate diet during one year of treatment (Gardner et al., 2007). A meta-analysis showed that carbohydrate-reduced diets achieved greater initial weight losses but no difference between diets could be demonstrated after one year (Nordmann et al., 2006).

Lowering the glycaemic index and load of the diet is a much touted and popular approach to weight loss, however, conflicting reports have appeared (Thomas et al., 2007; Aston et al., 2008). A recent meta-analysis showed that diets emphasising carbohydrates with a low glycaemic index improved weight loss by about 1 kg compared to higher glycaemic carbohydrates (Thomas et al., 2007). On the other hand, a well-controlled study in which most foods were provided to participants found no advantage of a diet with a low glycaemic index on weight loss, dietary intake or appetite ratings (Aston et al., 2008). Evidence does exist indicating that short-term weight loss and maintenance is improved when low fat protein sources of energy replace carbohydrates (Halton and Hu, 2004; Paddon-Jones et al. 2008). For improved long-term weight maintenance a reduction in fat intake has been shown to be effective in a number of studies (Howard et al., 2006; Phelan et al., 2006; Jakicic et al., 2008). However, in a well-controlled dietary intervention study a low-carbohydrate diet and a Mediterranean diet were superior to a low fat diet after two years (Shai et al., 2008). In this regard, it has to be noted that the low carbohydrate diet was not energy-restricted while the two other diets were restricted.

Despite a wide diversity of studies and opinions regarding the optimal amounts of fat and carbohydrate in weight reducing diets, a number of dietary regimens have been tried that emphasise an increased intake of vegetables and fruit (de Souza et al., 2008). Table 2 shows some beneficial effects of vegetables and fruit.

Table 2. Beneficial effects of increasing intake of vegetables and fruit

Energy density ⇩
Fibre ⇧
Vitamin C, folate ⇧
Potassium, magnesium ⇧
Antioxidants (i.e. carotenoids) ⇧
Hunger ⇩

Vegetables and fruit are rich in fibre, have low energy density and may reduce hunger (Ello-Martin et al., 2007). As an example, a randomized, clinical trial found that women given dietary advice to reduce fat and increase intakes of vegetables and fruit lost 1.5 kg more weight and reported less feelings of hunger, compared to women that were given advice to eat a low fat diet without increasing intake of vegetables and fruit (Ello-Martin et al., 2007).

1.2.3 The role of eating behaviour

Appetite is regulated by a complex interplay between physiological and psychological mechanisms. Psychological and behavioural factors such as dietary restraint, disinhibition, hunger and binge eating have been found to influence energy intake and body weight. Dietary restraint is defined as the tendency to consciously restrict food intake either to prevent weight gain or to promote weight loss by control over energy intake and types of food eaten (Pirke and Laessle, 1993). Disinhibition is the tendency to overeat in the presence of palatable foods or other disinhibitors such as emotional distress (Lowe and Maycock, 1988). Hunger has been technically defined as the susceptibility to perceived body symptoms that signal the need for food (Lowe and Maycock, 1988). Other psychological and behavioural factors as anxiety, depression, and social and cultural environments are not further discussed here.

Binge eating has been identified as a common problem among obese subjects seeking obesity treatment. Table 3 shows the diagnostic criteria for the binge eating disorder (American Psychiatric Association, 1994).

While the disorder has been reported with a prevalence of 30% among participants in weight loss programmes in earlier studies (Spitzer et al., 1992; Spitzer et al., 1993), more recent studies have reported prevalences of less than 10% (Vamado et al., 1997; de Man Lapidoth et al., 2006). However, in these studies about 20% of the participants reported binge eating without endorsing all criteria necessary to warrant a diagnosis of the disorder (Vamado et al., 1997; de Man Lapidoth et al., 2006). Obese subjects that do not meet all of the diagnostic criteria may binge eat less regularly. High scores for disinhibition and hunger have been associated with a high intake of fatty foods (Lindroos et al., 1997) and with a greater binge eating severity (Foster et al., 1998). During binge eating the energy intake per episode may be of thousands kcalories and high palatable, fatty foods are preferred (Marcus, 1993).

Table 3. The American Psychiatric Association DSM-IV-TR diagnosic criteria for binge eating disorder

A. RECURRENT EPISODES OF BINGE EATING

An episode of binge eating is characterized by both of the following:

1. Eating in a discrete period of time (e.g., within any two-hour period), an amount of food that is definitely larger than most people would eat in a similar period of time under similar circumstances.
2. A sense of lack of control over eating during the episode (e.g., a feeling that one cannot stop eating or control what or how much one is eating).

B. THE BINGE EATING EPISODES ARE ASSOCIATED WITH THREE (OR MORE) OF THE FOLLOWING:

1. Eating much more rapidly than normal
2. Eating until feeling uncomfortably full
3. Eating large amounts of food when not feeling physically hungry
4. Eating alone because of being embarrassed by how much one is eating
5. Feeling disgusted with oneself, depressed or very guilty after overeating

C. MARKED DISTRESS REGARDING BINGE EATING IS PRESENT

D. THE BINGE EATING OCCURS, ON AVERAGE, AT LEAST TWO DAYS A WEEK FOR SIX MONTHS

Note: The method for determining frequency differs from that used for Bulimia Nervosa; future research should address whether the preferred method of setting a frequency threshold is counting the number of days on which binges occur or counting the number of episodes of binge eating

E. THE BINGE EATING IS NOT ASSOCIATED WITH THE REGULAR USE OF INAPPROPRIATE COMPENSATORY BEHAVIOUR (e.g., purging, fasting, excessive exercise etc.) and does not occur exclusively during the course of Anorexia Nervosa or Bulimia Nervosa.

Increasing dietary restraint, and decreasing disinhibition, hunger and binge eating episodes are important interventions for improved weight maintenance after weight loss (Clark et al., 1994; Foster et al., 1998; Cooper et al., 2003; Vogels et al., 2005; Bulik et al., 2007). To improve eating behaviour, standard behaviour therapy is widely used in lifestyle

modification for obesity (Stunkard and Berthold, 1985; Fabricatore, 2007). Table 4 shows elements of standard behaviour therapy used in obesity treatment.

Table 4. Elements of standard behavioural therapy used in obesity treatment

- Can be delivered in individual sessions or in group sessions for 60 to 90 min
- The sessions are usually in pre-determined order
- Treatments are time-limited and problem-oriented
- Treatments are present and future-focused
    - Involve stimulus control
        - Shopping (i.e. shop after eating, shop from a list)
        - Involve making exact plans (i.e. plans for how to eat regularly, exercise or eat less in parties)
    - Involve skills for changing eating behaviour (i.e. eat slowly, do not eat when bored, sad or while watching TV)
    - Involve handling with reward (i.e. plan rewards other than eating for specific behaviour)
    - Involve self monitoring (i.e. diet diary, step counting, body weight)
    - Involve realistic goal setting for eating behaviour, exercise and weight
    - Involve problem-solving issues and prepare for relapse
    - Involve handling with dysfunctional thoughts (i.e. think about progress not shortcomings, have rational restatements of negative thoughts)
    - The patient is educated in basic nutrition (healthful eating pattern), body weight regulation, health effects on modest weight loss and physical activity

1.2.3.1 Questionnaires used to assess eating behaviour

The Three Factor Eating Questionnaire (TFEQ) (Stunkard and Messicks, 1985) and the Binge Eating Scale (BES) (Gormally et al., 1982) are recognized instruments to assess eating behaviour. The TFEQ is a 51-item instrument, which contains three subscales measuring dietary restraint, disinhibition and hunger. It consists of 36 closed questions with a forced, false/true answer format and 15 with a 4-point "Likert" scale (Stunkard and Messicks, 1985). Using a Likert scale, the participants are asked to respond to the most appropriate alternative

of almost never, seldom, usually or almost always. Examples of questions used for the assessment of the eating behaviours assessed in the TFEQ are shown in table 5.

Table 5. Examples of questions to measure the eating behaviors dietary restraint, disinhibition and hunger

Eating behaviour	*Questions*	*Scale*
Dietary restraint	How frequent do you avoid "stocking up" on tempting foods?	Almost never, seldom, usually or almost always
Disinhibition	When I feel anxious, I find myself eating.	False or true
Hunger	I am usually so hungry that I eat more than three times a day.	False or true

The BES consists of 16 sets of 3-4 statements relating to binge eating behaviour and the subject is asked to endorse the one item of each set which best describes the eating behaviour.

1.2.4 The role of pharmacological treatment

Drug treatment may be used as an adjunct to diet changes to improve treatment outcomes in subjects with BMI >30 or subjects whose BMI is in the range of 27-29.9 and who also have metabolic risk factors (Lau et al., 2007). The anti-obesity drugs orlistat, sibutramine and rimonabant are all approved in Europe for long-term treatment of obesity (Rucker et al., 2007). Here the focus will be on the pancreatic lipase inhibitor, orlistat.

Due to the pharmacological action of the drug, it has been thought that orlistat may have a policing influence on food intake. About 30% of dietary fat is not metabolised, but excreted in the faeces (Guerciolini, 1997). If the dietary intake of fat is higher than about 30% of energy, abdominal symptoms including oily spotting, faecal incontinency and flatus may ensue. In double blind, randomized placebo controlled trials including more than 10 000

participants treatment with orlistat was shown to enhance weight reduction by 2.9 kg (95% CI 2.5 -3.2) (Rucker et al., 2007). Moreover, orlistat has been shown to reduce the incidence of diabetes (Torgerson et al., 2004) and improve weight maintenance after weight loss (Richelsen et al., 2007).

The effect of anti-obesity drugs is likely to be counteracted by compensatory changes in food intake and energy expenditure to maintain a stable body weight over time, unless changes in lifestyle are made simultaneously (Speakman, 2004). However, the motivation to maintain lifestyle changes may be attenuated by the use of a drug known to reduce the intestinal absorption of fat. Subjects that are taking an active, effective drug may be less likely to be restrictive and comply with dietary recommendations than subjects taking placebo. Diet pills are often advertised with appetite suppressing, fat and carbohydrate blocking, metabolism boosting, fatigue reducing and fat burning properties. As an example an extract of a cactus was promised to induce a weight loss of 17.5 kg during four weeks. Due to the fibre component of the extract, malabsorption should occur. Weight loss was promised without any change in diet or physical activity. Some subjects might hope for weight loss independent of efforts to change lifestyle, while others might use pills as a tool to achieve changes in diet and physical activity. Thus, a better understanding of how a pill like orlistat affects eating behaviours and dietary intake during weight loss and maintenance, could be helpful to improve the clinical management of obesity.

1.3 Self report of dietary intake – the problem of underreporting

In nutritional research, in dietary counselling and in evaluation of the nutritional effects of the intervention data of dietary intake is needed. This is an area of research that is fraught with difficulties due to the tendency of human beings in Westernized societies to underreport their

energy intake. Dietary assessment methods may be retrospective such as the food frequency questionnaire (FFQ) and the 24-hour recall or prospective such as the dietary record (DR).

In general, the strength of the FFQ is its ability to assess the usual long-term intake and also to capture the intake of infrequently consumptions during the time period assessed. On the other hand, retrospective methods are subject to error because of poor recollection of past diet and difficulty estimating portion sizes. In the quantitative, prospective DR, all foods and beverages consumed are measured. However, DRs may be prone to reporting errors because of altered food choice and under-eating (Trabulsi and Scoeller, 2001). In this regard both the retrospective and the prospective dietary assessment instruments are thought to have uncorrelated errors, as well as a correlated error of underreporting.

Given the widespread tendency of misreporting, it is important to use biomarkers that have errors independent of the reported intake errors to validate the data. During weight stability, total energy expenditure (TEE)(Kniplis et al., 2001) and urinary nitrogen excretion (Bingham and Cummings, 1985) are established biomarkers for energy and protein intake, respectively. In addition plasma carotenoids (Al-Delaimy et al., 2005) are concentration biomarkers that reflects the reported intake of fruit and vegetables.

1.3.1.1 Validation of reported dietary intake against total energy expenditure

A measurement of TEE by the doubly labelled water (DLW) method is considered as the gold standard in free-living individuals (Coward and Prentice, 1985; Schoeller, 1988; Livingstone and Black, 2003). The method involves the administration of water containing enriched quantities of stable isotopes of deuterium and oxygen-18. An exact dose of the DLW is given to the subject and the concentration of the isotopes is measured in urine specimens. The concentration of the deuterium is eliminated in water. Most of the oxygen-18 is eliminated from the body in the form of water, but some is also eliminated in the form of carbondioxid.

The elimination rate of the oxygen-18 is therefore steeper than the rate for deuterium. The difference in eliminating rate between these two isotopes is a measure of carbondioxid production an indirect measure of the metabolic rate.

Despite the superior measurement of TEE, the usefulness of the DLW method in a clinical setting is limited by its high cost and technical complexity. TEE may also be estimated based on resting metabolic rate (RMR) and assessments of physical activity. Physical activity has been assessed by a number of questionnaires and different kinds of accelerometers. In the present ones, the ActiReg® method is used. ActiReg® is not an accelerometer, but an electromechanical monitor which records the main body positions (stand, sit, bent forward and lie) together with motion of the trunk and/or one leg each second. Figure 3 and 4 shows the ActiReg® monitor.

Figure 3. The ActiReg® unit.

The picture shows the close-up of the Actireg® unit. The apparatus has two pairs of position and motion sensors connected by cables to a battery operated storage unit.

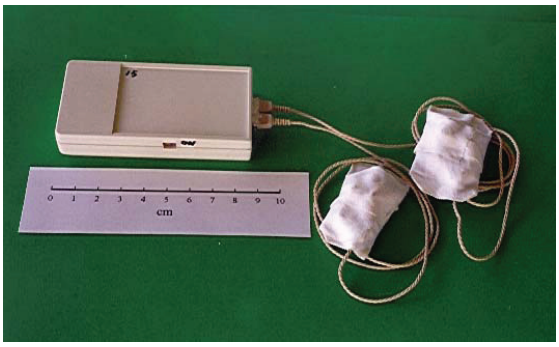
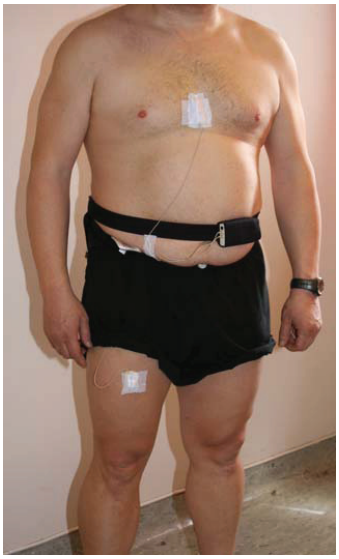


Figure 4. The Actireg® during registration.

The picture shows a person wearing the Actireg®. One pair of sensors is placed on the chest (sternum) while the other is placed on the right thigh. The sensors are attached to the skin with medical tape. The storage unit is placed in a small bag fixed to an elastic belt worn around the waist.



An estimate of RMR is an important part of the ActiReg® method to calculate TEE. RMR can be calculted by equations or measured by direct or indirect calorimety. Indirect calorimetry is based on the fact that when food is oxidized in the body, oxygen is used and carbondioxid is produced in proportion to the heat generated. Thus, measuring the volume of oxygen consumption and carbondioxid production, the energy expended can be calculated (Haugen et al., 2007).

A validation of the ActiReg® method against the DLW method in obese subjects is important for the usefulness of the method in a clinical setting. Furthermore, all established methods used in the dietary assessments rely on the subjects' self-reported intake. It has been shown that the report of dietary intake is influenced by a number of variables such as gender, age, BMI, socioeconomic status, physical activity, smoking status,

psychological factors, cultural context, dieting and eating behaviour (Livingstone and Black, 2003). Thus, a better understanding of the accuracy of reported intake data might be helpful to improve dietary counselling and evaluate effects of dietary interventions for sustained weight reduction.

2 AIMS AND RESEARCH QUESTIONS

The overall purpose of this doctoral dissertation is to inform clinical interventions promoting behaviour change (including diet and physical activity) for the purpose of weight reduction and the maintenance of weight loss in obese subjects with high risk of CVD. The dissertation focuses on assessment and lifestyle issues including the accuracy of dietary report and energy expenditure data, assessment of physical activity patterns, effects of dietary change and medication for weight loss. The independent scientific contributions consist of three papers using a cross-sectional design (of which two are validation studies) and three papers reporting two randomized controlled clinical trials. The individual aims of each paper are stated in their introductions. As this dissertation focuses on more general aspects of assessment and counselling, the research questions are specified below.

Research questions:

1. What is the accuracy of dietary intake data reported by obese subjects with components of metabolic syndrome? Paper 1
2. Does recording of physical activity obtained by ActiReg® contribute to a valid estimate of total energy expenditure in obese subjects with components of metabolic syndrome? Paper 2
3. What is the level of physical activity at baseline in obese subjects with components of metabolic syndrome who are seeking treatment for weight loss? Paper 3
4. What is the effect of dietary intervention to increase intake of vegetables and fruit on weight loss and CVD risk factors in obese subjects with SRBD? Paper 4
5. How does orlistat affect eating behaviour and dietary intake when used for long-term weight maintenance in obese subjects with components of metabolic syndrome? Papers 5 and 6

3 MATERIALS AND METHODS

3.1 Subjects

Table 6 shows an overview of the study population and design of the studies.

Table 6. An overview of the study population and design of the studies

Paper	*N*	*Population*	*Design*
Paper 1 and 2	50	Non-smoking men and women with components of metabolic syndrome (all subjects were included in Paper 3)	Cross-sectional, validation studies
Paper 3	130	Men and women with components of metabolic syndrome	Cross-sectional study
Paper 4	138	Men and women with sleep related breathing disorders	Randomized, controlled, intervention trial
Paper 5	306	Men and women with components of metabolic syndrome	Randomized, double-blinded, clinical trial (The SMOMS multi-center trial)
Paper 6	44	The Norwegian sample of the men and women with components of metabolic syndrome in the SMOMS trial (40 of the subjects were included in Paper 3)	Controlled, intervention study

3.1.1 Obese subjects with components of metabolic syndrome

The participants in Paper 1 and 2 were 27 women and 23 men, all non-smoking and aged 24 to 64 years with a mean BMI of 36 kg/m^2 (range 31-48). They were recruited by newspaper advertisement or referral to the Department of Preventive Cardiology at Ullevål University

Hospital. Inclusion criteria were two or more risk factors for metabolic syndrome according to the National Cholesterol Education Program: glucose ≥ 6.1 mmol/L or HDL-cholesterol ≤ 1.03 mmol/L for males or ≤ 1.29 mmol/L for females or serum triglycerides ≥ 1.69 mmol/L or waist circumference > 102 cm for males or > 88 cm for females or systolic blood pressure ≥ 130 mm Hg and diastolic blood pressure ≥ 85 mm Hg. Exclusion criteria were body weight > 135 kg, current dieting, cigarette smoking, history of eating disorder or other chronic diseases, drug or insulin treated diabetes mellitus and abuse of drugs or alcohol. In Paper 1, one subject did not complete the DR.

Paper 3 included 68 women and 62 men aged 24 to 64 years with a mean BMI of 37 kg/m^2 (range 30-50) and ≥2 components of metabolic syndrome according to criteria of the National Cholesterol Education Program (National Cholesterol Education Program, 2002). The 27 women and 23 men participating in the validation studies (Papers 1 and 2) were part of this sample as were 19 women and 21 men in Paper 6. All participants were recruited by newspaper advertisement or referral to the Department of Preventive Cardiology at Ullevål University Hospital.

In Paper 5 the participants were 149 men and 157 women aged 19 to 64 years with a mean BMI of 38 kg/m^2 (range 30-45) of which 197 (64.4%) remained in the study after three years. They were recruited from nine clinical research centres in Denmark, Finland, Norway, and Sweden in response to newspaper advertisements to participate in the Scandinavian Multicenter study of obese subjects with the Metabolic Syndrome (SMOMS). The main results from the study have been published (Richelsen et al., 2007). The inclusion criteria were abdominal obesity (waist circumference > 92 cm for females and > 102 cm for males) and at least one of the following metabolic risk factors: impaired fasting glucose (> 6.1 mmol/L and < 7.1 mmol/L), diet treated type 2 diabetes, dyslipidaemia (HDL-cholesterol < 1.1 mmol/L for females and 0.9 mmol/L for males) or triglycerides > 2.0 mmol/L. Subjects

were excluded if they had clinically relevant conditions that might affect the outcome of the trial (i.e. weight change of more than ± 4% for the last two months before inclusion, pregnancy, lactation, chronic diseases, weight loss surgery, bulimia, fat soluble vitamin deficiencies, abuse of drugs or alcohol).

In Paper 6 the subjects were the Norwegian participants in the SMOMS trial (Richelsen et al., 2007). Fifty-two subjects were randomized after weight loss, 44 subjects were examined after one year and 33 subjects were examined two months after the end of the trial.

3.1.2 Obese subjects with sleep related breathing disorders

The subjects in Paper 4 were 138 men and women diagnosed with SRBD of which 95 men and 30 women completed the study. The participants were consecutively referred from the Ear, Nose and Throat Department at Ullevål University Hospital or from primary care physicians to the Department of Preventive Cardiology for weight reduction. Their diagnosis was established by polysomnography in a sleep laboratory. They were aged between 28 and 72 years and had a mean BMI of 37 kg/m^2 (range 27-55). Exclusion criteria were any major non-cardiac disease expected to reduce life expectancy and abuse of drugs or alcohol.

Fifty of the subjects in Paper 4 tested the reproducibility of the FFQ. The reproducibility data are not published but will be briefly discussed in this overview.

3.2 Design of the studies

3.2.1 Papers 1 and 2

Papers 1 and 2 were methodological, validation studies using cross-sectional design initiated by Serena Tonstad and Mette Svendsen. In paper 1 we compared EI to TEE. EI data were obtained by a dietary interview based on a FFQ and by DR. TEE was measured by the doubly

labelled water (DLW) method. Accuracy was seen as the expected EI/TEE of 1.00. Subjects were classified as under reporters and non-under reporters by how much the EI/TEE ratio deviated from 1.00 by calculating the 95% confidence interval (CI) of the EI/TEE ratio according to the equation by Black & Cole, 2000:

$$95\%CI = \pm 2 \cdot \sqrt{[(CV_{TEE})^2 + (CV^2_{EI}/d)]}.$$

For the coefficient of variation $(CV)_{TEE}$ for repeated measurements for energy measurements by the DLW method 8% was used (Black & Cole, 2000). CV_{EI} is the within-subject CV for daily intake of energy and 23% was used (Bingham, 1987). The number of days was 90 for the FFQ and three for the DR.

In paper 2 TEE was calculated by the ActiReg® method based on a registration of physical activity and measured RMR. This result was compared to TEE measured by the DLW method.

3.2.2 Paper 3

Paper 3 was a cross-sectional study reporting the physical activity patterns of obese patients with components of metabolic syndrome before advice for change in diet or physical activity for weight loss purposes was given. Recording of physical activity with ActiReg® is described.

3.2.3 Paper 4

This is a randomized, controlled clinical trial initiated by Serena Tonstad and Mette Svendsen. The effects of an increased intake of vegetables and fruit were studied in subjects with SRBD. All subjects received dietary advice for weight loss before randomization. Subjects in the intervention group were given dietary counselling in a behavioural treatment program in eight sessions.

3.2.4 Papers 5 and 6

In paper 5, the effect of orlistat on dietary restraint, disinhibition, hunger and binge eating was examined in a randomized, multicentre clinical trial initiated by Aila Rissanen and supported by the manufacturer of orlistat. Subjects that achieved a weight loss of ≥5% during eight weeks of treatment with a liquid very low energy diet (VLED) were randomized to orlistat or placebo treatment for three years. The subjects were monitored monthly for the first 18 months and then every third month for the rest of the study.

In paper 6, dietary intake data was compared between Norwegian participants in the SMOMS study randomized to orlistat or placebo before the VLED, one year after randomization and in subjects that chose to take orlistat or not two months after the end of the trial.

3.2.5 Assessments of dietary intake

The main dietary assessment method in this overview is the FFQ used as a semi-structured interview lasting for 1 to 2 hours (Paper 1, 4 and 6). In the interview the subject reported the frequencies and the estimated amount of 174 food items and beverages that have been consumed over the last three months. Portion sizes were estimated with the used of an atlas of food portions, photographs, household measurements and ordinary models of sweets and snacks. In Paper 1, the dietary assessments were done immediately after the assessments of energy expenditure. In Paper 4, the dietary assessments were done at baseline and after three months of intervention. In Paper 6, the dietary intake was assessed before the VLED, one year after randomization to orlistat or placebo and two months after the end of the trial. The same registered dietician (Mette Svendsen) performed all the dietary interviews.

In Paper 1 weighed dietary records for three non-consecutive days were obtained about one month before the measurement of TEE. In Paper 6, a 24-hour dietary recall was conducted one year after randomization. The same dietician (Mette Svendsen) performed all the dietary recalls. Portion sizes were estimated with use of the same tools as for the FFQ. The results of the questionnaires, DRs and recalls were coded manually and energy and nutrient calculations were done with the same software program (Mat på data 3.0, 1996).

3.2.6 Measurement of energy expenditure and physical activity

3.2.6.1 The doubly labelled water method

In paper 1 and 2 the doubly labelled water method was used to assess total energy expenditure. The participants ingested 0.05 g deuterium-labelled water and 0.10 g oxygen-18-labelled water per kg body weight and this dose was planned to enrich body water by approximately 350 δ (delta per million) for deuterium and 60 δ (delta per million) for oxygen-18. Urine samples for analysis of the elimination of the isotopes were collected at day 2, 3, 4, 8, 13, 14 and 15 after ingestion of the isotope-enriched water at day 1. The analyses were done in triplicates with a mass spectrometer (Thermoquest Finnigan MAT, Bremen, Germany). The elimination rates for the isotopes were calculated by the multipoint method (International Dietary Energy Consultancy Group, 1990). The difference between the elimination rates of the isotopes was used as an indirect measure of carbondioxid production. The analyses and calculations were done at the Sahlgrenska Academy in Gøteborg, Sweden.

3.2.6.2 The ActiReg® method

In paper 2, 3 and 6 TEE was estimated by the ActiReg® method. A specially developed computer program (ActiCalc32®) was used to calculate TEE. The calculation model is based on the estimated energy cost of the body position and activity combined with the number of

position changes each minute. Table values (Food and Agricultural Organization/World Health Organization/united Nations University, 1985) for energy expenditure during different activities were used in according to corrected values for weight bearing activities. The corrected values for weight bearing activities were calculated from oxygen expired during a standardized, walking test on a treadmill at six different intensities (1-6 km/h) done at the National School of Sports, Oslo (Paper 2). The physical activity patterns are expressed as physical activity ratios (PAR) as is the ratio of energy expenditure to RMR. TEE is calculated by summarizing the calculated energy expenditure for each minute of the measurement period.

In Paper 2 the subjects recorded physical activity by the ActiReg® method during the first seven days of the DLW measurement period. In this overview not only TEE, but also the physical activity level (PAL) value (i.e. the ratio of TEE to RMR) will be discussed. Dr. Bo-Egil Hustvedt, University of Oslo, was responsible for the calculations of the calibrated values.

In Paper 3, physical activity patterns were assessed by an ActiReg® recording performed continuously for a mean of four days. Time spent on different levels of physical activity categorised as PAR levels was calculated using the ActiCalc32® program. Based on table values a PAR of 1.0-1.5 corresponded to lying down standing and sitting (Food and Agricultural Organization/World Health Organization/united Nations University, 1985) and based on the results of the treadmill test (Paper 2), a PAR 1.6-6.0 corresponded to strolling and walking and a PAR >6 corresponded to brisk walking and jogging at a speed of more than 4.5 km/h.

3.2.6.3 The resting metabolic rate

In Paper 2 we measured RMR indirectly with a standard portable ventilated hood system (Deltatrac® Metabolic Monitor; Datex Instrumentarium Corp., Helsinki, Finland). The measurements and calculations were done after standardized procedures (Wooley, 2006). Dr. Bo-Egil Hustvedt, University of Oslo, performed the RMR measurements and calculations. RMR may also be estimated by several equations (Paper 2). In Paper 3 and 6, the Mifflin equation was used to calculate RMR (Mifflin et al., 1990):

RMR (kcal) women: 9.99 · weight (kg) + 6.25 · height (cm) – 4.92 · age - 161

RMR (kcal) men: 9.99 · weight (kg) + 6.25 · height (cm) – 4.92 · age + 5

3.2.7 Assessment of eating behaviour

We used the TFEQ to assess the eating behaviours restraint, disinhibition and hunger in Papers 1 and 5. Binge eating was assessed with the BES in Paper 5. Scores were calculated and the ranges for possible scores were 0-21 for dietary restraint, 0-16 for disinhibition 0-14 for hunger and 0-46 for BES (Gormally, et al., 1982). Higher scores reflect a greater tendency to exhibit the particular eating behaviour characteristics.

3.2.8 Laboratory measurements

Fasting serum lipids and glucose were analysed at the Clinical Chemical Department of Ullevål University Hospital (Papers 1 and 4) or centrally for all the sites participating in SMOMS (Medilab, Copenhagen, Denmark) in Papers 5 and 6. In Paper 4 serum folate was analysed at the Clinical Chemical Department of Ullevål University Hospital and plasma carotenoids and the ferric reducing antioxidant power were analyzed in batches at the Institute for Nutrition Research, University of Oslo, Oslo.

3.2.9 Anthropometric measurements

Physicians at the Department of Preventive Cardiology performed the baseline measurements of weight, height, and waist- and hip circumference with standardized tools in Papers 1-4. The measurements were done by the same registered dietician (Mette Svendsen) for the rest of the study periods. In Paper 5 these measurements were done by the responsible staff at the different SMOMS sites.

Waist was measured with the subject unclothed, midway between lower costa and crista (Papers 5 and 6) or at the level of the umbilicus (Paper 4) while the subject was standing. Hip circumference was measured at the level of the greater trochanter. BMI was calculated as weight in kg divided by height in square meters. Blood pressure was measured by physicians or staff with a mercury or digital blood pressure monitor. After the subjects sat quietly for 5 minutes, blood pressure was measured in the right arm with an appropriately sized cuff.

Body composition was determined by DEXA in Paper 1, 2 and 3 at Spesialistsenteret Pilestredet Park, Oslo. The responsible physician was Johan Halse, a specialist in endocrinology.

3.2.10 Interventions

The intervention in Paper 4 was for subjects to increase the daily intake of vegetables and fruit to 400 g and 300 g, respectively. The overall goal was to replace vegetables and fruit for more energy dense food items to achieve energy deficiency. In Papers 5 and 6 weight loss was achieved with a commercially available VLED (≤ 800 kcal per day) administered for eight weeks. The dietary recommendations for the weight maintenance phase of the trial were to follow a diet consisting of <30% of energy from fat and to decrease the intake of saturated fat to <10% of energy while allowing for a small amount of unsaturated fat from fatty fish and

plant sources. The subjects were advised to increase their intake of fibre from vegetables, legumes, fruits and whole meal bread, to choose low fat diary products and lean meat or chicken instead of minced meat and sausages, and to reduce their intake of cakes/biscuits, ice cream, chocolate, sugar containing beverages and alcohol when re-introducing foods after they finished the VLED.

In Papers 4, 5 and 6, the dietary and behavioural interventions were mostly done in group sessions. All participants were given individual dietary advice before randomization in Paper 4 and the following sessions in the intervention group were done in groups with 10-18 subjects. In the group sessions practical implementations of increasing the daily intake of vegetables and fruit were focused. The subjects were given homework exercises and their experiences were discusses in the groups. Table 7 shows the contents of the sessions.

Table 7. The contents of the sessions.

Sessions	*Theme of the session*	*Practical exercise at the sessions*	*Homework exercise*
1.	The scientific background for the recommendation to increase vegetables and fruit for health and weight loss purpose	Exhibition showing the weight of some vegetables and fruit	
	Goal	Make plans to increase intake of vegetables and fruit	Try to eat 400 g vegetables and 300 g fruit
2.	How to increase intake of vegetables and fruit?	Make an eating plan with regular meals and snacks	Use the eating plan
	Increase intake of vegetables to dinner and lunch		Eat vegetables to the dishes
	Use fruit in stead of sweets, desserts and snacks		Make fruit available
	If necessary, use vegetables for the evening meal		Make salads or soups

3.	The plate model that includes 50% of the plate to be filled with vegetables	Make plans to use the plate model for lunch and dinner	Make shopping lists Prepare salads for lunch
4.	Increase the variability of vegetables and fruit No forbidden vegetables and fruit Use the vegetables and fruit of the season Use beans and lentils to increase satiety Use small amounts of oils and dressings to increase taste	Shopping in a vegetable and fruit store	The group make their own recipe book Participate in cooking-course
5.	Healthy eating; reduce intake of fatty diary and meat products, eat more fish, lean meat and chicken, use low fat diary products, use small amounts of oils, dressings and nuts, use whole-meal or rye bread, reduce intake of sweets, desserts and snacks	The group evaluted their dietary intake based on a 24-hour recall	Record your dietary intake for at least a week
6.	How to eat in parties?	Talk about how to deny politely Make plans for restricting cakes Plan to eat much vegetables	Bring fruits as a gift Make sure that salads and vegetables will be served
7.	How to eat during holidays?	Plan to eat regularly meals Plan to increase physical activity When and how to do?	Prepare to do your physical activity in any circumstances
8.	Body weight regulation Realistic goal setting of 7-10% weight reduction	Write the weight goal that you: Hope to achieve Will be satisfied with Will not be satisfied with	Think about what you have achieved, how does it feel in terms of eating healthy, easiness to move, pains

In papers 5 and 6, the subjects were offered dietary and behavioural therapy in groups for the first 18 months of the study and for the next 18 months the subjects met individually with a dietician. At the group session's exhibitions were used to illustrate the amount of fat in foods. Subjects were taught to read food labels, make a plan for mealtimes and plan menus. Subjects were instructed to use a plate model for hot meals. They were asked to use this model at different occasions including social eating and eating at restaurants. The subjects were asked to assess eating behaviour and plan specific strategies to avoid pitfalls. The behavioural program otherwise included features such as goal setting, stimulus control and cognitive restructuring (Melin & Rössner, 2003). In studies 5 and 6, the subjects that achieved a weight loss of more than 5% during the VLED, were randomized to orlistat 120 mg three times per day or to matching placebo three times per day.

3.2.11 Statistical analyses

In all papers descriptive data were presented as means (SD) or as medians with interquartile ranges for not normally distributed data. Mean and median differences between groups were tested with the unpaired Student's t-test and the Mann-Whitney rank test for normally distributed and skewed variables. Differences within the groups were tested with the paired t-test or Wilcoxon signed rank test as appropriate. The Chi squared test was used in comparisons of categorical variables. Pearson's correlation coefficients were calculated for normally distributed variables and Spearman's rank correlation coefficients were calculated for skewed variables.

In paper 1, simple regression analyses were conducted and factors that were statistically significant in the univariate analysis were entered into a multiple regression analyses to identify the most important factors correlated to reported accuracy of energy intake. In Paper 2, the agreement of the results obtained by the methods was tested by the

method of Bland Altman. In Paper 3, serum triglycerides and the amount of time spent at PAR >6 were log-transformed because of skewness. Variables that were significantly correlated with minutes per day spent at PAR>6 were entered into a multiple regression analysis and adjusted for confounders.

In Paper 4, we conducted multiple regression analyses to identify factors that determined weight change in all subjects as follows: To test for group homogeneity, we did a multiple regression analysis including the exposure factor (x_1), treatment variable (x_2) and the product variable of x_1 and x_2. When the product variable was found to be significant in the multiple regression analysis, the slopes of association were claimed to be different between treatment groups. In such a case simple regression analysis between change in weight and the exposure factor was done within each treatment group. Group homogeneity was tested for the presence of diabetes or CVD, use of antihypertensive or lipid-lowering medication, smoking habits and gender. The tests for homogeneity showed heterogeneity between the control and intervention group only in regard to use of antihypertensive medication. Because of the heterogeneity, use of antihypertensive medication was not included in the final multiple regression analysis.

In Papers 1 and 4, energy density was calculated for the whole diet minus drinks (coffee, tea, milk, juice, soft drinks and alcoholic beverages).

In Paper 5, analysis of variance using least square means with changes in weight as covariates was done to test the possible difference between the orlistat and placebo group with regard to changes in scores for eating behaviour at the measured time-points. Changes were calculated as the difference in scores between screening and subsequent measurements. To test the interaction effect between genders F-tests from ANCOVA were used. To test the association between eating behaviour and change in weight at month 33, we conducted multiple regression analyses with change in weight as the dependent variable and BMI at

screening, age, gender, treatment and the changes in scores for restraint, disinhibition, hunger and binge eating, respectively, as four separate independent variables. Analyses were done using the observed values (for which the data are shown), last observation carried forward values and for the completers. The statistician (Fredrik Hansson) performed the statistical procedures in Paper 5.

Statistical tests were considered significant at $P < 0.05$. Statistical analyses were performed using the Stat View 5.0.1 software in Papers 1, 3, 4, and 6. SPSS for windows (version 13.0.0) software was used in Paper 2 and SAS software (version 9.1.3) was used in Paper 5.

4 REVIEW OF THE PAPER RESULTS

I. **Accuracy of food intake reporting in obese subjects with metabolic risk factors.**

Mette Svendsen and Serena Tonstad.

We examined the determinants of reporting accuracy and explored which specific foods and eating behaviours were associated with underreporting. The DLW method was used to estimate TEE. Fifty women and men with a BMI of 36 kg/m^2 participated in a dietary interview based on a FFQ and completed weighed DR. TEE did not differ between underreporters (one-half of the sample) and non-underreporters. The accuracy was assessed by the ratio EI/TEE as is expected to be 1.00. The EI/TEE was 0.68 in underreporters and 1.01 in non-underreporters according to the FFQ. According to the DR the accuracy was 0.57 in underreporters and 0.85 in non-underreporters. Underreporters had lower median intakes of sweets/desserts/snacks than non-underreporters according to both methods. According to the FFQ, reported intake of bread and sweets/desserts/snacks were determinants of accuracy. According to the DR, sweets/desserts/snacks and dietary restraint were determinants of accuracy.

II. Validation of ActiReg® to measure physical activity and energy expenditure against doubly labelled water in obese persons.

Bo-Egil Hustvedt, Mette Svendsen, Arne Løvø, Lars Ellegård, Jostein Hallén and Serena Tonstad.

In this study we compared TEE estimated by the DLW method and TEE calculated from a seven-day recording of physical activity with ActiReg® in 50 obese subjects. We also compared RMR measured by indirect calorimetry and estimated by standardized equations. TEE estimated by the DLW method was 3322 kcal (SD 590 kcal) and total energy expenditure calculated from ActiReg® was 3219 kcal (SD 540 kcal), an underestimation of about 4%. RMR derived from standard equations based on weight, age and sex were overestimated while the RMR based on fat-free mass values in addition was underestimated.

III. Physical activity patterns of obese subjects with a high metabolic risk profile.

Mette Svendsen, Bo Egil Hustvedt and Serena Tonstad.

In this study we assessed physical activity patterns of 130 treatment seeking obese women and men with components of metabolic syndrome and examined the association between cardiovascular risk factors and time spent on moderate intensity physical activity. Physical activity was measured continuously with ActiReg® for a mean of four days. Time spent on physical activity was categorized as PAR levels expressed as the ratio of energy expenditure of different activities to RMR. The subjects daily spent a mean of 1065 minutes lying, standing and sitting (PAR 1.0-1.5), 348 minutes strolling and walking (PAR 1.6-6.0), and 26 minutes brisk walking and

jogging (PAR >6.0). The time spent on brisk walking and jogging was inversely associated with total cholesterol and triglycerides explaining about 6% and 8%, respectively of the variation in the lipid levels after adjustment for age and sex.

IV. The effect of an increased intake of vegetables and fruit on weight loss, blood pressure and antioxidant defence in subjects with sleep related breathing disorders.

Mette Svendsen, Rune Blomhoff, Ingar Holme and Serena Tonstad.

The effect of an increased intake of vegetables and fruit with regard to weight loss, blood pressure and antioxidant defence was studied in a randomized, controlled trial in 138 obese subjects with SRBD. The intervention to increase the intake of vegetables to 400 g per day and fruit to 300 g per day was conducted in a group based behaviour program. After three months, the intervention group achieved a 2% better weight loss and reduced systolic (-7 mmHg) and diastolic (-4 mmHg) blood pressure (mean difference between the groups), compared to the control group. A doubling in the intake of fruit and vegetables resulted in increased concentrations of plasma α-carotene and β-carotene, with no change in antioxidant defence measured with the ferric reducing antioxidant power assay. In a multiple regression analysis the change in intake of vegetables was a significant contributor to the change in weight.

V. Effect of orlistat on eating behaviour among participants in a three-year weight maintenance trial.

Mette Svendsen, Aila Rissanen, Bjørn Richelsen, Stephan Rössner, Fredrik Hansson and Serena Tonstad.

We studied the effect of orlistat on dietary restraint, disinhibition, hunger and binge eating and assessed the relation between changes in eating behaviour and weight maintenance in 306 abdominally, obese subjects included in the SMOMS trial. This was a three-year clinical trial of orlistat or placebo following an eight-week VLED. Compared to screening, dietary restraint was increased and disinhibition, hunger and binge eating were decreased in the orlistat and in the placebo group. These changes were similar in both groups with the exception of the hunger score. This score was reduced more in the placebo than in the orlistat group at the end of the study. In multivariate analyses scores for restraint, disinhibition and binge eating were associated with weight loss after adjustment for BMI, gender, age and treatment.

VI. The long-term influence of orlistat on dietary intake in obese subjects with components of metabolic syndrome.

Mette Svendsen, Merete Helgeland and Serena Tonstad.

In this study we assessed dietary intake in the Norwegian participates in the SMOMS trial. A dietary interview based on a FFQ was conducted in 44 subjects before weight reduction and after one year of treatment with orlistat or placebo. Two months after the end of the trial, dietary intake was assessed in 33 subjects that remained in the trial. At one year, dietary intake did not differ between the orlistat and placebo group. Energy percent from fat was reduced and energy percent from carbohydrate was increased within both groups. Two months after the end of the trial when the use of orlistat was optional, energy percent of fat was 32.6% in subjects that chose to take orlistat and 27.7% in subjects not taking orlistat.

5 DISCUSSION

5.1 General discussion

Conservative therapy for weight loss and maintenance consists of changes in diet, physical activity and eating behaviour. Pharmacological therapy is an adjunct to changes in lifestyle. This discussion will focus on these four elements of the clinical management of obesity.

- Assessments of dietary intake
- Measurements of energy expenditure and physical activity
- The effect of an increased intake of vegetables and fruit
- The influence of an anti-obesity drug on eating behaviour and diet

In this section methodological considerations and a discussion of the results will precede implications for treatment.

5.1.1 Methodological considerations

5.1.1.1 Assessment of dietary intake

In Paper 1 we validated reported EI against TEE measured by the DLW method, as is the gold standard. When using TEE as an objective measure of EI, we assume that the subjects were in energy balance over the measurement period of 14 days. The measurement period was according to standard procedures (International Dietary Energy Consultancy Group, 1990) but may still be too short to measure long-term energy expenditure. Weight changes during the study were minor and were not adjusted for (International Dietary Energy Consultancy Group, 1990).

Another methodological issue in Paper 1 was that the subjects recorded food intake for only three days about one month before the measurement of TEE. This was to reduce the burden on the participants. Ideally, the DR should have been performed during the measurement period. Usually a seven-day record is recommended for a valid estimate on

energy intake (Bingham, 1987). A longer period of recording may have increased the reported energy intake due to less day-to-day variability, especially within the non-underreporting group. However, within the underreporting group that showed a higher dietary restraint, a longer recording period may have been out weighted by more underreporting (Trabulsi and Schoeller, 2001).

Men and women were almost equally represented in the study. Due to the high cost of the method, the study population was small and we do not have a non-obese control group. The FFQ method has recently been shown to better capture obesity related reporting than 24-hour dietary recalls (Lissner et al., 2007).

5.1.1.2 Measurement of energy expenditure

In Paper 2, we compared TEE measured by the DLW method and by the ActiReg® method. A methodological consideration in this study was the difference in measurement period. To reduce the burden of the subjects, the ActiReg® recordings were only done during the first seven days of the DLW measurement period. The subjects may have been more active during the ActiReg® recording compared to the whole measurement period. If this was the case, more underestimation of TEE by ActiReg® compared to TEE measured by the DLW method would have been expected.

Another methodological issue is the PAR levels we used to summarize TEE. Data is sparse on energy expenditure during various intensities of weight bearing activities in the obese. The PAR level approach has been assumed to compensate for differences in BMI. However, table values are based on measurement in normal weight subjects (Food and Agricultural Organization/World Health Organization/United Nations University, 1985) and increasing PAR with increasing body weight has been shown (Kuriyan et al., 2006). In Paper 2 we reported that walking faster than 4.5 km/h corresponded to a PAR>6 as is higher than

reported in normal- and overweight subjects (Food and Agricultural Organization/World Health Organization/united Nations University, 1985; Kuriyan et al., 2006). If using normal weight PAR levels for weight bearing activities, the underestimation of TEE by the ActiReg® method compared to TEE measured by the DLW method would have been about 15% (Paper 2). Thus, using higher PAR levels in the obese seems appropriate.

5.1.1.3 Registration of physical activity

In paper 3, we assessed physical activity patterns with the ActiReg® method. The study was cross-sectional and cannot be used to determine causality. We used an objective measurement of physical activity, but the study has some methodological considerations. As discussed above, we used PAR>6 for walking with a speed higher than 4.5 km/h. If the higher energy cost of weight bearing activities in the obese had not been taken into consideration, time spent on PAR>6 would have been lower.

We measured physical activity for only four days. In the updated recommendations from the American College of Sports Medicine and American Heart Association, five days a week was identified as a minimum level of physical activity (American College of Sports Medicine and American Heart Association, 2007). Although the measurement period was within the recommended number of days for adults (Troiano, 2005), ideally the measurement period should have been for five days. It is a possibility that the subjects increased their physical activity in a shorter period of recording. Indeed, a higher PAL was seen among our subjects compared to the observed PAL in the studies that used ActiReg® for seven days (Mattiessen et al., 2008; Kurtze et al., 2007). Moreover, we did not separate between vigorous and moderate intensity. If some of the activity recorded was of vigorous intensity, then the calculated time spent on PAR>6 should have been higher. It has to be argued that in the NHANES 2003-2004 the time spent on vigorous physical activity was less than one minute

per day (Troiano, 2008). The level of physical activity at vigorous intensity probably would have been low, but this was not measured in our study. We also accumulated all bouts of time spent on PAR>6. In the 2007 recommendations it is clearly stated that the minimum length of the short bouts used to accumulate physical activity has to be at least 10 minutes (American College of Sports Medicine and American Heart Association, 2007). This may have been of minor influence because in the NHANES 2003-2004 the results of calculating bouts minutes per day were similar to the overall minutes per day (Metzger et al., 2008). Another consideration in this study was that we used estimated RMR based on the Mifflin equation instead of a measurement of RMR. Ideally, the RMR should have been measured, but when calculating PAL and PAR levels the RMR is both in the numerator and in the divider and the error in RMR will be outweighed.

5.1.1.4 The effect of an increased intake of vegetables and fruit

In the randomized, controlled dietary intervention study (Paper 4) we examined the effect of a dietary intervention during three months. Long-term adherence to dietary interventions is paramount in the clinical management of obesity. This study did not assess the long-term adherence to the diet because the study was a part of a longer study involving a three months weight loss period and a one-year weight maintenance period with orlistat. Only the three-month period is reported here (the result of the one year maintenance period will be reported elsewhere). Ideally the weight loss period should have lasted for six months (Franz et al., 2007), but three months was chosen because the control group had to wait for treatment. The subjects in the control group were waiting-list controls and we wanted to offer them treatment as soon as possible. Another consideration by the study design was that the treatments had unequal intensity between the intervention and the control group. However, the treatment offered to the control group reflected a usual setting of individual therapy with a dietician.

This study showed that the recommended amounts of vegetables and fruit were feasible to achieve in a daily life setting where subjects had to plan, purchase and prepare their own food. More than one-half of the subjects reported consumption of the recommended amount of vegetables and fruit. However, overreporting of fruit and vegetables in the intervention group may occur. We did not validate the energy intake data as will be discussed in the next section, but we used carotenoids as biomarkers for the reported intake of vegetables and fruit. β-chryptoxanthin, α-carotene and lycopene have been shown to predict intake of citrus fruit, carrots, and tomato products, respectively (Al-Delaimy et al., 2005). Indeed, we found reported intake of vegetables associated with α-carotene and intake of fruit associated with β-chryptoxanthin. Moreover, the concentrations of α-carotene, zeaxanthin and lycopene after three months in the intervention group were in line with the concentrations reported after increasing intake of vegetables and fruit by 450 g in a well-controlled feeding study (Brevik et al., 2004) (data not shown). Thus, based on the results of the carotenoid analyses we assume that the reported intake of vegetables and fruit showed adherence to the dietary intervention. The lower concentrations of some of the carotenoids may reflect differences in intake; not all vegetables and fruit are rich in the various carotenoids.

We used the ferric reducing antioxidant power assay to evaluate the effect of the dietary intervention on antioxidant defence and found no effect on antioxidant capacity. The lack of effect may be due to the fact that this assay is better suited for measurements of antioxidant status in plant products than in biological samples (Ghiselli et al., 2000). Carotenoids are lipid-phase antioxidants and are important to prevent and repair lipid peroxidation (Karlsen, 2008). Indeed, vegetables such as broccoli, carrots, onions and tomatoes that are high in carotenoids also contain compounds that may induce endogenous antioxidant effect (Blomhoff, 2005). Thus, the increased intake of vegetable and fruit may have improved the antioxidant capacity of the subjects in the intervention group due to the

increase in plasma concentrations of plasma α- and β- carotenoids despite no change in the ferric reducing antioxidant power assay.

The sample size and statistical power calculations were based on the assumed difference in weight between the intervention and control group. Unfortunately, the sample size was too small for us to perform sub-group analyses of subjects treated or not treated with CPAP.

5.1.1.5 The influence of orlistat on eating behaviour and diet

In paper 5, the influence of orlistat on eating behaviour was examined in more than three hundred obese subjects about equal distributed between genders. Thus, the statistical power was good. The subjects were followed for three years in a placebo controlled trial and the completion rate was relatively high. We were able to prospectively measure changes in eating behaviour at several time-points. However, we did not have any measurement of the eating behaviour at randomization, when the olistat or placebo treatment started. It was not clinically relevant to assess eating behaviour after an eight-week, liquid VLED before ordinary diets was restarted.

Eating behaviour was measured with the validated questionnaires TFEQ and BES (in Paper 1 the TTEQ was used). An important consideration is how questions that are not answered are handled. We scored unanswered questions as zero, meaning that the subject got a low score on the eating behaviour. If the subject rejected to answer the question because of embarrassment or neglect, the score on the eating behaviour would have been too low. Another consideration is that the Norwegian questionnaires were translated for the purpose of the SMOMS study. The translation was not back translated.

In Paper 6, dietary intake was assessed during long-term follow up after weight loss. We measured TEE to validate the intake data and showed that underreporting was obvious in

both groups. Only retrospective dietary methods are reported in this study and the underreporting could be due to difficulties with remembering all food eaten or estimating portion sizes. However, these subjects also recorded their diet for several times during the study (Richelsen et al., 2007) and were well aware of the eating habits.

Another consideration of the study was that we did not measure RMR. Ideally it should have been measured, but the recording was done when the subjects were supposed to be in energy balance before weight reduction and during weight maintenance.

5.1.2 The accuracy of reported dietary intake

We found that at the group level energy intake was under reported by 17.6% with the FFQ method and by 29.6% with the DR method (Paper 1). Tables 8 and 9 show that the difference between energy intakes estimated by the dietary methods and measured energy expenditure was 575 kcal (2.4 MJ) by the FFQ and 982 kcal (4.1 MJ) by the DR method. To achieve a weight loss of 0.5-1.0 kg per week, an energy deficit of about 500-1000 kcal (2.1-4.2 MJ) is recommended. Based on these findings it seems like self-reported dietary intake data may be of limited value for the purpose of counselling for energy deficient diets (tables 8 and 9).

Table 8. Energy intakes reported by the food frequency questionnaire and energy calculated from biomarkers[1]

	All subjects	Underreporters[2]	Non-underreporters[2]	
N	50	28	22	
	Mean (SD)	Mean (SD)	Mean (SD)	P[3]
Total energy expenditure, kcal[4]	3332 (590)	3355 (589)	3303 (603)	0.7594
Reported energy intake, kcal	2757 (909)	2296 (616)	3343 (893)	<0.0001
Difference energy intake – total energy expenditure, kcal	-575 (735)	-1059 (385)	40 (602)	<0.0001
Energy from protein biomarker[5], kcal	452 (112)	462 (112)	440 (114)	0.5022
Energy from protein by reported diet, kcal	422 (128)	369 (92)	489 (137)	0.0006
Difference protein by reported diet - protein biomarker, kcal	-31 (127)	-93 (105)	49 (109)	<0.0001

[1]Total energy expenditure measured by the doubly labelled water method was used as biomarker for energy intake and nitrogen excretion was used as a biomarker for protein intake.

[2] The subjects were classified as underreporters and non-underreporters by how much the ratio of energy intake to energy expenditure deviated from 1.00 by calculating the 95% confidence limits (CL) of the ratio of reported energy intake to total energy expenditure according to the equation:

$95\%CL = \pm 2 \times \sqrt{[(CV_{TEE})^2 + (CV^2_{EI}/d)]}$; $CV_{TEE} = 8$, $CV^2_{EI} = 23$ and $d = 90$.

[3] Differences between the underreporters and the non-underreporters were tested with un-paired t-tests.

[4] To convert kcal to kJ, multiply kcal by 4.184.

[5] Urinary excretion of nitrogen was converted to dietary nitrogen by dividing the urinary nitrogen by 0.81. Dietary nitrogen was converted to dietary protein by multiplying dietary nitrogen by 6.25

Table 9. Energy intakes reported by dietary records and energy calculated from biomarkers[1]

	All subjects	Underreporters[2]	Non-underreporters[2]	
N	49	26	23	
	Mean (SD)	Mean (SD)	Mean (SD)	P[3]
Total energy expenditure, kcal[4]	3316 (585)	3372 (667)	3254 (483)	0.4868
Energy intake, kcal	2334 (727)	1935 (479)	2784 (701)	<0.0001
Difference energy intake – total energy expenditure	-982 (693)	-1437 (451)	-470 (547)	<0.0001
Energy from protein estimated by urinary nitrogen[5], kcal	450 (112)	455 (114)	445 (113)	0.7577
Energy from protein by reported diet, kcal	362 (108)	323 (95)	406 (108)	0.0063
Difference protein by reported diet - protein biomarker	-88 (116)	-132 (101)	-39 (115)	0.0041

[1] Total energy expenditure measured by the doubly labelled water method was used as biomarker for energy intake and nitrogen excretion was used as a biomarker for protein intake.

[2] The subjects were classified as underreporters and non-underreporters by how much the EI/TEE ratio deviated from 1.00 by calculating the 95% confidence interval (CI) of the ratio of reported energy intake to total energy expenditure according to the equation:

$95\%CI = \pm 2 \times \sqrt{[(CV_{TEE})^2 + (CV^2_{EI}/d)]}$; $CV_{TEE} = 8$, $CV^2_{EI} = 23$ and $d = 3$.

[3] Differences between the underreporters and the non-underreporters were tested with un-paired t-tests.

[4] To convert kcal to kJ, multiply kcal by 4.184.

[5] Urinary excretion of nitrogen was converted to dietary nitrogen by dividing the urinary nitrogen by 0.81. Dietary nitrogen was converted to dietary protein by multiplying dietary nitrogen by 6.25.

According to the FFQ, underreporters had a ratio of EI/TEE below 0.83 and non-underreporters had a ratio equal or above 0.83. The corresponding numbers for the DR were a ratio of EI/TEE below 0.69 for underreporters and equal or above 0.69 for non-underreporters. About half of the subjects were classified as underreporters by both methods. However compared to the expected EI/TEE = 1.00 in weight stable subjects the FFQ showed superior accuracy compared to the DR (EI/TEE = 0.824 and EI/TEE = 0.704 according to the FFQ and DR, respectively, P=0.0003). Table 8 shows that according to the FFQ, EI was in accordance with TEE among the non-underreporters. Thus, it seems like the FFQ method better capture energy intake compared to three days of DR and a minority of subjects are able to report adequately by the method.

To further validate the dietary intake data we used the excretion of urinary nitrogen as a biomarker for the dietary intake of protein (Svendsen and Tonstad, 2008). Subjects were the 23 men and 27 women participating in the validation study in Paper 1. The subjects collected 24-hour urinary samples for three non-consecutive days. Completeness was ascertained using the para-aminobenzoic-acid method (Bingham and Cummings, 1983; Johansson et al., 1999). As seen in table 8 and 9, underreporters of total energy also underreported energy from protein with both dietary methods. However, the underreporting of energy from protein was smaller than the underreporting of total energy. The less underreporting of energy from protein than total energy may indicate a bias toward more underreporting of carbohydrate, fat, or alcohol. Unfortunately, biomarker-type measurement does not exist for other nutrients than protein. The protein data cannot be generalized to other nutrients. However, data suggest that under-reporters of energy also reports consuming less of all food groups but that the degree of underreporting can vary between foods (Krebs-Smith et al., 2000). This large-scale study found that low energy reporters omitted foods from their reports and substantially underreported portion sizes of many foods. This may have been the case in our study too, as

evidenced by a general underreporting of portion sizes and a specific underreporting of bread, sweets, desserts and snacks.

The results of our study showing the problem underreporting of dietary intake in the obese are in accordance with a number of other studies (Hill et al., 2001; Trabulsi and Schoeller, 2001; Subar et al., 2003). Thus, the results are generalisable to obese, middle-aged subjects that are non-smoking and highly motivated. It has to be mentioned that the subjects knew that the energy expenditure would be measured and they had been recording their dietary intake prior to the dietary interview. They may have been more aware of their dietary intake and eating habits and reported more accurately than subjects usually seen in a clinical setting. These results may be less generalisable to a smoking, obese population that are not as aware of the eating habits.

5.1.3 The validity of energy expenditure estimated by ActiReg®

In Paper 2 we showed that energy expenditure calculated by the ActiReg® method was underestimated by 103 kcal (431 kJ) compared to TEE measured by the DLW method. The study highlights the importance of considering the increased energy expended during weight-bearing activities in obese subjects. As seen in Paper 2, calculation of TEE with normal PAR levels the underestimation of TEE by the ActiReg® method was 507 kcal (2.1 MJ).

The small underestimation of TEE seen by the ActReg® system may be due to the fact that the energy expenditure of activities such as arm movements (i.e. snow shuffling), carrying loads, walking upstairs, muscle strength training, swimming and cycling or spinning is not captured. In light of the small underestimation it seems like activity measured by ActiReg® is closely in accordance with activity levels measured by the DLW in other studies (Tooze et al., 2007; Goris and Westerterp, 2008). In our study, PAL level assessed by the AciReg® method was 1.80 which is slightly higher than the mean PAL of 1.74 in obese

subjects reported by Goris and Westerterp in a recent review (Goris and Westerterp, 2008). In the OPEN study TEE measured with the DLW method was 2728 kcal (11.4 MJ) in obese women and 3576 kcal (15.0 MJ) in obese men (Tooze et al., 2007). According to the ActiReg® method, TEE was 2944 kcal (11.5 MJ) in women and 3541 kcal (14.9 MJ) in men. This may indicate that the ActiReg® method better capture energy expenditure in women than in men.

Energy expenditure at rest accounts for most of the expended energy during the day. RMR can be measured or predicted by various equations. In Paper 2 we used the formula adopted from Schofield in the FAO/WHO/UNU Report (Food and Agricultural Organization/World Health Organization/united Nations University, 1985), Mifflin (Mifflin et al., 1990), Cunningham (Cunningham, 1991) and Müller (Müller et al., 2004). Calculation of RMR using all these formulas was significantly different from the RMR measured by indirect calorimetry. In accordance with other studies performed among obese subjects (Frankenfiled et al., 2005), the Mifflin formula showed best agreement with the measured RMR. At the group level the predicted RMR by the Mifflin formula was 1889 kcal (SD 245 kcal), indicating overestimation by 4.6% (mean difference between predicted RMR and measured RMR was 87 kcal [364 kJ]). At the individual level the predicted RMR (Mifflin) was significantly correlated to measured RMR (r=0.876 [95%CI 0.815, 0.937]). The Cunningham formula underestimated RMR by 266 kcal (1.1 MJ) and it has to be noted that this formula is used in impedance measurements to estimate RMR.

We concluded in Paper 2, that ActiReg® may be used to measure TEE in obese subjects on two premises: RMR should be measured, and the increased energy expenditure during weight-bearing activities in obese subjects should be considered. Ideally a measurement of RMR should be performed in a clinical setting where predictions of RMR are widely used. A concern about predicting RMR is that a possible adoption of energy efficiency

reduces RMR. In a number of studies it has been shown that RMR is reduced during energy deficient states (Rosenbaum et al., 2003; Westerterp, 2003; Stiegler and Cunliff, 2006; Martin et al., 2007). A small but well-designed study by Martin et al. showed a mean reduction in RMR of 91 kcal (382 kJ) per day compared to controls who were not energy restricted after adjustment for fat free mass (Martin et al., 2007). However, recent studies have shown that during weight maintenance the reduction in RMR is explained by altered body composition (Das et al, 2003; Hunter et al., 2008). Hunter et al. reported no decrease in REE adjusted for fat free mass and fat mass during four weeks of weight maintenance after weight reduction (Hunter et al., 2008). In line with this, no change in RMR was seen in subjects after gastric bypass when change in fat mass and fat free mass were accounted for (Das et al., 2003). It seems like RMR is reduced during energy deficient states but when new equilibrium states are achieved no reduction in RMR is seen. This means that RMR may be predicted in weight stable individuals. Using the Mifflin formula an overestimation of about 5% in RMR may be expected.

The study population was the same as in Paper 1 and the results are generalisable to middle-aged, non-smoking, highly motivated, obese subjects that are moderately active (see below). The results may be less generalisable to smoking, younger or older adults or subjects with a weight above 135 kg. It has to be noted that recording of physical activity with the ActiReg® method require motivated participants. Being monitored for several days and having irritating cables attached to the body are some of the barriers to easy use of this method.

5.1.4 The level of physical activity before weight loss was initiated

In Paper 3 we found that obese subjects were moderately physically active for about 26 minutes per day. This amount is close to the recommended 30 minutes of physical activity

given by the American College of Sports Medicine and American Heart Association (American College of Sports Medicine and American Heart Association, 2007). It is also almost in accordance to the activity found in the NHANES 2003-2004 (Troiano et al., 2008). In this study PA was monitored for at least four days in almost 5000 US subjects of whom 30% were obese. The main results of the study were that males were more physically active than females, that physical activity declines with age and that only 5% of adults obtained physical activity at the recommended level (Troiano et al., 2008). In contrast, we found that about one-third of obese subjects seeking treatment for weight loss was physically active for more than 30 minutes per day. This may indicate that the excess weight does not exclude moderate physical activity. The overall PAL during the day was 1.85 that is slightly higher than the PAL of 1.71 reported in 138 Danish subjects (Matthiessen et al., 2008) and the PAL of 1.68 reported in 93 overweight, young Norwegian men (Kurtze et al., 2007).

To initiate weight reduction or maintain weight loss even more physical activity is probably necessary (Ross et al., 2000; Slentz et al., 2004; Stiegler and Cunliff, 2006; Johannsen et al., 2007; Catenacci et al., 2008). Obese men that spent 60 minutes per day on brisk walking achieved about a 7% reduction in body weight (Ross et al., 2000). Likewise, the Studies of Targeted Risk Reduction Interventions through Defined Exercise showed that physical activity corresponding to 4-5 km of daily walking had salutary effects on body weight (Slentz et al., 2004). Assuming a speed of 4.5 km per hour, our subjects would have had to walk for 60 minutes per day to reach this level of activity. Adherence to 60 minutes or more of physical activity may be a challenge. In a recent study only 12 of 202 subjects maintained a daily physical activity level of more than 60 minutes per day for 18 months (Tate et al., 2007).

In addition, individual variations in the effect of an exercise-induced weight loss program may occur (King et al., 2008). This was shown in a study of 35 obese men and

women that participated in a well-controlled exercise program for 12 weeks. The mean weight loss was 3.7 kg (SD 3.6 kg) as was predicted by the increase in energy expenditure. However, when dividing the subjects in two groups according to their actual versus predicted weight loss, it was seen that some individuals (compensators) compensated the energy expended during exercise with increased efficiency of energy expenditure and increased energy intake. The compensators lost about 1.5 kg of weight compared to 6.3 kg lost by non-compensators. In compensators RMR was reduced whereas EI at a test meal and subjective hunger ratings were increased. In non-compensators EI was decreased and change in hunger ratings were not reported (King et al., 2008). Thus, prescriptions of exercise may benefit from considering dietary and behavioural therapy and vice versa.

A recent review concluded that changes in physical activity appear to result in less weight loss than expected (Stiegler and Cunliff, 2006). This may be due to reduced RMR during energy deficient states (Rosenbaum et al., 2003; Stiegler and Cunliff, 2006; Martin et al., 2007) and more energy efficient muscle contractility (Rosenbaum et al., 2003). With regard to weight reduction an increase in TEE and overall PAL is necessary. In a recent study it was shown that overall PAL decreased in the energy restricted group with no such decrease in the energy restricted plus exercise group (Martin et al., 2007). Thus there are good reasons to increase the level of overall physical activity both during energy deficient states and during weight maintenance. Indeed, a high level of physical activity has been shown among subjects that sustained weight loss for long-term (Jakicic et al., 2008; Catenacciet al., 2008). Participation in physical activity for 48 minutes per day was reported among the 25% of subjects that maintained a weight loss of more than 10% during two years of follow-up (Jakicic et al., 2008). Likewise, the National Weight Control Registry found that subjects having successfully maintained a weight loss of at least 13.6 kg for more than one year participated in 60 to 75 minutes of moderate physical activity per day. However, the standard

deviation was high suggesting a wide range in the amount of performed physical activity (Catenacci et al., 2008).

The result of our study showing that the subjects participated in moderately physical activity for 26 minutes per day at least four days of the week may be generalised to middle-aged obese men and women with risk factors for metabolic syndrome. The study was small but the results are is in line with the 27 minutes of physical activity monitored in a subgroup of more than 2000 healthy subjects aged 20 to 69 years in the NHANES 2003-2004 study (Troiano, 2008).

5.1.5 The effect of increasing intake of vegetables and fruit on weight loss and cardiovascular risk factors

In paper 4 targeted dietary counselling to increase the intake of vegetables and fruit contributed to weight reduction and decreased systolic and diastolic blood pressure in subjects with SRBD but had no effect on blood lipids or antioxidant status measured with the ferric reducing antioxidant power assay. The dietary intervention resulted in a doubled intake of vegetables and fruit in the intervention group compared to the control group. Energy density, energy percent of fat and energy percent of saturated fat were decreased and energy percent of carbohydrate, fibre, potassium, magnesium, vitamin C, folate, α-tocopherol and β-carotene were all increased in the intervention group compared to control group. The multiple regression analysis indicates that increased intake of vegetables contributed to the weight loss of about 1 kg per month. A similar monthly weight reduction was seen in the PREMIER clinical trial (Appel et al., 2003). The PREMIER study lasted for six months, included adults with above optimal levels of blood pressure and tested the effect of established recommendations for blood pressure reduction (weight loss, sodium restriction, increased physical activity and limited alcohol intake) compared to the effect of these recommendations

plus the DASH diet rich in fruit, vegetables and low fat diary products. In the PREMIER study, both interventions reduced blood pressure compared to the control group. In the group that included the DASH diet, the reduction in systolic and diastolic blood pressure was 4 and 3 mmHg, respectively (Appel et al., 2003). Like ours, the study was conducted in a clinical setting and no food was provided.

It seems like a dietary and behavioural intervention to increase intake of vegetables and fruit was effective with regard to weight loss, blood pressure reductions and increased concentrations of plasma antioxidants, but had no effect on serum lipids. This may be due to the obesity status of the participants. In a feeding study Lefevre et al. showed that the reduction in LDL cholesterol decreased as the percentage of body fat, BMI and insulin increased (Lefevre et al., 2005). In addition, the intervention could have been more tailored to favourable effects on lipid levels. The subjects in this study were included on the basis of their diagnosis of SRBD but they also experienced a high metabolic risk profile including components of the metabolic syndrome. In dietary treatment of the metabolic syndrome it is recommended to decreased intake of saturated fat to less than 7% of total energy (Grundy et al., 2005). Indeed, the intake of fatty meat product decreased and the intake of lean meat products increased in the intervention group compared to the control group (data not shown). However, given the beneficial effect on apolipoprotein-B and triglyceride containing lipids including more protein in the diet (Furtado et al., 2008), maybe targeted advice to increase the intake of low-fat diary products, legumes and nuts may have been beneficial. To decrease intake of saturated fat also more emphasis should have been given to reduction of the intake of buns, cakes, biscuits, ice cream, chocolate and snacks. Very low fat diets however, are not preferable in individuals with depressed HDL-cholesterol and elevated triacylglycerol concentrations. An eating pattern like the Mediterranean-style diet, which is rich in fibre and

has moderate amounts of unsaturated fat, has been shown to reduce metabolic risk factors (Esposito et al., 2004).

The reported dietary intake of saturated fat decreased, especially in the intervention group. However, the energy intake was underreported. In this study we did not validate the energy intake data by measured TEE, but EI can be validated as suggested by Goldberg et al. (Goldberg et al., 1991). With this technique recorded EI is compared with presumed energy expenditure (i.e. EI is expressed as a multiple of RMR). Using the Mifflin formula to calculate RMR, the EI/RMR showed underreporting in both groups at baseline (EI/RMR= 1.40 in the control group versus EI/RMR=1.38 in the intervention group) and after three months (EI/RMR= 1.38 in the control group versus EI/RMR=1.29 in the intervention group). This may have been expected, given the above discussion. In addition, unpublished data with regard to the reproducibility of the FFQ as was tested in 50 subjects with SRBD participating in two dietary interview separated by one month, showed more underreporting at the second interview compared to the first (EI at the first FFQ was 2551 kcal and EI at the second FFQ was 2329 kcal). Moreover, participation in a weight-loss program may further increase underreporting (Johnson et al., 2005). Taken the underreporting into consideration, the intake of saturated fat may have been higher than reported.

Conducting a dietary intervention in subjects diagnosed with SRBD, gave some unexpected challenges. Despite CPAP treatment, many of the subjects were tired, exhausted and felt asleep during the sessions. Not all subjects managed to do their homework exercises and issues that were important for the subjects were discussed in the group when they came up. As an example the subjects wishes for new recipes and tips for variation of the diet preceded the visit to the vegetables and fruit store, as was a success. The behavioural treatment program was group based, but at every visit the subjects were weighed individually.

This time was used to briefly assess individual barriers to the intervention. If suitable, the barriers were discussed more broadly in the group sessions.

About generalisability, this study included patients diagnosed with SRBD and consecutively referred to the Department of Preventive Medicine. The majority of the subjects used CPAP. The results of this study may be less generalisable to subjects with SRBD not treated with CPAP. The study also included a small number of women due to pervasiveness of SRBD in male gender. However, a number of other trials have investigated the effect of an increased intake of vegetables and fruit in women (Apple et al., 1997; Howard et al., 2006; Ello-Martin et al., 2007). After three years, the Women's Health Initiative Dietary Modification Trial showed that weight loss was achieved with a decrease in intake of fat and increased intake of vegetables and fruit. Furthermore, the DASH trial that included both women and men, showed reduction in diastolic blood pressure (Apple et al., 1997).

5.1.6 The influence of orlistat with regard to eating behaviour and dietary intake

5.1.6.1 Eating behaviour

In Paper 5 we found no substantial differences between the effect of orlistat or placebo on eating behaviour measured with the TFEQ and BES during three years of weight maintenance following a VLED. We observed that the placebo group showed a lower score for hunger after 33 months, but this difference in hunger may be of minor clinical relevance given the overall reduction in hunger in both groups. Golay et al have recently reported this. They followed 55 severely obese subjects for five years after a six-week in-hospital weight loss program and found emotional liability, depression and anxiety rather than hunger as reasons for overeating. Moreover, the subjects that regained weight during follow up reported higher rates of behaviour disorder, psychological difficulties and a poor dietary structure (Golay et al., 2004).

All subjects in our study were instructed to take orlistat or placebo to their main meals. In this regard orlistat may be important to enable subjects to have a regular meal structure. The optimal meal structure has not been established for weight loss and weight maintenance. However, most of the successful weight maintainers in the National Weight Control Registry reported regularly eating breakfast every day of the week (Wyatt et al., 2002). A regular meal plan may be important in preventing impulsive eating and over-eating episodes. Over-eating tendency was a good predictor for energy intake in Type 2 diabetes patients followed for four years (Van Strien and Van der Laar, 2008) and impulsivity has been associated with disinhibition (Yeomans et al., 2008). Moreover, in a retrospective study in 535 women habitual disinhibition was the strongest correlate of weight gain during 20 years (Hays and Roberts, 2008). A reduction in disinhibition together with an increased score for restraint has been taken to be an expression of successful dieting (Yeomans et al., 2008). In our study we found that increased score for restraint and decreased scores for disinhibition and binge eating were associated with changes in weight in the orlistat group. It can be speculated that orlistat have influenced these eating behaviours in a subgroup of individuals prone to overeat in response to different disinhibitors.

In multivariate analyses, increased restraint and decreased disinhibition and binge eating were predictors of sustained weight maintenance in all participants adjusted for BMI, age, gender and treatment. Thus, changes in eating behaviour in the orlistat group are mostly due to quantified eating behaviour similar to the way placebo operates. In fact, dietary restraint increased and disinhibition, hunger and binge eating decreased during the VLED period. It seems like the weight reduction itself contributed to these changes in eating behaviour and that these changes sustained whether or not taken an active anti-obesity drug. This may be due to the dietary and behavioural treatment program as was organized both in group and in individual sessions and included weight monitoring, dietary diaries, skills

training, problem solving issues, relapse prevention and dealing with emotional states. These were the same elements emphasized in the Look AHEAD study an intervention study that is designed to determine the effect of weight reduction on cardiovascular morbidity and mortality in overweight subjects with type 2 diabetes (Wadden et al., 2006).

The results of our study are generalisable to men and women with metabolic risk factors that have achieved an initial weight loss and use orlistat treatment for weight maintenance in a behaviour modification program. The results may be less generalisable to subjects with eating disorders.

5.1.6.2 Dietary intake

In paper 6 we have two findings. First, the use of orlistat did not lead to a lower fat intake compared to placebo in obese subjects consuming a low fat diet and participating in a structured program for weight loss maintenance. Second, subjects that chose to take orlistat when its use was optional did not comply with the dietary fat recommendations.

One concern about the use of an-antiobesity drug is that the use will attenuate the motivation to maintain dietary changes. Like others (Hill et al., 1999; Franson and Rössner, 2000), we did not find differences in dietary intake between the orlistat and placebo groups. The orlistat group reduced their dietary intake of fat and increased their intake of carbohydrates just like the placebo group and both groups reported to consume a low fat diet. In this regard, the use of orlistat seems not to influence the dietary intake when the subjects are participating in a behavioural treatment program and follow dietary recommendations. In these subjects, the pharmacological effect of the drug will improve weight loss and maintenance (Richelsen et al., 2007; Rucker et al., 2007).

In contrast, the subjects that chose to take orlistat when it's use was optional may have been aware that they will not or can not adhere to the dietary recommendations and this may

hamper the effect of the drug (Ullrich et al., 2003; Golay et al., 2005). In our study, these subjects achieved an initial weight loss of more than 5%, they participated in a behavioural treatment program and were scheduled for 30 visits with a dietician during three years of follow up. In addition they were weighed at the visits and monitored their diet on four occasions. Despite of this treatment program the subjects were not able to sustain the dietary recommendations that were given. These subjects may need follow-up by a psychologist or specialist in behavioural therapy to work with other barriers of weight loss. Other dietary composition approaches may also have been more beneficial for these subjects. A recent study showed that weight loss was sustained for two years in three dietary regimens: 3 kg by a low fat diet, 4 kg by a Mediterranean diet and 5 kg by a low carbohydrate diet (Shai et al., 2008). It may be easier for some subjects to reduce carbohydrate than fat. May be better individualized tailoring of different diets based on preferences and experiences in the nutrition counseling could increase compliance with dietary interventions. Enhanced compliance may also be achieved by more focus on self-monitoring of weight. Consistent daily or weekly self-monitoring of weight has been reported to be beneficial for weight maintenance (Wing et al., 2006; Butryn et al., 2007).

The use of the VLED may be questioned. However, the method has been recommended for weight loss purposes, providing the diet is followed by a 1-2 years integrated weight maintenance programme consisting of lifestyle interventions involving dietary change, nutrition education and behavioural therapy (Astrup & Rössner, 2000) as was given in the SMOMS trial (Richelsen et al., 2007). Given the overall result of the SMOMS trial, the method seems safe and effective and improvement in weight maintenance may be achieved with the use of orlistat. It has to be mentioned that the subjects were followed weekly during the VLED period and behavioural treatment were included in the sessions. The subjects were recommended to consume the VLED portion at regular meal times and eat

vegetables in the amounts of 100 kcal per day. After the VLED period, the subjects were very restrictive and interested to make change in their diet to sustain weight reduction. During the long-term follow up, the diet deteriorated slightly. This was observed despite an obvious underreporting of dietary intake. Two months after the end of the trial, the reported intake of saturated fat was above the recommended level of 10% of energy in the orlistat group.

The study population in this study was small and care must be taken with regard to generalisability of the three years data. More research is needed on dietary intake of subjects that chose to take an anti-obesity drug for long-term weight maintenance after weight loss.

5.2 Implications for treatment

A majority of obese subjects with metabolic risk factors underreported dietary intake to such a degree that dietary counselling with the aim of energy restriction was difficult. This may be a general underreporting of portion sizes and a more specific underreporting of sweets, desserts and snacks. Hence, it seems important to ask pointedly about these items in dietary interviews. The dietary interview based on a FFQ provided more accurate data than the DR. This may be due to the fact that FFQ reflected dietary intake during the last three months and included holidays. Moreover, restrained eating behaviour did not influence reporting accuracy according to the FFQ. Thus is seems like long-term assessments of dietary intake better capture irregular eating occasions that increase energy intake in the obese.

A measurement of TEE with the ActiReg® method gives a valid estimate of TEE despite a small underestimation. Improvements of the ActiReg® system with regard to actually calculating energy used during vigorous exercise, carrying loads and resistance training activities are needed. In obesity research a measurement of RMR is preferable in assessments of TEE especially in subjects that are not weight stable. Given the poor reporting accuracy of dietary intake among the obese, prescribing energy restriction 500 kcal to1000

kcal below TEE is another approach that can be used. In this approach individually meal plans based on the subjects' food preferences may be prescribed at the correct energy intake.

Obese subjects performed moderate physical activity for 26 minutes at least four days of the week, before advice for weight loss was given. This level of physical activity is almost in accordance with recommended levels for health benefits. For weight loss and maintenance, the amount of exercise probably has to be more than doubled. However, the individual variation in level of physical activity is high and an objective assessment of physical activity may be a tool for tailoring exercise prescriptions.

A doubling of the intake of vegetables and fruit induced weight loss, decreased blood pressure and increased carotenoid concentrations in obese subjects with SRBD. The subjects participated in a behavioural treatment program conducted of a registered dietician. Minor weight loss was seen in the control group that only were seen at a usual care practice by the same dietician. This implies that dietary interventions to increase intake of vegetables and fruit in free-living obese subjects are achievable when the subjects are offered a behavioural treatment program of eight sessions during three months.

In motivated subjects a substantial, initial weight loss was achieved with a VLED in a behavioural treatment program. Orlistat did not influence eating behaviour or dietary intake other ways than the placebo did during long-term weight maintenance after a VLED for eight weeks. Some subjects that chose to take orlistat may be aware that they do not or cannot follow dietary recommendations and this may hamper the effect of the drug. In these subjects various dietary interventions, more intensive programs including consistent self-monitoring, additional psychological therapies, other anti-obesity drugs or surgery need to be considered.

6 CONCLUSIONS

1. A majority of obese subjects with metabolic risk factors underreported dietary intake to such a degree that dietary advice to achieve moderate energy restriction cannot be based on dietary assessments. The mean difference between reported energy intake and energy expenditure was about 500 kcal and 1000 kcal as measured by the FFQ and the DR, respectively.
2. Despite a small underestimation of 100 kcal, measurement of TEE with the ActiReg® method gives a valid estimate of TEE in obese subjects with metabolic risk factors when RMR is measured and the increased energy during weight bearing activities is considered**.** This is a non-invasive way of measuring TEE that may deserve further clinical study and application.
3. Obese subjects seeking treatment spent a mean of 26 min per day in moderate physical activity measured with ActiReg® before advice for weight loss was given. This suggests that for weight loss and maintenance purposes the level of physical activity has to be increased.
4. Targeted dietary advice to increase the daily intake of vegetables and fruit given in a behavioural group program for three months resulted in better weight loss, decreased systolic and diastolic blood pressure and increased plasma levels of lipid soluble antioxidants (α-carotene, β-carotene) in the intervention group compared to controls.
5. When included as part of a behavioural program for weight loss maintenance orlistat did not influence dietary restraint, disinhibition or binge eating in other ways than placebo. Hunger was reduced more in the placebo group than in the orlistat group, but this seems not to be of clinical importance giving the overall reduction in hunger in the orlistat group.

6. When included in a dietary treatment program orlistat did not influence dietary intake of fat differently than placebo. Subjects that chose to take orlistat after the end of the program had higher than recommended intake of fat and this may hamper the effectiveness of the drug.

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Erratata list

Page 5 line 9: Journal of Physical activity & Health has been changed to J Phys Act Health.
Page 10 line 20: maintained has been changed to maintain.
Page 30 line 11: between groups have been included.
Page 30 line 12: signed have been deleted from the description of the Mann-Whitney rank test.
Page 30 line 14: the words matched pairs and sum has been deleted in the description of the test.
Page 34 line 5 and 9: defense has been changed to defence.
Page 37 line 8: However, has been deleted.
Page 47 line 10: per hour has been deleted.
Page 58 line 10: β-chryptoxanthin has been deleted.
Page 61 line 7: the reference Cooper Z et al., 2003 has been moved from line 20.
Page 61 line 13: the reference Couillard C et al., 2001 has been moved from line 22.
Page 77: Appendices has been changed to Appendixes.

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British Journal of Nutrition (2006), **95**, 640–649 DOI: 10.1079/BJN20051662

Accuracy of food intake reporting in obese subjects with metabolic risk factors

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(Received 4 May 2005 – Revised 13 October 2005 – Accepted 7 November 2005)

The aim of the present study was to determine the accuracy of reported energy intake according to a food-frequency questionnaire (FFQ) and dietary records (DR) in obese subjects with metabolic syndrome risk factors. Subjects were twenty-three men and twenty-seven women with mean BMI of 35·7 (range 30·5–43·8) kg/m^2 who participated in a dietary interview based on a FFQ and completed weighed DR. Total energy expenditure was measured with the doubly labelled water method. Total energy expenditure, measured RMR and physical activity level did not differ between under-reporters (50 % of the sample) and non-under-reporters. Under-reporters had lower median intake of sweets, desserts and snacks than non-under-reporters (100 *v.* 161 g/d (P=0·0008) and 61 *v.* 128 g/d (P=0·0002) according to the FFQ and DR, respectively). The DR also showed lower energy density (6·7 (SD 1·3) *v.* 7·9 (SD 1·6) kJ/g; P=0·0064), lower intake of sugary drinks (0 *v.* 167 g/d; P=0·0063) and higher scores for dietary restraint (9·0 (SD 5·0) *v.* 6·1 (SD 3·5); P=0·0285) in under-reporters. Energy density was associated with accuracy according to the FFQ (Spearman's rank correlation coefficient (R_S) 0·406; P=0·0034) and the DR (R_S 0·537; $P<0·0001$). In multivariate analysis, consumption of bread and sweets, desserts and snacks measured by the FFQ was positively associated with accuracy (R^2_{adjusted} 0·46 (95 % CI 0·32, 0·70)). According to the DR, consumption of sweets, desserts and snacks was also associated with accuracy, as was dietary restraint (inversely) (R^2_{adjusted} 0·67 (95 % CI 0·54, 0·83)). In obese subjects with metabolic risk factors, intake of sweets, desserts and snacks, bread and dietary restraint were determinants of reporting accuracy.

Energy intake: Under-reporting: Eating behaviour: Doubly labelled water

In 2001, the National Cholesterol Education Program defined the metabolic syndrome as a constellation of at least three of five risk factors (increased waist circumference, hypertriacylglycerolaemia, low HDL-cholesterol, high blood pressure, and high fasting glucose) that increase the risk of CHD and recommended that the metabolic syndrome should be a target of risk-reduction therapy after the primary target, level of LDL-cholesterol, is met (Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults, 2001). One of the primary methods of risk reduction is weight loss. Studies have shown that even a modest degree of weight loss induced by dietary and lifestyle change in individuals with characteristics of the metabolic syndrome is associated with clinical benefits (Tuomilehto *et al.* 2001). However, counselling for weight loss is hampered by the difficulty in obtaining accurate dietary data (Lissner, 2002). The development of the doubly labelled water (DLW) method to estimate total energy expenditure (TEE) has led to general acceptance of the notion that dietary self-reporting substantially underestimates energy intake (EI), and data have accumulated for nearly two decades showing that obese individuals are more likely to under-report than others (Prentice *et al.* 1986, 1996; Braam *et al.* 1998; Hill & Davies, 2001).

Studies on the accuracy of dietary self-reporting have considered a number of factors in addition to a high BMI that may influence the likelihood of under-reporting. These include sex, age, socio-economic status, eating behaviour and dieting, physical activity, non-smoking, psychological factors and the cultural context as reviewed recently (Livingstone & Black, 2003). Under-reporting is accentuated when repeated assessment of dietary intake is conducted (Caan *et al.* 2004). Furthermore, certain foods and nutrients are more likely to be under-reported than others, primarily carbohydrates, fats, snacks, and foods or drinks that are considered to be unhealthy (Bingham *et al.* 1995; Heitmann & Lissner, 1995; Poppitt *et al.* 1998; Tonstad *et al.* 1999; Goris *et al.* 2000; Hill & Davies, 2001). Such selective under-reporting may lead to spurious associations between dietary components and biological markers or risk factors (Lissner *et al.* 1998). Poor accuracy of dietary data may be more pronounced and may mislead public policy, in particular when subjects at high risk for disease are considered, for example, individuals with the metabolic syndrome, as indeed has been demonstrated recently (Rosell *et al.* 2003). In this study, under-reporters had a higher prevalence of the metabolic syndrome than other subjects and associations between the diet and the components of the metabolic syndrome differed between under-reporters and the remaining subjects (Rosell *et al.* 2003).

The aim of the present investigation was to examine the determinants of self-reporting of energy in a sample of obese men and women with the metabolic syndrome or at least two of its risk factors and to explore the specific foods, eating patterns and behaviour that are associated with under-reporting using the DLW method to estimate TEE. The overall goal was to inform

Abbreviations: CL, confidence limit; DLW, doubly labelled water; DR, dietary record; EI, energy intake; FQ, food quotient; FFQ, food-frequency questionnaire; IQR, interquartile range; OPEN, Observing Protein and Energy Nutrition; TEE, total energy expenditure.

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the dietary advice that is appropriate for this group of individuals. Because cigarette smoking may confound the relationship between nutrient intakes and under-reporting (Dallongeville *et al.* 1998), we chose to include only non-smokers in the study.

Subjects and methods

Subjects

Fifty non-smoking, obese men and women with a mean age of 43·2 (SD 10·3; range 24–64) years and a mean BMI of 35·7 (SD 3·3; range 30·5–43·8) kg/m^2 and two or more risk factors for the metabolic syndrome according to the National Cholesterol Education Program (glucose $\geq$6·1 mmol/l or HDL-cholesterol $\leq$1·03 mmol/l for males or $\leq$1·29 mmol/l for females or serum triacylglycerols $\geq$1·69 mmol/l or waist circumference $>$102 cm for males or $>$88 cm for females or systolic blood pressure $\geq$130 mmHg and diastolic blood pressure $\geq$85 mmHg) were recruited by newspaper advertisement and referral to the Department of Preventive Cardiology at Ullevål University Hospital (Oslo, Norway). Subjects were screened via blood chemistry and a medical examination done by a physician to assess risk factors and eligibility to the study. Fifteen subjects had two risk factors, twenty-seven subjects had three risk factors and eight subjects had four risk factors; thus 60 % had the full metabolic syndrome. Exclusion criteria were body weight $>$135 kg, current dieting, cigarette smoking, history of eating disorder or chronic disease, suspected non-compliance due to abuse of drugs or alcohol, drug- or insulin-treated diabetes mellitus, migraine requiring intermittent medication, use of thyroxin, diuretics or weight-reducing agents and use of inhaled or oral β-agonists or corticosteroids. The educational level of each subject was determined according to the number of years of education and categorised as completed primary school, high school or a university degree. The Ethical Committee (region 1 in Norway) approved the protocol and all participants gave their written informed consent. The study was conducted between October 2001 and October 2003.

Measurements

Height was measured with a standardised wall measuring stick scale to the nearest 0·5 cm. Subjects were weighed (in underwear) with a digital weight (SECA, Hamburg, Germany) to the nearest 0·1 kg. Weight was measured at the screening and baseline visits and on day 1 and day 15 of the DLW measurement period. Weight changes during the DLW period were calculated as the difference between day 15 and day 1 and weight changes during the 3-month study as the difference between day 15 of the DLW period and baseline. Waist was measured in the standing position at the level of umbilicus (unclothed) and hip circumference was measured at the level of the greater trochanter. Body composition was determined by dual-energy X-ray absorptiometry (Lunar Expert 1116; Lunar Corp., Madison, WI, USA). The measurement was done in 15 min. The CV for the dual-energy X-ray absorptiometry measurements was 3–4 %. RMR was measured with a standard portable ventilated hood system (Deltatrac® Metabolic Monitor; Datex Instrumentarium Corp., Helsinki, Finland). The Deltatrac® was calibrated by automatic standard gas calibration at the start of each measurement. The subjects slept at home the night before the measurement. On the day of the measurement the subjects took a taxi to the site. The subjects fasted during the last 12 h before the measurement and were instructed not to eat or drink anything but water on the day of the measurement. After changing clothes and mounting the equipment, the subjects relaxed for 30 min in the recumbent position before the head was covered with the canopy. Measurements were done at 1 min intervals for 20–25 min. A mean value of at least a 10 min period at a stable level of energy expenditure was defined to be the RMR.

Doubly labelled water method

Energy expenditure by the DLW method was measured over a period of 14 d and used as a measure of habitual energy expenditure. Sample analyses and calculation procedures have been described in detail elsewhere (Slinde *et al.* 2003). First a baseline urine sample was collected for the determination of the background isotope enrichment (day 1). Then a weighed mixture of ^{2}H-labelled and oxygenated water, corresponding to 0·05 g ^{2}H-labelled water and 0·10 g ^{18}O-labelled water/kg body weight, was ingested. The percentage enrichment of the waters was 99·9 % for ^{2}H and 10·0 % for ^{18}O. The dose was planned to enrich body water with approximately 350 parts per thousand (δ per mil) for ^{2}H and 60 parts per thousand (δ per mil) for ^{18}O. Urine samples were collected from the second voiding during day 2, day 3, day 4, day 8, day 13, day 14 and day 15. The mean time interval between drinking dose and the first post-dose urine sample was 22 (SD 3; range 12–30) h. The participants were instructed to collect the urine spot, register exact voiding time and freeze the samples at home. Participants were called every voiding day to ensure compliance with the procedure. When the samplings were completed, the urine samples were stored at $-$75°C until transportation to the laboratory on dry ice.

Analysis of the isotopic enrichment was determined in triplicates with a Thermoquest Finnigan MAT Delta plus isotope-ratio mass spectrometer with a water/H_2-CO_2 equilibrating device (Thermoquest Finnigan MAT, Bremen, Germany). The precision defined as standard error in triplicate samples is 0·26 for ^{3}H and 0·10 for ^{18}O. Tap water was collected and analysed for background measurements and all TEE calculations were corrected for the content of isotopes in the drinking water. TEE was calculated by the multi-point method using linear regression as suggested by the International Dietary Energy Consultancy Group (1990). All elimination curves were checked for major or diverging residuals. The CV for the elimination constants was on average 3·2 % for hydrogen and 2·7 % for oxygen. The mean N_o:N_d ratio was 1·033 (SD 0·008; range 1·007–1·049). We used the relationship between pool size of ^{2}H (N_d) and pool size of ^{18}O (N_o) as a quality measurement for the DLW. The mean food quotient (FQ) determined from the food-frequency questionnaire (FFQ) was 0·85 (SD 0·016; range 0·81–0·89). The individual N_o:N_d ratio and FQ of the participants were used in the calculation of the energy equivalence of the produced CO_2 as suggested by the International Dietary Energy Consultancy Group (1990).

Food-frequency questionnaire

A FFQ was used as the basis of an interview with a registered dietitian (M. S.), lasting between 1 and 2 h. The FFQ was designed to assess the food intake during the last 3 months and based on two

previously developed FFQ (Lindroos *et al.* 1993; Andersen *et al.* 1999). The questionnaire elicited frequencies and consumption of 174 individual food items or constellations of items grouped together according to the typical Norwegian meal pattern. Specifically the consumption of soft drinks and alcoholic beverages, sweet baked goods, cookies, cakes, ice cream, desserts, sweets, chocolate and snacks as nuts, potato crisp and popcorn were asked for. The FFQ also included twenty-one summary questions and seven dietary supplement questions. An atlas of food portions as well as photographs, household measurements and ordinary models of sweets and snacks was used to estimate portion sizes. Particular attention was given to extra layers of bread spread, food eaten during cooking, fat used in frying and extra portions of dinner and dinner leftovers. The FFQ interview was done immediately following the DLW measurement period.

Weighed dietary records

Participants were provided with food scales and instructed to weigh each individual food item using a digital scaled weight and provide notes on ingredients of composite dishes with approximate quantities. When weighing was not appropriate, the subject used household measurements and pictures to record portion sizes. Forty-nine subjects completed dietary records (DR) for three non-consecutive days. One male subject was not able to complete the DR due to personal problems. The records were done with 3–4 d between each recording and all days of the week were about equally represented. On average the DR were completed a mean of 34 (SD 25) d before the DLW measurements.

We assessed eating frequency by counting the number of eating occasions recorded in the DR. An eating occasion was defined as a food or snack (solid or liquid) containing energy with an interval of >1 h separating the occasions (Farshchi *et al.* 2005).

Assessment of eating behaviour

Forty-nine subjects completed the Norwegian version of the Three Factor Eating Questionnaire. One male refused to fill in the questionnaire. The Three Factor Eating Questionnaire was developed to measure cognitive and behavioural components of eating (Stunkard & Messick, 1985). The scale contains subscales for restraint (possible scores 0–21), disinhibition (0–16) and hunger (0–14). The restraint subscale assesses the intent to control food intake to achieve and maintain a desired body weight. The disinhibition scale assesses overeating in response to a variety of situations associated with loss of control of food intake, while the hunger subscale assesses subjective feeling of hunger and food cravings.

Definition of under-reporters and non-under-reporters of energy

Subjects were identified as under-reporters, accurate reporters and over-reporters of energy based on the 95 % confidence limits (CL) of the expected EI:TEE ratio of 1·00. The 95 % CL between the two measurements were calculated from the published equation (Black & Cole, 2000):

$$95\,\%\mathrm{CL} = \pm 2 \times \sqrt{((\mathrm{CV_{TEE}})^2 + (\mathrm{CV_{EI}^2}/\mathrm{d}))}.$$

$\mathrm{CV_{TEE}}$ for repeated measurements for energy measurements by the DLW method was 8 % (Black & Cole, 2000). $\mathrm{CV_{EI}}$ is the within-subject CV for daily intake of energy and 23 % was used (Bingham, 1987). The number of days was 90 for the FFQ and 3 for the DR.

According to this calculation, subjects were classified as under-reporters, accurate reporters and over-reporters, depending on how much EI:TEE deviated from the expected ratio of 1·00. Subjects defined as accurate-reporters$_{\mathrm{FFQ}}$ had an $\mathrm{EI_{FFQ}}$:TEE ratio within the 95 % CL (0·83, 1·17), under-reporters$_{\mathrm{FFQ}}$ had an $\mathrm{EI_{FFQ}}$:TEE ratio below the lower CL ($<0{\cdot}83$) and over-reporters$_{\mathrm{FFQ}}$ had an $\mathrm{EI_{FFQ}}$:TEE ratio above the upper CL ($>1{\cdot}17$). Accurate-reporters$_{\mathrm{DR}}$ had an $\mathrm{EI_{DR}}$:TEE ratio within the 95 % CL (0·69, 1·31), under-reporters$_{\mathrm{DR}}$ had an $\mathrm{EI_{DR}}$:TEE ratio below the lower CL ($<0{\cdot}69$) and over-reporters$_{\mathrm{DR}}$ had an $\mathrm{EI_{DR}}$:TEE ratio above the upper CL ($>1{\cdot}31$). According to the FFQ, two males and two females were classified as over-reporters; while, according to the DR, one female was an over-reporter. Because the number of over-reporters was minor, accurate reporters and over-reporters were grouped as non-under-reporters.

Calculations and statistics

The FFQ and the DR were coded manually for calculations of total energy, energy density, energy-yielding nutrients and food items with a computer program based on the Norwegian food composition table (Rimestad *et al.* 1995; National Association for Nutrition and Health, 1996). Because of the low median intake of specific foods, we grouped food items into the following categories: fruit, berries, juice and jam; milk; bread; vegetables; boiled potatoes, rice and pasta; fatty meats, minced meat and sausages; chicken and meat with less fat; fish and fish products; oil, butter and margarine; fatty cheese; sweets, desserts and snacks; sugary drinks. The calculated dietary intake of energy assessed by FFQ and DR was compared directly to the estimated TEE from the DLW measurement. Energy density was calculated for the whole diet minus all drinks (coffee, tea, milk, juice, soft drinks and alcoholic beverages).

The results are presented as means and standard deviations, or medians with interquartile ranges (IQR) for non-normally distributed data. Mean and median differences were tested with the unpaired Student's t test and the Mann–Whitney signed rank test, respectively.

Anthropometrics, daily intake of nutrients and food and eating scores for restraint, disinhibition and hunger were correlated with the EI:TEE ratio calculated by the FFQ and the DR. Pearson's correlation coefficients were calculated for normally distributed variables and Spearman's rank correlation coefficients (R_{S}) were calculated for skewed variables. Simple regression analyses were conducted to assess the relation of reporting accuracy to percentages of energy from protein, fat, and carbohydrate and food intakes to determine whether there was selective under-reporting. Factors that were statistically significant in the univariate analysis were entered into a multiple regression analysis to identify the most important factors correlated to reported accuracy of EI. Because the intake of food is reflected in the calculated diet composition, we chose to enter the reported intake of food instead of diet composition in the multivariate regression analyses.

The tests were considered significant at $P<0{\cdot}05$. Statistical analyses were performed using the StatView 5.0.1 software (SAS Institute Inc., Cary, NC, USA).

Results

Characteristics of participants according to sex are shown in Table 1. Subjects had high concentrations of triacylglycerols, diastolic blood pressure and waist circumference in accordance with the inclusion criteria. Restraint and hunger scores were similar for men and women, but women scored higher than men on the disinhibition factor.

Eating characteristics of under-reporters and non-under-reporters

Relative to TEE, both men and women under-reported EI in both dietary assessment methods. EI among male subjects was under-reported by 14·1 (SD 18·9; range −36·0 to 41·6) % according to the FFQ and by 27·9 (SD 14·4; range −3·1 to 51·7) % according to the DR compared with the measured TEE. Female subjects under-reported EI by 20·6 (SD 24·1; range −35·2 to 62·9) and 31·0 (SD 22·0; range −44·6 to 69·0) % according to the FFQ and the DR, respectively. No significant difference by sex was seen in the reported EI relative to TEE. More than 50 % of the subjects were classified as under-reporters of EI according to both methods (Table 2).

Anthropometrics, energy expenditure, energy density, daily intakes of energy-yielding nutrients and foods and scores for eating behaviour in under-reporters and non-under-reporters according to the FFQ and DR are shown in Tables 3 and 4, and Tables 5 and 6, respectively. Eating frequency assessed by the DR is also shown in Table 6. According to the DR, under-reporters had a lower energy density of the diet and reported a lower intake of 'sweets, desserts and snacks' and sugary drinks, as was reflected in a higher percentage of energy from protein and a lower percentage of energy from sugar among under-reporters compared with non-under-reporters. However, the reported absolute amount of protein was lower in under-reporters compared with non-under-reporters according to both dietary assessment methods (92·2 (SD 23·0) *v.* 117·2 (SD 30·9) g; $P=0·0018$ according to the FFQ and 80·7 (SD 23·7) *v.* 97·5 (SD 20·0) g; $P=0·0116$ according to the DR). Energy-adjusted intake of sweets, desserts and snacks (median) was lower among under-reporters than non-under-reporters, respectively (7·7 (interquartile range (IQR) 4·9–10·9) *v.* 11·0 (IQR 7·6–14·7) g/MJ; $P=0·0275$) according to the DR. According to the FFQ the same trend was seen, although not statistically significant (11·1 (IQR 7·0–13·7) *v.* 12·2 (IQR 9·9–14·8) g/MJ; $P=0·2256$). Sweets, desserts and snacks intake was inversely related to percentage energy from protein according to the FFQ (R_S −0·51; $P=0·0004$) and the DR (R_S −0·48; $P=0·0008$). According to the DR, the energy density of the diet was associated with sweets, desserts and snacks (R_S 0·553; $P=0·0001$) and according to the FFQ the association was not significant (R_S 0·272; $P=0·573$).

No significant differences were seen in eating behaviour scores in under-reporters compared with non-under-reporters, with the exception of restraint scores, which were higher in under-reporters than non-under-reporters according to the DR (Table 5).

During the DLW period, the mean weight change in all participants was 0·1 (SD 1·0; range −3·6 to 1·8) kg. No significant weight difference was seen between under-reporters and non-under-reporters (0·01 (SD 0·97) *v.* 0·22 (SD 1·11) kg; $P=0·4768$ according to the FFQ and 0·01 (SD 1·12) *v.* 0·29 (SD 0·82) kg; $P=0·3299$ according to the DR). There was no significant difference in the weight change between under-reporters and non-under-reporters in the entire 3-month period of the study (1·24 (SD 1·56) *v.* 1·49 (SD 1·57) kg; $P=0·5769$ according to the FFQ and 1·50 (SD 1·50) *v.* 1·21 (SD 1·65) kg according to the DR).

Reporting accuracy

The relationship of reporting accuracy to anthropometrics, energy density, diet composition, intakes of food and eating behaviour scores is shown in Table 7. According to the FFQ, reporting accuracy was inversely correlated to the percentage energy from protein and positively to the percentage energy from sugar and the energy density of the diet. Accuracy was positively associated with the intake of the following: sweets, desserts and snacks; fatty cheese; bread; milk; oil, butter and margarine. Scores for restraint were inversely and scores for hunger were positively

Table 1. Characteristics of participants*
(Mean values and standard deviations)

	Men (*n* 23)		Women (*n* 27)		
	Mean	SD	Mean	SD	*P*
Age (years)	44	10	42	11	0·5543
Height (m)	1·82	0·07	1·69	0·06	<0·0001
Weight (kg)	115·1	13·8	104·5	12·1	0·0058
BMI (kg/m^2)	34·6	2·9	36·6	3·4	0·0290
Tissue fat (%)	41·5	7·5	53·3	5·6	<0·0001
Waist (cm)	118·4	10·0	106·4	9·4	<0·0001
Hips (cm)	110·0	7·6	117·6	9·6	0·0036
Waist:hip ratio	1·1	0·1	0·9	0·1	<0·0001
Systolic blood pressure (mmHg)	128	11	123	15	0·1824
Diastolic blood pressure (mmHg)	85	8	85	10	0·9143
Total cholesterol (mmol/l)	6·2	1·1	6·2	1·3	0·9217
LDL-cholesterol (mmol/l)	3·8	0·7	4·1	1·1	0·4256
HDL-cholesterol (mmol/l)	1·1	0·2	1·3	0·3	0·0044
Triacylglycerols (mmol/l)	2·7	1·6	1·9	0·8	0·0184
Glucose (mmol/l)	5·4	0·6	5·3	0·5	0·8167

* Differences between sexes were tested with unpaired *t* tests.

Table 2. Percentage of participants classified as under-reporters, accurate reporters and over reporters of energy intake by the food-frequency questionnaire and the dietary records

	Food-frequency questionnaire (*n* 50)			Dietary records (*n* 49)		
	Percentage	Male (*n*)	Female (*n*)	Percentage	Male (*n*)	Female (*n*)
Under-reporters of energy	56	12	16	53	11	15
Accurate reporters of energy	36	9	9	45	11	11
Over-reporters of energy	8	2	2	2	0	1

associated with accuracy. According to the DR, the percentage energy from protein and scores for restraint were inversely correlated to accuracy, while the percentage energy from fat, energy density, eating frequencies, intakes of 'sweets, desserts and snacks', sugary drinks, 'fatty meat, minced meat and sausages', and fatty cheese and hunger were positively associated with accuracy. Reporting accuracy was not significantly correlated to educational level (R 0·156; P=0·2781).

The multiple regression analysis of the reporting accuracy of energy according to the FFQ is shown in Table 8. 'Sweets, desserts and snacks', energy density, fatty cheese, bread, 'oil, butter and margarine', and restraint were entered into the model. 'Sweets, desserts and snacks' and bread (R^2_{adj} 0·46 (95 % CI 0·32, 0·70)) were significant predictors. The multiple regression analysis of the reporting accuracy of energy according to the DR is shown in Table 9. 'Sweets, desserts and snacks', energy density, sugary drinks, restraint, eating frequency and hunger were entered into the multivariate model and 'sweets, desserts and snacks' and restraint were significant predictors of the variation in the reporting accuracy of energy (R^2_{adj} 0·67 (95 % CI 0·54, 0·83)). Eating frequency was positively correlated to sweets, desserts and snacks (R_S 0·54; P=0·0002) and sugary drinks (R_S 0·30; P=0·0360). Individual items in the category of sweets, desserts and snacks showed the same trends as the entire category (data not shown).

Discussion

The present study focused on subjects with the metabolic syndrome or two risk factors for the metabolic syndrome. The reported consumption of sweets, desserts and snacks was lower among under-reporters than non-under-reporters and showed a significant association with reporting accuracy according to both methods of dietary assessment (FFQ and DR) in the multiple regression analyses. In addition, bread consumption assessed by the FFQ was a significant contributor to reporting accuracy. Dietary restraint was an additional significant contributor to accuracy assessed by DR.

About half of this obese population with metabolic risk factors under-reported EI. This finding is in accordance with the Observing Protein and Energy Nutrition (OPEN) study that included 484 subjects of whom 142 had BMI $\geq 30\,kg/m^2$, and 57 % of the

Table 3. Anthropometry, energy expenditure, energy intake, energy density, macronutrient composition and eating scores in under-reporters (UR) and non-under-reporters (non-UR) according to the food-frequency questionnaire*

(Mean values and standard deviations)

	UR (*n* 28)		Non-UR (*n* 22)		
	Mean	SD	Mean	SD	*P*
Tissue fat (%)	49·1	8·7	46·2	9·0	0·2489
Waist:hip ratio	1·0	0·1	1·0	0·1	0·8777
BMI (kg/m^2)	36·3	3·6	35·0	2·7	0·1522
Total energy expenditure (kJ/d)	14 038	2464	13 819	2524	0·7594
RMR (kJ/d)	7606	898	7452	1178	0·6016
Physical activity level†	1·84	0·17	1·86	0·21	0·7401
Energy intake (kJ/d)	9573	2571	13 938	3723	<0·0001
Energy intake:total energy expenditure	0·68	0·12	1·01	0·18	<0·0001
Energy density (kJ/g)	6·49	1·06	7·35	2·13	0·0699
Diet composition (% energy)					
Fat	34·5	3·9	35·5	5·9	0·4793
Protein	16·3	2·4	14·8	2·4	0·0287
Carbohydrate	45·2	5·5	46·6	7·0	0·4349
Sucrose	5·4	2·9	8·6	8·3	0·0698
Alcohol	2·7	2·9	2·3	4·5	0·7038
Eating scores‡					
Restraint	8·5	4·8	6·3	3·9	0·0929
Disinhibition	8·1	3·2	8·2	3·2	0·9180
Hunger	5·3	2·9	6·0	3·4	0·4625

* Differences between UR and non-UR were tested with unpaired *t* tests.
† Physical activity level is calculated by dividing total energy expenditure by RMR.
‡ The results of one male were missing.

Table 4. Intake of food in under-reporters (UR) and non-under-reporters (non-UR) according to the food-frequency questionnaire*

(Medians and interquartile ranges)

	UR (*n* 28)		Non-UR (*n* 22)		
Food (g/d)	Median	IQR	Median	IQR	*P*
Fruit, berries, juice and jam	246	140–387	305	78–614	0·6182
Milk	202	55–338	302	154–604	0·0451
Bread	152	114–180	213	125–286	0·0837
Vegetables	190	116–269	197	123–332	0·8298
Boiled potatoes, rice and pasta	153	119–210	190	119–208	0·4117
Fatty meat, minced meat and sausages	71	47–82	66	32–161	0·9820
Chicken and meat with less fat	69	43–103	68	28–93	0·5909
Fish and fish products	58	36–92	82	28–105	0·8298
Oil, butter and margarine	50	28–67	67	29–91	0·1112
Fatty cheese	24	9–48	35	18–72	0·1594
Sweets, desserts and snacks	100	64–135	161	121–196	0·0008
Sugary drinks	63	0–211	150	18–378	0·2146

IQR, interquartile range.

* Differences in reported intake of food in UR and non-UR were tested with the Mann–Whitney signed rank test.

Table 5. Anthropometry, energy expenditure, energy intake, energy density, macronutrient composition and eating scores in under-reporters (UR) and non-under-reporters (non-UR) according to the dietary records*

(Mean values and standard deviations)

	UR (*n* 28)		Non-UR (*n* 22)		
	Mean	SD	Mean	SD	*P*
Tissue fat (%)	49·2	8·2	45·9	8·7	0·2613
Waist:hip ratio	1·0	0·1	1·0	0·1	0·7869
BMI (kg/m^2)	36·4	3·2	35·0	3·4	0·1531
Total energy expenditure (kJ/d)	14 107	2791	13 613	2021	0·4866
RMR (kJ/d)	7598	990	7394	1022	0·4836
Physical activity level†	1·85	0·22	1·84	0·15	0·9143
Energy intake (kJ/d)	8071	1999	12 756	4112	<0·0001
Energy intake: total energy expenditure	0·57	0·09	0·85	0·16	<0·0001
Energy density (kJ/g)	6·70	1·34	7·90	1·59	0·0064
Diet composition (% energy)					
Fat	34·8	6·5	36·2	5·5	0·4396
Protein	16·8	3·3	14·7	2·3	0·0151
Carbohydrate	46·3	7·5	46·6	6·2	0·9789
Sucrose	4·9	3·3	9·3	7·0	0·0063
Alcohol	1·3	2·8	2·1	4·5	0·4819
Eating scores‡					
Restraint	9·0	5·0	6·1	3·5	0·0285
Disinhibition	7·7	3·2	8·9	3·0	0·2184
Hunger	4·8	2·9	6·4	3·2	0·0769

* Differences between UR and non-UR were tested with unpaired *t* tests.

† Physical activity level is calculated by dividing total energy expenditure by RMR.

‡ The results of one male were missing.

obese participants were classified as under-reporters of energy according to the FFQ (Subar *et al.* 2003). In the present study, men under-reported EI by 14 % and women by 21 % according to the FFQ. According to the DR, men and women under-reported EI by 28 and 31 %, respectively. In comparison, Goris *et al.* (2000) found a 37 % under-reporting of energy by male subjects with a BMI similar to that of subjects in the present study.

A novel finding in the present study was that consumption of bread according to the FFQ was a significant contributor to accuracy. This finding may be partly explained by the association between bread consumption and an irregular or frequent meal pattern. In Norway bread-based meals are typically consumed two to three times per d. In addition, bread is often consumed as a between-meal snack. The extra bread eaten at irregular meals and snacks may be forgotten or not reported. In contrast, consumption of bread was not associated with accuracy measured by DR. However, under eating may be a major problem with DR as discussed below.

Consumption of sweets, desserts and snacks was robustly associated with reporting accuracy, and was the only multivariate determinant of accuracy in addition to bread consumption according to the FFQ. These two variables explained almost half of the multivariate variance in accuracy. Likewise, reported intake of sweets, desserts and snacks was associated with accuracy

Table 6. Daily intake of food and eating frequency in under-reporters (UR) and non-under-reporters (non-UR) according to the dietary records*

(Medians and interquartile ranges)

	UR (*n* 28)		Non-UR (*n* 22)		
Food (g/d)	Median	IQR	Median	IQR	*P*
Fruit, berries, juice and jam	253	110–354	195	75–388	0·9680
Milk	126	0–292	127	54–370	0·5090
Bread	154	121–213	198	121–235	0·3890
Vegetables	115	75–149	103	57–158	0·8100
Boiled potatoes, rice and pasta	118	85–168	100	58–202	0·6025
Fatty meat, minced meat and sausages	52	8–121	119	38–164	0·1158
Chicken and meat with less fat	71	20–100	34	9–63	0·2033
Fish and fish products	44	0–137	47	4–122	0·7800
Oil, butter and margarine	31	17–56	40	21–79	0·2662
Fatty cheese	23	13–34	28	8–47	0·3781
Sweets, deserts and snacks	61	38–93	128	108–185	0·0002
Sugary drinks	0	0–110	167	0–335	0·0057
Eating frequency (times/d)	4·7	3·3–5·0	5·0	4·3–6·0	0·2333

IQR, interquartile range.

* Differences in reported intake of food in UR and non-UR were tested with the Mann–Whitney signed rank test.

according to the DR. The finding of low reports of sweets, desserts and snacks is in accordance with a number of previous studies. In a study by Livingstone *et al.* (1990), snacks were named as the most onerous and irritating aspect of the recording procedure and subjects admitted having omitted or simplified some measurements. In a Norwegian survey, Johansson *et al.* (1998) reported that under-reporters had a lower intake of cakes, potato chips, chocolate, sweets and sugar-containing soft drinks. Likewise, among non-obese women, Bingham *et al.* (1995) found a lower intake of breakfast cereals, cakes and sugars and confectionery in under-reporters compared with accurate reporters and consumption of sugar and sweet foods was also lower in under-reporters in two other large surveys (Rothenberg *et al.* 1997; Cook *et al.* 2000). Based on data from a number of studies Heitmann & Lissner (1995) concluded that snack-type foods might preferably be forgotten or suppressed when obese subjects omit food items in dietary reporting. This notion has been directly substantiated in an elegant study. Poppitt *et al.* (1998) covertly measured the food intake of obese and non-obese women confined to a metabolic facility and allowed *ad libitum* food intake. Food consumed during a meal was reported accurately, but the between-meal snack food was under-reported by over one-third. With the exception of the study by Livingstone *et al.* (1990), under-reporters were identified according to calculated EI:RMR ratios, N excretion or directly measured in a metabolic facility. One strength of the present study is that we quantified the contribution of under-reporting of sweets, desserts and snacks to accuracy in free-living, obese subjects with the DLW method according to both the DR and the FFQ.

Table 7. Relation of accuracy according to the food-frequency questionnaire (FFQ) and dietary records (DR) to energy density, macronutrient composition, food, eating frequency and eating scores

(Correlation coefficients)

	FFQ		DR	
	EI:TEE	*P*	EI:TEE	*P*
Energy density*	0·406	0·0034	0·537	<0·0001
Diet composition (% energy)*				
Fat	0·224	0·118	0·292	0·0414
Protein	−0·438	0·0015	−0·403	0·0041
Carbohydrate	0·051	0·7273	0·191	0·1882
Sucrose	0·283	0·0462	0·251	0·0824
Alcohol	0·049	0·7342	0·220	0·1283
Food (g/d)†				
Fruit, berries, juice and jam	0·135	0·3445	0·040	0·7826
Milk	0·333	0·0197	0·007	0·9630
Bread	0·387	0·0067	0·262	0·0694
Vegetables	0·024	0·8690	−0·005	0·9735
Boiled potatoes, rice and pasta	0·201	0·1584	−0·013	0·9286
Fatty meat, minced meat and sausages	0·218	0·1270	0·300	0·0380
Chicken and meat with less fat	−0·093	0·5166	−0·237	0·1009
Fish and fish products	0·114	0·4250	0·002	0·9917
Oil, butter and margarine	0·379	0·0080	0·256	0·0758
Fatty cheese	0·398	0·0053	0·295	0·0407
Sweets, desserts and snacks	0·513	0·0003	0·698	<0·0001
Sugary drinks	0·214	0·1336	0·490	0·0007
Eating frequency (times/d)†	NA	NA	0·360	0·0125
Eating scores*				
Restraint	−0·373	0·0098	−0·388	0·0140
Disinhibition	0·038	0·7913	0·118	0·4203
Hunger	0·301	0·0369	0·318	0·0304

EI, energy intake; TEE, total energy expenditure, NA, not analysed.

* Correlation coefficients were calculated with Pearson's correlations.

† Correlation coefficients were calculated with Spearman rank correlations.

Table 8. Multiple regression analyses of reporting accuracy of energy according to the food-frequency questionnaire*

(Standardised regression coefficients with their standard errors)

	β	SE	*P*
Sweets, desserts and snacks	0·354	0·0002	0·0043
Energy density	0·031	0·0170	0·8113
Bread	0·380	0·0003	0·0163
Fatty cheese	0·093	0·0010	0·4434
Oil, butter and margarine	0·139	0·0010	0·3022
Restraint	−0·079	0·0060	0·5159

* Reporting accuracy is indicated by the energy intake:total energy expenditure ratio.

Table 9. Multiple regression analyses of reporting accuracy of energy according to the dietary records*

(Standardised regression coefficients and their standard errors)

	β	SE	*P*
Sweets, desserts and snacks	0·612	0·0002	< 0·0001
Energy density	0·053	0·0130	0·6298
Sugary drinks	0·104	0·0001	0·1508
Restraint	− 0·212	0·0040	0·0444
Eating frequency	0·154	0·0170	0·1450
Hunger	0·005	0·0060	0·9610

* Reporting accuracy is indicated by the energy intake:total energy expenditure ratio.

Despite a strong statistical association between reporting accuracy and sweets, desserts and snacks in the multivariate analyses, the difference in reported intake of sweets, desserts and snacks was only 60 g/d representing about 1 MJ/d. The energy difference between under-reporters and non-under-reporters was 4·5 MJ/d, thus less than one-quarter was accounted for. It appears that the underestimation is more general as seen in the energy density of the diet, at least according to the DR, and in the macronutrient composition. The percentage energy from protein was higher but the actual intake of protein was lower among under-reporters than non-under-reporters. Excretion of urinary N also indicated that protein intake was lower among under-reporters of energy (data not shown).

The percentage of energy from protein in the diet was strongly inversely related to the reported intake of sweets, desserts and snacks (data not shown). This and other studies (Heitmann & Lissner, 1995; Pryer *et al.* 1997; Livingstone & Black, 2003) suggest that when sweet and fatty foods are under-reported, the percentage of energy from protein increases. In the largest study using urinary N and DLW methodologies, protein density was similar for men, but slightly overestimated for women (Subar *et al.* 2003). Heitmann & Lissner (1995) found a positive association between under-reporting of protein (and total energy) and the degree of obesity among 323 lean and obese men and women. Compared with total energy, protein was over-reported by the obese subjects. Other studies have reported preferential under-reporting of fat (Bingham *et al.* 1995; Macdiarmid *et al.* 1998; Goris *et al.* 2000). Discrepancies between the studies may be explained by differences between populations due to different cultural attitudes or habits, temporal trends, chance or other factors. What foods are considered not healthy, and thus are under-reported, may be affected by societal expectations and messages from the media (Blundell, 2000). In the present study involving obese individuals with characteristics of the metabolic syndrome we speculate that under-reporters may be prone to under-report carbohydrate and fatty food used as sweets, desserts and snacks because of the influence of ongoing debates about low-glycaemic-index diets.

The optimal number of eating occasions to facilitate weight reduction has been debated and it has long been believed that a 'nibbling meal pattern' could be beneficial for the purpose of weight reduction. However, a recent review concluded that weight loss was not facilitated by a high meal frequency (Bellisle, 2004). An association between frequent snacking and EI was also seen in a Swedish study among 4259 obese subjects (Bertéus Forslund *et al.* 2005). Our findings tend to support these findings since reported energy was, not surprisingly, related to the frequency of eating occasions. However, we did not find a statistically significant difference between the frequency of eating between under-reporters and non-under-reporters. The same was also seen in a study by Livingstone *et al.* (1990), and may be due to small sample size, which is also a limitation of the present study.

Dietary restraint, the self-imposed practice of consciously attempting to restrict EI with the purpose of preventing weight gain or promoting weight loss, is a common determinant of accuracy (Bathalon *et al.* 2000), though some conflicting data have been reported. For example, Taren *et al.* (1999) found no association between restraint and reporting accuracy in overweight individuals using 3 d DR to assess reported EI. Lindroos *et al.* (1997) found strong associations between EI, disinhibition and hunger, but a weaker association between EI and restraint in obese women. However, reporting accuracy was not considered in that study. In the OPEN study, restrained eating predicted reporting accuracy only when EI was assessed by the 24 h recall and only among men (Tooze *et al.* 2004). Furthermore, Bingham *et al.* (1995) could not differentiate restrained eating from the known effects of obesity on reporting accuracy. In a simple comparison of under-reporters and non-under-reporters, the present study clearly showed that under-reporters in this population had higher dietary restraint scores and restraint was a significant predictor of accuracy according to the DR. Dietary restraint did not contribute significantly to accuracy according to the FFQ. Hunger scores were associated with accuracy in the univariate but not in the final multiple analyses. Intercollinearity between several of the measured variables may explain these observations. Scores for restraint were inversely associated with the consumption of bread, milk and 'oil, butter and margarine' according to the FFQ (data not shown). Of these variables, bread consumption significantly contributed to accuracy in the multivariate analyses.

Methodological issues

When using DLW as an objective measure of EI, we assume that the subjects were in energy balance over the period of the measurement. The weight change during the DLW period was minor and in accordance with the weight change seen in the Dutch men (Goris *et al.* 2000). We could have adjusted for the weight change in the calculations, but according to the International Dietary Energy Consultancy Group (1990) the difficulty of accurately estimating change in body composition over the short period of DLW measurement is such that little advantage is gained. We could have adjusted for the increase in weight during the 3-month period resulting in a greater degree of underestimation. However, the 3-month study period that included holidays may not adequately represent long-term weight change. The DLW measurement was done during 14 d and may not measure the actual long-term energy expenditure (Livingstone & Black, 2003).

In the calculations of the TEE, we used the individually calculated FQ from the reported intakes according to the FFQ. The under-reporting of carbohydrate could be a concern. However, this probably does not affect the energy calculations because of the high homogeneity of FQ. Fat and carbohydrate intake may vary over a fairly wide range and still the variation in FQ is small (Black *et al.* 1986). The mean FQ was 0·85 in

the present study, in agreement with a typically Western diet (Black *et al.* 1986).

While the FFQ method is prone to under-reporting due to difficulties in remembering eating occasions and estimating portions sizes, under-reporting according to weighed DR may be due both to under-eating and under-recording. We are not able to differentiate between under-eating and under-reporting because a 3 d food record has too short a time period to follow weight changes. It may be that subjects with high scores for dietary restraint actually under-eat when they record food intake to achieve weight reduction, but it has also been shown that restrained eaters as a group do not report all food they consume (Bathalon *et al.* 2000).

Dietary restraint became less obvious in the FFQ that covers a much longer time period including holiday seasons for almost all the participants, and most individuals do not restrict eating during holidays. Limitations of the present study were that we did not collect data on the temporal distribution of eating. Furthermore, the DR was collected for only 3 d and was done a mean of 34 d before the DLW measurement period. We chose the 3 d DR to minimise the burden on the participants in accordance with the suggestion by Trabulsi & Schoeller (2001), that in subjects defined as dietary resistant and obese, any precision gained through a long dietary recording period is outweighed by a larger magnitude of under-reporting. To minimise the burden of the participants was also the reason for obtaining the DR before the DLW measurement period. However, the results of the DR were largely in accordance with the results of the FFQ that covered the DLW measurement period.

In contrast to others (Johansson *et al.* 1998; Cook *et al.* 2000), we did not find any relationship between sex or education level and reporting accuracy. This may be due to the limited size of our sample or to differences between populations. Furthermore, in our sample of obese individuals, BMI, percentage of body fat and fat distribution did not predict reporting accuracy. This may be due to the small variation of BMI or that BMI levels out as a predictor for reporting accuracy of energy at BMI levels above 35 kg/m^2 as was observed in the OPEN study (Tooze *et al.* 2004). The study included only non-smokers and may not be applicable to obese smokers.

Implications

An understanding of the foods and meal patterns that are associated with errors in self-reported data is critical. Individuals often report their usual dietary intake and forget to report extra meals and snacks or more frequent eating patterns. These items must be particularly emphasised in dietary interviews. It was not our objective to determine which of the dietary assessment methods showed superior accuracy in this obese sample. However, it seems that the FFQ is less influenced by the restraint eating behaviour, while the DR may be an important tool to achieve change in eating behaviour because of increased attention. Because of their high risk, obese subjects with the metabolic syndrome or risk factors for the metabolic syndrome are prime targets for dietary advice to achieve weight reduction. While weight-stable, individuals that report high dietary restraint, low consumption of bread, sweets, desserts and snacks and a low frequency of eating may require particular attention when a dietary assessment is conducted.

Acknowledgements

We thank Bo-Egil Hustvedt for the RMR measurements, Tor Ole Klemsdal for some of the medical examinations and Lars Ellegård and the staff at the Department of Clinical Nutrition at Sahlgrenska University Hospital, Sweden for the DLW analyses. We also thank the subjects who took part in the study. Both authors contributed to the development of the protocol and wrote the manuscript. M. S. developed the FFQ, performed all the dietary assessments and analysed the data. None of the authors had any personal or financial conflicts of interest.

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British Journal of Nutrition (2008), **100**, 219–226 doi:10.1017/S0007114507886363

Validation of ActiReg® to measure physical activity and energy expenditure against doubly labelled water in obese persons

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(Received 22 March 2007 – Revised 28 September 2007 – Accepted 6 November 2007 – First published online 15 January 2008)

ActiReg® is an instrument that uses combined recordings of body position and motion to calculate energy expenditure (EE) and physical activity (PA). The aim of the study was to compare mean total energy expenditure (TEE) measured by ActiReg® and doubly labelled water (DLW) in obese subjects. TEE was measured by the DLW method during a period of 14 d in fifty obese men and women with metabolic risk factors. During the same period ActiReg® recordings were obtained for 7 d. RMR was measured by indirect calorimetry and also estimated by standardized equations. Because EE may be disproportionately increased in obese subjects during weight-bearing activities, we established a new set of physical activity ratios (PAR). These ratios were based on oxygen uptake measurements during treadmill walking. The mean TEE according to the DLW was 13·94 (SD 2·47) MJ/d. Mean TEE calculated from the ActiReg® data and measured RMR was 13·39 (SD 2·26) MJ/d, an underestimation of 0·55 MJ (95 % CI 0·13, 0·98; $P=0·012$) or 3·9 %. RMR derived from standard equations based on weight, age and sex were overestimated while the RMR based on fat-free mass values in addition was underestimated. Despite slight underestimation ActiReg® may be used to measure TEE in obese subjects on two premises: RMR should be measured, and the increased EE during weight-bearing activities in obese subjects should be considered.

Energy expenditure: Physical activity: Activity pattern

Obesity (BMI $> 30\,kg/m^2$) is associated with type 2 diabetes, CHD, stroke, increased morbidity and early mortality. In order to plan optimal treatment for subjects at risk, validated methods to measure total energy expenditure (TEE) and physical activity (PA) are essential.

Although the doubly labelled water (DLW) method is clearly the most accurate measure of TEE, its widespread use is limited by the high cost of the labelled water and the requirement of highly specialized and expensive equipment for analysis. The need for precise quantification of TEE and PA during usual living conditions has led to the development of several measurement methods[1]. We have recently described a novel instrument called ActiReg®, a validated position-and-movement monitor[2]. The ActiReg® system uses RMR combined with calculated physical activity ratio (PAR) values as the basis for energy and activity calculations. RMR may be measured or estimated from predictive equations. These equations are usually based on body weight, body height, age and sex and/or fat-free mass (FFM)[3–6]. However, the most widely used predictive equations may not be well suited in obese populations, because the source materials on which they are based include very few if any obese individuals[6–9]. Choice of prediction method for the estimation of RMR may therefore be important. To date the PAR values used by the ActiCalc32® program to calculate EE are published reference values for people with normal body weight[2,6]. Due to the relative increase of adipose tissue mass in obese individuals, predictive equations for RMR based on body weight and developed mainly for a normal-weight population may lead to overestimation of RMR. Likewise the energy cost of weight-bearing activities such as walking and standing is related to body weight, and is therefore increased in obesity[10–14]. Therefore PAR values developed for weight-bearing activities in lean individuals may not be appropriate for obese subjects[15]. As both these factors are the main contributors to the calculation model, it is important to establish reliable and validated values for obese individuals.

The aim of the present study was to calculate TEE from the ActiReg® recordings and compare this to TEE measured by DLW. In order to achieve this aim we established a set of mean PAR values for obese subjects during weight-bearing activity. Finally, we asked whether RMR could be estimated using predictive equations rather than directly measured by indirect calorimetry to simplify the procedure.

Abbreviations: DEXA, dual-energy X-ray absorptiometry; DLW, doubly labelled water; EE, energy expenditure; FFM, fat-free mass; LPA, Low Physical Activity; MHPA, Moderate-High Physical Activity; PA, physical activity; PAR, physical activity ratio; TEE, total energy expenditure; VLPA, Very Low Physical Activity.

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Subjects and methods

Subjects

Fifty non-smoking, obese men and women (BMI ≥ 30·0 kg/m^2) with two or more risk factors for the metabolic syndrome were recruited by newspaper advertisement and referral to the Department of Preventive Cardiology at Ullevål University Hospital. The characteristics of the group are shown in Table 1. As this was part of a broader study of subjects with two or more risk factors for metabolic syndrome subjects were screened via blood chemistry and a medical examination done by a physician to assess risk factors and eligibility to the study[16]. Exclusion criteria were body weight > 135 kg, current dieting, cigarette smoking, history of eating disorder or chronic disease, suspected non-compliance due to abuse of drugs or alcohol, drug- or insulin-treated diabetes mellitus, migraine requiring intermittent medication, use of thyroxin, diuretics or weight-reducing agents, and use of inhaled or oral β-agonists or corticosteroids. The educational level of each subject was determined according to the number of years of education and categorized as completed primary school, high school or a university degree. The Ethical Committee (region 1 in Norway) approved the protocol and all participants gave their written informed consent. The study was conducted between October 2001 and October 2003.

Experimental schedule

The total duration of the experiment was 4 weeks. At baseline (week 1) the participant underwent physical examination including measurement of height, body weight, waist–hip ratio, sitting blood pressure, and collection of plasma and serum samples for different blood parameters[16]. During this week lean body mass was determined by dual-energy X-ray absorptiometry (DEXA). At week 2, RMR and energy expenditure (EE) during weight-bearing activities were measured. Later in the same week the DLW measurement, which lasted for 14 d, was initiated. At the same time the subjects attached the ActiReg® instrument for recording for 7 consecutive days, i.e. the first 7 d of the 14 d DLW period. At week 4, the DLW measurement was terminated and the final urine spot samples were delivered. A dietary assessment using a FFQ was administered.

Methods

Height was measured with a standardized wall measuring stick scale to the nearest 0·5 cm. Subjects were weighed (in underwear) with a digital weight (Seca, Germany) to the nearest 0·1 kg. Weight was measured at the screening and baseline visits and on day 1 and day 15 of the DLW measurement period. Weight changes during the DLW period were calculated as the difference between day 15 and day 1. Body composition was determined by DEXA (Lunar Expert 1116). The measurement was done in the course of 15 min. The CV for the DEXA measurements was 3–4 %. RMR was measured with a standard portable ventilated hood system (Deltatrac® Metabolic Monitor; Datex Instrumentarium Corp., Helsinki, Finland). The Deltatrac® was calibrated by automatic standard gas calibration at the start of each measurement. The subjects slept at home the night before the measurement. On the day of the measurement the subjects took a taxi to the site. The subjects fasted during the last 12 h before the measurement and were instructed not to eat or drink anything but water on the day of the measurement. After changing clothes and mounting the equipment, the subjects relaxed for 30 min in the recumbent position before the head was covered with the canopy. Measurements were done at 1 min intervals for 20–25 min. A mean value of at least a 10 min period at a stable level of EE was defined to be the RMR. After completion of the RMR measurements the subjects were offered a sugar-containing drink prior to the start of a standardized treadmill test. This was done because they all had fasted for more than 12 h. The treadmill test consisted of walking at increasing speeds (1, 2, 3, 4, 5 and 6 km/h) at an inclination of 1 % for periods of 5 min at each velocity, while their O_2 uptake and CO_2 output were measured with spirometry (Jaeger Oxicon®). The treadmill test was performed in order to obtain calibration values for the EE related to different weight-bearing PA.

The doubly labelled water method

EE by the DLW method was measured over a period of 14 d and used as a gold measure of habitual EE. Sample analyses and calculation procedures have been described in detail elsewhere[17]. First a baseline urine sample was collected for the determination of the background isotope enrichment (day 1). Then a weighted mixture of deuteriated and oxygenated water, corresponding to 0·05 g ^2H_2O and 0·10 g $H_2^{18}O$ per kg body weight, was ingested. The percentage enrichment of the waters was 99·9 % for ^2H and 10·0 % for ^{18}O. The dose was planned to enrich body water with approximately 350 δ (delta per mill) for ^2H and 60 δ (delta per mill) for ^{18}O. Urine samples were collected from the second voiding during days 2, 3, 4, 8, 13, 14 and 15. The mean time interval between drinking dose and the first post-dose urine sample was 22 (SD 3) h (range 12–30 h). The participants were instructed to collect the urine spot, register exact voiding

Table 1. Physical characteristics of the participants

	Males (*n* 23)			Females (*n* 27)			
	Mean	SD	Range	Mean	SD	Range	*P**
Age (years)	44·5	10·0	27·7	42·6	10·7	41·0	0·5322
Height (m)	1·82	0·07	0·30	1·69	0·06	0·24	0·0000
Weight (kg)	116·2	14·8	52·6	105·0	12·2	48·1	0·0053
BMI (kg/m^2)	34·9	3·1	11·5	36·6	3·3	12·8	0·0645

* Two-sample *t* test assuming equal variances.

Table 2. Mean RMR measured by indirect calorimetry and calculated by different prediction equations

Type of measurement/estimation	RMR (MJ/d) Mean	SD	Difference	Range	95 % CI	P*
Measured by indirect calorimetry	7·75	1·05		4·53		
Mifflin *et al.*[4]†	7·94	1·06	− 0·19	4·61	− 0·33, − 0·05	< 0·007
FAO/WHO/UN University[6]‡	8·20	1·19	− 0·45	4·44	− 0·62, − 0·29	< 0·000
Müller *et al.*[3]§	8·25	1·09	− 0·50	4·53	− 0·63, − 0·36	< 0·000
Cunningham[5], fat-free mass from DEXA¶	6·65	0·97	1·11	4·17	0·90, 1·32	< 0·000

DEXA, dual-energy X-ray absorptiometry.
* Paired samples test on the mean difference between the measured value and predicted values. Significance level $P < 0{\cdot}05$.
† Calculation formula: RMR (kcal/d) = 9·99 × weight (kg) + 6·25 × height (cm) − 4·92 × age + 166 × sex (males = 1; females = 0) − 161 (result converted to MJ/d by multiplication by 4·184).
‡ Calculation formula: Males (age 30–60): RMR (MJ/d) = 0·0485 × weight (kg) + 3·67; females (age 30–60): RMR (MJ/d) = 0·0364 × weight (kg) + 3·64.
§ Calculation formula (BMI ≥ 30): RMR (MJ/d) = 0·05 × weight (kg) + 1·103 × sex (males = 1; females = 0) − 0·01 586 × age + 2·924.
¶ Calculation formula: RMR (kcal/d) = 370 + 21·6 × fat-free mass (kg) (result converted to MJ/d by multiplication by 4·184).

time and freeze the samples at home. Participants were called every voiding day to ensure compliance with the procedure. When the samplings were completed, the urine samples were stored at −75°C until transportation to laboratory on dry ice.

Analysis of the isotopic enrichment was determined in triplicates with a Thermoquest Finnigan MAT Delta plus isotope-ratio mass spectrometer with water/H_2–CO_2 equilibrating device (Thermoquest Finnigan MAT, Bremen, Germany). The precision defined as standard error in triplicate samples is 0·26 δ for ^2H and 0·10 δ for ^{18}O. Tap water was collected and analysed for background measurements and all TEE calculations were corrected for the content of isotopes in the drinking water. TEE was calculated by the multi-point method using linear regression as suggested by the International Dietary Energy Consultancy Group[18]. All elimination curves were checked for major or diverging residuals. The CV for the elimination constants was on average 3·2 % for hydrogen and 2·7 % for oxygen. We used the relationship between pool size of ^2H (N_d) and pool size of ^{18}O (N_o) derived from the antilog intercept on the *y*-axis of the elimination curves as a quality measurement for the DLW as suggested by the International Dietary Energy Consultancy Group[18]. The mean food quotient determined from the FFQ was 0·85 (SD 0·016; range 0·81–0·89). The individual N_o/N_d ratio and food quotient of the participants were used in the calculation of the energy

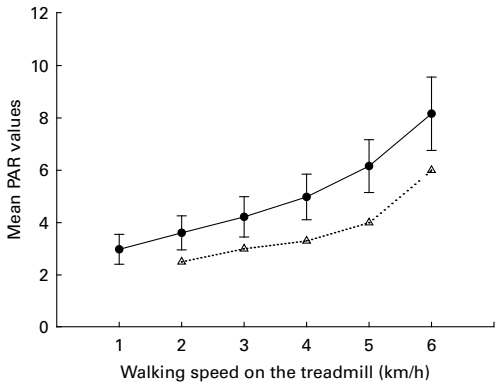

Fig. 1. Treadmill walking test for the combined group of obese subjects (both sexes, *n* 50). The physical activity ratio (PAR) is the measured energy expenditure divided by RMR at each speed. Values are means with their standard errors depicted by vertical bars. (●), Mean PAR for the combined obese group (both sexes); (△), for comparison, table values of PAR for normal-weight subjects[6,23,24].

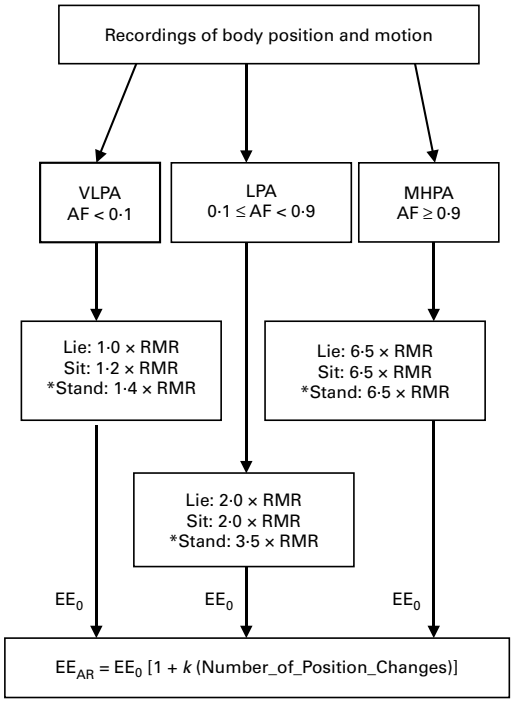

Fig. 2. The calculation procedure for energy expenditure based on ActiReg® data (EE_{AR}). In the first step the data are distributed into the three activity levels: Very Low Physical Activity (VLPA), Low Physical Activity (LPA) and Moderate-High Physical Activity (MHPA)[2]. The calculation within each level is based on the estimated energy cost for the actual body position, expressed as the RMR-factors. The result of this first calculation step is denoted EE_0. The second step takes the number of body position changes into account by applying the algorithm shown, where EE_{AR} is the final result for the actual minute. The constant $k = 0{\cdot}025$ determines the weight given to the number of body position changes, here designated as 'Number_of_Position_Changes'. AF, activity factor; * Stand, standing position including the bent forward position.

equivalence of the produced carbon dioxide as suggested by the International Dietary Energy Consultancy Group[18]. The mean N_o/N_d ratio was 1·033 (SD 0·008; range 1·007–1·049).

ActiReg®

ActiReg® is an electromechanical device which records the main body positions (stand, sit, bent forward and lie) together with motion of the trunk and/or one leg each second[2]. The position (tilt switches) and motion sensors are fixed to plastic brackets. During registration the subjects attached the ActiReg® (actual size of the box is 8·5 cm × 4·5 cm × 1·5 cm) to a belt while the sensors were connected to the box with thin lines. The brackets were attached by medical tape to the subject's chest (on sternum) and on the front of the right thigh approximately midway between the knee and the hip. The tilt switches were oriented so that they would be in the vertical position when the subject was standing. A specially developed computer program (ActiCalc32®) calculated EE and activity pattern from the collected information and calibration data[2].

The ActiReg® system uses a combined second-to-second recording of body position and motion to calculate EE and PA. The apparatus has two pairs of position and motion sensors connected by cables to a battery-operated storage unit fixed to a waist belt. Each pair of sensors is attached by medical tape to the chest and the front of the right thigh, respectively. The collected data are transferred to a PC and processed by a dedicated program ActiCalc32®. More details about the method are published elsewhere[2]. The calculation model used by ActiCalc32® is based on the estimated cost of the actual body position and activity expressed as PAR values (i.e. EE/RMR) combined with the number of position changes within each minute.

As described by Hustvedt *et al.* [2] the data from the ActiReg® was categorized into three levels of physical activity defined as Very Low Physical Activity (VLPA), Low Physical Activity (LPA) and Moderate-High Physical Activity (MHPA). The calculation within each level was based on the estimated energy cost for the actual body position, expressed as RMR-factors (PAR values) for subjects with normal body weight and taken from published reference values (Annex 5 of FAO/UN University/WHO[6]). In the VLPA range, the following factors were selected: lie still: 1·0 × RMR; sit still: 1·2 × RMR; stand still/bent forward: 1·4 × RMR. The LPA range extended from moving very slowly to walking at about 3 km/h and 2·5 × RMR is chosen as the average energy cost of standing activities. This is the energy cost given for 'walking around or strolling'. The factor for sitting and lying activities, which are non-weight-bearing activities, was set somewhat lower, at 2·0 × RMR. The dominant activity in the MHPA range during the daily life of most people is walking. The reported energy cost of 'walking: at normal pace' is 3·2 × RMR. In addition, a variable amount of more energy-requiring activities is expected, such as walking on stairs or uphill, walking while carrying loads, and performing exercise. The factor 5·0 × RMR was therefore chosen as the average energy cost of all MHPA activities. It was applied for all body positions. The treadmill experiments performed by Hustvedt *et al.* [2] showed that walking will fall in the MHPA range and that no body position changes were recorded until the walking speed exceeded 5 km/h. At ≥ 7 km/h, where the number of position changes increased, some minutes with the body positions 'sit' or 'lie' were also recorded. These recordings show that the state of the position sensors as well as the movement sensors is influenced by acceleration forces during rapid movement, such as running, in addition to the effect of the position angle. When walking/running speed increases a rising number of body position changes is recorded which is used to discriminate between higher levels of PA. The calculation procedure for EE_{AR} (EE based on ActiReg® data) utilizes the combined information about PA level, body position and the number of position changes. The EE_{AR} of all MHPA are therefore not calculated according to a PAR value of 5·0 but by an increased value proportional to the number of position changes as described by Hustvedt *et al.* [2].

However, for obese subjects we expected that the PAR values (RMR-factors) for weight-bearing activities would be

Table 3. Mean total energy expenditure (TEE) from the doubly labelled water (DLW) measurements and those calculated from the ActiReg® data based on the different RMR values and the difference between results calculated by ActiReg® and DLW values

	TEE (MJ/d)		Difference (MJ)			
Calculation method*	Mean	SD	Mean	SD	95 % CI	*P*†
DLW	13·94	2·47				
AR-RMR-measured	13·39	2·26	−0·55	1·49	−0·98, −0·13	0·012
AR-RMR-Mifflin	13·73	2·32	−0·21	1·65	−0·68, 0·25	0·358
AR-RMR-FAO/WHO/UNU	14·18	2·59	0·24	1·78	−0·26, 0·75	0·341
AR-RMR-Müller	14·26	2·42	0·32	1·71	−0·17, 0·80	0·199
AR-RMR-Cunningham-FFM-DEXA	11·48	2·05	−2·46	2·00	−3·03, −1·90	0·000
AR-RMR-measured-normal-PAR	11·81	1·87	−2·13	1·36	−2·52, −1·74	0·000

DEXA, dual-energy X-ray absorptiometry; FFM, fat-free mass; PAR, physical activity ratio.

* AR-RMR-measured: calculated by ActiReg® with measured RMR. AR-RMR-Mifflin: calculated by ActiReg® with RMR predicted from the equation of Mifflin *et al.*[4]. AR-RMR-FAO/WHO/UNU: calculated by ActiReg® with RMR predicted from FAO/WHO/UN University equation[6]. AR-RMR-Müller: calculated by ActiReg® with RMR predicted from the equation of Müller *et al.*[3]. AR-RMR-Cunningham-FFM-DEXA: calculated by ActiReg® with RMR predicted from the equation of Cunningham, with FFM from DEXA[5]. AR-RMR-measured-normal-PAR: calculated by ActiReg® with measured RMR combined with calculation parameters for normal-weight subjects. For comparison, the last row gives the result obtained by using the measured RMR values combined with PAR values for normal-weight subjects, i.e. no correction made for increased energy expenditure during weight-bearing activities.

† Paired samples test on the mean difference between the TEE calculated from ActiReg® data and those measured by DLW.

somewhat increased. In this investigation these PAR values were based on mean values obtained during the treadmill experiments.

Statistics

The agreement between the results obtained by two different methods was tested by the method of Bland and Altman[19]. Paired two-sample *t* tests were used to evaluate the difference between the groups (SPSS for Windows version 13.0.0; SPSS Inc., Chicago, IL, USA). The significance level was set at $P<0{\cdot}05$. The correlation of linear regression is given as r^2.

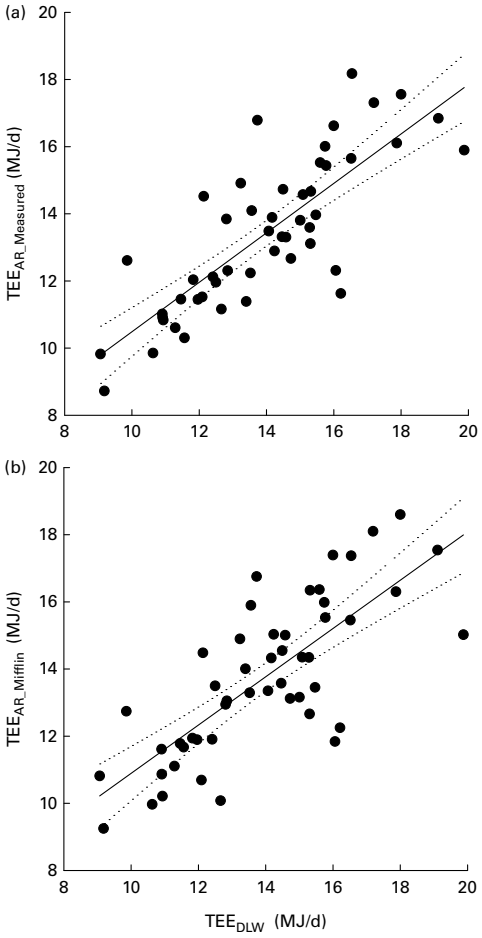

Fig. 3. (a), Linear correlation between the mean total energy expenditure measured by ActiReg® based on measured RMR ($TEE_{AR\_Measured}$) and the mean total energy expenditure from doubly labelled water measurements (TEE_{DLW}). The linear regression line shows a positive correlation ($y = 0{\cdot}736 + 3{\cdot}122$, r^2 0·64, P=0·000). (b), Correlation between by TEE_{DLW} and the mean total energy expenditure from the same ActiReg® data but here the calculations are based on predicted RMR from equations developed by Mifflin *et al.*[4] ($TEE_{AR\_Mifflin}$). The linear regression line shows a positive correlation ($y = 0{\cdot}718 + 3{\cdot}704$, r^2 0·585, P=0·000). —, Linear regression line; ·····, 95 % confidence limits.

Results

Subject characteristics according to sex are shown in Table 1. There was no significant difference in mean age between males and females.

TEE from DLW measured over 14 d was not significantly different from the value extracted from 7 d, and the mean value during 14 d was chosen as a more reliable measurement based on more data points.

Table 2 shows the mean RMR combined for both sexes obtained by indirect calorimetry and corresponding results using different predictive equations for RMR. The results of all predictive methods differed significantly from the measured RMR value. The equation proposed by Cunningham based on FFM significantly underestimated RMR[5]. The equations proposed by Müller *et al.*[3], Mifflin *et al.*[4] and the FAO/WHO/UN University[6] all led to overestimation of RMR.

Figure 1 show the results of the treadmill experiments. It will be seen that the mean PAR value for the total group (both sexes) is increased compared to the table values of PAR for normal-weight subjects at all walking speeds between 2 and 6 km/h. This implies that the basic PAR values used during calculation of EE_{AR} should be increased accordingly during weight-bearing conditions in order to follow the same logic as used for normal-weight subjects. The logic of the calculation model is described earlier[2].

The calculation procedure established for the obese group is shown in Fig. 2. This implies that the PAR value for the weight-bearing body positions, i.e. standing and bent forward at LPA (which corresponds to a walking speed of about 3 km/h) is increased from 2·5 to 3·5, while it is increased from 5·0 to 6·5 at MHPA (corresponding to a PA of walking 4·0–5·0 km/h) for all body positions.

The mean TEE from the DLW measurements as well as those calculated from the ActiReg® data based on different RMR values are presented in Table 3. There was no significant difference between the mean TEE_{DLW} and those calculated from ActiReg® data based on RMR values from the FAO/WHO/UN University, Mifflin and Müller predictive equations (Table 3). However, the difference between the mean TEE_{DLW} and the mean TEE_{AR} based on RMR values measured by indirect calorimetry was statistically significant. The TEE_{AR} value calculated from RMR values based on the predictive equation using FFM instead of body weight significantly underestimated the mean TEE. Also the mean TEE_{AR} based on measured RMR but using PAR values for 'normal-weight' subjects grossly underestimated mean TEE.

Fig. 3 shows the linear correlation between $TEE_{AR\_Measured\_RMR}$ and TEE_{DLW} (Fig. 3 (a)) and $TEE_{AR\_Mifflin\_RMR}$ and TEE_{DLW} (Fig. 3 (b)) with r^2 0·64 (P=0·00) and r^2 0·585 (P=0·00), respectively.

In Figs. 4 and 5 the results are compared with Bland–Altman plots. The difference between the calculated TEE_{AR} and the TEE_{DLW} are plotted against their average values. The limits of agreement of the mean difference (i.e. ±2 SD) are indicated by the dotted lines. Fig. 4 includes results calculated from measured RMR, and predicted from

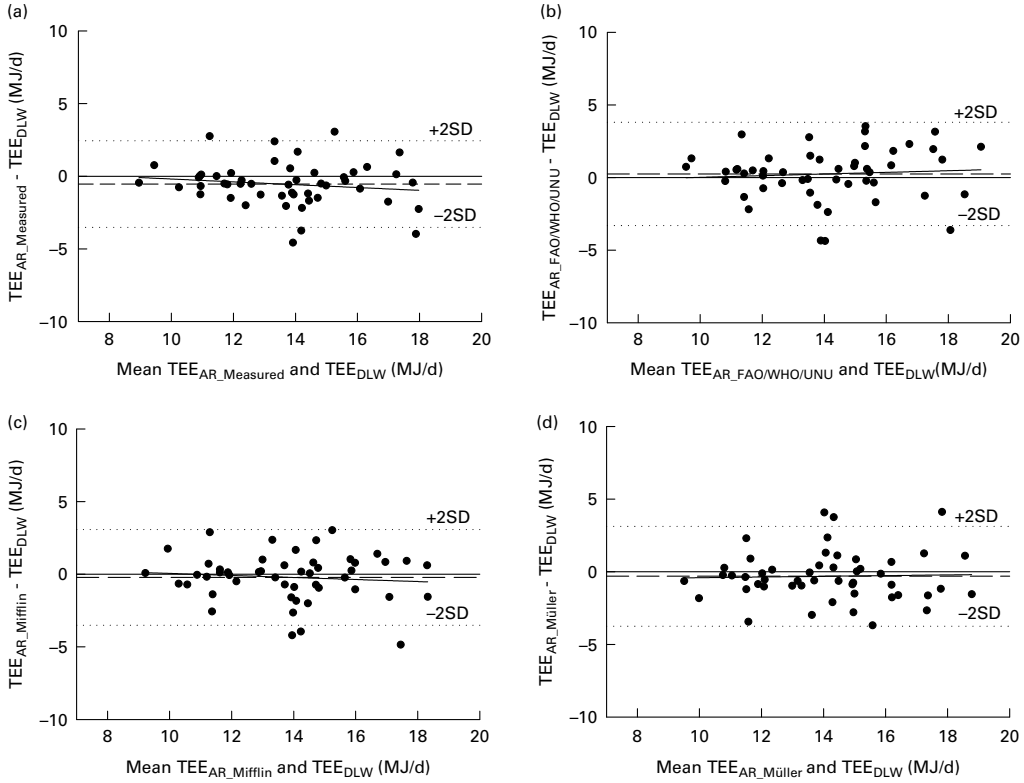

Fig. 4. The results are compared in Bland–Altman plots. The difference between the calculated total energy expenditure measured by ActiReg® (TEE_{AR}) and the total energy expenditure from doubly labelled water measurements (TEE_{DLW}) are plotted against the average value of them. The results are based on measured RMR (a), and RMR predicted from the equations of the FAO/WHO/UN University[6] (b), Mifflin *et al.*[4] (c) and Müller *et al.*[3] (d). – – –, Mean difference; ·····, limits of agreement of the mean difference (±2SD); —, zero difference and the linear regression lines.

FAO/WHO/UN University[6], Mifflin[4] and Müller[3] equations. For all four graphs in this figure the differences are evenly distributed throughout the range of the measurements and the linear regression lines are almost parallel to the *x*-axis. Fig. 5 presents the corresponding results based on RMR values calculated from FFM by the Cunningham equation[5] and measured RMR using PAR values for normal-weight people. The graphs in Fig. 5 show serious underestimation of mean TEE, but also a tendency to increased underestimation at higher levels of TEE. This is shown by the negative trend of the linear regression line and is most pronounced when PAR values for normal-weight people are employed.

Discussion

The present study compared measurements of TEE by ActiReg® and DLW in a group of obese subjects. The results show that with the use of increased PAR values for weight-bearing activities, mean TEE calculated from the ActiReg® data was underestimated by less than 4 % compared to DLW, a statistically significant but minor difference for most purposes. Despite this underestimation we propose that ActiReg® may be used to measure TEE in obese subjects on two premises: RMR should be measured, and the increased EE during weight-bearing activities in obese subjects should be considered.

The calculation model used by the ActiReg® system is based on the product of RMR (kJ/min) and the PAR value for each minute of the registration period, i.e. the factorial principle. The PAR values for each specific minute is estimated from the combined information of body position, motion and number of position changes. Reliable values of RMR and PAR values in different body positions and activity levels are therefore a prerequisite for an optimal estimate of EE. The results of using the prediction equations based solely on anthropometric data, age and sex led to significant overestimation for this group of obese subjects (Table 2). This is in accordance with previous reports[3,9,11,20–22]. The Schofield[8] equations which have been adopted by WHO for general use in predicting RMR are linear in weight (Table 2)[6]. The overestimation between measured and predicted RMR may be explained in part by composition of the database and biological factors. These equations are based on analysis of data collected from 114 previous studies made

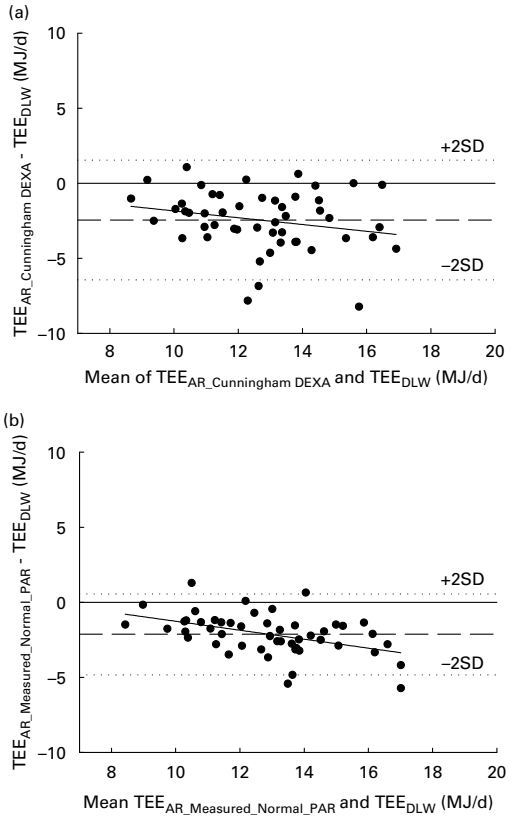

Fig. 5. Bland–Altman plots comparing the calculated total energy expenditure measured by ActiReg® (TEE_{AR}) results based on RMR values calculated from fat-free mass obtained from dual-energy X-ray absorptiometry (DEXA) by the Cunningham[6] equation (a) and measured RMR values using physical activity ratio (PAR) values for normal-weight people (b) with total energy expenditure from doubly labelled water measurements (TEE_{DLW}). – – –, Mean difference; ·····, limits of agreement of the mean difference (±2SD); —, zero difference and the linear regression lines.

in persons belonging to different races. In addition, one-third of the reference population had BMI $< 20\,kg/m^2$, but very few were obese. Also the total distribution of body weights within this population is quite different from the normal distribution for subjects living in modern affluent societies[3,9]. It is well documented that RMR increases with increased body weight and with increasing BMI, but the increase is not linear or directly proportional to body weight. RMR increases more slowly at heavier weights, and to ignore this will lead to overestimation of RMR in the obese. When the body gets fatter, a greater ratio of fat to lean tissue is deposited and as the metabolic rate of adipose tissue is low compared to that of lean tissue, RMR will not increase linearly by weight[9].

Mifflin *et al.*[4] derived new prediction equations based on a data set of 498 men and women that also incorporated a significant number of obese subjects (Table 2). More recently Müller *et al.*[3] developed equations based on an actual German database for different BMI groups of which the equation for BMI ≥ 30 has been used in this paper (Table 2). Because most of the values included in the development of these prediction equations fell within the normal weight range, it is reasonable that they will overestimate RMR in the obese because they all are linear with respect to body weight. The RMR values calculated by the general prediction equation proposed by Cunningham[5] are based solely on the amount of FFM (Table 2). When we apply this equation to calculate RMR in our obese subjects this will underestimate RMR compared to measured values.

In the present study, reliable PAR values for obese subjects during weight-bearing activities were obtained by treadmill walking and indirect calorimetry (Fig. 1). Based on the mean PAR values for the whole group (both sexes) at LPA and MHPA the PAR values in the calculation model were set to 3·5 and 6·5, respectively, compared to 2·5 and 5·0 for normal-weight people. The LPA level extends from moving very slowly to walking at about 3 km/h which is equivalent to 'walking around or strolling'. A reasonable value for this activity for the obese is therefore set to 3·5, a value that we chose empirically.

Walking is the dominant activity in the MHPA range during the daily life of most people. The reported PAR value of 'walking at normal pace' (4–5 km/h) is 3·2 for normal-weight people.

In addition, there will be a variable amount of more energy-requiring activities, such as walking on stairs or uphill, walking while carrying loads, and performing exercise. Based on the treadmill measurements a PAR value of 6·5 is therefore chosen as the average energy cost of all MHPA activities. The same PAR value is applied for all body positions, since the body position recording may be erroneous during high activity[2].

Comparison of the results of mean TEE from the ActiReg® recordings based on different RMR values (measured and estimated) and the DLW measurements shows that results based on the anthropometric data age and sex are not significantly different from the DLW values. This is likely to be due to overestimation of RMR and underestimation of PA by ActiReg® while the results based on measured RMR underestimate TEE by − 0·55 MJ on average (Table 3). The reason for the underestimation based on the measured RMR may be due to variation in variables other than pure anthropometrical data.

The mean TEE values obtained by using RMR based on FFM calculated by the Cunningham equation seriously underestimate TEE compared to TEE_{DLW}.

The correlation between $TEE_{AR\_Measured\_RMR}$ and TEE_{DLW} (Fig. 3 (a)) and $TEE_{AR\_Mifflin\_RMR}$ and TEE_{DLW} (Fig. 3 (b)) are of the same magnitude, i.e. r^2 0·645 and r^2 0·585, respectively. The difference between them is small also when the results based upon RMR values from the anthropometrical data and measurements are compared in Bland–Altman plots (Fig. 4). However, the limits of agreement for the measurement based upon measured RMR are narrower than for those based upon predicted RMR values. This is the most likely reason why this value is different compared to DLW. It will be seen that the mean and the standard deviation of the differences are constant throughout the range of measurements and normality tests show that the differences are evenly distributed and the linear regression line is almost parallel to the *x*-axis. A close

look at the frequency distribution plot of the difference values based upon the RMR value from FFM shows that these are less evenly distributed and exhibit an increased tendency to underestimation as TEE increases (Fig. 5 (a)). This is also demonstrated by the negative trend of the linear regression. The reason for underestimation of TEE in this plot is solely due to underestimated RMR because all other calculation parameters are equal. (Measured RMR is closely correlated to body weight also in this group (r 0·82) while the corresponding values for $RMR_{Cunningham\_DEXA}$ is 0·50.)

In Fig. 5 (b) the calculation has been performed using the measured RMR values but employing the calculation parameters (PAR values) for normal-weight subjects. The underestimation can be seen clearly, and in addition this underestimation increases with higher TEE. This clearly demonstrates the significant impact on TEE of the increased EE due to body weight during weight-bearing activities which are not compensated for when using the PAR values for normal-weight subjects. The only difference in this calculation compared to that in Fig. 4 (a) is the application of lower PAR values.

In conclusion, ActiReg® is a simple and cheap method to estimate TEE compared to DLW. The present study shows ActiReg® to give good estimates of mean TEE in obese subjects as validated by DLW with a mean underestimation of only 0·55 MJ. The performance of ActiReg® in obese subjects is comparable to that previously shown in normal-weight subjects[2].

Acknowledgements

The authors thank Svein Leirstein and Jan Erlend Hem for measurement of oxygen uptake during the treadmill walking. All authors have contributed to study design and writing of the paper, and none of them have any personal or financial conflicts of interest. S. T. and M. S. were in charge of the clinical examinations and the practical measurements and collection of data with ActiReg® and DLW, while L. E. was responsible for the analysis and evaluation of DLW data. B.-E. H. and A. L. have developed the ActiReg® system. B.-E. H. performed the RMR measurements and has been mainly responsible for data processing and manuscript preparation.

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III

Physical activity patterns of obese subjects with a high metabolic risk profile

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ABSTRACT

Background: We assessed physical activity (PA) pattern and examined the association between cardiovascular risk factors and PA in treatment seeking obese subjects.

Methods: Sixty-eight women and 62 men aged 24-64 years who were obese and had a high metabolic risk profile were referred for treatment. They recorded PA continuously with a position and movement recorder (ActiReg®) for a mean of 4 days. Time spent on PA categorized as physical activity ratios (PAR) were calculated. Blood tests were obtained for fasting lipids.

Results: The subjects spent a mean±s.d. of 1065±107 min lying, standing and sitting (PAR 1.0-1.5), 348±100 min strolling and walking (PAR 1.6-6.0), and 26±17 min brisk walking and jogging (PAR >6.0) daily. The time spent on brisk walking and jogging was inversely associated with total cholesterol (R^2_{adj}=0.056 [95%CI 0.013, 0.186], P=0.0161) and triglycerides (R^2_{adj}=0.080 [95%CI 0.027, 0.208], P=0.0039) after adjustment for age and sex.

Conclusion: Obese subjects with metabolic risk factors engaged in activities corresponding to brisk walking and jogging for a mean of 26 min per day, before weight loss was attempted. This level of PA was inversely associated with lipid concentrations. Thus higher levels of activity than 30 min daily may be needed to facilitate weight loss.

Introduction

The relation between obesity and several of its co-morbidities such as premature atherosclerosis and type 2 diabetes is primarily mediated by risk factors including increased abdominal obesity, high blood pressure, impaired glucose metabolism, and dyslipidemia.[1,2] The metabolic syndrome is a cluster of these risk factors that increases the risk of cardiovascular disease (CVD) and all cause mortality.[2, 3] One of the primary methods to reduce the risk associated with the metabolic syndrome is weight loss.[4] Studies have shown that even a modest degree of weight reduction induced by change in diet and physical activity (PA) reduced the risk for developing type 2 diabetes.[5, 6] Physically active women and men of all ages have a lower mortality rate compared to inactive individuals.[7, 8] Moreover, increased leisure time PA in middle-aged men was associated with decreased risk of developing metabolic syndrome nearly 30 years later.[9] However, obesity has been inversely associated with physical activity in observational studies. [10]

In 2007 the American College of Sports Medicine and American Heart Association updated their recommendations for PA and public health. To promote and maintain health it was recommended that all healthy adults aged 18 to 65 years should engage in a minimum of 30 min of moderate-intensity (endurance) physical activity on five days each week as well as activities that maintain and increase muscular strength and endurance a minimum of two days each week.[11] Furthermore, for individuals that achieve this level of activity but remain overweight, a further increase in PA was stated to be a reasonable component of strategies to lose weight.[11] Data from the 1999-2002 NHANES showed that only 20% of obese subjects that wanted to loose weight participated in moderate- to vigorous-intensity physical activity for more than 150 min per week. [12] Clearly, this is an area where adherence is a challenge. To be able to design appropriate exercise prescriptions in a clinical setting, a quantified assessment of the activity pattern of obese individuals i.e. the amount time spent in inactivity,

light activity and moderate to vigorous activity is useful. However, data regarding the physical activity pattern of severely obese subjects is sparse.

Though PA has been assessed in numerous ways, accurate and reliable monitors for assessing PA are still lacking.[13] ActiReg®, a portable device that records positions and movements [14], has been shown to give valid estimates of total energy expenditure (TEE) in obese subjects.[15] In addition, activity patterns can be assessed from the recording by calculating the physical activity ratio (PAR) of different physical activities. The PAR is the ratio that expresses the energy expended in a particular activity, as multiples of the resting metabolic rate (RMR). [16]

This study's primary aim was to assess PA patterns recorded using ActiReg®, in treatment seeking obese women and men with a high risk of CVD due to the presence of risk factors for the metabolic syndrome. Furthermore, we examined the associations between moderate intensity PA and cardiovascular and metabolic risk factor levels.

Methods

Subjects

Subjects were 68 women and 62 men consecutively referred to the Department of Preventive Medicine at Ullevål University Hospital for weight loss and cardiovascular risk reduction or who were seeking obesity treatment in response to newspaper advertisements. The present study was performed before any dietary advice or advice to increase physical activity was given. Potential subjects were included if they had two or more components of the metabolic syndrome according to the National Cholesterol Education Program.[17] Exclusion criteria were body weight > 135 kg, suspected non-compliance because of drug or alcohol abuse, any major non-cardiac disease expected to interfere with the study, weight change of more than ± 4%

and/or the use of appetite suppressants or weight reducing medication within the last three months.

The subjects were aged between 24 and 64 years and had a mean ± s.d. body mass index (BMI; kg/m^2) of 36.4 ± 4.3 (range 30-50). They were screened via blood chemistry and a medical examination to assess risk factors and eligibility. The Ethical Committee (region 1) in Norway approved the protocol and all subjects provided written informed consent. The study was conducted between November 1998 and September 2003.

Anthropometrics and biochemical measurements

The subjects were weighed (in underwear) with a digital weight (Seca, Germany) to the nearest 0.1 kg. Height was measured with a standardized wall measuring stick scale to the nearest 0.5 cm. Waist circumference was measured at the umbilicus with the subject unclothed and in the standing position. Blood pressure was measured in the right arm with a digital blood pressure monitor (OMRON Hem-705 CP).

Body composition (total fat mass, trunk fat mass and lean mass) was determined by dual energy X-ray absorptiometry (Lunar Expert 1116). The coefficient of variation (CV) for the dual energy X-ray absorptiometry measurements was 3-4%. Due to technical problems, 2 subjects were missing data for total body fat and lean body mass. In addition, trunk fat was not calculated for 3 subjects.

Fasting (10 h overnight) blood samples were analyzed for serum lipids and glucose with conventional enzymatic methods. Values of serum triglycerides and glucose were missing for 1 male subject due to insufficient material for analysis.

Assessment of physical activity

PA was assessed continuously with ActiReg® for a mean ± s.d. of 4 ± 2 days. All days of the week were represented in the measurements. ActiReg® is not waterproof and the device was taken off when the subjects took a shower or were swimming. A few subjects experienced skin reactions due to the medical tape used to attach the sensors, and were allowed to take off the device during sleep at night.

A detailed description of the ActiReg® system has been described elsewhere. [14] In short, ActiReg® is an electromechanical device, which records the main body positions (stand, sit, bent forward and lie) together with motion of the trunk and/or one leg each second. During registration the subjects wear the ActiReg® monitor in a belt and two sensors are connected to the monitor with thin cables. The motion sensors are attached to the subject's chest (on sternum) and on the front of the right thigh. A dedicated computer program (ActiCalc32®) calculates TEE and activity patterns from the collected information.[14] The calculation model used by ActiCalc32® is based on the estimated cost of the actual body position and activity expressed as PAR values (i.e. EE/RMR) combined with the number of position changes within each minute. [15]

We used PAR values between 1-1.5 for activities such as lying down, standing and sitting. As described elsewhere [15], 50 of the subjects did a treadmill test where they walked at increasing speeds as follows: 0.6, 1.2, 1.8, 2.4, 3.0 and 3.6 mph (1, 2, 3, 4, 5 and 6 km/h). According to this test, PAR values between 1.6-6.0 corresponded to strolling and walking at a speed of 0.6-2.7 mph (1.0-4.5 km/h) and PAR >6.0 corresponded to brisk walking at a speed of >2.7 mph (4.5 km/h). While the PA pattern is expressed in terms of walking at different speeds in correspondence to the treadmill test, the actual activity correspondent to PAR 1.6-6.0 included light housework, gardening, vacuuming, golf, dancing and bicycling. The actual activity correspondent to PAR >6.0 included brisk walking, heavy yard work, snow

shoveling, cross-country skiing, aerobics, fast bicycling, walking uphill, climbing stairs, walking with loads and jogging.

TEE was calculated by summing the energy expended on the different PAR levels. RMR was calculated with the Mifflin formula [18] as this formula has shown good predictibility of RMR in obese subjects. [19, 20]

RMR (kcal) = 9.99·weight (kg) + 6.25·height (cm) – 4.92·age + 166·sex (men;1, women;0) [18]

To assess the overall activity during the day, we calculated the physical activity level (PAL) as the ratio of TEE to RMR. Compared to TEE measured with the doubly labelled method, the ActiReg® method has shown a small, but clinically insignificant underestimation of TEE.[15]

Statistics

Data are presented as mean ± s.d. or median (25^{th}, 75^{th} percentile) values. Differences between sexes were tested with an unpaired t-test and the Mann-Whitney two-sample rank test as appropriate for normally distributed and skewed variables. Associations between single, continuous variables were tested with Pearson's correlation. Serum triglycerides and the amount of time spent at PAR >6 were log-transformed because of skewness but are presented as untransformed values. One female subject did not record any activity at PAR >6. The variable (ln (min at PAR>6 + 1 min)) was used in the analyses. Variables that were significantly correlated with min per day spent at PAR>6 were entered into a multiple regression analysis and adjusted for confounders. In these analyses min per day spent at PAR>6 was the dependent variable and sex and age and total cholesterol, HDL cholesterol or triglycerides were the independent variables. The tests were considered statistically

significant at $P<0.05$. Statistical analyses were performed using Stat View 5.0.1 (Abacus concepts, Berkeley, CA).

Results

Characteristics of the subjects are shown in Table 1. The subjects had a high mean waist circumference, high systolic and diastolic blood pressures and high concentrations of total cholesterol and triglycerides. Of the total, 16 subjects (12%) had diabetes type 2 and 98 (75%) had the full metabolic syndrome. Women had a lower mean waist circumference and lean body mass and higher total fat mass than men.

Table 2 shows the levels of energy expenditure and activity pattern separated by sex. Women spent a median (25^{th}, 75^{th} percentile) of 23 (12, 32) min per day at PAR >6 while men spent a median (25^{th}, 75^{th} percentile) of 25 (16, 36) min per day at this PAR level (P= 0.3512).

Pearson's correlations between cardiovascular risk factors and min spent at PAR >6 are shown in Table 3. Serum concentrations of total cholesterol (r=-0.23, P=0.002), HDL-cholesterol (r=-0.18, P=0.042) and triglyceride (r=-0.26, P=0.003) were associated with min spent at PAR >6. The trends were similar in women and men (data not shown).

In multiple regression analyses the number of min per day spent at PAR >6 was inversely associated with total cholesterol (R^2_{adj}=0.056 [95%CI 0.013, 0.186], P=0.0161) and triglycerides (R^2_{adj}=0.080 [95%CI 0.027, 0.208], P=0.0039), but not HDL-cholesterol R^2_{adj}=0.091 [95%CI 0.030, 0.231], P=0.0794) after adjustment for sex and age. Including BMI in the multivariate regression analyses did not change the model (data not shown).

Discussion

Our primary finding was that obese men and women seeking treatment spent about 26 min in moderate intensity PA corresponding to brisk walking or jogging (PAR >6) per day. Futhermore, nearly 6 h were spent on strolling and walking and nearly 18 h per day lying down, standing and sitting. The time spent at moderate intensity PA (PAR >6) was inversely associated with serum total cholesterol and triglycerides explaining 6% and 8%, respectively, of the variation in these lipid levels after adjustment for age and sex.

Unexpectedly, we found that these obese subjects with high risk for CVD due to metabolic syndrome risk factors were moderately active even before formal advice to increase physical activity was given as part of a weight reduction program. One-third of the subjects recorded more than 30 min of moderate activity per day. The mean of 26 min per day spent on activities such as brisk walking or jogging was close to the recommendations of the American College of Sports Medicine and American Heart Association. [11] Moreover, the mean overall PAL of 1.7 is in accordance with the mean PAL observed earlier among obese subjects. [21] The intensity of PAR > 6 corresponds with a walking speed above 2.7 mph, (about 3 metabolic equivalents), and is considered to be moderate intensity PA. [11, 22] However, the mean time spent on moderate PA was about half of the time that may be needed to reduce cardiovascular risk [23] and to manage body weight. [24] In the Womens' Health Initiative Observational Study [23], subjects in the highest quintile of PA walked about 60 min per day at a speed corresponding to a PAR >6. This amount of physical activity was associated with a reduction in the risk of cardiovascular disease by about 30% compared to the women in the lowest quintile. [23] Obese men that spent 60 min per day on brisk walking have achieved about a 7% reduction in body weight. [25] Likewise, the Studies of Targeted Risk Reduction Interventions through Defined Exercise showed that PA corresponding to 2.4-3.0 miles (4-5 km) of daily walking had salutary effects on body weight.[26] Assuming a speed of 2.7 mph

(4.5 km/h), our subjects would have had to walk for 60 min per day to reach this level of activity. Adherence to 60 min or more of PA may be a challenge. A recent study showed that only 12 of 202 subjects maintained a daily PA level of >60 min for 18 months. [27] However, moderate weight reduction of about 7-10% as suggested for treatment of the metabolic syndrome [4] may be achievable with individualized advice to slightly increase the level of PA to the minimum of 30 min of moderate intensity PA five days a week in a program including dietary and behavioral therapy.

Intervention studies have shown that increased physical activity alone or in combination with change in diet and weight loss improves most metabolic risk factors, in particular those associated with the metabolic syndrome. [5, 6, 28, 29, 30, 31] A recent meta-analysis quantified the effect of exercise for the purpose of weight loss on diastolic blood pressure (-0.2 mm Hg), triglycerides (-0.2 mmol/L) and fasting glucose (-2 mmol/L) but did not find an effect of exercise on total cholesterol and HDL cholesterol. [31] However, data on the effects of physical activity on metabolic risk factors in very obese individuals is sparse. [26, 31] In line with the meta-analysis we found inverse associations between time spent at moderate intensity PA (PAR >6) and serum concentrations of triglycerides. The time spent at PAR >6 were also inversely correlated with serum total cholesterol concentrations. Unexpectedly HDL cholesterol was inversely associated with time spent at PAR >6. The amount of PA in our study may have been insufficient to increase HDL cholesterol [29] or exercise may vary in its effects on lipids in very obese subjects. A relatively high heritability factor has been reported for the change in HDL cholesterol after endurance training. [32]

The study has several limitations. The sample size was limited and may explain the lack of association between blood presure and serum glucose concentrations and physical activity. Moreover, in a cross-sectional study as the present one, causality cannot be determined. We measured PA for only 4 days to assure compliance. This amount of recording

is consistent with the 3-5 days that are recommended for PA measurements in adults, [33] however, recording for a longer period may have given a better estimate of usual PA. The subjects may have changed their activity pattern during this short period of time.

Conclusions

We observed that very obese subjects with metabolic risk factors engaged in close to the recommended amount of moderate physical activity before weight loss interventions were initiated. This activity was associated with lower lipid levels, but intervention to increase this level of PA may be needed to achieve weight loss.

Acknowledgement

We thank Tor Ole Klemsdal for some of the medical examinations and Ingar Holme for statistical assistance.

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Table 1 Characteristics of the subjects

Variables	Women (N=68)	Men (N=62)	P[1]
Age (y)	44±10	48±10	0.0640
Height (m)	1.7±0.1	1.8±0.1	<0.0001
Weight (kg)	105.3±13.4	114.5±15.0	0.0003
Body mass index (in; kg/m^2)	37.6±4.4	35.4±3.8	0.0029
Waist (cm)	109.8±10.3	119.1±10.2	<0.0001
Total body fat mass (kg)	56.1±13.8	47.9±13.1	0.0008
Trunk fat mass (kg)	29.3±7.7	29.4±8.6	0.9276
Lean body mass (kg)	47.8±5.1	65.1±7.6	<0.0001
Systolic blood pressure (mmHg)	133±17	139±18	0.0446
Diastolic blood pressure (mmHg)	85±10	89±11	0.0521
Glucose (mmol/L)	5.9±1.8	6.0±1.7	0.6681
Total cholesterol (mmol/L)	6.1±1.1	6.1±1.1	0.8178
HDL cholesterol (mmol/L)	1.2±0.3	1.1±0.2	0.0069
Triglycerides (mmol/L)	2.1±0.8	2.6±1.7	0.2078

NOTE. Values are means and standard deviations.

[1]Unpaired t-test and the Mann-Whitney two sample rank test were used to examine differences between sexes.

Table 2 Energy expenditure and physical activity patterns

Values	Women (N=68)	Men (N=62)	P[1]
Resting metabolic rate (MJ/d)	7.2±0.7	8.5±0.8	<0.0001
Total energy expenditure (MJ/d)	12.5±1.8	14.8±2.4	<0.0001
Physical activity level	1.7±0.2	1.7±0.2	0.9638
Physical activity ratio 1.0-1.5 (min/d)[2]	1070±113	1060±101	0.6098
Physical activity ratio 1.6-6.0 (min/d)[2]	347±107	349±92	0.7779
Physical activity ratio >6.0 (min/d)[2]	25±17	27±17	0.3512

NOTE. Values are means and standard deviations.

[1] Unpaired t-test and the Mann-Whitney two sample rank test were used to examine differences between sexes.

[2] Physical activity ratio 1.0-1.5 corresponds to laying, standing and sitting; physical activity ratio 1.6-6.0 corresponds to strolling and walking with a speed of 0.6-2.7 mph (1.0-4.5 km/h) and physical activity ratio >6.0 corresponds to brisk walking or jogging with a speed of >2.7 mph (4.5 km/h).

Table 3 Correlations between metabolic risk factors and minutes spent at physical activity ratio>6[1] in all subjects (N=130)

Cardiovascular risk factors	Correlation coefficient[2]	P	95% CI
Waist (cm)	-0.08	0.402	-0.25, 1.00
Fat trunk (kg)	-0.03	0.782	-0.20, 0.15
Diastolic blood pressure (mmHg)	-0.11	0.214	-0.28, 0.06
Systolic blood pressure (mmHg)	-0.06	0.499	-0.23, 0.11
Serum total cholesterol (mmol/L)	-0.23	0.002	-0.42, -0.10
Serum HDL-cholesterol (mmol/L)	-0.18	0.042	-0.34, -0.07
Serum triglycerides (mmol/L)[3]	-0.26	0.003	-0.41, -0.09
Serum glucose (mmol/L)[4]	-0.07	0.456	-0.25, 0.12

[1]Physical activity ratio >6 corresponds to brisk walking or jogging with a speed of >2.7 mph (4.5 km/h).

[2] Pearson's correlation. Min spent at PAR>6 and concentrations of serum triglycerides were skewed variables and log transformed. Because one subject did not record any min at PAR>6 the variable (ln (min PAR>6 + 1 min)) was used in the correlation analyses.

[3] 1 man was missing value for serum triglycerides.

[4] 7 women and 9 men with diabetes type 2 were excluded from the analysis. 1 man was missing value for serum glucose.

IV

European Journal of Clinical Nutrition (2007) 61, 1301–1311

0954-3007/07 $30.00
www.nature.com/ejcn

ORIGINAL ARTICLE

The effect of an increased intake of vegetables and fruit on weight loss, blood pressure and antioxidant defense in subjects with sleep related breathing disorders

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Objective: To assess the effect of an increased consumption of vegetables and fruit on body weight, risk factors for cardiovascular disease (CVD) and antioxidant defense in obese patients with sleep-related breathing disorders (SRBD).
Design: Randomized, controlled trial of an intervention to increase the intake of vegetables to 400 g/day and fruit to 300 g/day. Dietary intake was calculated from a food frequency questionnaire. Antioxidant status was assessed with the ferric-reducing/antioxidant power (FRAP) assay. Plasma carotenoids were biomarkers for the intake of vegetables and fruit.
Setting: A hospital clinic preventing risk factors for CVD.
Subjects: Subjects were 103 men and 35 women with a body mass index of $36.7 \pm 5.8\,kg/m^2$ of which 57 (86%) in the control and 68 (94%) in the intervention group completed the study.
Intervention: Group-based behavioral program during 3 months.
Results: The mean between group differences in body weight was −2.0% (95% CI −3.6, −0.5), $P<0.0001$. The mean between group difference in systolic and diastolic blood pressure (BP) was −7.1 mm Hg (95% CI: −11.6, −2.6), $P=0.0022$ and −3.9 mm Hg (95% CI: −7.0, −0.9), $P=0.0120$, respectively. The mean change in daily intake of vegetables and fruit was 12 g (95% CI: −33, 57) and −4 g (95% CI: −79, 71) versus 245 g (95% CI: 194, 296) and 248 g (95% CI: 176, 320) in the control and intervention groups, respectively. This was reflected in higher concentrations of α-carotene and β-carotene. No change in FRAP was seen. In a multiple regression analysis the change in intake of vegetables was a significant contributor ($R^2_{adj}=0.073$ (95% CI: 0.019, 0.214)) to the change in weight.
Conclusion: Targeted dietary advice to increase the intake of vegetables and fruit among subjects with SRBD contributed to weight reduction and reduced systolic and diastolic BP, but had no effect on antioxidant defense measured with FRAP.
Sponsorship: None.
European Journal of Clinical Nutrition (2007) **61,** 1301–1311; doi:10.1038/sj.ejcn.1602652; published online 31 January 2007

Keywords: sleep apnea; treatment; dietary advice; risk reduction; carotenoids; FRAP (ferric reducing/antioxidant power)

Introduction

Sleep-related breathing disorders (SRBD), including habitual snoring, increased upper airway resistance syndrome and obstructive sleep apnea (OSA) are increasingly common causes of morbidity and mortality among overweight and obese individuals. It is estimated that one of five white adults has at least mild disease and one of 15 has at least moderate disease (Caples *et al*., 2005). There is a graded increase in OSA prevalence with increasing body mass index (BMI) and waist to hip ratio. Observational studies indicate that a decrease in body weight of 10% is associated with improvement in the apnea–hypoapnea index (AHI), a widely used measure of the frequency of disordered breathing events (Peppard *et al*., 2000). Thus, weight loss is universally recommended to overweight or obese patients with SRBD. However, the response to weight loss is variable, weight loss is difficult to maintain long-term and SRBD may recur even after

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Received 3 July 2006; revised 7 December 2006; accepted 12 December 2006; published online 31 January 2007

surgically induced weight loss (Pillar *et al*., 1994; Strobel and Rosen, 1996). Furthermore, studies of weight loss are severely deficient in regard to their design and follow-up (Strobel and Rosen, 1996; Shneerson and Wright, 2001). As a result, the use of continuous positive airway pressure (CPAP), which appears to be the most effective therapy for symptoms of OSA, has become the treatment of choice. Treatment with CPAP may additionally cause weight loss (Loube *et al*., 1997).

SRBD have been associated with hypertension and an increased risk of cardiovascular disease (CVD) in clinical and epidemiological reports for nearly three decades. This association is confounded by other comorbid conditions, most notably obesity, and to date the question of whether SRBD independently cause CVD remains controversial (Lavie, 2004; Stradling, 2004). Although the exact mechanisms of any atherogenic effects of SRBD have not been established, a number of hypotheses have been proposed. For example, increased sympathetic activity, endothelial dysfunction and inflammatory processes have been suggested to act as mediators of cardiovascular morbidity in patients with SRBD and have been shown to be associated with SRBD in clinical studies. Thus, patients with SRBD may exhibit evidence of sympathetic overactivity and increased oxidative stress, insulin resistance and a prothrombotic state (Shamsuzzaman *et al*., 2002; Fletcher, 2003; Lavie, 2003; Punjabi *et al*., 2004). Most convincing in this regard is evidence that treatment with CPAP reduces blood pressure (BP), at least in the short term (Becker *et al*., 2003).

Alhough the exact independent relation of SRBD to CVD is still not clear, there is widespread acceptance of the idea that patients with SRBD are at high risk of CVD because of concomitant factors, including the pervasiveness of male gender, age, central obesity, hypertension, diabetes and dyslipidemia (Newman *et al*., 2001). Dietary modification is a primary cornerstone of lifestyle changes that aim to prevent CVD, even though weight reduction sufficient to reduce the need of CPAP is probably very difficult to achieve in the long term. A diet rich in vegetables and fruits has been consistently associated with a reduced risk of cardiovascular disease (Dauchet *et al*., 2006), and is widely recommended to facilitate weight reduction (Krauss *et al*., 1996). The feasibility and efficacy of such a diet with the aim of CVD risk factor reduction in patients with SRBD has not been previously studied, to our knowledge. The specific purpose of this study was to test the effects of dietary advice given as a behavioral, group session-based intervention aimed to increase the consumption of vegetables and fruit compared with a single session of dietary advice, in free living overweight or obese subjects with SRBD. The primary outcome was weight change from baseline to 3 months. Changes in established risk factors for CVD including BP, lipids and glucose at 3 months were secondary outcomes. The third outcome was change in oxidative defense measured with ferric reducing/antioxidant power (FRAP) assays at 3 months.

Subjects and methods

Subjects

Participants included 103 men and 35 women diagnosed with SRBD and a mean $\pm$s.d. age and BMI of 48.2 ± 9.0 year and $36.7 \pm 5.8\,kg/m^2$, respectively. The Ear, Nose and Throat department at the hospital or primary care physicians referred the subjects to the Department of Preventive Cardiology at Ullevål University Hospital for weight reduction. The diagnosis was verified during polysomnography in a sleep laboratory, mostly done at the Ullevål University Hospital. Of the total, 64 subjects had severe OSA (AHI >30 mean episodes per hour of sleep), 34 subjects had moderate OSA (AHI 15–30), 23 subjects had mild OSA (AHI 5–15), five subjects only had SRBD (AHI <5) and AHI could not be calculated for one subject after the polysomnography owing to technical problems, but the oxygen saturation was 85% and the sleep pattern was in accordance with OSA. The actual AHI measurement could not be located for 11 subjects owing to moving of the Ear, Nose and Throat department to another location. Of the total, 93 subjects were treated with therapeutic or subtherapeutic continuous positive airways pressure CPAP and eight had undergone surgical treatments. No significant difference in AHI was seen between subjects treated with CPAP or who had undergone surgical treatment and subjects without treatment (32.3 ± 25.7 versus 39.8 ± 27.7, $P = 0.1640$).

Study design

All participants underwent baseline examinations for eligibility, including a medical history and physical examination as well as CVD risk factor assessment (including cardiovascular risk factors, BP and blood chemistry). Inclusion criteria were men and women between the ages of 21 and 72 with BMI $\geqslant 27\,kg/m^2$ and diagnosis of OSAS made during polysomnography in a sleep laboratory and based on standard criteria. Exclusion criteria were suspected non-compliance because of drug or alcohol abuse or lack of motivation, participating in another trial, any major non-cardiac disease expected to reduce life expectancy or interfere with the study and the use of appetite suppressants or weight-reducing medication within the last 3 months. The Ethical Committee (region 1) in Norway approved the protocol and all participants provided written informed consent. The study was conducted between May 2000 and December 2003.

The study design was a randomized, parallel group controlled comparison of a group-based nutritional and cognitive-behavioral intervention aimed to increase the intake of vegetables and fruit and simple dietary advice given only at baseline (control). The dietitian opened sealed, consecutively numbered, opaque envelopes containing the randomization number and group assignment (control or intervention). The dietitian then allocated the next available number to each screened and eligible subject. The randomization list was based on a table of random numbers set up by

an investigator, who had no contact with the subjects during the study period. Before randomization, all participants were given written individualized dietary advice aimed to achieve a 5% weight reduction. The advice was based on the subjects' usual diet assessed with a dietary interview based on a food frequency questionnaire (FFQ) as described below.

The control group
After the randomization, the participants in the control group were not given further dietary advice or counseling on behavior change. The subjects had no further contact with the dietitian until the 3 months examination.

The intervention group
Participants randomized to the intervention group were consecutively assigned to six groups with 10–18 participants in each group. Because of holidays, the meeting frequency for each group during the 3-month period was a minimum of six and a maximum of 10 sessions.

The primary goal of the intervention was to increase the consumption of vegetables (to at least 400 g/day) and fruit (to at least 300 g/day). The subjects were told the weights of various vegetables and fruit and encouraged to use a scaled weight at home to become familiar with the amount of vegetables and fruit that was necessary for goal achievement. Participants were instructed to use a plate model for hot meals and advised to fill one-half of the plate with vegetables, one-fourth with potatoes, rice or pasta and one-fourth with meat, chicken, fish and/or legumes. When the plate model was used, the amount of vegetables increased and the portion size of meat and/or potatoes, rice and pasta decreased. Substitution of vegetables and fruit for high-fat, high-energy food was otherwise emphasized. The dietary sessions focused on how to implement an increased intake of vegetables and fruit in daily life settings and on problem solving issues. Recipes and tips to encourage compliance were provided.

Dietary assessments
The subjects participated in a dietary interview based on a FFQ before randomization and at the 3-month examination. The interview was done with a registered dietitian and lasted between 1 and 2 h. The FFQ was designed to assess the food intake during the last 3 months and has been validated and described in details elsewhere (Svendsen and Tonstad, 2006). In short, the questionnaire elicited frequencies and consumption of 174 food items (of these 28 vegetables and 29 fruits and berries). Portion sizes were estimated with the use of a photographic atlas, photographs, ordinary food items and standardized units. The FFQ was coded manually for the calculation of total energy, energy yielding nutrients and food items using a software program (Mat på data 3.0, 1996) based on the Norwegian food composition table (Rimestad *et al*., 1995). Two subjects in the control group and one subject in the intervention group did not take part in the interview at the 3-month visit but completed the other examinations.

Use of supplements was assessed in the dietary interview. Fifteen subjects reported daily use of low-dose multivitamin supplements and two subjects used high-dose multivitamin supplements at baseline. As intake of vitamins and minerals from supplements was low, we did not include supplements in the calculated dietary intake or in the statistical analyses.

Anthropometry and blood pressure measurements
Subjects were weighed (in underwear) with a digital weight (Seca, Germany) to the nearest 0.1 kg. Height was measured with a standardized wall measuring stick scale to the nearest 0.5 cm. Waist circumference was measured at the umbilicus with the subject unclothed and in the standing position and hip circumference was measured at the greater trochanter.

After the subjects sat quietly for 5 min, BP was measured automatically in the right arm with a digital BP monitor (OMRON Hem-705 CP) and an appropriately sized cuff.

Laboratory measurements
Fasting (10 h overnight) blood samples were collected at baseline and after 3 months. All samples except those for the analysis of lipids and glucose were kept frozen at $-70^{\circ}C$ for batch analyzes of the variables. Serum lipids and glucose were analyzed with conventional enzymatic methods and folate was analyzed with a chemoluminescence detection technology method (Advia Centaur immunoassay, Bayer AG, Leverkusen, Germany).

FRAP was determined as described elsewhere (Benzie and Strain, 1999). The carotenoids lutein, zeaxanthin, β-krytpoxanthin, α-carotene, β-carotene and lycopene were determined in plasma by high-performance liquid chromatography. Proteins were precipitated and removed by the addition of a 4.5 volume of isopropanol followed by centrifugation at 3000 g at $4^{\circ}C$ for 15 min. The internal standard astaxanthin was added with the isopropanol and 25 μl of the clear supernatant were used for analysis. The mobile phases consisted of A: 20% water and 24% acetone in ethanol and B: acetone. The gradient conditions were as follows: From 2 to 100% B within 20 min, followed by 100% B for 15 min. Detection was performed at 453 nm using a variable wavelength detector. Plasma calibrators quantified against the National Institute of Standards and Technology and 968c standard reference material were used as standards.

Values of the laboratory parameters (lipids and glucose) were missing for 1–5 in the control group and 2–4 subjects in the intervention group. Values of carotenoids and FRAP were missing for eight subjects and three subjects in the control and intervention group, respectively. Values of serum folate were missing for 13 subjects in the control group and 13 subjects in the intervention group. In all cases missing samples were due to insufficient material for analysis.

Statistical analysis

The results are presented as means ± s.d., means (95% CI) or median and 25th, 75th percentiles. Mean differences within and between groups were tested with paired- and unpaired *t*-tests, respectively. Differences between the groups for skewed variables were tested with the Mann–Whitney two-sample rank test. The χ^2 test was used in comparisons of categorical variables. Changes were calculated as the difference between 3 months and baseline. Energy density was calculated for the whole diet minus all drinks (coffee, tea, milk, juice, soft drinks and alcoholic beverages).

The association between single, continuous variables was explored by Pearson or Spearman correlations as appropriate for normally distributed or skewed variables. We conducted multiple regression analyses to identify factors that determined weight change in all subjects as follows. To test for group homogeneity, we did a multiple regression analysis including the exposure factor (x_1), treatment variable (x_2) and the product variable of x_1 and x_2. When the product variable was found to be significant in the multiple regression analysis, the slopes of association were claimed to be different between treatment groups. In such a case simple regression analysis between change in weight and the exposure factor was done within each treatment group. Group homogeneity was tested for the presence of diabetes or CVD, use of antihypertensive or lipid-lowering medication, smoking habits and gender. The tests for homogeneity showed heterogeneity between the control and intervention group only in regard to the use of antihypertensive medication ($P=0.0165$). Because of the heterogeneity, use of antihypertensive medication was not included in the final multiple regression analysis.

The tests were considered significant at $P<0.05$. Statistical analyses were performed using the Stat View 5.0.1 software (Abacus concepts, Berkeley, CA, USA).

Results

A total of 125 subjects participated in the 3-month examination. There were nine dropouts in the control group and four in the intervention group. In the control group three subjects withdrew because of a high workload, three subjects did not show up after three reminders, one subject wanted another treatment, one subject was disappointed with the treatment and one moved to another city. In the intervention group one subject withdrew because of a divorce, one because of a hip operation, one because of alcohol abuse and one quit attending the group sessions and did not show up at the 3-month examination after three reminders.

The ranges for age and BMI were 28–72 years and 27.2–55.4 kg/m^2, respectively. Other characteristics of participants are shown in Table 1. There was no statistically significant difference in BMI between the groups.

At baseline, there were no statistically significant differences in weight, BP, serum concentrations of lipids, glucose and FRAP or dietary intake of vegetables and fruit between the groups (Tables 2 and 3). However, serum folate was higher in the intervention group.

Outcome effects

The mean ± s.d. % weight loss was 0.9 ± 4.3% in the control group and 3.0 ± 4.6% in the intervention group ($P<0.0001$). In the control group eight subjects (14%) achieved a weight loss of ⩾5% compared to 21 subjects (31%) in the intervention group ($P=0.01$).

Systolic and diastolic BP was reduced in the intervention group (Table 2). The mean change in systolic BP was 2.7% (95% CI: −0.3, 5.7) versus −3.2% (95% CI: −5.5, −1.0) and the mean change in diastolic BP was 1.3% (95% CI: −1.7, 4.3) versus −3.5% (95% CI: −5.8, −1.2) in the control group

Table 1 Baseline characteristics of participants in the control and intervention group

	Control group (n = 57)	*Intervention group (n = 68)*
Gender		
Male, *n* (%)	42 (74)	53 (78)
Female, *n* (%)	15 (26)	15 (22)
Age (years)	49.2 ± 10.3[a]	47.1 ± 7.5
Cardiovascular disease, *n* (%)	7 (12)	9 (13)
Diabetes mellitus, *n* (%)	3 (5)	8 (12)
Hypertensive[b], *n* (%)	31 (54)	38 (59)
Antihypertensive medication, *n* (%)	18 (32)	18 (26)
Lipid lowering medication, *n* (%)	11 (19)	15 (22)
Smokers, *n* (%)	19 (33)	17 (25)
CPAP[c] treatment, *n* (%)	34 (60)	43 (63)
Height (m)		
Male	1.79 ± 0.06	1.81 ± 0.06
Female	1.66 ± 0.07	1.64 ± 0.06
Weight (kg)		
Male	120.0 ± 23.7	115.5 ± 17.6
Female	103.2 ± 21.5	97.2 ± 16.3
BMI (kg/m^2)		
Male	37.5 ± 6.4	35.5 ± 5.1
Female	37.6 ± 7.5	36.0 ± 5.5
Waist (cm)		
Male	124 ± 16	119 ± 12
Female	113 ± 12	107 ± 14
Hip (cm)		
Male	114 ± 12	111 ± 9
Female	114 ± 12	116 ± 11
Waist-to-hip ratio	1.1 ± 0.1	1.0 ± 0.1

Abbreviations: BMI, body mass index; CPAP, continuous positive airway pressure.
[a]$\bar{\chi}$ ± s.d. (all such values).
[b]Blood pressure ⩾140/90 mm Hg or current uses of antihypertensive medications.

Table 2 Intervention outcomes at baseline and at 3 months

	Control group (n = 57)	P[a]	*Intervention group (n = 68)*	P[a]	P[b]
Weight (kg)					
Baseline	115.6±24.2[c]	—	111.5±18.8	—	0.2867
3 months	114.7±25.2	—	108.1±18.0	—	—
Change	−0.9±4.8	0.1425	−3.4±5.3	<0.0001	0.0074
Waist (cm)					
Baseline	121±16	—	116±14	—	0.0601
3 months	120±18	—	113±14	—	—
Change	−1.6±5.5	0.0365	−3.0±5.4	<0.0001	0.1541
Systolic blood pressure (mm Hg)					
Baseline	127±18	—	130±14	—	0.2286
3 months	130±18	—	126±16	—	—
Change	2.7±13.0	0.1278	−4.4±12.1	0.0044	0.0022
Diastolic blood pressure (mm Hg)					
Baseline	81±10	—	84±9	—	0.1692
3 months	82±11	—	80±9	—	—
Change	0.6±8.6	0.5843	−3.3±8.3	0.0019	0.0120
Total cholesterol (mmol/l)					
Baseline	5.5±1.0	—	5.6±1.2	—	0.6390
3 months	5.4±1.1	—	5.4±1.2	—	—
Change	−0.0±0.8	0.7540	−0.2±0.8	0.0660	0.3219
HDL cholesterol (mmol/l)					
Baseline	1.2±0.2	—	1.2±0.2	—	0.7216
3 months	1.2±0.2	—	1.2±0.2	—	—
Change	0.0±0.1	0.5692	0.0±0.1	0.1430	0.5853
Triacylglycerols (mmol/l)					
Baseline	2.1±0.1	—	2.4±2.0	—	0.3394
3 months	2.2±1.2	—	2.0±1.5	—	—
Change	0.1±1.1	0.7519	−0.4±1.4	0.0260	0.0638
Glucose (mmol/l)					
Baseline	6.1±1.1	—	5.9±2.1	—	0.6520
3 months	6.0±1.3	—	5.8±2.3	—	—
Change	−0.1±1.1	0.6587	−0.2±1.6	0.3905	0.6887
Serum folate (nmol/l)					
Baseline	12.7±5.5	—	16.3±9.1	—	0.0209
3 months	13.6±6.0	—	16.9±7.7	—	—
Change	0.9±5.1	0.2508	0.6±6.2	0.4792	0.8021
FRAP[d] (μmol/l)					
Baseline	1369±209	—	1368±235	—	0.9835
3 months	1347±221	—	1376±243	—	—
Change	−22±111	0.1692	7±203	0.7702	0.3596

[a]Differences between baseline and 3 months within the control group and the intervention group (paired *t*-test).
[b]Differences between the control group and the intervention group (unpaired *t*-test).
[c]$\bar{\chi}$±s.d. (all such values).
[d]FRAP, ferric reducing/antioxidant power.

and intervention group, respectively. The between-group difference in BP remained significant after adjustment for baseline BP and BMI (mean change systolic BP: −6.6 mm Hg (95% CI: −11.0, −2.2) and diastolic BP: -3.6 mm Hg (95% CI: −6.4, −0.8)).

No differences in lipid or glucose concentrations in between-group comparisons were seen, neither did we see any statistically significant difference in antioxidant defense measured with FRAP within or between the groups (Table 2).

Effects of intervention on diet and serum biomarkers

The intake of vegetables and fruit (including berries and juice) was doubled in the intervention group which was a

Table 3 Intervention outcomes for vegetables, fruit and nutrients at baseline and 3 months

	Control group (n = 57)	*Intervention group (n = 68)*	P[a]
Vegetables (g/day)			
Baseline	226 ± 178[b]	223 ± 159	0.9717
3 months	238 ± 144	457 ± 240	
Change	12 (−33, 57)[c]	245 (194, 296)	<0.0001
Fruit, juice and berries (g/day)			
Baseline	327 ± 266	242 ± 199	0.0626
3 months	322 ± 258	486 ± 285	
Change	−4 (−79, 71)	248 (176, 320)	<0.0001
Energy (kJ/day)			
Baseline	11334 ± 4220	10461 ± 3943	0.2322
3 months	9875 ± 3114	8982 ± 2243	
Change	−1563 (−2216, −910)	−1463 (−2300, −626)	0.8499
Energy density (kJ/g)[d]			
Baseline	6.62 ± 1.25	6.63 ± 1.48	0.8081
3 months	6.29 ± 1.43	4.91 ± 1.14	
Change	−0.33 (−0.64, −0−01)	−1.76 (−2.08, −1.44)	<0.0001
Protein (% of energy)			
Baseline	16.3 ± 2.8	16.7 ± 2.6	0.4118
3 months	16.6 ± 3.1	17.4 ± 2.6	
Change	0.4 (−0.5, 1.3)	0.6 (−0.2, 1.5)	0.6978
Fat (% of energy)			
Baseline	34.0 ± 5.6	33.4 ± 7.0	0.5997
3 months	32.2 ± 5.6	28.0 ± 5.9	
Change	−1.8 (−3.5, −0.1)	−5.2 (−7.1, −3.4)	0.0078
Saturated fat (% of energy)			
Baseline	12.8 ± 3.1	12.4 ± 3.1	0.4505
3 months	11.8 ± 2.9	8.9 ± 2.9	
Change	−0.9 (−1.7, −0.2)	−3.4 (−4.2, −2.5)	<0.0001
Carbohydrate (% of energy)			
Baseline	45.3 ± 6.4	44.8 ± 7.1	0.6682
3 months	47.3 ± 6.0	49.6 ± 6.3	
Change	1.8 (0.2, 3.3)	4.8 (3.2, 6.4)	0.0091
Alcohol (% of energy)			
Baseline	3.2 ± 4.4	3.9 ± 4.0	0.3717
3 months	3.0 ± 3.6	3.9 ± 4.5	
Change	−0.13 (−0.93, 0.66)	−0.03 (−0.10, 0.63)	0.8432
Fiber (g/day)			
Baseline	30 ± 12	27 ± 12	0.1762
3 months	29 ± 9	36 ± 12	
Change	−2 (−5, 1)	9 (6, 13)	<0.0001
Cholesterol (mg/day)			
Baseline	337 ± 133	304 ± 125	0.1547
3 months	292 ± 122	239 ± 95	
Change	−46 (−75, −18)	−65 (−94, −36)	0.3631
Sodium (mg/day)			
Baseline	3593 ± 1674	3485 ± 1278	0.6847
3 months	3065 ± 1182	2801 ± 909	
Change	−543 (−861, −225)	−609 (−1070, −380)	0.5208
Potassium (mg/day)			
Baseline	4751 ± 1591	4309 ± 1477	0.1358
3 months	4339 ± 1283	4904 ± 1126	
Change	−422 (−749, −95)	642 (337, 947)	<0.0001
Calcium (mg/day)			
Baseline	1134 ± 584	1015 ± 509	0.2258
3 months	999 ± 414	900 ± 463	
Change	−138 (−255, −21)	−103 (−221, 15)	0.6763
Magnesium (mg/day)			
Baseline	474 ± 170	437 ± 144	0.1899
3 months	443 ± 142	459 ± 108	
Change	−35 (−64, 6−)	25 (−5, 55)	0.0063
α-tocopherol (mg/day)			
Baseline	9.5 ± 3.9	8.0 ± 3.3	0.0223
3 months	8.7 ± 3.5	9.0 ± 3.3	
Change	−0.9 (−1.9, 0.1)	1.1 (0.1, 2.1)	0.0065
Vitamin C (mg/day)			
Baseline	215 ± 438	130 ± 76	0.1128
3 months	151 ± 83	240 ± 107	
Change	−68 (−190, 53)	113 (85, 141)	0.0019
Folate (μg/day)			
Baseline	303 ± 115	272 ± 101	0.1053
3 months	287 ± 102	337 ± 101	
Change	−20 (−49, 8)	71 (42, 100)	<0.0001
β-Carotene (μg/day)			
Baseline	2996 ± 1870	3037 ± 2281	0.9156
3 months	3600 ± 2729	7086 ± 4528	
Change	588 (−81, 1257)	4140 (2948, 5334)	<0.0001

Abbreviation: CI, confidence interval.
[a]Differences between the control group and the intervention group (unpaired *t*-test).
[b]$\bar{\chi}$ ± s.d. (all such values).
[c]$\bar{\chi}$; 95% CI in parentheses (all such values).
[d]Energy density was calculated for the whole diet minus tea, coffee, water and soft drinks.

significant increase compared to controls and resulted in lower energy density of the diet, a reduction in energy percent from total and saturated fat, higher intake of energy percent from carbohydrate and higher intake of fiber, potassium, magnesium, α-tocopherol, Vitamin C, folate and β-carotene (Table 3). No significant change was seen in the intake of calcium within the intervention group. However, the dietary intake of diary products was decreased (−62 g (95% CI −143, 19) and −77 g (95% CI −151, −3), $P = 0.8023$ in the control and intervention group, respectively).

The increased intake of vegetables and fruit was reflected in increased plasma concentrations of α- and β-carotene in between-group comparisons (Table 4). The mean between-group difference of the percentage change in serum levels of α-carotene, β-carotene, β-cryptoxanthin, lycopene, lutein and zeaxanthin were 166.2% (95% CI 59.5, 273.0), 61.3% (95% CI 31.5, 91.1), 47.2% (95% CI 7.2, 87.86), 56.1% (95% CI −29.9, 142.0), 21.0% (95% CI −7.1, 49.9) and −3.0% (95% CI −65.7, 59.7), respectively.

Table 4 Changes in plasma carotenoid values from baseline to 3 months in the control and intervention group

	Control group (n = 49)	*Intervention group (n = 65)*	P[a]
Lutein (μM)	−0.017 (−0.039, 0.005)[b]	0.005 (−0.013, 0.023)	0.0555
Zeaxanthin (μM)	−0.010 (−0.020, 0.005)	−0.002 (−0.062, 0.058)	0.0844
β-Cryptoxanthin (μM)	0.026 (−0.004, 0.056)	0.055 (0.021, 0.089)	0.0516
α-Carotene (μM)	−0.002 (−0.016, 0.020)	0.046 (0.026, 0.066)	0.0023
β-Carotene (μM)	−0.029 (−0.077, 0.019)	0.086 (0.036, 0.150)	0.0031
Lycopene (μM)	0.014 (−0.004, 0.068)	0.073 (0.009, 0.137)	0.3015

Abbreviation: CI, confidence interval.
[a]Differences between groups (the Mann–Whitney two sample rank test).
[b]$\bar{\chi}$; 95% CI in parentheses (all such values). Eight subjects in the control group and three in the intervention group were missing values.

Table 5 Correlations between antioxidant markers and change in intakes of vegetables and fruit between baseline and 3 months and intakes at 3 months in the intervention group

	Changes in intake (n = 64)[a]				*Intake at 3 mo (n = 65)*[b]			
	Vegetables		*Fruit*		*Vegetables*		*Fruit*	
	R[c]	P	r	P	r	P	r	P
Lutein	0.190	0.1318	0.094	0.4569	−0.001	0.9971	0.045	0.7208
Zeaxanthin	−0.096	0.9609	0.023	0.8532	−0.127	0.3105	−0.016	0.8996
β-Kryptoxanthin	0.108	0.3926	−0.041	0.7442	0.203	0.1038	0.270	0.0308
β-Carotene	0.072	0.5688	−0.018	0.8887	0.160	0.2010	0.233	0.0620
α-Carotene	0.323	0.0103	0.153	0.2238	0.213	0.0887	0.426	0.0006
Lycopene	0.038	0.7626	−0.004	0.9722	0.085	0.4973	−0.124	0.3219
FRAP[d]	0.135	0.2831	0.060	0.6322	−0.083	0.5092	0.001	0.9933

[a]One subject was missing dietary data and three subjects were missing serum values for carotenoids and FRAP.
[b]One subject was missing dietary data and two subjects were missing serum values for carotenoids and FRAP.
[c]Spearmann correlations (all such values).
[d]FRAP, the ferric reducing/antioxidant power.

At 3 months, nine subjects (16%) in the control group and 37 subjects (55%) in the intervention group reported an intake of ⩾400 g/day of vegetables ($P = 0.001$). Furthermore, 27 subjects (49%) in the control group and 46 subjects (69%) in the intervention reported an intake of fruit ⩾300 g/day ($P = 0.05$). Compared to controls, the increased intakes of vegetables and fruit in the intervention group were also reflected in higher calculated intakes of β-carotene and folate (Table 3).

Correlation analyses

Correlation analyses conducted among all subjects showed that changes in intake of vegetables and fruit were positively correlated to change in body weight ($r_S = 0.284$, $P = 0.0015$ and $r_S = 0.209$, $P = 0.0207$, respectively) but not to changes in systolic or diastolic BP (data not shown). Within the intervention group, weight changes were significantly correlated to changes in systolic BP ($r = 0.304$, $P = 0.0137$) not to diastolic BP ($r = 0.232$, $P = 0.0631$).

Tables 5 and 6 show correlations between antioxidant markers (lutein, zeaxanthin, β-kryptoxanthin, β-carotene, α-carotene, lycopene and FRAP) and changes in the intake of vegetables and fruit and the correlations between antioxidant markers and the intake at 3 months in the intervention group and among all subjects. The marker that showed best correlation in all analyses was α-carotene (Tables 5 and 6). Changes in intake of β-carotene were significantly correlated to changes in serum concentrations of β-carotene ($r_S = 0.234$, $P = 0.0135$).

To explore the most important factor for weight reduction during the intervention we included change in the intake of vegetables, change in the intake of fruit and treatment group in a multiple regression analysis. As shown in Table 7 change in the intake of vegetables was the only significant factor in the model ($R^2_{adj} = 0.073$ (95% CI 0.019, 0.214)). Including CPAP did not improve the model ($R^2_{adj} = 0.062$ (95% CI 0.018, 0.210)) nor did the inclusion of age, gender and BMI at baseline ($R^2_{adj} = 0.051$ (95% CI 0.020, 0.216)).

Discussion

Our main finding was that an ∼500 g increase in the total consumption of vegetables and fruit resulted in weight loss and BP reduction in the intervention group, but did not

Table 6 Correlations between antioxidant markers and change in intakes of vegetables and fruit between baseline and 3 months and intakes at 3 months in all subjects

	Changes in intake (n = 112)[a]				*Intake at 3 months (n = 114)*[b]			
	Vegetables		*Fruit*		*Vegetables*		*Fruit*	
	r[c]	P	r	P	r	P	r	P
Lutein	0.255	0.0073	0.121	0.2016	0.270	0.0041	0.156	0.0965
Zeaxanthin	0.103	0.2776	0.138	0.1490	0.126	0.1812	0.123	0.1911
β-Kryptoxanthin	0.112	0.2366	0.067	0.4775	0.233	0.0133	0.319	0.0008
β-Carotene	0.229	0.0159	0.134	0.1581	0.315	0.0008	0.235	0.0126
α-Carotene	0.422	<0.0001	0.221	0.0198	0.399	<0.0001	0.349	0.0002
Lycopene	0.117	0.2176	0.119	0.2115	0.207	0.0280	−0.054	0.5693
FRAP[d]	0.030	0.7484	0.046	0.6270	0.063	0.5005	0.069	0.4631

Abbreviation: FRAP, the ferric reducing/antioxidant power.
[a]Three subjects were missing dietary data and 10 subjects were missing serum values for carotenoids and FRAP.
[b]Three subjects were missing dietary data and eight subjects were missing serum values for carotenoids and FRAP.
[c]Spearmann correlations (all such values).

Table 7 Multiple regression analysis of change in weight in all participants ($n = 122$)[a]

	Standardized regression coefficients	P
Changes in intake of vegetables	0.213	0.0443
Changes in intake of fruit	0.106	0.2813
Treatment	0.063	0.5565

[a]Dietary data were missing for three subjects.

influence antioxidant defense measured with FRAP. Compared to the control group, the intervention group reported a doubled intake of vegetables and fruit, a reduced intake of saturated fat and increased intake of fiber, vitamins and minerals known to lower BP.

The effects that we observed in these obese subjects with SRBD were achieved with dietary advice to increase the intake of vegetables and fruit primarily. Even though no food was provided in our study, more than 50% of the participant reported achieving the dietary goals. A design with no food provided was also used in the PREMIER clinical trial (Appel *et al*., 2003). The study lasted for 6 months and tested the effect of established recommendations for BP reduction (weight loss, sodium restriction, increased physical activity and limited alcohol intake) compared to the effect of these recommendations plus the DASH diet among adults with above optimal levels of BP. As in our study the achieved weight reduction was about 1 kg per month and the reduction in systolic and diastolic BP was 4 and 3 mm Hg, respectively. In comparison, the DASH study, which was a feeding study, showed that a diet that was high in vegetables and fruit, low-fat dairy product, nuts, fish and otherwise low in total and saturated fat was associated with highly significant reductions of 6 and 3 mm Hg in systolic and diastolic BP, respectively, after 8 weeks (Appel *et al*., 1997). Furthermore, Nowson *et al*. (2005) showed that specific targets to increase the foods used in the DASH diet resulted in a greater decrease in BP than did an ordinary low-fat diet without dietary targets (Nowson *et al*., 2005). As in the study by Nowson *et al*. (2005), we had specific targets for the intake of vegetables and fruit. Even more than seven servings of vegetables and fruit as we used in our study have been used to achieve BP reductions. For example, in the Optimal Macronutrient Intake Trial to Prevent Heart Disease trial the total servings of vegetables and fruit were nine to 11 servings per day (Appel *et al*., 2005). However, this regimen may be difficult to achieve (Weinberger, 2005). In a daily life setting in which persons have to plan, purchase and prepare their own meals, a more moderate intake of vegetables and fruit will probably be more realistic and feasible.

In contrast to the DASH study (Appel *et al*., 1997), the primary aim of our study which was performed in obese subjects with SRBD was to study the effect of increased intake of vegetables and fruit on weight reduction. We did not emphasize increasing the intake of low-fat diary products. Vegetables and fruit were recommended in exchange for other food groups. Therefore, in contrast to the DASH study we observed a small reduction in the intake of diary products in the intervention group.

The clinical relevance of the weight loss achieved in these obese subjects with SRBD is reflected in the BP reduction. In a study conducted in general practice among nearly 300 hypertensive subjects, prompt sheets for high fruit, vegetable, fiber and low fat resulted in a weight reduction of 1.2 kg during 6 months, but did not reduce blood pressure (Little *et al*., 2004). In our study, despite a 2% difference in body weight we did not see any difference in serum concentrations of lipids or glucose between the groups. Both the weight reduction and the reduced intake of total and saturated fat are expected to act favorably on concentrations of total and LDL cholesterol. In a recent controlled feeding study, the reduction in LDL cholesterol concentrations decreased as the percentage of body fat, BMI and insulin concentrations increased (Lefevre *et al*., 2005). Because our

subjects were obese, this observation may partly explain the lack of effect of weight reduction on lipids in our study. Furthermore, the amount of weight loss needed to influence lipids or glucose concentrations are thought to be at least 5% (World Health Organization (1997)).

Obese subjects and subjects with SRBD may exhibit increased oxidative stress (Schultz *et al*., 2000; Dandona *et al*., 2001; Dyugovskaya *et al*., 2002), though a recent study questioned this notion (Svatikova *et al*., 2005). After dietary restriction and weight loss a decrease in reactive oxygen species generation by leukocytes and oxidative damage to lipids, protein and amino acids has been shown (Dandona *et al*., 2001). We measured fasting serum FRAP as a measure of antioxidant defense but this parameter was largely unaffected by our intervention. Similar findings were reported in a recent dietary study by Dragsted *et al*. (2004). This study showed that markers of oxidative damage, oxidative capacity or antioxidant defense were largely unaffected by an increase in vegetables and fruit. However, the diet was not based on vegetables and fruit known to have high FRAP values. For example, blue grapes, berries, dog roses, pomegranates, Brussels sprouts, kale, red cabbage, herbs, green tea, dark chocolate, seeds, nuts and oils have high FRAP values (Halvorsen *et al*., 2002; Dragland *et al*., 2003). This may also have been the case in our study. The participants chose their own vegetables and fruit based on preferences and primarily consumed carrots, tomatoes, apples and bananas (data not shown), which are not particularly high in FRAP. In contrast, increased levels of FRAP were demonstrated in 12 obese, hypertensive subjects that followed the DASH diet (Lopes *et al*., 2003). A limitation of our study was that we did not measure postprandial FRAP. It has been proposed that food items with very high amounts of specific plant phenols may have postprandial effects (Dragsted *et al*., 2004). In contrast to a number of other studies we did not include a depletion period and this may have minimized the effects of the diet. Furthermore, baseline concentrations of FRAP were high compared to other studies (Lopes *et al*., 2003; Dragsted *et al*., 2004). Indeed, we observed a small trend toward an increase in FRAP in the intervention group, but based on these small changes, we would require approximately 500 participants in each group to achieve statistical significance (data not shown) and the clinical significance of such a small change has not been proven.

Study limitations

We included all subjects in the analyses and did not separate subjects using CPAP and subjects not using CPAP due to reduced sample size and power. However, in the multiple regression analysis, CPAP treatment was not a significant contributor to the weight change observed.

In the multivariate analysis, changes in intake of vegetables only explained 7% of the variation in weight loss. This may be owing to weight reduction, which also depends on other factors such as motivation to reach goal weight and participants expectations of weight loss (Foster *et al*., 1997). These factors were not considered in the multivariate model. Because the intervention only lasted for 3 months the data do not address the feasibility and efficacy of the dietary advice for a longer time period. Another limitation is underreporting of energy, as commonly noted in dietary studies, particularly among obese individuals (Tooze *et al*., 2004). Despite a greater weight loss in the intervention group, there was no statistically significant difference in reported energy intake at 3 months between the control and the intervention groups. The intervention group may have been more aware of their dietary intake and reported more accurately. However, the primary goal of our intervention was to increase the consumption of vegetables and fruit. Over-reporting of vegetables and fruit may have occurred in the intervention group because of social desirability bias (Tooze *et al*., 2004) and because of the large number of vegetables, fruit and berries that were included in the FFQ (about 50). The reported difference in the intake of these items was highly statistically significant between the groups and was mirrored in increased serum concentrations of lutein, β-kryptoxanthin, α-carotene and β-carotene in the intervention group. The relatively small correlation coefficients seen may be explained by the homogeneous population, the emphasis on all vegetables and fruit (not all vegetables and fruit are high in carotenoids) and the fact that concentration biomarkers often shows low correlation coefficients with reported dietary intake (Al-Delaimy *et al*., 2005).

We did not include specific targets to increase the intake of low-fat diary products in our intervention. In the DASH study a more beneficial effect was seen in the group that ate more low-fat diary products compared to the group that only increased the intake of vegetables and fruit. However, the subjects in the DASH study had a low baseline intake of calcium (Appel *et al*., 1997). Our subjects had a high baseline intake of calcium. Further studies would be needed in obese subjects with SRBD to examine the effect of increasing low-fat diary products in the dietary intervention.

A minor limitation is that we did not exclude subjects taking high doses of multivitamin supplements; however, only two subjects in the intervention group took such doses at baseline. This may be reflected in the difference in serum folate between the groups at baseline. These subjects stopped taking the supplements during the study. We have no reasons to believe that this has influenced our results.

Implications

We have shown that simple dietary advice to increase intake of vegetable and fruit in a group session based behavior treatment was effective for moderate weight reduction and in reducing systolic and diastolic BP in a population of symptomatically treated subjects with SRBD. Vegetables and fruit are water-rich foods that are low in energy density and

hence allow big portion sizes while reducing energy intake. A behavioral program with targeted goals to increase the intake of vegetables appears to be important for weight loss and BP reduction. If no advice is given about the specific choice of food items known to be high in FRAP, antioxidant defense does not appear to be affected.

Acknowledgements

We thank Tor Ole Klemsdal for some of the medical examinations and Hanne Dyrendal for the dietary calculations. We also thank the subjects who took part in this study. ST and MS contributed to the development of the protocol. MS performed all the dietary assessments and analyzed the data. IH assisted with the statistical analyses and RB was responsible for the FRAP and caretenoid analyses. None of the authors had any personal or financial conflicts of interest.

Funding source: None.

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V

Effect of Orlistat on Eating Behavior Among Participants in a 3-year Weight Maintenance Trial

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Objective: To examine the effect of orlistat on dietary restraint, disinhibition, hunger, and binge eating and to understand the relation between changes in eating behavior and weight maintenance.

Methods and Procedures: Subjects were 306 women and men (age: 19–45 years; BMI: 37.5 ± 4.1 kg/m^2) included in the Scandinavian Multicenter study of Obese subjects with the Metabolic Syndrome, a 3-year clinical trial of orlistat or placebo following an 8-week very low energy diet (VLED). Outcomes were changes in weight and in the Three Factor Eating Questionnaire (TFEQ) and Binge Eating Scale (BES) between screening and 17 and 33 months after randomization. As reported previously, weight gain following VLED was lower in subjects treated with orlistat than with placebo.

Results: Compared to screening results, dietary restraint was increased and disinhibition, hunger, and binge eating were decreased in both groups. These changes were similar in both groups with the exception of the hunger score at month 33 that was reduced more in the placebo than in the orlistat group (difference between groups –1.1 (95% CI (–2.0, –0.2)) $P = 0.014$). In multivariate analyses, scores for restraint, disinhibition and binge eating were associated with weight loss after adjustment for BMI, gender, age, and treatment (all $P \leq 0.002$, model $R^2 = 0.12$–0.17).

Discussion: Orlistat did not affect eating behavior differently in any substantial way than the placebo did in this long-term weight maintenance trial. The results indicate that increased restraint and decreased disinhibition and binge eating are important for sustained weight maintenance in obese subjects with the metabolic syndrome.

Obesity (2008) **16,** 327–333. doi:10.1038/oby.2007.82

INTRODUCTION

The relation between obesity and several of its comorbidities such as type 2 diabetes and premature atherosclerosis seems to be mediated by risk factors characterized by the metabolic syndrome (1,2). The metabolic syndrome is closely associated with abdominal obesity (3,4). Weight loss achieved by changes in eating behavior, diet and physical activity are the cornerstones in the treatment of the metabolic syndrome. However, weight control methods often produce short-term success, while sustained weight maintenance is difficult to achieve (5,6). Thus, pharmacological therapy has been proposed as an adjunct to diet and lifestyle changes to improve the maintenance of weight loss. Orlistat is a pancreatic lipase inhibitor that reduces the intestinal absorption of fat (7,8). Because gastrointestinal symptoms may occur after the intake of fatty food, orlistat is thought to act as a policing influence in daily food choices.

Psychological and behavioral factors including dietary restraint, disinhibition, hunger, and binge eating may also influence dietary intake and body weight. Dietary restraint is defined as the tendency to consciously restrict food intake either to prevent weight gain or to promote weight loss by control over energy intake and types of food eaten (9). Disinhibition is the tendency to overeat in the presence of palatable foods or other disinhibitors such as emotional distress (10). Hunger has been defined as the susceptibility to perceived body symptoms that signal the need for food (10). The Three Factor Eating Questionnaire (TFEQ) (11) and the Binge Eating Scale (BES) (12) are recognized instruments for assessing these eating behaviors.

Increasing dietary restraint, and decreasing disinhibition and hunger are important for improved weight maintenance (13–17). A better understanding of how eating behaviors influence weight maintenance during treatment with orlistat, a drug that may influence daily food choices, could be helpful in the clinical management of obesity.

We recently reported the results of the Scandinavian Multicenter study of Obese subjects with the Metabolic Syndrome, a placebo-controlled clinical trial aimed at investigating the efficacy of orlistat in the maintenance of weight loss following a very low energy diet (VLED) in 309 abdominally obese women and men. The VLED initiated a weight loss

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Received 16 January 2007; accepted 10 July 2007. doi:10.1038/oby.2007.82

of 14.4 kg. After 18 months the maintained weight loss was 11.7 kg in the orlistat group and 9.6 kg in the placebo group and after 3 years the maintained weight loss was 9.4 kg and 7.2 kg respectively, in the orlistat and placebo groups (all $P < 0.05$). Thus, the gain in body weight after VLED at month 36 was less in the orlistat group compared to the placebo group (4.6 kg vs. 7.0 kg, $P < 0.02$) (18).

The objective of this study was to examine the effect of orlistat on dietary restraint, disinhibition, hunger and binge eating at month 17 and month 33 after an initial weight loss and to understand the relation between eating behavior and weight maintenance in subjects participating in the Scandinavian Multicenter study of Obese subjects with the Metabolic Syndrome trial.

METHODS AND PROCEDURES

Subjects

Subjects were recruited at nine clinical research centers in Scandinavia (Denmark, Finland, Norway, and Sweden) to participate in the Scandinavian Multicenter study of Obese subjects with the Metabolic Syndrome as described elsewhere (18). Men and women were eligible for the trial if they met the following criteria: (i) abdominal obesity (waist circumference >92 cm for females and >102 cm for males) (ii) at least one of the following metabolic risk factors: impaired fasting glucose (>6.1 mmol/l and <7.1 mmol/l), diet treated type II diabetes, dyslipidemia (HDL cholesterol <1.1 mmol/l for females and 0.9 mmol/l for males) and/or triglycerides >2.0 mmol/l. Subjects were excluded if they had clinically relevant conditions that might affect the outcome of the trial (i.e., weight change of more than ±4% for the previous 2 months before inclusion, women of child-bearing potential who were not on any accepted method of birth control or were lactating, uncontrolled hypertension (systolic >180 mm Hg, diastolic >115 mm Hg), significant cardiac, respiratory, renal, neurological, gastrointestinal, or endocrine disease, pre-existing cholelithiasis, pancreatic disease, history of gastrointestinal surgery for weight loss purposes, active gastrointestinal disease, drug treated diabetes mellitus, HbA_1c >10%, serum triglycerides ≥10 mmol/l, serum creatinine >150 µmol/l, psychiatric disorders requiring medication that may impact upon the requirements of the protocol, history or presence of carcinoma, history or presence of bulimia or laxative abuse, clinical symptoms of fat soluble vitamin deficiencies, use/dependence of any substance of abuse, unable or unwilling to comply with the demands of the protocol, or subjects who had received any investigational drug within the previous 2 months). All participants gave their written informed consent. The study was conducted according to the Helsinki Declaration and the Ethical Committee in each of the four countries approved the protocol. The study was conducted between November 1998 and October 2002.

Study design

Before randomization the participants followed a VLED for 8 weeks with the aim of achieving a weight loss of ≥5%. The VLED provided 2.5–3.3 MJ (600–800 kcal) per day. One of two liquid protein preparations depending on commercial availability (Modifast; Novartis, Basel, Switzerland or Nutrilett; Nycomed Pharma, Oslo, Norway) was used in the study, provided free of charge. Of 383 participants, 309 achieved a ≥5% weight loss and were randomly assigned to 36 months of treatment with orlistat capsules (120 mg three times daily) or matching placebo capsules. Of these, 306 completed screening assessments of eating behavior and were eligible for this study, while three subjects refused to fill in the questionnaires.

After the VLED, participants were instructed to follow a diet reduced in energy by 2.5 MJ (600 kcal), mostly focused on reducing the fat content and following as closely as possible the prescription of 30% of total kcal from fat, 50% from carbohydrate, and 20% from protein. The energy deficit was individually determined and subtracted from an estimate of total energy expenditure based on the WHO formula (19) for calculating resting metabolic rate and was corrected for the physical activity level. Resting metabolic rate was estimated based on the subjects' weight after the VLED prior to randomization. According to the protocol a physical activity level of 1.3 or 1.5 was used for middle to moderate or heavy daily activity, respectively. A dietitian helped the participants to plan a diet at the appropriate energy level according to the abovementioned recommendations and the food preferences of the subjects. To standardize the dietary counseling, the 20 dietitians involved in the study were trained at two investigator meetings prior to the study. Based on individual preferences, the counseling was provided individually or in groups in sessions lasting 30 min. A dietitian who was blind to the drug assignment monitored patients every month for the first 18 months and then at 3-month intervals. The behavioral treatment was based on a structured program that included assignments to be completed at home (20). The subjects assessed problematic eating habits and were then given opportunities to suggest behavioral changes that were discussed with the dietitian or group participants. If the patients did not have suggestions for behavioral changes themselves, the dietitians suggested changes. The behavioral changes concerned food choices and the organization of meals as well as dealing with emotional states or specific situations and eating occasions.

Assessments

The body weight, waist circumference, and blood pressure were measured by standardized techniques (18). Fasting (10 hour overnight) blood samples were collected according to the protocol of the trial and laboratory parameters were analyzed centrally (Medilab, Copenhagen, Denmark).

Eating behavior was assessed with the TFEQ and the BES at screening (i.e., before VLED treatment) and at month 1, month 8, month 17, month 21, and month 33 after randomization. The TFEQ is a 51-item instrument, which contains three subscales measuring dietary restraint, disinhibition, and hunger. It consists of 36 closed questions with a forced, false/true answer format and 15 with a 4-point "Likert" scale (11). Dietary restraint is measured with questions like:

> How frequently do you avoid "stocking up" on tempting foods? Using a Likert scale, the participants are asked to respond to the most appropriate alternative of almost never, seldom, usually or almost always.

Disinhibition is measured using a false/true format including items such as:

> When I feel anxious, I find myself eating.

Hunger is measured using a false/true format, including items such as:

> I am usually so hungry that I eat more than three times a day.

The BES consists of 16 sets of 3–4 statements relating to binge eating behavior and the subject is asked to endorse the one item from each set which best describes the eating behavior. Scores were calculated and the ranges for possible scores were 0–21 for dietary restraint, 0–16 for disinhibition 0–14 for hunger and 0–46 for BES (12). Higher scores reflect a greater tendency to exhibit the particular eating behavior characteristics.

Statistical analyses

Descriptive statistics are presented as means ± s.d. and means (95% CI). Scores for eating behavior were calculated at screening time (i.e., before VLED treatment) and at months 1, 8, 17, 21, and month 33. Analyses of variance using least square means with changes in weight as covariates was done to test the possible difference between the orlistat and placebo group with regard to changes in scores for eating behavior at

the measured time-points. Changes were calculated as the difference in scores between screening and subsequent measurements at months 1, 8, 17, 21, and 33. To test the interaction effect between treatment and gender, *F*-tests from ANCOVA were used. Student's *t*-test was used to test differences between gender and the Chi squared test was used in comparisons of categorical variables.

Pearson's correlations were done between changes in weight and changes in the scores for eating behavior within each treatment group at month 17 and month 33. To test the association between eating behavior and change in weight at month 33, we conducted multiple regression analyses with change in weight as the dependent variable and BMI at screening, age, gender, treatment, and the changes in scores for restraint, disinhibition, hunger, and binge eating, respectively, as four separate independent variables. Analyses were done using the observed values (for which the data are shown), last observation carried forward (from month 8) values and for the completers. The statistician (Fredrik Hansson) chose to use month 8 from screening (month 6 from randomization) because a reasonable test of the study medication required that it had to be taken for a relatively long period of time. The tests were considered significant at $P < 0.05$. Statistical analyses were performed using SAS software (version 9.1.3; SAS Institute, Cary, NC).

Power calculations was based on the main hypothesis of the study that the proportion of subjects with a maintained weight loss of at least 5% after the VLED was greater with orlistat than placebo treatment. The sample size calculation was performed using a formula from Lachin (21) $N = (Z_\alpha + Z_\beta)^2 4 \cdot P(1-P)/(P_c - P_e)^2$ where Z_α is the significance level set to be 0.05, Z_β is the power of the study set to be 90%, P_c is the proportion of treatment success in the orlistat group set to be 0.60, P_e is the proportion of treatment success in the placebo group set to be 0.35 and P is the overall treatment success rate. With this, the total number of subjects needed would be 168. With a drop out rate of 40% the number of subjects required would be 280. The statistical power of the study to achieve this goal was 92%.

RESULTS

Of the 306 subjects whose eating behavior was assessed at screening, 251 (82.0%) and 197 (64.4%) subjects remained in the trial and completed eating behavior assessments at months 17 and 33, respectively. Within each group, 1 subject was unwilling to complete the BES following screening. The last observation carried forward population consisted of 140 subjects in the orlistat group and 137 subjects in the placebo group and the completer population included 103 subjects and 98 subjects in the orlistat and placebo group, respectively. Table 1 shows the subject characteristics. These were similar in the orlistat and placebo groups.

Table 1 Characteristics of the participants in the orlistat and placebo groups before weight reduction (screening)

	Orlistat ($N = 153$)	Placebo ($N = 156$)	*P* value[a]
Female, *N* (%)	77 (50.3)	80 (51.3)	0.8666
Age, *y*	47.2 ± 8.6[b]	46.7 ± 9.4	0.5998
Smokers, *n* (%)	33 (21.6)	40 (25.6)	0.3991
Weight (kg)			
Female	101.6 ± 13.5	104.1 ± 12.7	0.2294
Male	119.1 ± 19.8	119.6 ± 15.3	0.8552
Height (m)			
Female	1.64 ± 0.06	1.65 ± 0.06	0.5599
Male	1.79 ± 0.07	1.80 ± 0.06	0.3386
BMI (kg/m^2)			
Female	37.6 ± 3.8	38.4 ± 4.2	0.2456
Male	37.1 ± 4.3	36.8 ± 3.9	0.6689
Waist (cm)			
Female	114 ± 12	115 ± 10	0.6436
Male	123 ± 11	124 ± 10	0.8603
Hip (cm)			
Female	123 ± 10	124 ± 10	0.3351
Male	117 ± 10	118 ± 10	0.6548
Waist-to-hip ratio	1.0 ± 0.1	1.0 ± 0.1	0.6957
Diastolic blood pressure (mm Hg)	91 ± 12	91 ± 10	0.9235
Systolic blood pressure (mm Hg)	144 ± 19	144 ± 17	0.8872
Glucose (mmol/l)	6.44 ± 1.83	6.27 ± 1.54	0.4046
Total cholesterol (mmol/l)	5.91 ± 1.26	6.02 ± 1.08	0.3760
HDL cholesterol (mmol/l)	1.13 ± 0.26	1.15 ± 0.26	0.5473
Triacylglycerols (mmol/l)	2.36 ± 1.24	2.50 ± 1.41	0.3406

[a]Differences between the orlistat and placebo group were tested with the Student's *t*-test for all variables except from gender and smoking that was tested with the Chi square test.
[b]Mean ± s.d. and all such values.

Eating behavior

Table 2 shows the eating behavior scores at screening and the changes in eating behaviors at month 1, 8, 17, 21, and 33 in the orlistat and placebo groups. No difference was seen between the groups before weight reduction. The mean scores from the BES tended to be low.

The main changes in eating behavior occurred between screening and month 1 and remained quite stable thereafter (Table 2). There was no significant difference between genders with regard to change in dietary restraint, disinhibition and binge eating (data not shown). Dietary restraint increased, while disinhibition, hunger, and binge eating decreased with no between-group differences with the exception of the score for hunger. The scores for hunger were reduced more in the placebo than in the orlistat group at month 33 and this was statistically significant in men (between-group difference −1.1 (95% CI −1.2, 0) $P = 0.0493$). This tendency was similar in women in numerical terms but did not achieve statistical significance (between-group difference −1.0 (95% CI −2.2, 0.3) $P = 0.1395$). There was no statistically significant interaction effect between treatment and gender (Table 2). However, scores for restraint increased more in men than in women (7.0 (95% CI 6.3, 7.8) vs. 5.0 (95% CI 4.3, 5.7), respectively; $P = 0.0002$) but there was no difference between the genders at month 33 (5.8 (95% CI 4.9, 6.6) vs. 5.0 (95% CI 4.2, 5.8), $P = 0.2$).

Change in weight was significantly associated with change in restraint, disinhibition and binge eating at month 17 and

Table 2 Scores before weight reduction (screening) and changes in eating behavior in the orlistat and placebo group; the observed population

	Orlistat		Placebo		Between-group difference	P^a	P^a treatment sex
	N		*N*				
Restraint							
Before weight reduction (screening)	151	8.0 ± 4.0[b]	155	7.8 ± 4.2			
Difference: month 1 after randomization—screening	150	6.9 (6.2, 7.6)[c]	152	7.2 (6.4, 8.0)	0.3 (–0.7, 1.3)	0.576	0.715
Difference: month 8 after randomization—screening	140	6.0 (5.4, 6.7)	137	6.5 (5.7, 7.3)	0.6 (–0.4, 1.6)	0.240	0.984
Difference: month 17 after randomization—screening	128	5.9 (5.2, 6.6)	122	6.2 (5.3, 7.0)	0.6 (–0.5, 1.6)	0.297	0.985
Difference: month 21 after randomization—screening	121	5.6 (4.8, 6.3)	109	5.9 (5.1, 6.7)	0.9 (–0.2, 2.0)	0.103	0.920
Difference: month 33 after randomization—screening	104	5.2 (4.4, 6.1)	93	5.4 (4.5, 6.3)	0.5 (–0.7, 1.7)	0.407	0.457
Disinhibition							
Before weight reduction (screening)	151	9.1 ± 3.4	155	9.2 ± 3.1			
Difference: month 1 after randomization—screening	150	–2.8 (–3.3, –2.3)	152	–2.8 (–3.3, –2.3)	0.3 (–0.7, 1.3)	0.576	0.715
Difference: month 8 after randomization—screening	140	–2.3 (–2.8, –1.8)	137	–2.5 (–3.0, –2.0)	0.6 (–0.4, 1.6)	0.240	0.984
Difference: month 17 after randomization—screening	128	–2.4 (–2.9, –1.8)	122	–2.1 (–2.6, –1.5)	0.0 (–0.7, 0.8)	0.929	0.348
Difference: month 21 after randomization—screening	121	–2.3 (–2.8, –1.8)	109	–2.4 (–3.0, –1.8)	0.9 (–0.2, 2.0)	0.103	0.920
Difference: month 33 after randomization—screening	104	–2.3 (–2.9, –1.7)	93	–2.6 (–3.2, –2.0)	–0.5 (–1.3, 0.4)	0.289	0.419
Hunger							
Before weight reduction (screening)	151	6.3 ± 3.3	155	6.8 ± 3.4			
Difference: month 1 after randomization—screening	150	–2.4 (–2.9, –2.0)	152	–2.9 (–3.4, –2.3)	0.3 (–0.7, 1.3)	0.576	0.715
Difference: month 8 after randomization—screening	140	–2.0 (–2.4, –1.5)	137	–2.7 (–3.2, –2.1)	0.6 (–0.4, 1.6)	0.240	0.984
Difference: month 17 after randomization—screening	128	–2.1 (–2.6, –1.7)	122	–2.5 (–3.1, –1.9)	–0.5 (–1.3, 0.2)	0.160	0.445
Difference: month 21 after randomization—screening	121	–1.4 (–2.1, –0.7)	109	–2.8 (–3.4, –2.1)	0.9 (–0.2, 2.0)	0.103	0.920
Difference: month 33 after randomization—screening	104	–1.8 (–2.4, –1.3)	93	–2.9 (–3.6, –2.2)	–1.1 (–2.0, –0.2)	0.014	0.921
Binge eating							
Before weight reduction (screening)	150	14.7 ± 7.8	154	14.8 ± 7.2			
Difference: month 1 after randomization—screening	149	–4.8 (–5.8, –3.8)	151	–5.7 (–6.8, –4.6)	0.3 (–0.7, 1.3)	0.576	0.715
Difference: month 8 after randomization—screening	139	–4.8 (–5.8, –3.7)	136	–5.3 (–6.5, –4.1)	0.6 (–0.4, 1.6)	0.240	0.984
Difference: month 17 after randomization—screening	127	–4.6 (–5.7, –3.5)	122	–5.3 (–6.5, –4.1)	–1.2 (–2.7, 0.3)	0.123	0.871
Difference: month 21 after randomization—screening	121	–4.4 (–5.5, –3.4)	109	–6.1 (–7.4, –4.8)	0.9 (–0.2, 2.0)	0.103	0.920
Difference: month 33 after randomization—screening	104	–4.5 (–5.7, –3.3)	93	–5.1 (–6.5, –3.7)	–0.9 (–2.7, 1.0)	0.358	0.615

[a]Differences between the groups before weight reduction was tested with Students *t*-test. Differences in changes between the groups were covariance analyses with weight reduction as covariate. *F*-tests from ANCOVA were used to test the interaction effect between treatment and gender.
[b]Mean ± s.d. and all such values.
[c]Mean (95% CI) and all such values.

33 and with hunger at month 17 (all $P < 0.01$), but not with hunger at month 33 ($P = 0.09$). The same trends were seen in the last observation carried forward analyses and in the analyses of completers (data not shown).

Table 3 shows the correlations between change in weight and eating behavior within each group in the observed population. In the orlistat group, change in weight was inversely correlated to change in restraint and positively correlated to change in disinhibition and binge eating. In the placebo group, change in weight was inversely correlated to change in restraint. In addition change in hunger and binge eating was positively correlated to change in weight at month 17. The correlations observed in the completer analyses as well as the last observation carried forward analyses were very similar.

The results of the multiple linear regression analyses for restraint, disinhibition, hunger and binge eating, each adjusted for treatment, BMI at screening, gender, and age are shown in **Table 4**. Changes in restraint, disinhibition, and binge eating were significantly associated with weight loss (all $P \leq 0.002$), explaining 12–17% of the variation in weight predicted for month 33. Changes in hunger tended to be associated with weight loss ($P = 0.0534$) explaining 9% of the variation. We also included smoking as an independent variable, but smoking was not a significant predictor in the models (data not shown).

DISCUSSION

In this 3-year, placebo-controlled clinical trial of weight maintenance following a VLED we found no substantial differences

Table 3 Correlations between change in weight and eating behavior in the orlistat and placebo group (observed population)

	Orlistat			Placebo		
	N	Correlation coefficient	*P*[a]	*N*	Correlation coefficient	*P*
Restraint						
Changes month 17	126	–0.1888	0.0342	119	–0.3017	0.0009
Changes month 33	103	–0.2983	0.0022	92	–0.3536	0.0005
Disinhibition						
Changes month 17	126	0.2994	0.0007	119	0.1444	0.1171
Changes month 33	103	0.3888	<0.0001	92	0.0473	0.6541
Hunger						
Changes month 17	126	0.0733	0.4145	119	0.2653	0.0035
Changes month 33	103	0.1188	0.2319	92	0.1402	0.1826
Binge eating						
Changes month 17	125	0.3898	<0.0001	119	0.2980	0.0010
Changes month 33	103	0.3157	0.0012	92	0.1179	0.2630

[a]Correlation were calculated with Pearson's correlation.

Table 4 Multiple regression analyses of change in eating behavior scores as predictors for weight change at month 33 in 197 subjects in the observed population

	Correlation coefficient	s.e.	*P*	R^2 (95% CI)
Restraint	–0.603	0.128	<0.0001	0.168 (0.075, 0.261)
Treatment	–2.476	1.116	0.0276	
Gender	1.772	1.135	0.1199	
BMI	–0.423	0.143	0.0035	
Age	0.065	0.064	0.3062	
Disinhibition	0.591	0.187	0.0018	0.117 (0.035, 0.199)
Treatment	–2.571	1.150	0.0266	
Gender	2.326	1.161	0.0465	
BMI	–0.421	0.147	0.0048	
Age	0.059	0.066	0.3672	
Hunger	0.372	0.194	0.0534	0.088 (0.015, 0.162)
Treatment	–2.794	1.185	0.0194	
Gender	2.264	1.182	0.0568	
BMI	–0.453	0.150	0.0029	
Age	0.052	0.067	0.4372	
Binge eating	0.304	0.088	0.0007	0.125 (0.041, 0.209)
Treatment	–2.592	1.145	0.0248	
Gender	2.558	1.156	0.0282	
BMI	–0.480	0.147	0.0013	
Age	0.049	0.065	0.4557	

between the effect of orlistat or placebo on eating behavior measured with the TFEQ and BES. Increased restraint and decreased disinhibition and binge eating were predictors of sustained weight maintenance in these participants; the participants were obese men and women with metabolic syndrome risk factors. Thus, changes in eating behavior in the orlistat group are mostly due to quantified eating behavior similar to the way the placebo operates. We are not aware of previous reports that have examined the effect of long-term orlistat treatment on eating behavior following a VLED.

Compared to scores at screening, dietary restraint was increased and disinhibition, hunger and binge eating were decreased in both the orlistat and placebo groups. An improvement in restraint, disinhibition and hunger has been shown in other trials of obese subjects taking sibutramine, an obesity drug that influences appetite (22,23). In the study by Hainer *et al.* 80 women were followed for 1 year and a drop in the disinhibition score was the most important factor associated with the decrease in BMI (22). In the study by Bauer *et al.*, the improvement in eating behavior was mostly attributed to the cognitive weight loss program (23). This may have been the case in our study, too, as all the subjects took part in a dietitian-managed behavioral program.

We found no differences between the treatment with orlistat or with the placebo with regard to restraint, disinhibition and binge eating. Thus, this form of drug treatment appears to be safe with regard to eating behavior amongst obese adults. Notably the placebo group showed a greater reduction in hunger scores than the orlistat group at 33 months. This difference may be due to chance or to the greater degree of weight loss in orlistat-treated subjects though a ~2 kg difference between the groups seems unlikely to affect regulators of appetite. Energy balance and appetite regulation are controlled by a number of hormones and peptides (24). Decreased levels of leptin may increase the release of orexigenic signals and thereby induce hunger (25). Unfortunately, we did not measure leptin levels in this study. The difference in hunger between the orlistat and the placebo group may be of minor clinical relevance given the overall reduction in hunger.

Increase in dietary restraint was inversely correlated to weight maintenance in both groups. However, the associations between change in weight and other eating behaviors

differed somewhat within the orlistat and placebo groups. Lower scores for disinhibition and binge eating were associated with changes in weight only in the orlistat group. High scores for disinhibition and hunger have been associated with a high intake of fatty foods (26) and with a greater binge eating severity (14,27,28). During binge eating the energy intake per episode may be of several thousand kJ and highly palatable, fatty foods are preferred (29). Participants taking orlistat may have experienced gastric side effects when eating palatable and fatty food, consistent with its mechanism of action. Though the overall percentage of energy from fat did not differ between the groups (18,30), a subgroup of participants may have reduced the intake of energy yielding fatty foods and improved their weight maintenance because of that. Indeed, better weight loss and reduced binge eating has been reported in obese binge eaters treated with orlistat (31). Other contributors to weight loss maintenance than those measured here could be significant predictors of success. Despite the blinded design of the study, some participants may have guessed that they received the placebo. This may have increased their motivation to reach weight goals or maintain a certain amount of weight loss (32).

We further sought to understand the role of eating behavior in long-term weight maintenance. After adjustment, increase in restraint and decrease in disinhibition and binge eating were all associated with improved weight maintenance, as expected, given that any dietary treatment of obesity implies increased restraint. Obese individuals who are not in treatment tend to score similarly to non-obese individuals as regards cognitive restraint; however, their scores increase during treatment (33). Likewise, obese subjects score higher than non-obese subjects with regard to disinhibition but their scores fall during treatment (13). Moreover, scores for disinhibition have been shown to correlate with the severity of binge eating (34). The relative importance of restraint, disinhibition, hunger, and binge eating as predictors for weight maintenance has been shown in a number of other studies (16,17,27,35,36). In the study of Pekkarinen *et al.* obese subjects were followed for 2 years after a weight loss program of 17 sessions including an 8-week VLED (16). Subjects that maintained an increase in restraint and decreases in disinhibition and hunger also were most successful with their weight reduction (16). Among subjects who were followed for 1 year after a VLED-induced initial weight loss, success was associated with increased dietary restraint and lesser feelings of hunger (17). In a study by Westenhoefer *et al.* 7407 men and women participated in a computer-based weight loss program. Successful weight reduction was associated with higher baseline scores for restraint that increased further during treatment and a lower baseline score of disinhibition that was further reduced (36). Changes in BES were not measured in these studies.

During weight maintenance it may be of special importance to focus on increasing dietary restraint and reducing disinhibition. This is challenging in an environment where there is an abundance of fatty foods and snacks, but inter-personal group approaches for dealing with stimuli control and disinhibitors such as lack of time, social eating, stress, anxiety, distress, and loneliness may be helpful (11).

Our study has several strengths. With over three hundred obese individuals and about equal numbers of men and women, its statistical power was good. The subjects were followed for 3 years in a placebo-controlled trial and the completion rate was relatively high. We were able to prospectively measure changes in eating behavior at several time-points. One of the limitations is that we did not perform the assessments of eating behavior at randomization, after the end of the VLED. It was not considered to be clinically relevant to measure eating behavior in subjects at the end of a VLED fast before a normal diet was restarted. Thus, we were unable to assess changes during the VLED. However, the changes in restraint, disinhibition and hunger were in accordance with others (16,17,35). Moreover, no difference in weight loss was seen between the orlistat and placebo groups after VLED (18).

In conclusion, our novel finding was that, in a long-term weight maintenance trial, orlistat did not affect eating behavior in a substantially different way than did the placebo. These findings indicate that orlistat does not impair the effect of a behavioral program and that changes in eating behavior seem to be important determinants of success during drug treatment.

ACKNOWLEDGMENTS

We thank the subjects for participating and the staff who were involved in this study. Hoffman La Roche financed the trial. Serena Tonstad has received honoraria for lectures from Hoffman La Roche, the manufacturer of orlistat.

DISCLOSURE

Serena Tonstad has received honoraria for lectures from Hoffman La Roche, the manufacturer of orlistat.

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VI

The long-term influence of orlistat on dietary intake in obese subjects with components of metabolic syndrome

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Abstract

Background Orlistat is a lipase inhibitor that reduces the intestinal absorption of fat and may enhance the effects of dietary and behavioural therapy on weight loss and maintenance. This study examined the effect of orlistat on dietary intake, especially fat intake, during long-term weight maintenance.

Methods Subjects were 44 men and women (age: 18-63 years; BMI: 37.5 ± 4.3 kg m^{-2}) included in the Scandinavian Multicenter study of Obese subjects with the Metabolic Syndrome, a three-year clinical trial of orlistat or placebo following an eight-week very low energy diet (VLED). Two months after the end of the trial when the use of orlistat was optional, 33 subjects remained in the study. A dietary interview based on a validated food frequency questionnaire was conducted before the VLED, after one year of treatment with orlistat or placebo and two months after the end of the trial.

Results At one year, dietary intake did not differ between the orlistat and placebo group. Energy percent (E%) fat was reduced and E% carbohydrate was increased within both groups. Two months after the end of the trial, E% fat was 32.6% (SD 6.2%) in subjects that chose to take orlistat and 27.7% (SD 5.5%) in subjects not taking orlistat (between group difference -5.0% [95%CI -9.2 to -0.7]; P=0.021).

Conclusion Use of orlistat compared with placebo in a lifestyle modification program does not appear to influence dietary intake. Subjects that chose to take orlistat after the end of the program did not comply with dietary recommendations and this may hamper the effect of the drug.

Introduction

Given the worldwide increase in obesity and its health consequences, efficient strategies for its prevention and treatment are important. It has been recommended that weight reduction programs focus on achieving a modest weight loss of 7% to 10% of the initial weight (Grundy, 2005). Behavioural and dietary therapies are the cornerstones of weight reducing programs. However, long-term adherence to the diet is difficult and pharmacological therapy has been proposed as an adjunct to diet and lifestyle changes to improve treatment outcomes.

Orlistat has been shown to improve weight loss compared to placebo in behavioural treatment programs (Rucker *et al*., 2007). The drug is a pancreatic lipase inhibitor that reduces the intestinal absorption of fat. About 30% of dietary fat is not metabolised, but excreted in the faeces (Guerciolini, 1997). If the dietary intake of fat is higher than recommended, abdominal symptoms including oily spotting, faecal incontinency and flatus ensue. As part of a dietary and behavioural treatment program, orlistat may promote weight loss in two ways: 1) by the loss of energy as fat in the stools due to the pharmacological effect of the drug and 2) by improving adherence to a low fat diet. Because gastrointestinal symptoms may occur after the intake of fatty food, orlistat is thought to act as a policing influence in daily food choices. However, this notion has not been substantiated in studies conducted to date (Franson & Rössner, 2000; Krempf *et al*., 2003).

The effect of anti-obesity drugs is likely to be counteracted by compensatory changes in food intake and energy expenditure to maintain a stable body weight over time, unless changes in lifestyle are made simultaneously (Speakman, 2004). However, the motivation to maintain lifestyle changes may be attenuated by the use of a drug (or its placebo) for weight reduction. For example, studies have shown only minor weight reductions in the placebo arm of recent studies of anti-obesity drugs (Pi-Sunyer *et al.,* 2006). Subjects that are taking an active, effective drug may be less likely to comply with dietary advice than subjects taking

placebo. Moreover, data is sparse with regard to the dietary intake of individuals that take anti-obesity drugs for weight maintenance compared to those that choose not to use such drugs.

The results of the Scandinavian Multicenter study of obese subjects with the Metabolic Syndrome (SMOMS) have been reported elsewhere (Richelsen *et al*., 2007). In short, the study was a double-blinded, randomized clinical trial comparing the effects of orlistat and placebo on weight maintenance after weight loss following an eight-week very low energy diet (VLED) in subjects with components of metabolic syndrome. After the VLED, the subjects were randomized to orlistat or placebo for three years. During weight maintenance, all the subjects participated in a dietary and behavioural treatment program and special emphasis was given to the reduction of fat intake. The main result of the study was that after three years, participants receiving orlistat maintained 2 kg more of their weight loss compared with participants receiving placebo (Richelsen *et al*., 2007). The present study included all subjects that participated in SMOMS at the Oslo centre.

The aims of the study were to:

1. Examine whether there were differences in dietary intake, in particular fat intake, among subjects treated with orlistat or placebo one year after randomization.
2. Examine whether there were differences in dietary intake among participants that chose to take or not to take orlistat two months after the end of the trial when the use of orlistat was optional.

Subjects and methods

Subjects with a body mass index (BMI) >30 kg m^{-2} were recruited to take part in a placebo-controlled, multi-centre trial of orlistat for weight maintenance following a VLED. Subjects were eligible for the trial if they met the following criteria for the metabolic syndrome:

abdominal obesity (waist circumference > 92 cm for females and > 102 cm for males) and at least one or more metabolic risk factor including an impaired fasting glucose (> 6.1 mmol L^{-1} and < 7.1 mmol L^{-1}) or diet treated type 2 diabetes and/or dyslipidaemia (HDL-cholesterol <1.1 mmol L^{-1} for females and 0.9 mmol L^{-1} for males) and/or triglycerides >2.0 mmol L^{-1}. The study was initiated before the National Cholesterol Education Program (NCEP) proposed definitions of the metabolic syndrome in 2001 (Expert Panel, 2001). In the NCEP definition at least three of the metabolic risk factors have to be present to define the metabolic syndrome. Thus, some of the subjects in this study did not have the full metabolic syndrome according to the NCEP criteria. Therefore the phrase "components of the metabolic syndrome" is used. Subjects were excluded if they had clinically relevant conditions that might affect the outcome of the trial. The Ethical Committee (region 1 in Norway) approved the protocol and all participants gave written informed consent. The study was conducted between November 1998 and January 2003.

Study design

After the screening visit, all subjects initiated a VLED (800 kcal [≤3.4 MJ] day^{-1}) for eight weeks. Immediately following the VLED subjects were randomized to orlistat 120 mg three times day^{-1} or to matching placebo three times day^{-1} for three years. Subjects were scheduled to attend 24 clinical visits during the first 18 months and every third month during the next 18 months for a total of 30 visits. Weight was monitored, tablets were counted and adverse events were reported at every visit. At the last visit in the trial, subjects chose to take or not to take orlistat as treatment to facilitate weight maintenance after the end of the trial.

At every visit the participants met with a registered dietitian. For the first 18 months the dietary behaviour program consisted of scheduled group sessions for four to six subjects. Individual sessions were offered only when subjects were unable to attend the scheduled

visits. For the last 18 months of the study, only individual sessions were offered. Each of the individual and group sessions lasted for about 30 min.

During the dietary and behavioural treatment program, the subjects were instructed to follow a diet consisting of <30% of energy from fat and to decrease the intake of saturated fat to <10% of energy while allowing for a small amount of unsaturated fat from fatty fish and plant sources. The subjects were to increase the intake of fibre from vegetables, legumes, fruits and whole meal bread, to choose low fat diary products and lean meat or chicken in stead of minced meat and sausages, and to reduce the intake of cakes/biscuits, ice cream, chocolate, sugar containing beverages and alcohol to achieve a energy deficit of 600 kcal (2.5 MJ) day^{-1} giving the possibility for further weight reduction when the subjects were highly motivated. Subsequently the subjects increased the energy intake.

In the group session's exhibitions were used to illustrate the amount of fat in foods. Subjects were taught to read food labels, make a plan for mealtimes and plan menus. Subjects were instructed to use a plate model for hot meals and advised to fill one-half of the plate with vegetables, one-fourth with potatoes, rice or pasta and one-fourth with meat, chicken, fish and/or legumes. The plate model was recommended at occasions such as eating with friends, at parties and at restaurants. The subjects were given exercises to assess eating behaviour and to plan specific strategies to avoid pitfalls (Melin & Rössner, 2003). The behavioural program otherwise included features such as goal setting, stimulus control and cognitive restructuring.

Dietary assessment

The dietician assessed dietary intake by an interview based on a food frequency questionnaire (FFQ)(Andersen *et al.*, 1999; Lindroos *et al.*, 1993). The first dietary interview was conducted immediately before the VLED period. The second dietary interview was

conducted 11 ± 2 months after randomization to orlistat or placebo. The third dietary interview was conducted 10 ± 4 weeks after the end of the trial. Dietary data was missing for two subjects at the one-year examination. As a supplementary check of dietary compliance a 24 h dietary recall was conducted in 33 subjects (15 subjects in the placebo group and 18 in the orlistat group) one year after randomization.

The dietary interview was conducted by a registered dietician and lasted between one and two hours. The FFQ was designed to assess habitual food intake during the last three months. The questionnaire elicited frequencies and consumption of 174 individual food items or constellations of items grouped together according to the typical Norwegian meal pattern. Particular attention was given to extra layers of bread-spread, food eaten during meal preparation, fat used in frying and extra portions of dinner and dinner leftovers. The between meal intake of sweet baked goods, cookies, cakes, ice cream, desserts, sweets, chocolate and snacks as nuts, potato crisp and popcorn was specifically queried. An atlas of food portions as well as photographs, household measurements and ordinary models of sweets and snacks was used to estimate portion sizes. The FFQ was coded manually for the calculation of energy intake (EI), energy yielding nutrients and food items using a software program (Mat på data 3.0, Landsforeningen for kosthold og helse, Oslo, Norway, 1996) based on the Norwegian food composition table (Rimestad *et al*., 1995). The FFQ has been validated against doubly labelled water (Svendsen & Tonstad, 2006). Despite underestimation of energy intake the FFQ method showed better reporting accuracy assessed by the ratio of EI to total energy expenditure (TEE) compared to three days of dietary records (EI/TEE = 0.824 according to the FFQ and EI/TEE = 0.704 according to the dietary records, $P<0.001$).

Because underreporting may impair the validity of dietary reports (Subar *et al.*, 2003) total energy expenditure was measured to validate the dietary intake data. Physical activity was recorded for three consecutive days by ActiReg®, an electromechanical device which

records the main body positions (standing, sitting, bent forward and lying down) together with motion of the trunk and/or one leg each second. The ActiReg® method uses a combination of position changes and movements not acceleration, to estimate the energy cost of the activity. An especially developed computer program (ActiCalc®) was used to calculate total energy expenditure. The calculation model is based on the estimated energy cost of the body position and activity expressed as physical activity ratios (the ratio of energy expenditure to resting metabolic rate) combined with the number of position changes each minute. Total energy expenditure was calculated by summarizing the calculated energy expenditure for each minute of the measurement period (Hustvedt *et al.*, 2004). Total energy expenditure calculated by the ActiReg® system has shown valid estimates in obese subjects compared to total energy expenditure measured by the doubly labelled water method (Hustvedt *et al.*, 2008). The resting metabolic rates were calculated with the Mifflin formula (Mifflin *et al.*, 1990) as this equation has shown valid estimates of resting metabolic rate in obese subjects (Frankenfield *et al*., 2005; Tooze et al., 2007). The physical activity level was calculated as the ratio of total energy expenditure to resting metabolic rate. In weight stable subjects energy intake equals energy expenditure (i.e. EI/TEE = 1). The EI/TEE was calculated before the VLED, at one year after randomization and at the final assessment.

The ActiReg® recording was missing for four subjects before weight reduction due to technical problems. At one year and at the final visit, five and six subjects, respectively, were unwilling to take part in the procedure.

Anthropometrics and biochemical measurements

Participants were weighed wearing light indoor clothing and no shoes with a digital scale at every visit. The average of two measurements was recorded. Height was measured once using a standard scale. Blood pressure was measured automatically with a digital blood

pressure monitor (OMRON Hem-705 CP) in sitting position. Waist was measured midway between lower costa and crista while the participant was unclothed and standing and hip circumference was measured at the level of the greater trochanter.

Fasting (10 hour overnight) blood samples were collected according to the protocol of the SMOMS trial and laboratory parameters (serum total cholesterol, high density lipoprotein cholesterol, triglycerides, and glucose) were analyzed centrally in the SMOMS trial (Medilab, Copenhagen, Denmark).

Statistical analysis

Descriptive statistics were performed and presented as means (SD), means (95% CI), median ($25^{th.}$,$75^{th.}$ percentile). For normally distributed variables mean differences within and between groups were tested with paired- and unpaired t-tests, respectively. For skewed variables differences within each group were tested with the Wilcoxon signed rank test and differences between groups were tested with the Mann-Whitney two-sample rank test. The Chi squared test was used in comparisons of categorical variables. Pearson's correlation coefficient was used to test agreement between the dietary methods. The tests were considered significant at $P<0.05$. Statistical analyses were performed using Stat View 5.0.1 (Abacus concepts, Berkeley, CA, USA).

Results

Fifty-two subjects were randomized after the VLED period to orlistat or placebo. Six subjects dropped out before one year. One subject refused to participate in the dietary assessment and one subject had received formal dietary counselling just before entering the trial and was excluded. Thus 44 subjects were available at one year. There were 10 further dropouts leaving 34 subjects at the end of the trial. At this time-point nine of 17 subjects in the orlistat

group chose to continue with orlistat and five of 16 subjects in the placebo group started orlistat giving a total of 14 subjects who were taking orlistat two months after the end of the trial. Eleven subjects in the placebo group continued without orlistat and eight subjects in the orlistat group stopped treatment giving a total of 19 subjects who were not taking orlistat after the end of the trial. One final subject chose sibutramine and was excluded from the end of trial population. Table 1 shows the characteristics of the subjects before starting the VLED.

Dietary intake before the VLED and one year after randomization

The placebo and orlistat groups achieved almost similar dietary changes with no differences between the groups. The energy percent from fat and sucrose decreased and the energy percent from carbohydrate increased. The reduction in energy percent from fat was 9.6% (95% CI –12.6 to -6.6) in the orlistat group and 10.2% (95% CI -13.0 to -7.4) in the placebo group (P = 0.8 between the groups). The energy percent from fat ranged from 19.7% to 39.9% in the orlistat group and from 14.6% to 37.9% in the placebo group. The between group change in energy percent from fat was -0.6% (95% CI - 4.9 to 3.7). This was confirmed by the 24 h dietary recall as showed that the energy percent from fat was 30.6% (SD 9.1%) in the orlistat group and 29.4% (SD 9.1%) in the placebo group (P = 0.5). Energy percent from fat calculated by the two methods showed significant correlations (r = 0.513, P = 0.010). EI/TEE ratio was similar in both groups (Table 2) as were the intakes of fat-soluble vitamins, fibre and cholesterol (data not shown). The intake of butter and cream was higher in the orlistat group than in the placebo group (Table 3).

Dietary intake two months after the end of the placebo controlled trial

Subjects that did take orlistat reported a higher percent of energy from fat than subjects that did not take orlistat two months after the end of the trial (Table 4). In subjects that took orlistat the energy percent from fat was 32.6% (SD 6.2%) and in subjects that did not take orlistat the energy percent from fat was 27.7% (SD 5.5%) (mean between group difference -5.0% [95% CI -9.2 to -0.7]; P = 0.021). The energy percent from fat ranged from 25.1% to 46.3% among subjects taken orlistat and from 16.9% to 40.8% among subject not taking orlistat. The EI/TEE ratio was similar in both groups (Table 4) as were the intakes of fat-soluble vitamins, fibre and cholesterol (data not shown). The reported intake of food items between the groups did not differ significantly (data not shown).

Change in weight during the placebo controlled trial

The eight-week VLED resulted in a weight loss of 13% (SD 3%) with no difference between the orlistat or placebo groups (-13.0% in the orlistat group vs. –12.7% in the placebo group, P=0.8). At one year the mean weight had increased by 2% (SD 7%) in the orlistat group versus 3% (SD 7%) in the placebo (P = 0.5). The overall weight reduction during the study was 7.2 kg (SD 8.1 kg) in the orlistat group and 3.9 kg (SD 7.4 kg) in the placebo group (P = 0.2).

Adverse events

The tablet counting showed that one subject in the placebo group and one subject in the orlistat group reported less than 60% compliance with the study drug. The results are shown for the intent-to-treat population, but very similar trends were seen when the non-compliant subjects were excluded from the analyses with one exception. The difference in reported intake of butter and cream only achieved borderline significance (P=0.053). Sixteen subjects (16/21) in the placebo group and 20 (20/23) in the orlistat group (P = 0.4) reported adverse

events possibly or probably related to the drug once or more during the trial. Both groups reported abdominal pain, increased defecation, decreased defecation, soft stool, liquid stool, faecal urgency and flatulence. Subjects in the placebo group were the only ones that reported nausea and faecal incontinency, while subjects in the orlistat group were the only ones that reported frequent stool, vomiting, fatty evacuation and oily spotting (data not shown).

Discussion

In obese subjects with metabolic risk factors the use of orlistat did not change dietary intake during long-term weight maintenance compared to placebo. After the end of the study, dietary intake of fat was higher in subjects taking orlistat compared to subjects that did not choose to take orlistat. During the entire study the subjects participated in a behavioural treatment program and were monitored in a clinical trial. The physical activity level did not differ between the groups. Hence any differences in weight maintenance between the orlistat and placebo groups were potentially due to orlistat and/or the diet. The findings of this study suggest that orlistat did not reduce dietary fat intake beyond that due to the structured behavioural treatment programme for weight reduction and maintenance. The novel finding of this study was that dietary intake of fat was higher than recommended in subjects that chose to take orlistat when it's use was optional compared to subjects not taking orlistat.

In the first part of the study the group randomised to orlistat did not achieve further reduction in fat intake compared to subjects randomised to placebo. The mean intake of dietary fat in both the orlistat and placebo groups was reduced to well below the recommended limit of 30% of energy from fat after one year, a reduction of about 10% compared to the subjects' habitual diet prior to the VLED. Intakes above this level are likely to increase the gastrointestinal adverse events according to the manufacturer (Summary of Product Characteristics, 2005). Indeed, a reduced intake of fat was seen in the orlistat group

compared to the placebo group in binge eaters eating a high fat diet (Golay *et al.*, 2005). The present study confirmed the finding of Hill *et al.* showing no difference between the orlistat and placebo group when dietary intake of fat was low (Hill *et al.*, 1999). It seems like subjects taking orlistat did not need a further reduction in fat intake to avoid gastrointestinal events.

In the second part of the study, the dietary intakes of subjects who chose to take orlistat were compared to the dietary intake of the subjects that did not take orlistat after the end of the trial. Subjects who chose to take orlistat after the end of the trial appeared not to comply with the dietary recommendations. They reported a higher energy percent from total fat compared to those that did not take the drug. The total fat intake was within the limits of 25% to 35% that is generally recommended for weight maintenance (McManus *et al.*, 2001; Wing RR *et al*., 2005; Schoeller & Bucholz, 2005). However, increasing fat intake has been associated with a lesser efficacy of orlistat (Ulrich *et al.,* 2003). The mean intake of saturated fat was above the recommended level, reflecting a lack of compliance with the dietary instruction that was given. It can be hypothesised that subjects who chose to take orlistat may have been less likely than subjects that did not choose orlistat to follow dietary instructions because of awareness of the action of orlistat on fat loss in the stools. Furthermore, the subjects may have chosen to take orlistat because of non-compliance with the diet.

The dietary methods used in this study were retrospective and the subjects may not have remembered foods and estimated portion sizes accurately. However, in previous studies, the FFQ showed better agreement with energy expenditure measured with the doubly labelled water method when compared to dietary records (Svendsen & Tonstad, 2006). Nevertheless, underreporting of energy intake was obvious in both groups. This is in accordance with numerous other studies showing that obesity accentuates this phenomenon (Hill & Davies, 2001; Livingstone and Black, 2003; Subar *et al*., 2003; Svendsen & Tonstad, 2006) and that

the obese selectively underreport sweets and fatty foods (Goris *et al.*, 2000; Svendsen & Tonstad, 2006). In the present study underreporting was assessed by estimating total energy expenditure based on a recording of physical activity with ActiReg®. This method has shown good agreement with total energy expenditure measured by the doubly labelled water method in obese subjects with metabolic syndrome (Hustvedt *et al*., 2008). However, total energy expenditure may be slightly underestimated by the ActiReg® method. Thus, the actual intake of fatty foods may have been higher than reported in this study. Despite an obvious underreporting of dietary intake, the diet deteriorated during long-term weight maintenance therapy.

Limitations of the study were the small sample size and the number of dropouts. High dropout rates are typical of long-term studies of obesity (Pi-Sunyer *et al*., 2006; Richelsen *et al*., 2007). The overall maintained weight loss during the three-year trial was about 4 kg in the placebo group and 7 kg in the orlistat group (NS). In the entire study population, the difference between the groups was statistically significant (Richelsen *et al*., 2007), but was not statistically significant at the Norwegian centre, due to the small sample size. Total energy expenditure was reduced in the orlistat group compared to the placebo group. This may be explained by a reduction in resting metabolic rate due to lower body weight and a slightly reduction (not statistically significant) in physical activity level. More research is needed on dietary intake of subjects that chose to take an anti-obesity drug for long-term weight maintenance after weight loss.

The use of the VLED may be questioned. However, the method has been recommended for weight loss purposes, providing the diet is followed by a 1-2 years integrated weight maintenance programme consisting of lifestyle interventions involving dietary change, nutrition education and behavioural therapy (Astrup & Rössner, 2000) as was given in the SMOMS trial (Richelsen *et al*., 2007). Given the overall result of the SMOMS

trial, the method seems safe and effective and improvement in weight maintenance may be achieved with the use of orlistat.

Conclusion

The use of orlistat did not lead to a lower fat intake compared to placebo in obese subjects participating in a structured program for weight loss and maintenance. The drug did not show a policing effect on daily food choices in subjects consuming a low fat diet. Subjects that chose to take orlistat did not comply with dietary fat recommendations and this may hamper the effect of the drug.

Acknowledgements

We thank the subjects for participating in the trial, Ragnhild Kleve for the blood samplings and Bo-Egil Hustvedt for support with the Actireg® recordings.

Source of Funding

This clinical trial was investigator initiated and sponsored by Roche Scandinavia. The sponsor had no involvement in the design, collection, analysis and interpretation of the data or in the writing of the manuscript.

Conflict of Interest

MS and MH declare that they have no conflicts of interests. ST has received honoraria for lectures and consulting from Roche and other pharmaceutical companies.

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Table 1 Characteristics of the participants before weight loss.

	Orlistat group	Placebo group
Male/female, n	11/12	10/11
Age (years)	49.1 (8.2)	46.6 (10.9)[†]
Cigarette smokers, n	5	5
BMI (kg m^{-2})	37.0 (4.0)	38.1 (4.6)
Waist (cm)		
Male	122.8 (7.7)	122.4 (10.6)
Female	106.4 (8.9)[*]	115.4 (9.1)
Waist-to-hip ratio	1.0 (0.1)	1.0 (0.1)
Systolic blood pressure (mmHg)	141 (20)	140 (15)
Diastolic blood pressure (mmHg)	86 (11)	85 (10)
Total cholesterol (mmol L^{-1})	6.0 (1.3)	6.3 (0.8)
HDL cholesterol (mmol L^{-1})	1.1 (0.3)	1.1 (0.2)
Triglycerides (mmol L^{-1})	2.2 (0.8)	2.9 (2.2)
Glucose (mmol L^{-1})	6.4 (2.0)	7.1 (3.0)

[†] Mean (SD) and all such values.

[*] $p<0.05$; Difference between the orlistat group and the placebo group (unpaired t-test).

Table 2. Body weight, daily energy expenditure and dietary intake data before the very low energy diet (VLED) and one year after randomization to orlistat or placebo.

	Orlistat				Placebo				
	Before VLED		After one year		Before VLED		After one year		
	n	Mean (SD)	n	Mean (SD)	n	Mean (SD)	n	Mean (SD)	P value[†]
Weight (kg)	22	108.3 (17.6)	21	94.8 (16.4)****	18	112.8 (18.0)	18	102.9 (15.9)****	0.127
Resting metabolic rate (kcal)[‡]	22	1823 (270)	21	1673 (249)****	18	1904 (272)	18	1778 (252)****	0.202
Total energy expenditure (kcal)	22	3186 (571)	21	2749 (438)***	18	3322 (796)	18	3185 (676)	0.020
Physical activity level[§]	22	1.76 (0.25)	21	1.65 (0.16)	18	1.74 (0.32)	18	1.79 (0.30)	0.059
Energy intake (kcal)	23	2507 (696)	22	1937 (621)***	21	2888 (820)	20	2013 (710)***	0.716
Energy intake/energy expenditure	22	0.82 (0.28)	20	0.71 (0.22)	18	0.86 (0.25)	17	0.61 (0.22)	0.142
E% fat (%)	23	38.4 (7.7)	22	29.1 (5.7)****	21	35.8 (3.7)	20	25.8 (6.3)****	0.075
E% saturated fat (%)	23	15.3 (3.5)	22	10.2 (2.2)****	21	14.8 (2.1)	20	9.3 (3.1)****	0.275
E% monounsaturated fat (%)	23	13.2 (3)	22	10.7 (2.6)***	21	12.4 (1.6)	20	9.2 (2.8)****	0.067
E% polyunsaturated fat (%)	23	6.6 (2.8)	22	5.5 (1.8)*	21	5.8 (1.7)	20	4.8 (1.7)	0.217
E% protein (%)	23	15.7 (2.7)	22	16.1 (3.1)	21	16.3 (3.5)	20	17.1 (3.8)	0.350
E% carbohydrate (%)	23	42.0 (9.0)	22	48.9 (8.6)***	21	44.2 (6.8)	20	50.6 (8.6)**	0.525
E% sucrose (%)	23	5.7 (4.7)	22	4.2 (3.3)*	21	6.5 (6.5)	20	3.1 (2.5)**	0.231
E% alcohol (%)	23	3.1 (3.8)	22	3.1 (4.5)	21	3.0 (5.1)	20	3.8 (4.9)	0.637

E%, energy percent.

[†] Differences between the orlistat and placebo group (unpaired t-test).

[‡] To convert kcal to kJ, multiply kcal by 4.184. [§] The physical activity level was calculated as the ratio of total energy expenditure to resting metabolic rate.

[*] P<0.05, [**] P<0.01, [***] P<0.001, [****] P<0.0001; Within group differences (paired t-test).

Table 3. Food intake before the very low energy diet (VLED) and one year after randomization to orlistat or placebo.

	Orlistat (n=23) Before VLED	(n=22) After one year	Placebo (n=21) Before VLED	(n=20) After one year	P value[†]
Food	Median (25th, 75th percentiles)		Median (25th, 75th percentiles)		
Bread and cereals (g)	190 (134, 252)	174 (120, 246)	190 (119, 262)	174 (130, 201)	0.960
Milk (g)	59 (22, 379)	107 (48, 249)	202 (84, 565)	105 (39, 158)**	0.400
Butter and cream (g)	18 (8, 30)	11 (6, 12)**	13 (7, 28)	1 (0,10)*	0.036
Fatty cheese (g)	19 (2, 78)	0 (0, 19)**	40 (29, 93)	7 (0, 21)**	0.237
Fatty meat[‡] (g)	108 (57, 142)	48 (26, 76)**	112 (89, 174)	50 (15, 72)***	0.696
Poultry and lean meat (g)	36 (28, 54)	48 (21, 78)	28 (14, 44)	63 (45, 88)**	0.174
Fish (g)	56 (28, 68)	62 (38, 96)	75 (43, 122)	53 (35, 77)	0.497
Potatoes, rice, pasta (g)	191 (150, 266)	143 (105, 209)***	155 (116, 248)	162 (99, 213)	0.890
Vegetables (g)	302 (247, 497)	548 (366, 714)***	354 (225, 445)	635 (244, 767)*	0.900
Fruit[‡] (g)	172 (86, 363)	321 (210, 435)*	262 (114, 342)	222 (143, 513)	0.623
Sweets/cookies/desserts (g)	38 (22, 64)	32 (20, 53)	52 (31, 101)	32 (10, 61)*	0.588
Oil[‡] (g)	13 (10, 44)	13 (4, 20)	25 (11, 42)	17 (1, 30)	0.546
Nuts, olives and avocado (g)	0 (0, 13)	7 (0, 2)	0(0, 8)	1 (0, 7)	0.082

[†] Differences between orlistat group and the placebo (the Mann-Whitney two-sample rank test).

[‡] Fatty meat includes minced meat, sausages, pork chops, salami; Fruit includes orange juice, berries and berry jam; Oil includes original and fat reduced soft margarine, mayonnaise and salad-dressings. * $P< 0.05$, ** $P< 0.01$, *** $P< 0.001$;Within group comparisons (the Wilcoxon signed rank test) before the VLED and one year after randomization to orlistat or placebo year.

Table 4. Weight, daily energy expenditure and dietary intake data in subjects taking orlistat and subjects not taking orlistat two months after the end of the placebo controlled trial.

	Orlistat		No orlistat		
	n	Mean (SD)	n	Mean (SD)	P value[†]
Weight (kg)	11	104.1 (14.6)	16	102.1 (19.5)	0.560
Resting metabolic rate (kcal)[‡]	11	1851 (277)	16	1764 (275)	0.428
Total energy expenditure (kcal)	11	3037 (498)	16	3059 (529)	0.915
Physical activity level[§]	11	1.64 (0.16)	16	1.75 (0.25)	0.240
Energy (kcal)	14	2293 (538)	19	2104 (695)	0.404
Energy intake/Total energy expenditure	11	0.76 (0.17)	16	0.72 (0.21)	0.645
E% fat (%)	14	32.6 (6.2)	19	27.7 (5.5)	0.021
E% saturated fat (%)	14	12.4 (4.0)	19	10.1 (2.6)	0.050
E% monounsaturated fat (%)	14	11.7 (2.2)	19	10.0 (2.5)	0.053
E% polyunsaturated fat (%)	14	5.7 (1.4)	19	4.8 (1.2)	0.046
E% protein (%)	14	16.2 (2.6)	19	17.5 (3.9)	0.291
E% carbohydrate (%)	14	46.2 (7.3)	19	51.4 (8.2)	0.073
E% sucrose (%)	14	3.9 (3.3)	19	4.8 (3.5)	0.419
E% alcohol (%)	14	4.0 (3.8)	19	2.4 (3.1)	0.184

E%, energy percent.
[†] Differences between the groups (unpaired t-test).
[‡] To convert from kcal to kJ, multiply by 4.184.
[§] The physical activity level was calculated as the ratio of total energy expenditure to resting metabolic rate.

Appendix 1

Matvaner og matvarevalg

Navn:

Har du endret matvanene dine i løpet av de siste tre månedene?

Begrenser du matinntaket for øyeblikket?

1. MÅLTIDSRYTME

Med mellommåltid mener vi et lite måltid som f.eks. kake, frukt, sjokolade eller snacks etc. Drikke uten tilbehør regnes ikke som måltid.

	Gang per måned					Gang per uke					Klokkeslett
	0	<1	1	2	3	1	2	3	4-5	6-7	
Frokost	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Mellommåltid formiddag	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Lunsj	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Mellommåltid ettermiddag	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Middag	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Mellommåltid kveld	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Kveldsmat	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Nattmat	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____

Hvordan fordeler du maten i løpet av døgnet?

Jevnt fordelt på måltider ☐ Mest til middag ☐ Mest til lunsj ☐ Mest utover kvelden/natten ☐

2. HVOR MYE BRØD PLEIER DU Å SPISE?

Legg sammen det du bruker til alle måltider (1/2 rundstykke = 1 skive)

	antall skiver x antall dager per uke				**Sum**
	Frokost	Lunsj	Middag	Kvelds	
Fint brød (loff, baguett, pita, fine rundstkykker)					
Mellomgrøvt brød (helkorn, kneip, lys hjembakt, kneiprundstykker)					
Grovt brød (fiberkneip, grovt hjembakt, grove rundstykker)					
Lyst knekkebrød (kavring)					
Grovt knekkebrød (skonrokk)					
Flatbrød					
Smørbrødkjeks					

Sum antall skiver/enheter per uke: _____

Hvor tykke brødskiver skjærer du? Bilde _____

3. SMØREFETT

Hvor mye smør/margarin bruker du per skive? Bilde _____

Hva pleier du å smøre på brødet?

Smør	☐☐	antall	Vita-lett	☐☐	antall
Bremyk	☐☐	antall	Soft light	☐☐	antall
Brelett	☐☐	antall	Olivero	☐☐	antall
Soya soft	☐☐	antall	Omega 3	☐☐	antall
Vita	☐☐	antall	Annet _____	☐☐	antall

4. PÅLEGG

Tenk deg din brødmat i løpet av en uke. Hvor mange skiver har du med følgende pålegg?

	Antall skiver per uke											Kommentar
	0	½	1	2-3	3-5	6-7	8-10	11-14	15-21	21-28	29-35	
Brun ost; helfet	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Brun ost; halvfet, prim	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Hvit ost > 27% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Hvit ost; 27% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Hvit ost; 16% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Magerost; <10% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Kjøttpålegg >20% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Kjøttpålegg 10%-20% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Kjøttpålegg <10% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Leverpostei	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Lett-leverpostei	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Kaviar, rognleverpostei	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Fet fisk som pålegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Røkt, gravet fisk	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Reker, krabbe	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Fiskepudding, crabstick	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Syltetøy, marmelade	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Lett-syltetøy, frysetøy	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Honning, sirup	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Sjokoladepålegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Peanøttsmør	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Grønnsaker som pålegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Frukt som pålegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Majonessalat	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Lett-majonessalat etc.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Majones over/under	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Lett-majones over/under	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Ikke noe pålegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____

Pålegg spist uten brød i forbindelse med tillaging, ekstra lag, pynt osv.

	Antall skiver per uke											Kommentar
	0	½	1	2-3	3-5	6-7	8-10	11-14	15-21	21-28	29-35	
Brun ost; helfet	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Brun ost; halvfet, prim	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Hvit ost > 27% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Hvit ost; 27% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Hvit ost; 16% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Magerost; <10% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Kjøttpålegg >20% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Kjøttpålegg 10%-20% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Kjøttpålegg <10% fett	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Leverpostei	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Lett-leverpostei	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Kaviar, rognleverpostei	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Fet fisk som pålegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Røkt, gravet fisk	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Reker, krabbe	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Fiskepudding, crabstick	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Syltetøy, marmelade	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Lett-syltetøy, frysetøy	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Honning, sirup	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Sjokoladepålegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Peanøttsmør	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Grønnsaker som pålegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Frukt som pålegg	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Majonessalat	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Lett-majonessalat etc.	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____
Annet	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	_____

5. EGG

Hvor ofte spiser du egg? Stekt, kokt, eggerøre, omelett etc.

Antall per måned					Antall per uke					Antall per dag					
0	<1	1	2	3	1	2	3-4	5-6	7	1	2	3	4	5	6
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

6. GRYN, FLAK OG KORNBLANDINGER MED TILBEHØR OG YOGHURT

Hvor ofte spiser du gryn flak, kornblandinger?

Gang per måned					Gang per uke				
0	<1	1	2	3	1	2-3	4-5	6-7	8-10
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Hvor ofte spiser du havregrøt?

Gang per måned					Gang per uke					Mengde						
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	Bilde	1	2	3	4	5	6
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐		☐	☐	☐	☐	☐	☐

Hvor ofte spiser du havregryn, usøtet müsli, Go´dag, Bran flakes?

Gang per måned					Gang per uke					Mengde						
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	bilde	1	2	3	4	5	6
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐		☐	☐	☐	☐	☐	☐

Hvor ofte spiser du søt müsli f.eks Tropisk müsli, Solfrokost?

Gang per måned					Gang per uke					Mengde						
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	bilde	1	2	3	4	5	6
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐		☐	☐	☐	☐	☐	☐

Hvor ofte spiser du Cornflakes, havrenøtter, japansk ris, kavring?

Gang per måned: 0 <1 1 2 3 | Gang per uke: 1 2-3 4-5 6-7 8-10 | Mengde: bilde 1 2 3 4 5 6

Sukker til frokostkorn, gryn og grøt

Gang per måned: 0 <1 1 2 3 | Gang per uke: 1 2-3 4-5 6-7 8-10 | Mengde: ss 1 2 3 4 5 6

Syltetøy til frokostkorn, gryn og grøt

Gang per måned: 0 <1 1 2 3 | Gang per uke: 1 2-3 4-5 6-7 8-10 | Mengde: ss 1 2 3 4 5 6

Helmelk, kefir

Gang per måned: 0 <1 1 2 3 | Gang per uke: 1 2-3 4-5 6-7 8-10 | Mengde: dl 1 2 3 4 5 6

Lettmelk, Cultura, Drikkeyoghurt, Biola melk

Gang per måned: 0 <1 1 2 3 | Gang per uke: 1 2-3 4-5 6-7 8-10 | Mengde: dl 1 2 3 4 5 6

Ekstra lett lettmelk

Gang per måned: 0 <1 1 2 3 | Gang per uke: 1 2-3 4-5 6-7 8-10 | Mengde: dl 1 2 3 4 5 6

Skummet melk, Skummet kultur

Gang per måned: 0 <1 1 2 3 | Gang per uke: 1 2-3 4-5 6-7 8-10 | Mengde: dl 1 2 3 4 5 6

Yoghurt, naturell og frukt

Gang per måned: 0 <1 1 2 3 | Gang per uke: 1 2-3 4-5 6-7 8-10 | Mengde: dl 1 2 3 4 5 6

Lettyoghurt

Gang per måned: 0 <1 1 2 3 | Gang per uke: 1 2-3 4-5 6-7 8-10 | Mengde: dl 1 2 3 4 5 6

7. KAFFE, TE OG KAKAO

	Antall kopper per uke: 1	2	3	4	5	6	Antall kopper per dag: 1	2	3-4	5-6	7-8	9-10	11-12
Kaffe, kokt	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kaffe, trakte/filter	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Cappucino	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Te	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Grønn te	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Iste	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
Kakao	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Tilbehør:

Antall per kopp: ½ 1 2 3 4 5 | Kopper per uke: 1 2 3 4 5 6 | Kopper per dag: 1 2 3-4 5-6 7-8

Sukker til kaffe	(ts)
Sukker til te	(ts)
Fløte i kaffe	(ss)
Helmelk i kaffe/te	(ss)
Lettmelk i kaffe/te	(ss)
Skummet melk i kaffe/t	(ss)
Krem i kakao	(ss)
Suketter	(stk)
Sitron i te	

8. DRIKKE

Helmelk, kefir

Gang per måned | Gang per uke | Mengde

0 <1 1 2 3 | 1 2-3 4-5 6-7 8-10 | glass 2 dl 1/2 1 2 3 4 5 6 7 ≥8

Lettmelk, Cultura, Milkshake, Sjokomelk, Biola melk

Gang per måned | Gang per uke | Mengde

0 <1 1 2 3 | 1 2-3 4-5 6-7 8-10 | glass 2 dl 1/2 1 2 3 4 5 6 7 ≥8

Skummet melk, skummet kultur

Gang per måned | Gang per uke | Mengde

0 <1 1 2 3 | 1 2-3 4-5 6-7 8-10 | glass 2 dl 1/2 1 2 3 4 5 6 7 ≥8

Ekstra lett lettmelk

Gang per måned | Gang per uke | Mengde

0 <1 1 2 3 | 1 2-3 4-5 6-7 8-10 | glass 2 dl 1/2 1 2 3 4 5 6 7 ≥8

Vann

Gang per måned | Gang per uke | Mengde

0 <1 1 2 3 | 1 2-3 4-5 6-7 8-10 | glass 2 dl 1/2 1 2 3 4 5 6 7 ≥8

Grønnsakjuice, tomatjuice, gulrot juice

Gang per måned | Gang per uke | Mengde

0 <1 1 2 3 | 1 2-3 4-5 6-7 8-10 | glass 2 dl 1/2 1 2 3 4 5 6 7 ≥8

Appelsinjuice

Gang per måned | Gang per uke | Mengde

0 <1 1 2 3 | 1 2-3 4-5 6-7 8-10 | glass 2 dl 1/2 1 2 3 4 5 6 7 ≥8

Grapefruktjuice, druejuice

Gang per måned | Gang per uke | Mengde

0 <1 1 2 3 | 1 2-3 4-5 6-7 8-10 | glass 2 dl 1/2 1 2 3 4 5 6 7 ≥8

Eplenektar, tropisk m.m.

Gang per måned | Gang per uke | Mengde

0 <1 1 2 3 | 1 2-3 4-5 6-7 8-10 | glass 2 dl 1/2 1 2 3 4 5 6 7 ≥8

Saft

Gang per måned | Gang per uke | Mengde

0 <1 1 2 3 | 1 2-3 4-5 6-7 8-10 | glass 1/2 1 2 3 4 5 6 7 ≥8

☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ 2 dl ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐

Lettsaft

Gang per måned					Gang per uke					Mengde									
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	glass	1/2	1	2	3	4	5	6	7	≥8
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	2 dl	☐	☐	☐	☐	☐	☐	☐	☐	☐

Brus

Gang per måned					Gang per uke					Mengde									
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	glass	1/2	1	2	3	4	5	6	7	≥8
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	2 dl	☐	☐	☐	☐	☐	☐	☐	☐	☐

Lettbrus

Gang per måned					Gang per uke					Mengde									
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	glass	1/2	1	2	3	4	5	6	7	≥8
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	2 dl	☐	☐	☐	☐	☐	☐	☐	☐	☐

Farris

Gang per måned					Gang per uke					Mengde									
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	glass	1/2	1	2	3	4	5	6	7	≥8
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	2 dl	☐	☐	☐	☐	☐	☐	☐	☐	☐

Alkoholfritt øl, lettøl, vørterøl

Gang per måned					Gang per uke					Mengde									
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	glass	1/2	1	2	3	4	5	6	7	≥8
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	3.3 dl	☐	☐	☐	☐	☐	☐	☐	☐	☐

Pils

Gang per måned					Gang per uke					Mengde									
0	<1	1	2	3	1	2-3	4-5	6-7	8+	glass	1/2	1	2	3	4	5	6	7	≥8
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	0.5 l	☐	☐	☐	☐	☐	☐	☐	☐	☐

Rødvin

Gang per måned					Gang per uke					Mengde									
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	glass	1/2	1	2	3	4	5	6	7	≥8
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	1.2 dl	☐	☐	☐	☐	☐	☐	☐	☐	☐

Hvitvin

Gang per måned					Gang per uke					Mengde									
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	glass	1/2	1	2	3	4	5	6	7	≥8
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	1.2 dl	☐	☐	☐	☐	☐	☐	☐	☐	☐

Likør

Gang per måned					Gang per uke					Mengde									
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	glass	1/2	1	2	3	4	5	6	7	≥8
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	2.5 cl	☐	☐	☐	☐	☐	☐	☐	☐	☐

Sprit (brennevin, konjakk, snaps, whisky, rusbrus)

Gang per måned					Gang per uke					Mengde									
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	glass	1/2	1	2	3	4	5	6	7	≥8
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	4.0 cl	☐	☐	☐	☐	☐	☐	☐	☐	☐

9. VARMRETTER, SMÅRETTER OG MIDDAGSRETTER

Hvor ofte spiser du varmretter, småretter og middagsretter?

Gang per måned					Gang per uke					Gang per dag					
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	1	2	3	4	5	6
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

Hvor ofte spiser du varmrett på kafé/kantine?

Gang per måned					Gang per uke					Gang per dag					
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	1	2	3	4	5	6

☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du på restaurant?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Gang per dag: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du fast food som wienerpølse, hamburger, kebab fra gatekjøkken etc.?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Stk.: 1 ☐ 2 ☐
Stor ☐ Brød ☐ Kommentar: _____
Liten ☐ Lompe ☐
Medium ☐

Hvor ofte spiser du pølser, kjøttpudding, farsekaker?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde ☐ stk. ☐
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du lett-pølser, kalkun- og lette kjøttprodukter?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde ☐ stk. ☐
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du hamburger, karbonadekaker, kjøttkaker, kjøttboller, løvbiff m.m?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde ☐
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐
Karbonadedeig ☐
Viltkarbonade ☐

Hvor ofte spiser du kjøttsaus, gryterett, Lasagne m.m.?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde ☐
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐
kjøttdeig ☐
karbonadedeig ☐

Hvor ofte spiser du bacon, flesk?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: g ☐
10 ☐ 20 ☐ 30 ☐ 40 ☐ 50 ☐ 60 ☐

Hvor ofte spiser du nakkekotlett, ribbe, entrecote, T-bone steak, Flintstek, spekeskinke etc.?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde ☐
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐
Ant. g _____
Med fett ☐
Uten fett ☐

Hvor ofte spiser du biff, stek, skinke, lam, vilt?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde ☐
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐
Ant. g _____

Hvor ofte spiser du lever?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde ☐
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐
Ant. g _____

Hvor ofte spiser du gryterett med renskåret rent kjøtt, lapskaus, kjøttsuppe?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde ☐
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du snitzel, steaklets, vårull m.m.?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde ☐
1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐
Egenpanering ☐

☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ stk. ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du kylling, kalkun, høne?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: 1/4 ☐ 1/2 ☐ 3/4 ☐ 1/1 ☐
Uten skinn ☐ Med skinn ☐ Gryte ☐

Hvor ofte spiser du torsk, sei, kolje?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐
Kokt ☐ Gryte ☐ Stekt ☐

Hvor ofte spiser du fiskekaker, -boller, -grateng, -pudding?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du skalldyr, sushi m.m.?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde med skall: hg 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du makrell, sild, kveite, laks, ørret?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐
Gryte ☐ Stekt ☐

Hvor ofte spiser du panert fisk, fiskepinner?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du pizza?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐
Med skinke ☐ Med kjøttdeig ☐ Vegetar ☐

Hvor ofte spiser du risgrøt/melkegrøt?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐
Smørøye ☐ Sukker ☐ Kommentar:_____

Hvor ofte spiser du pannekaker?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: stk 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐
Syltetøy ☐ Sukker ☐ Kommentar:_____

Hvor ofte spiser du suppe?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: dl 1-2 ☐ 3-4 ☐ 5-6 ☐ 7-8 ☐ 9-10 ☐ 10+ ☐
Med grønnsaker ☐
Type:_____

Hvor ofte spiser du salat som hovedrett til middag?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐
Type: _____

Hvor ofte spiser du salat til lunsj?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐
Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐
Mengde: bilde 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Type: _____

Hvor ofte spiser du vegetarretter til middag?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Type: _____

10. POTETER, RIS OG PASTA

Hvor ofte spiser du kokte poteter?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde stk.: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ Str. _____

Hvor ofte spiser du kokt ris?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du makaroni, spaghetti og pasta?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du pommes frites?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ gatekjøkken ☐ uten fett ☐

Hvor ofte spiser du stekte poteter?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du potetmos?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du fløtegratinerte poteter/grønnsaker?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du potetsalat?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

11. ROTFRUKTER, GRØNNSAKER OG BELGFRUKTER

Hvor ofte spiser du grønnsaker?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Gang per dag: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser rå gulrot? 1 stk.

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Gang per dag: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ Stor ☐ Middels ☐ Liten ☐

Hvor ofte spiser kokte gulrøtter?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du grønnsakblanding?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hermetisk ☐
Frossen ☐
Kommentar:_____

Hvor ofte spiser du tomater?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: ¼ ☐ ½ ☐ 1 ☐ 2 ☐ 3 ☐ 4 ☐

Pålegg ☐ Stor ☐
I salat ☐ Middels ☐
Stekt ☐ Liten ☐

Hvor ofte spiser du salat?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Issalat ☐ Rosso ☐
Kinakål ☐
Bladsalat ☐

Hvor ofte spiser du råkost av grovere grønnsaker som kålrot, gulrot, hodekål?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du brokkoli?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

I salat ☐ Rå ☐
Kokt ☐ I gryter ☐

Hvor ofte spiser du rosenkål?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du spinat?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

I salat ☐ Rå ☐
Kokt ☐ I gryter ☐

Hvor ofte spiser du blomkål?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

I salat ☐ Rå ☐
Kokt ☐ I gryter ☐

Hvor ofte spiser du hodekål?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Stuing ☐ Surkål ☐
Kokt ☐ I gryter ☐

Hvor ofte spiser du kålrot?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Rotmos ☐ Rå ☐
Kokt ☐ I gryter ☐

Hvor ofte spiser du aspargesbønner, asparges, sukkererter?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

I salat ☐ Rå ☐
Kokt ☐ I gryter ☐

Hvor ofte spiser du squash?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐ | Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐ | Mengde bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐ | I salat ☐ Rå ☐ Stekt ☐ I gryter ☐

Hvor ofte spiser du aubergine?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐ | Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐ | Mengde bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐ | I salat ☐ Rå ☐ Stekt ☐ I gryter ☐

Hvor ofte spiser du løk, hvitløk, purre?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐ | Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐ | Mengde bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐ | I salat ☐ Rå ☐ Stekt ☐ I gryter ☐

Hvor ofte spiser du sopp?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐ | Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐ | Mengde bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐ | I salat ☐ Rå ☐ Stekt ☐ I gryter ☐

Hvor ofte spiser du paprika?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐ | Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐ | Mengde bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐ | I salat ☐ Pålegg ☐ I gryter ☐

Hvor ofte spiser du mais?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐ | Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐ | Mengde bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐ | I salat ☐ Frisk ☐ Hermetisk ☐

Hvor ofte spiser du avocado?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐ | Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐ | Mengde bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐ | I salat ☐ Egen rett ☐ Pålegg ☐

Hvor ofte spiser du tomatbønner, bønner, linser, tørkede erter?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐ | Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐ | Mengde bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du tyttebær, sylteagurk, rødbet?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐ | Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐ | Mengde ss: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du andre grønnsaker?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐ | Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐ | Mengde bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐ Type: _____

12. TILBEHØR

Hvor ofte spiser du seterrømme, creme fraiche?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐ | Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐ | Mengde bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du lettrømme?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐ | Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐ | Mengde bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du oljebasert dressing/remulade m.m.?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ Type: _____

Hvor ofte spiser du fløtesaus?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du smørbaserte sauser og smeltet smør/margarin?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ Type: _____

Hvor ofte spiser du tomatbaserte sauser, tomatpure, tomatketchup?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Mengde bilde: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

13. MATLAGNINGSFETT

I løpet av 1 uke, hvor ofte tilbereder du:

kokt, microbølget, bakt? ☐ stekt/ fett i tillagningen ☐ Wok'et/ lite fett i tilagningen ☐

Når du steker/bruker fett i matlagningen, hvor mye fett bruker du? Bilde ☐

Hvilke type fett bruker du i matlagningen?

Olivenolje	Maisolje	Soyaolje	Olivero	Vita	Soya	Bremyk	Smør	Melange	Flytende margarin
Rapsolje	Solsikkeolje								
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

14. FRUKT OG BÆR

Hvor ofte spiser du frukt?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Gang per dag: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐

Hvor ofte spiser du sitrusfrukter (appelsiner, klementiner, grapefrukt)?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Gang per dag: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ | Stor ☐ Middels ☐ Liten ☐

Hvor ofte spiser du epler?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Gang per dag: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ | Stor ☐ Middels ☐ Liten ☐

Hvor ofte spiser du pærer?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Gang per dag: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ | Stor ☐ Middels ☐ Liten ☐

Hvor ofte spiser du bananer?

Gang per måned: 0 ☐ <1 ☐ 1 ☐ 2 ☐ 3 ☐ | Gang per uke: 1 ☐ 2-3 ☐ 4-5 ☐ 6-7 ☐ 8-10 ☐ | Gang per dag: 1 ☐ 2 ☐ 3 ☐ 4 ☐ 5 ☐ 6 ☐ | Stor ☐ Middels ☐ Liten ☐

Hvor ofte spiser du druer?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du plommer?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk per gang: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du moreller/kirsebær?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk per gang: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du melon?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Skiver per gang: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Kantaluppmelon ☐
Vannmelon ☐
Honningmelon ☐

Hvor ofte spiser du fersken/nektariner?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk per gang: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du eksotiske frukter (papaya, mango, sharon, ananas, pasjonsfrukt, litchi, guawa, kumquat, granateple)?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk per gang: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Type: _____

Hvor ofte spiser du kiwi?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk per gang: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du jordbær, bringebær, rips, molter m.m.

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Sukker ☐
Fløte ☐

Hvor ofte spiser du tørket frukt (rosin, aprikos, fiken, banan, eple)?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk per gang: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Type: _____

15. DESSERT

Hvor ofte spiser du fruktsalat, fruktgrøt, hermetisk frukt, gele?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Kommentar: ____

Hvor ofte spiser du iskrem?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Kroneis ☐
Softis ☐
Ispinne ☐

Hvor ofte spiser du yoghurtis/lettis?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Saftis ☐

Hvor ofte spiser du dessertpudding f.eks sjokolade-, karamell-, mandelkjernepudding m.m.?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du vaniljesaus?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du kremfløte?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Pisket ☐

16. KAKER OG KAFFEMAT

Hvor ofte spiser du kaker/kjeks?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Gang per dag: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du wienerbrød?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Gang per dag: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Stort ☐
Middels ☐
Lite ☐

Hvor ofte spiser du kremkake, bløtkake, Napoleonskake?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Marsipankake ☐

Hvor ofte spiser du sjokoladekake, nøttekake, formkake, eplekake m.m.?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Bilde: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor ofte spiser du søt gjærbakst som boller, kringle, julekake, skolebrød?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk per gang: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Stor ☐
Middels ☐
Liten ☐

Hvor ofte spiser du småkaker, kransekake, søte kjeks?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk. per gang: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Fylt kjeks ☐ Sjokolade ☐
Enkel kjeks ☐

Hvor ofte spiser du vafler?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Plate: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Smør ☐ Syltetøy ☐
Lettrømme ☐ Brunost ☐

Hvor ofte spiser du lefser, lomper etc.?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk.: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Fylt ☐

17. GODTERI OG SNACKS

Hvor ofte spiser du søtsaker og sjokolade?

Gang per måned: 0 <1 1 2 3
Gang per uke: 1 2-3 4-5 6-7 8-10
Gang per dag: 1 2 3 4 5 6

☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐ ☐

Hvor mange små sjokolader (<50g) spiser du?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk.: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Ren ☐ Kjeks ☐ Sukkerfyll ☐ Nøttefyll ☐

Hvor mange normalstore (50-100g) sjokolader spiser du?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk.: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Ren ☐ Kjeks ☐ Sukkerfyll ☐ Nøttefyll ☐

Hvor mange store sjokoladeplater (100-200g) spiser du?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk.: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Ren ☐ Kjeks ☐ Sukkerfyll ☐ Nøttefyll ☐

Hvor mange konfektbiter/småsjokolader spiser du?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk.: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor mye marsipan spiser du?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk. á 50 g: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Marsipanbrød ☐ Sjokoladetrekk ☐

Hvor mye søtsaker som karameller, lakris, vingummi, seimenn etc. spiser du?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Pose á 300g: 1/4 1/3 1/2 3/4 1/1 1,5 2 ☐ ☐ ☐ ☐ ☐ ☐ ☐

Hvor mye drops/pastiller spiser du?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Eske: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐
Sukkerfri ☐

Hvor ofte spiser du snacks og nøtter?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stk.: 1 2 3 4 5 6 ☐ ☐ ☐ ☐ ☐ ☐

Hvor mye potetgull spiser du?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Stor pose: 1/4 1/2 3/4 1/1 1,5 2 ☐ ☐ ☐ ☐ ☐ ☐

Hvor mye ostepop, popcorn o.l. spiser du?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Pose: 1/4 1/2 3/4 1/1 1,5 2 ☐ ☐ ☐ ☐ ☐ ☐

Hvor mye nøtter (peanøtter, mandler, hasselnøtter, valnøtter, cashewnøtter, pistasjnøtter) spiser du?

Gang per måned: 0 <1 1 2 3 ☐ ☐ ☐ ☐ ☐
Gang per uke: 1 2-3 4-5 6-7 8-10 ☐ ☐ ☐ ☐ ☐
Pose: 1/4 1/2 3/4 1/1 1,5 2 ☐ ☐ ☐ ☐ ☐ ☐
saltet ☐

☐☐☐☐☐ ☐☐☐☐☐ ☐☐☐☐☐☐

Stk.

1-5 6-10 10-15 15-20 20-30 30+

☐ ☐ ☐ ☐ ☐ ☐

Hvor mye oliven spiser du?

Gang per måned					Gang per uke					Stk.					
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	1-5	6-10	10-15	15-20	20-30	30+
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

18. TILSKUDD

Hvor ofte tar du tran?

Gang per måned					Gang per uke					Mengde							
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	ss	1/2	1	2	3	4	5	vinter ☐ periodevis ☐
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	stk	☐	☐	☐	☐	☐	☐	kommentar: _____

Hvor ofte tar du omega-3 tilskudd?

Gang per måned					Gang per uke					Mengde							
0	<1	1	2	3	1	2-3	4-5	6-7	8-10	ss	1/2	1	2	3	4	5	vinter ☐ periodevis ☐
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	stk	☐	☐	☐	☐	☐	☐	kommentar: _____

Hvor ofte tar du multivitamintabletter?

Gang per måned					Gang per uke					Mengde							
0	<1	1	2	3	1	2-3	4-5	6-7	8-10		1	2	3	4	5	6	vinter ☐ periodevis ☐
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	stk.	☐	☐	☐	☐	☐	☐	kommentar: _____

Hvor ofte tar du vitamintabletter?

Gang per måned					Gang per uke					Mengde							
0	<1	1	2	3	1	2-3	4-5	6-7	8-10		1	2	3	4	5	6	vinter ☐ periodevis ☐
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	stk.	☐	☐	☐	☐	☐	☐	kommentar: _____

Hvor ofte tar du mineraltilskudd?

Gang per måned					Gang per uke					Mengde							
0	<1	1	2	3	1	2-3	4-5	6-7	8-10		1	2	3	4	5	6	vinter ☐ periodevis ☐
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	stk.	☐	☐	☐	☐	☐	☐	kommentar: _____

Hvor ofte spiser du soyaproteinpulver?

Gang per måned					Gang per uke					Mengde							
0	<1	1	2	3	1	2-3	4-5	6-7	8-10		1	2	3	4	5	6	vinter ☐ periodevis ☐
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	ss	☐	☐	☐	☐	☐	☐	kommentar: _____

Hvor ofte spiser du Nutribars?

Gang per måned					Gang per uke					Mengde							
0	<1	1	2	3	1	2-3	4-5	6-7	8-10		1	2	3	4	5	6	vinter ☐ periodevis ☐
☐	☐	☐	☐	☐	☐	☐	☐	☐	☐	stk.	☐	☐	☐	☐	☐	☐	kommentar: _____

20. PERSONALIA

Vekt: Dato: Antall personer i husstanden:

Appendix 2

Patient initials _____

Patient number _____

Center number _____

Date of visit ☐☐ ☐☐ ☐☐☐☐

TFEQ

VEILEDNING:

Vennligst besvar de følgende spørsmål ved å krysse av for riktig eller galt. R = Riktig G = Galt

Del I

1. Når jeg kjenner duften av stekt biff eller ser et saftig stykke kjøtt, finner jeg det meget vanskelig å la være å spise, selv om jeg nettopp har avsluttet et måltid.	☐ R	☐ G
2 Jeg spiser vanligvis for mye ved sosiale anledninger, som i selskaper og ved utflukter.	☐ R	☐ G
3. Jeg er som oftest så sulten at jeg spiser mer enn tre ganger om dagen.	☐ R	☐ G
4. Når jeg har spist min kalori kvote, er jeg som regel flink til å la være å spise mer.	☐ R	☐ G
5. Slanking er så vanskelig for meg fordi jeg simpelthen blir så alt for sulten.	☐ R	☐ G
6. Jeg tar bevisst små porsjoner som et middel til å holde vekten min nede.	☐ R	☐ G
7. Noen ganger smaker det så godt at jeg fortsetter å spise, selv om jeg ikke er sulten lenger.	☐ R	☐ G
8. Siden jeg ofte er sulten, ønsker jeg noen ganger at en ekspert ville fortelle meg, mens jeg spiser, at nå har jeg fått nok, eller at jeg kan spise mer.	☐ R	☐ G
9. Når jeg er urolig må jeg spise noe.	☐ R	☐ G
10 Livet er for kort til å bekymre seg om slanking.	☐ R	☐ G
11. Siden vekten min går opp og ned, har jeg gått på slankekur mer enn en gang.	☐ R	☐ G
12. Jeg føler meg ofte så sulten at jeg bare må spise.	☐ R	☐ G
13 Når jeg er sammen med noen som spiser for mye, så spiser jeg som oftest også for mye	☐ R	☐ G
14. Jeg har en ganske god idé om antall kalorier i vanlig mat.	☐ R	☐ G
15 Noen ganger når jeg begynner å spise, klarer jeg bare ikke å stoppe.	☐ R	☐ G
16 Jeg har ikke vanskelig for å legge igjen noe på tallerkenen.	☐ R	☐ G
17 Jeg blir sulten på bestemte tider på dagen, fordi jeg er blitt vant til å spise da	☐ R	☐ G

Patient initials: _____

Patient number _____

Center number _____

18 Hvis jeg, under en slankekur, spiser mat som ikke er tillatt, spiser jeg bevisst mindre i en periode for å rette opp skaden.	☐ R	☐ G
19 Å være sammen med noen som spiser ofte, gjør meg sulten nok til at jeg også spiser.	☐ R	☐ G
20 Når jeg er nedfor, spiser jeg ofte for mye.	☐ R	☐ G
21 Jeg er alt for glad i å spise til å ødelegge det med å telle kalorier eller passe på vekten min.	☐ R	☐ G
22. Når jeg ser noe riktig lekkert, blir jeg ofte så sulten at jeg må spise straks.	☐ R	☐ G
23. Jeg slutter ofte å spise, før jeg er helt mett. Dette er en bevisst måte å begrense den mengde jeg spiser	☐ R	☐ G
24. Jeg blir så sulten at magen min ofte kjennes som et bunnløst hull.	☐ R	☐ G
25. Vekten min har nesten ikke endret seg i det hele tatt i de siste ti årene.	☐ R	☐ G
26. Jeg er alltid sulten, så det er vanskelig for meg å slutte å spise før tallerkenen min er tom.	☐ R	☐ G
27. Når jeg føler meg ensom, trøster jeg meg med å spise.	☐ R	☐ G
28. Jeg behersker meg bevisst ved måltidene for ikke å legge på meg.	☐ R	☐ G
29. Jeg blir noen ganger veldig sulten sent på kvelden eller om natten.	☐ R	☐ G
30. Jeg spiser alt jeg har lyst på, når jeg vil.	☐ R	☐ G
31. Uten en gang å tenke på det bruker jeg lang tid på å spise.	☐ R	☐ G
32 Jeg teller bevisst kalorier for å kontrollere vekten min.	☐ R	☐ G
33. Jeg unngår visse matsorter, fordi jeg blir tykk av dem.	☐ R	☐ G
34. Jeg er alltid sulten nok til å spise når som helst.	☐ R	☐ G
35 Jeg er meget oppmerksom på endringer i min figur.	☐ R	☐ G
36 Hvis jeg, under en slankekur, spiser noe som ikke er tillatt, kaster jeg meg ofte ut i det og spiser annen høykalori mat.	☐ R	☐ G

Patient initials _____

Patient number _____

Center number _____

Del II

FREMGANGSMÅTE:

Vennligst besvar de følgende spørsmål ved å tegne en sirkel rundt tallet over det svaret som passer for deg.

37. Hvor ofte er du på slankekur med den bestemte hensikt på å få vekten din under kontroll?

1	2	3	4
sjelden	noen ganger	ofte	alltid

38. Ville en vektforskjell på +/- to til tre kilo ha noen innvirkning på hvordan du lever ditt liv?

1	2	3	4
ingen	litt	en del	stor

39. Hvor ofte føler du deg sulten ?

1	2	3	4
bare til måltidene	i blant mellom måltidene	ofte mellom måltidene	nesten alltid

40. Hjelper din skyldfølelse deg til å få kontroll med vekten din når du spiser for mye?

1	2	3	4
aldri	sjelden	ofte	alltid

41. Hvor vanskelig ville det være for deg å slutte å spise når du var halvferdig med middagen, og så ikke spise noe i de neste fire timene?

1	2	3	4
lett	litt vanskelig	ganske vanskelig	meget vanskelig

Patient initials _____

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42 Hvor bevisst er du på hva du spiser?

1	2	3	4
slett ikke	litt	ganske	meget

43. Hvor ofte klarer du å la være å "lage" fristende mat?

1	2	3	4
nesten aldri	sjelden	ofte	nesten alltid

44. Hvor stor er mulighetene for at du kjøper lavkalori mat?

1	2	3	4
meget små	ganske små	ganske store	meget store

45. Spiser du fornuftig når noen ser deg, og fråtser når du er alene?

1	2	3	4
aldri	sjelden	ofte	alltid

46. Hvor sannsynlig er det at du bevisst spiser sakte for å redusere mengden du spiser?

1	2	3	4
usannsynlig	ikke helt usannsynlig	nokså sannsynlig	meget sannsynlig

47 Hvor ofte sløyfer du desserten, fordi du ikke lenger er sulten?

1	2	3	4
nesten aldri	sjelden	minst en gang i uken	nesten hver dag

Patient initials _____

Patient number _____

Center number _____

48. Hvor sannsynlig er det at du bevisst spiser mindre enn du har lyst på?

1	2	3	4
usannsynlig	ikke helt usannsynlig	nokså sannsynlig	meget sannsynlig

49. Fyller du deg med mat, selv om du ikke er sulten?

1	2	3	4
aldri	sjelden	noen ganger	minst en gang i uken

50. Hvilket utsang passer på deg? Sett kryss:

0.	Du spiser hva du vil, når du vil	
1.	Vanligvis spiser du hva du vil, når du vil	
2.	Du spiser ofte hva du vil, når du vil	
3.	Du begrenser ofte det du spiser, men du gir ofte etter	
4.	Du begrenser ofte det du spiser, og gir sjelden etter	
5.	Du begrenser ofte det du spiser, og gir aldri etter	

51. I hvilken grad beskriver følgende uttalelser dine spisevaner?
" jeg begynner med slanking om morgenen, men på grunn av forskjellige ting som hender i løpet av dagen, har jeg gitt opp om kvelden og spiser hva jeg vil, men lover meg selv å starter med slankingen igjen i morgen."

1	2	3	4
ikke lik meg	litt lik meg	ganske god beskrivelse av meg	beskrivelsen passer perfekt på meg

Appendix 3

BES

VEILEDNING:
På de følgende sider er det grupper av nummererte uttalelser. Les alle uttalelser i hver gruppe og sett et kryss ved den uttalelse i hver gruppe som best beskriver dine følelser i forhold til de problemer du har med å kontrollere dine spisevaner.

1

1.	☐	Jeg er ikke flau over vekten min eller størrelsen på kroppen min når jeg er sammen med andre.
2.	☐	Jeg tenker på hvordan andre ser meg, men det gjør meg normalt ikke skuffet over meg selv.
3.	☐	Jeg blir flau over mitt utseende og vekten min, og det gjør meg skuffet over meg selv.
4.	☐	Jeg er veldig flau over vekten min og jeg føler ofte dyp skam og avsky for meg selv. Jeg prøver å unngå kontakt med mennesker, fordi jeg er så flau.

2

1.	☐	Jeg har ingen vanskeligheter med å spise behersket og sakte.
2.	☐	Selv om jeg later til å "sluke" maten, føler jeg meg ikke overmett fordi jeg har spist for mye.
3.	☐	Noen ganger har jeg en tendens til å spise fort, og da føler jeg meg ubehagelig mett etterpå.
4.	☐	Jeg har for vane å sluke maten, uten å tygge den ordentlig. Når jeg gjør det, føler jeg meg som regel ubehagelig overmett, fordi jeg har spist for mye.

3

1.	☐	Jeg føler at jeg kan beherske min spisetrang, når jeg vil.
2.	☐	Jeg har en følelse av at jeg er dårligere til å beherske spisingen min enn gjennomsnittsmenneske.
3.	☐	Jeg føler meg helt hjelpeløs når det gjelder å beherske min spisetrang.
4.	☐	Fordi jeg føler meg så hjelpeløs når det gjelder å beherske spisingen min, er jeg blitt helt desperat for å prøve å få kontroll.

4

1.	☐	Jeg har ikke for vane å spise, når jeg kjeder meg.
2.	☐	Iblant spiser jeg, når jeg kjeder meg, men ofte klarer jeg å "finne på " noe for å få tankene bort fra mat.
3.	☐	Jeg har for vane å " kjedespise " men det hender at jeg kan foreta meg noe for å få tankene vekk fra å spise.
4.	☐	Jeg har en innbitt vane med å "kjedespise". Ingenting synes å hjelpe meg til å bli kvitt denne vanen.

5

1.	☐	Som regel er jeg fysisk sulten når jeg spiser noe.
2.	☐	Noen ganger spiser jeg noe helt impulsivt, selv om jeg egentlig ikke er sulten.
3.	☐	Jeg har den uvane å stadig spise mat som egentlig ikke smaker meg for å tilfredsstille en sultfølelse, enda jeg ikke trenger maten rent fysisk.
4.	☐	Selv om jeg ikke er fysisk sulten, får jeg en følelse av sult i munnen, som bare ser ut til å kunne tilfredsstilles hvis jeg spiser noe mat, f.eks. et stykke smørbrød som fyller munnen min. Noen ganger, når jeg spiser mat for å tilfredsstille munnsulten, spytter jeg ut maten for ikke å legge på meg.

6

1.	☐	Jeg føler ikke skyld eller selvforakt etter at jeg har spist for mye.
2.	☐	Når jeg har spist for mye føler jeg iblant skyld eller selvforakt.
3.	☐	Nesten hele tiden føler jeg skyld eller selvforakt når jeg har spist for mye.

7

1.	☐	Jeg mister ikke helt kontrollen med spisingen min under en slankekur, selv etter perioder hvor jeg har spist for mye.
2.	☐	Noen ganger når jeg spiser mye " forbudt " mens jeg er på slankekur, føler jeg at nå har jeg ødelagt alt og så spiser jeg enda mer.
3.	☐	Jeg sier ofte til meg selv, når jeg har spist for mye under en slankekur: " nå har jeg ødelagt det, så nå kan jeg like gjerne fortsette. " Når det hender, spiser jeg enda mer.
4.	☐	Jeg starter regelmessig på en streng slankekur, men jeg bryter kuren ved å begynne et "etegilde". Mitt liv ser ut til å være enten et "etegilde" eller en sultekur.

12

1.	☐	Det synes som om jeg spiser akkurat like mye når jeg er sammen med andre (familie, i selskaper) som når jeg er alene.
2.	☐	Noen ganger, når jeg er sammen med andre, spiser jeg ikke så mye som jeg har lyst til, fordi jeg er flau over spisingen min.
3.	☐	Ofte spiser jeg bare litt, når det er andre tilstede, fordi jeg er så veldig flau over spisingen min.
4.	☐	Jeg skammer meg sånn over den overdrevne spisingen min at jeg velger å "ete" på tider, da jeg vet at ingen ser meg. Jeg føler meg som en "skap-eter".

13

1.	☐	Jeg spiser tre måltider om dagen og tar bare iblant et mellommåltid.
2.	☐	Jeg spiser tre måltider om dagen, men jeg spiser normalt også litt mellom måltidene.
3.	☐	Når jeg småspiser for mye vender jeg meg til å hoppe over ordentlige måltider.
4.	☐	Det er hele perioder, hvor jeg later til å spise uavbrutt, uten noen planlagte måltider.

14

1.	☐	Jeg tenker ikke mye på å prøve å beherske min uønskede spisetrang.
2.	☐	Jeg føler i det minste noe av tiden, at tankene kretser om å prøve å beherske min spisetrang.
3.	☐	Jeg føler at jeg ofte bruker mye tid på å tenke på hvor mye jeg spiste eller på å prøve å ikke spise mer.
4.	☐	Jeg synes at jeg mesteparten av mitt våkne liv er opptatt med tanke om å spise eller ikke spise. Jeg føler det som om jeg stadig kjemper for ikke å spise.

15

1.	☐	Jeg tenker ikke særlig på mat.
2.	☐	Jeg har sterke anfall av trang til mat, men de er kortvarige.
3.	☐	Det er dager, hvor det virker som om jeg ikke kan tenke på annet enn mat.
4.	☐	Det meste av min tid synes å være opptatt med tanker på mat. Jeg føler at jeg lever for å spise.

16

1.	☐	Jeg vet normalt om jeg er fysisk sulten eller ikke. Jeg spiser en passende porsjon for å bli mett.
2.	☐	Det hender at jeg er usikker på om jeg er fysisk sulten eller ikke. Da er det vanskelig å vite hvor mye mat jeg skal spise for å bli mett.
3.	☐	Selv om jeg vet hvor mange kalorier jeg bør spise, har jeg ikke noen ide om hva som er "normal" mengde mat for meg.