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Effects of Cognitive Behavioural Therapy Targeting Eating Behaviour (CBT-TEB):

A novel obesity treatment

Lisbeth Stahre

Karolinska Institutet

FROM THE DEPARTMENT OF CLINICAL NEUROSCIENCE, DIVISION OF PSYCHIATRY

KAROLINSKA INSTITUTET, STOCKHOLM, SWEDEN

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By

Lisbeth Stahre

Principal Supervisor:
Professor emeritus Tore Hällström
Karolinska Institutet
Department of Clinical Neuroscience
Centre of Psychiatry Research

Opponent:
Professor Maria Tillfors
Örebro Universitet
Department of Psychology and Social work
Centre for Health and Medical Psychology

Examination Board:
Docent Erik Hemmingsson
Gymnastik- och Idrottshögskolan
Karolinska Institutet
Department of Medicine

Docent Claes Norring
Karolinska Institutet
Department of Clinical Neurosciences
Stockholm Centre for Eating Disorders

Docent Jarl Torgerson
University of Gothenburg
Sahlgrenska Academy
Department of Medicine
Division of Molecular and Clinical Medicine

Never stop fighting for what you believe in

To my children and grandchildren

ABSTRACT

Objective. The overall objective was to evaluate the outcome of the 10-week (20/30 h) CBT-TEB treatment programme for persons with obesity. The evaluation took place in three studies.

Paper I (Study I)

Method. One hundred and five obese women from an obesity unit`s waiting list participated in a randomised controlled study. Of these, 62 took part in the CBT-TEB programme and 43 served as controls. Follow-up was 6-,12- and 18-months post-treatment, without any booster treatment. *Results*. Weight change for the CBT-TEB patients after therapy was - 8.5 kg (SD 16.1) (per-protocol). Weight change at the follow-up for the CBT-TEB patients 18 months after the end of therapy was - 10.4 kg (SD 10.8) (per-protocol) and - 5.7 kg (SD 9.5) (ITT). Weight change (per-protocol) for the controls at the 18-month follow-up was + 2.3 kg (SD 7.0) and +1.6 kg (SD 6.0) (ITT). About two thirds still fulfilled the widely held success criterion of having lost 5% or more of initial weight at 1.5 years after the end of therapy.

Paper II (Study II)

Method. Fifty-four obese women from a child care centre participated in a randomised controlled trial involving the CBT-TEB programme and a conventional weight loss programme. Of these, 16 women took part in the CBT-TEB programme and 26 in the control programme. Follow-up was 6-,12- and 18-months post-treatment, without any booster treatment. *Results.* Weight change for the CBT-TEB patients after therapy was -8.6 kg (SD 2.9) (per-protocol) and -7.7 kg (SD 3.8) (ITT). Weight change for the CBT-TEB patients 18 months after the end of therapy was – 5.9 kg (SD 5.4) (per-protocol) and -5.5 kg (SD 5.5) (ITT). Weight change for the participants in the control programme after the end of therapy was -0.7 kg (SD 1.2) (per-protocol) and -1.4 kg (SD 1.6) (ITT). Weight change for the participants in the control programme at follow-up 18 months after end of therapy was +0.3 kg (SD 4.3) (per-protocol) and -0.6 kg (SD 5.5) (ITT).

Paper III (Study III)

Method. Eighty-three primary care patients (74 females and nine males) started the CBT-TEB programme. Follow-up was at 6 months. An objective in this study was also to evaluate change in waist circumference (WC) and waist-to-hip ratio (WHR) after therapy. Eating behaviour was determined by the Three Factor Eating Questionnaire and obesity-related problems by the Obesity-Related Problems Scale. *Results.* Weight change (ITT) for the CBT-TEB patients after therapy was -4.5 kg (SD 3.9), and at 6 months -4.4 kg (SD 4.9). Emotional and uncontrolled eating decreased and cognitive control increased during therapy. Obesity-related problems decreased.

Conclusions

The results demonstrate a very satisfactory efficacy for the CBT-TEB programme. It was feasible to implement the programme in primary healthcare, using ordinary personnel as group leaders after a short period of training.

LIST OF SCIENTIFIC PAPERS

I. Stahre L, Hällström, T. A short-term cognitive group treatment program gives substantial weight reduction up to 18 months from the end of treatment:a randomized controlled trial. Eat Weight Disord. 2005 Mar;10(1):51-8.

II. Stahre L, Tärnell B, Håkanson CE, Hällström T. A randomized controlled trial of two weight-reducing short-term group treatment programs for obesity with an 18-month follow-up. Int J Behav Med. 2007;14(1):48-55.

III. Stahre L, Blomstrand A, Hällström T. Observational study on implementation and effectiveness of Cognitive Behavioural Therapy Targeting Eating Behaviour for patients with abdominal obesity in an ordinary primary health care setting. Article.

CONTENTS

LIST OF ABBREVIATIONS

BMI	Body Mass Index
BED	Binge Eating Disorder
CT	Cognitive Therapy
CBT	Cognitive Behavioural Therapy
CBT-TEB	Cognitive Behavioural Therapy Targeting Eating Behaviour
CI	Confidence interval
CR	Cognitive Restraint
DSM-IV	Diagnostic and Statistical Manual of Mental Disorders. Fourth Edition
EE	Emotional Eating
EDPEX	Eating Eating Disorder Patient´s Expectations and Experiences of Treatment Questionnaire
GAS	General Adaptation Syndrome
HDL-C	High-density lipoproteins
LDL-C	Low-density lipoproteins
ORPS	The Obesity Related Problems Scale
RAB	Rating of Anorexia and Bulimia
SAD	Sagittal abdominal diameter
SCB	Statistiska Centralbyrån
SOS	Swedish Obese Subject
SRM	Standardized Response Mean (Effect size)
TFEQ	Three-Factor Eating Questionnaire
TFEQ EE	Three-Factor Eating Questionnaire Emotional Eating
TFEQ CR	Three-Factor Eating Questionnaire Cognitive Restraint
TFEQ UE	Three-Factor Eating Questionnaire Uncontrolled Eating
UE	Uncontrolled Eating
VLCD	Very Low Calorie Diet
WC	Waist circumference
WHR	Waist-to-hip ratio
WHO	World Health Organisation

PREFACE

In the 1990s I was engaged as a counsellor/therapist by the social insurance agencies in the Västmanland and Stockholm municipalities. My task was to treat people who were on long term sick leave with the diagnosis of fatique and depression. The aim was to help them recover from their depression, with the objective of returning to full time or part time work. The treatment, which was carried out on an individual basis, was based on cognitive psychotherapy. As the demand for interventions increased I developed group-related treatments for the target group which involved 2 hours per week for 10 weeks. The group format proved to be a positive form of treatment for a patient whose diagnosis is often associated with an inability to handle stress, a negative self-image and dysfunctional thought patterns.

The participants, who were on sick leave for burnout, included several women who were substantially overweight. These had previously undergone treatments for their obesity and had tried a number of diets. At some point after the programme ended a spokesman for the participants said that the cognitive programme provided them with knowledge which they found clarified the causes of their overeating. They also believed that the programme equipped them to deal with these causes, which led to a reduction in weight. The experiences of the participants made me interested in developing methodology for a cognitive treatment programme for people with obesity.

My ambition was to create a cognitive model that could be used in practical clinical treatment, for both health care personnel and for the obese patients. The hypothesis was that it is possible to successfully initiate a structured cognitive treatment programme as a method to use in health care. The treatment programme I designed was included as one of three alternatives for the treatment of obesity in a collaborative project between the county council and the social insurance agencies in Västmanland in 1996-97. The other alternatives comprised a programme with VLCD products and a programme which was in accordance with standard healthcare practice: knowledge of diet and exercise. A total of 54 individuals took part in the project, all with a BMI >30. All programmes lasted for 10 weeks, with three follow-up meetings. At the final meeting, 2 years after the end of the programme, it was observed that only those who had participated in the cognitive programme showed weight loss. The results aroused my interest in carrying out a scientific assessment of the effect of the cognitive treatment programme on weight patterns in individuals with obesity. This paper describes the manualised method I developed for treating obesity, as well as the method's efficacy and effectiveness in the group treatment of individuals diagnosed with obesity.

1 INTRODUCTION

1.1 Obesity – a worldwide health problem

Globally

In 1977 Obesity was described by the World Health Organization (WHO) as a "global epidemic" and was classified as a disease (1). Since then, the prevalence of obesity has increased globally and between 1980 and 2014 it has doubled (2). In 2014 there were more than 1.9 billion adults aged 18 years and older who were overweight or obese. Of these over 600 million were obese. About 13% of the world's adult population (11% of men and 15% of women) were obese at this time, while 39% of adults aged 18 years and over (38% of men and 40% of women) were overweight. Despite the fact that obesity is a major health problem very common in the Western industrialized countries, affecting all levels of society, the obesity problem is not confined to the western world; most of the world's population live in countries where being overweight or obese results in more deaths than being underweight (2).

Obesity amongst women has increased throughout the world. In North America, the USA is noticeable for its high prevalence of obesity, with 31.6% of men and 33.9% of women being obese. In fourteen countries in Central and Latin America the prevalence of obesity in women is greater than 20%, while the highest prevalence of obesity is observed among South African women: 42.0% in 2013. More than 50% of the obese individuals in the world live in just 10 countries. The USA, China and India are the leading countries. In 2013 the USA accounted for 13% of obese people in the world, with China and India jointly accounting for another 15%. A long-term study conducted over three decades suggested that there has been no slowing in the increases in in countries that already had higher rates of obesity in 1980 (3). Also, the proportion of people in Asia who are overweight or obese, which was previously low, is now growing rapidly. During the period 1992-2002 the overall prevalence of overweightness and obesity amongst Chinese people increased by 38.6% and 80.6% (4).

Sweden

According to SCB's study in 2007, which was based on self-reported figures, the proportion of men and women with obesity increased from 5 to 10 percent over the period 1980-2005. The increase in obesity was primarily in young women and by 2005 obesity in women was as common as in men. The study showed that being overweight was significantly more common amongst men than women; whereas around half of the men were overweight or had varying degrees of obesity, the corresponding proportion in the women was one third. Obesity has increased in all socioeconomic groups, but most of all amongst male workers (5).

The proportion of people who are overweight or obese has been seen to stabilise since the turn of the century (6,7). However, the studies are based on self-reporting of weight which makes the information uncertain because of the general tendency towards under-reporting. (8). For example, studies in Gothenburg and northern Sweden (the MONICA studies) show a

greater prevalence of obesity than is recorded in the self-reported figures of Statistics Sweden (Statistiska centralbyrån) (9,10).

In 2014 the proportion of people in Sweden who were obese increased to 12% for both women and men, while 28% of women and 42% of men were overweight. Obesity has increased most amongst young women and is now almost as common as obesity amongst young men. The proportion of middleaged women (aged 45-64) with obesity has fallen, whereas the proportions amongst both younger and older women have increased. A total of nearly a million Swedish women and men are now obese, while 2.8 million women and men are overweight (11).

1.2 Definitions of obesity

Body Mass Index (BMI)

Obesity is characterized by an excess of body fat (12). The proportion fat in the body varies between men and women and between younger and older people. Whereas the mass of body fat in middle aged men is between 10 and 15 kg, the normal body fat mass in women of an equivalent age is circa 50 % higher. Body fat mass is however difficult to measure and the body mass index, BMI, is used as the most common way to define obesity and to measure both body fat and muscle mass. BMI is defined as body weight (kg)/height^2 (m^2). The WHO Expert Committee has proposed a classification according to BMI (Table 1) (13).

Table 1. BMI classification (WHO)

BMI classes	Nutritional status
Below 18.5	Underweight
18.5–24.9	Normal weight
25.0–29.9	Pre-obesity (Overweight)
30.0–34.9	Obesity class 1
35.0–39.9	Obesity class 2
40.0 and above	Obesity class 3

However, the ranges given by WHO for normal weight, overweightness and obesity are quite arbitrary. They are the same for both men and women despite the fact that men have consistently higher BMIs than women (14). Nor do the risks associated with excessive body fat depend solely on the amount of fat but also on how it is distributed throughout the body (15). The value of BMI measurements is also limited by the fact that they does not differentiate between fat and muscles. In one study some individuals had a high BMI but a low waist measurement, which can be explained by the fact that BMI does not differentiate

between the weight of fat and the weight of muscles (16). The risks for women and men with high BMI are highest below the age of 50 (17).

Abdominal obesity

Studies emphasize the need to use waist measurements to a greater extent because this value is well correlated with abdominal obesity and therefore increased risk of cardiovascular disease (18,16). Abdominal obesity, amongst other things, poses an increased risk of developing type 2 diabetes and cardiovascular disease (19, 20). The measurement of waist circumference, waist-to-hip ratio and abdominal height (Table 2) are increasingly common methods for assessing obesity. Because both men and women tend to put on extra weight around the waist, the waist-to-hip-ratio (WHR) is used to evaluate abdominal obesity. However, studies show that waist measurement is better for recording the amount of fat around the waist region and gives fewer false readings than the waist-to-hip ratio, as the latter combines two values (21,18).

Table 2. Measures and threshold values for abdominal obesity

Measurement	Men	Women
1.Waist circumference (WC)		
Threshold values (increased risk)	94 cm	80 cm
Threshold values (substantially increased risk)	102 cm	88 cm
2. Waist-to-hip ratio (WHR)		
Threshold values	1.0	0.85
3. Sagittal abdominal diameter (SAD)		
Threshold values abdominal height	22 cm	20 cm

1. Measuring abdominal fat. A circumference above 80 cm for women and 94 for men should be a signal to not gain any further weight (22).

2. Measuring waist size in relation to hip size. The ratio is obtained by measuring the waist circumference and dividing by the circumference of the hips (23).

3. Measuring fat within the abdomen. Abdominal height is measured with the patient lying on his/her back; it is the distance between the flat surface and the highest point on the abdomen (24).

1.3 Causes and risk factors

Genetics

Obesity is generally considered to be linked to lifestyle: the association between diet and exercise. However, this assumption is not necessarily correct: some people become fatter than others despite having roughly the same lifestyle. According to Stunkard and colleagues most researchers are agreed that the causes of obesity also include strong genetic influences, meaning inherited susceptibility to developing obesity (25). Studies of twins show that identical twins, who have grown up in different environments after adoption, have major similarities in body weight and body fat mass, despite differences in the eating habits and degrees of obesity of their adopted families. According to a Finnish twins study, genes account for circa 30-50 % of the development of obesity (26). Another Finnish study with twins (27) showed genetic involvement in fondness for sweet and fatty foods in emotional eating and eating in response to stress.

It is known that a number of genes are associated with obesity. One of these is the FTO gene. Circa 16 % of all Europeans have two copies of an obesity-risk variant of the FTO gene and a further 47 % have a single copy of this variant (28). A Swedish study based on extensive material from the so-called Malmö Kost Cancer (Malmö Diet and Cancer) study shows that individuals who both eat fatty foods and have two copies of the obesity-risk variant of the FTO gene have more than double the risk of becoming fat than those without the obesity-risk variant. However, the absolute effect is only modest. An individual with the double FTO gene obesity-risk variant weighs circa 3 kg more than an individual without this variant. (29). Another study found several unknown risk genes and molecular mechanisms which link the risk of obesity with the risk of disease. 49 different risk gene variants were found which could be directly linked to obesity measured around the waist and hips. 19 of them displayed a stronger effect in women. (30).

Demographic factors

- Age. Obesity increases with age up to at least the age of 55 in men and 70 in women (31).
- Ethnicity. There are large, usually unexplained variations between ethnic groups (31).
- Marital status. Obesity usually increases after marriage (31).

Lapidus et al. describe a type of woman who is generally obese in as follow: she belongs to a low social class, has a low standard of education and is often married. Her health status is generally good except that she has a higher incidence of gall stones. She has no psychiatric or behavioural problems, smokes less and consumes less alcohol (32).

Socio-economic factors in Sweden

There is a difference in the prevalence of obesity between the populations of small towns and those of cities. The proportion of people with obesity is lower in cities and suburban

municipalities than in smaller municipalities: 9.2 % and 14.6 %, respectively (11). Communities with a higher proportion of people on benefits also have a higher proportion of people with obesity (33). However, obesity has generally increased in all socio-economic groups during the 2000s but has fallen amongst senior and middle-ranking white-collar workers as well as in people with higher education (34, 35).

A study in Västerbotten showed that the incidence of obesity over the period 2002-2007 was virtually twice as high in individuals with less than nine years of education than in those who had undergone higher education (33). Kark et al. noted that mean BMI and the prevalence of overweightness and obesity was higher among young men whose mothers had only a low standard of eduction than among those whose mothers had a high standard of education (36). An association between obesity and education could also be seen in 1980 in Skaraborg County amongst individuals with low or high levels of education, though the gap between them had decreased by 2002 (35). By comparison, SCB showed that in 2014-2015 obesity was more common amongst individuals with only basic education (14%) than amongst those with higher education (8.5%) (11). Obesity is therefore is almost twice as common in individuals with less than nine years of education than in those with higher education.

Parity

More than half of all Swedish women with morbid obesity have said that pregnancy was the overriding factor in their weight gain (37). Several studies have shown that pregnancy presents an increased risk of lasting weight gain after childbirth. One study shows that 73% of women with obesity had retained 10 kg of extra weight after pregnancy (38). Similar results were recorded in another study (39) and at the same time yet another study showed that women who had not returned to their original weight after childbirth had an increased risk of remaining at their new weight (40).

Drugs

Weight gain is a potential side effect with a number of drugs. A systematic review and meta-analysis has summarized information on a number of antiepileptics, neuroleptics, antidepressants and blood glucose lowering agents which can contribute to weight gain and weight reduction (41). Amongst antiepileptics, Gabapentin caused a weight gain of 2.2 kg after 1.5 months, whereas Lamotrigine was weight neutral. The following neuroleptics resulted in weight gain within an average of three months: olanzapine (2.4 kg), quetiapine (1.1 kg) and risperidone (0.8 kg). Weight loss was observed with bupropion (1.3 kg) and fluoxetine (13 kg). Lithium was weight neutral. Antidepressant treatment with amitriptyline (1.8 kg) and mirtazapine (1.5 kg) caused weight gain. A number of drugs are not included in this collation because of a lack of information about their effect on weight. For example, it does not include clozapine and insulin products, with which weight gain is a known clinical problem. These studies also have the disadvantage that the follow-up periods were often short. Also, they do not give any information about whether weight gain persists after treatment is ended (41).

Stress

There is no generally accepted definition of stress. The concept of "stress" takes many different forms in both everyday speech and scientific contexts. Selye, who created a detailed model of stress, defined the concept as the body's general, non-specific response to challenging stimuli and considered that this non-specific response followed a definite general pattern, which he termed "general adaptation syndrome" (GAS) (42). GAS comprises three phases: 1. the alarm phase, with physiological changes such as hormone release; 2. the resistance stage, when stress hormones remain at high levels. If the stressors become too great then 3. the exhaustion stage, is entered, in which the body's defence can no longer deal with any demands without stress symptoms appearing and illness can then develop.

Selye divided the concept of stress into negative stress (distress/feelings of anger and aggression) and positive stress (eustress/empathy, stimulation and positive energy.) The theory of distress and eustress has only weak empirical support as positive stress, if it is long term, can also have negative effects on health. A stress reaction can be triggered by biological, social and psychological stressors. It may also be either positive or negative. In daily life we often use the term "stress" to describe negative situations.

Another common misconception is that stress is synonymous with rushing around. However, biological differences have been found between hormonal reactions to positive and negative stress (43). In a demanding but positive situation, in which one feels in control, it is mainly noradrenaline that is released from the adrenal glands, and the body quickly returns to normal. However, in demanding situations where one does not feel in control, more adrenaline is released than noradrenaline and cortisol is also secreted from the adrenal cortex. According to the theory, which is based on the demand-control model, the combination of high demands with only small possibilities for exercising control over one's work situation, can be associated with health risks (44). High cortisol levels have been shown to be associated with feelings of helplessness and dejection. This negative stress can also be generated by situations with low demand and low stimulation, combined with low control, such as extremely monotonous, routine work tasks (43,45).

In the 1990s in Sweden, working life became more demanding while at the same time the potential for workers to control their workplace situations decreased. More and more workers were signed off work with burnout and around half of women found their work to be mentally exhausting (46). During the same period an increase was observed in the diagnosis of obesity. Since the start of the 1980s the proportion of obese individuals has increased in all groups of society from 5% to 10% in 2005, and over the same period the proportion of Swedish women who were overweight increased from 22% to 26%, with the proportion in men rising from 30% to 41%. (5). Stress can cause people who are overweight or obese to eat too much and, according to Björntorp et al, there is a reason why it is assumed that stress in modern society contributes to obesity, which develops through an increase in cortisol and leptin concentrations, which in turn increases the risk of cardiovascular disease, type 2 diabetes and stroke (47, 56).

How we understand and interpret what happens to us in our everyday lives reflects both our experience of stress and how we deal with it. A stress reaction often leads to an experience of pressure and inadequacy, which in turn can lead to a loss of control in situations which concern both private life and work. It is therefore easy to find oneself in a vicious circle and to subconsciously develop compensatory strategies: eating too much, drinking too much, smoking too much or devoting oneself to excessive physical training. Studies show that individuals who binge eat to a greater extent than others employ avoidance strategies such as overeating, instead of resolving the problem that is causing them stress (48).

In a longitudinal population study which included 1355 men and women, Block et al. examined the relationship between psychosocial stress and weight gain in obese subjects (49). The results show that in men weight gain was associated with increasing levels of psychosocial stress associated with job-related demands and economic problems. Among women, weight gain was associated with job-related demands and economic problems, but also with strains in family relationships. The authors therefore concluded that interventions to address psychosocial stress may restrict weight gain amongst overweight and obese men and women (49).

To examine the relationship between psychosocial working conditions and weight gain amongst employees, a study analysed data from postal questionnaires sent to 40- to 60-year-old women (n=7093) and men (n=1799) employed by the City of Helsinki in 2000-2002, containing questions about weight gain over the last 12 months. 25% of the women and 19% of the men reported weight gain. Work, tiredness and overtime were associated with weight gain in both sexes, whereas the dissatisfaction of women with the combination of paid employment and family life meant an increased risk of weight gain (50). Epel et al. considered that stress also caused increased food intake, but that this was only the case when the stress was followed by a neuroendocrine reaction which raised cortisol levels and contributed to abdominal obesity (51). Cross-sectional studies by Overgaard et al. reported only a weak association between psychosocial stress and BMI (52). However, another study which followed the weight patterns of 1355 individuals from baseline and in follow-ups nine years later found that in men and women with high baseline body mass index, psychosocial stress led to weight gain in the years between baseline and follow-up. The authors concluded that "interventions to address psychosocial stress may limit weight gain among overweight and obese men and women" (53). Even though studies have shown contradictory results regarding the question of whether people generally eat more in psychologically stressful situations, most agree that the mental and biological strain involved in stress can cause the development of obesity, particularly the harmful form of abdominal obesity (54). This is due to cortisol activating the uptake of fat into adipose depots and at the same time, in the presence of insulin, inhibiting fat release from these depots (55).

Stress triggers several types of reaction, the most common of which is activation of the sympathetic nervous system and the hypothalamic–pituitary–adrenal axis (55, 56). At the same time as cortisol increases the appetite for fatty and sweet foods, it also contributes to

excess weight, particularly in the abdomen (visceral fat). Comfort food, on the other hand, contributes to an increase in the secretion of opioids which reduce activity in the HPA axis and moderate the stress reaction (57, 58,). In my clinical practice I have often met patients who claimed that stress produced a strong desire for "comfort food", and that it was precisely this type of food that reduced their experiences of stress. In studies in rats Pecoraro et al. were able to show that our patients were right: stress creates a preference for comfort food, at the same time as comfort food also appears to reduce experiences of stress (59, 60).

There was previously a common and popular perception that obesity was linked to psychopathology (61). However, this notion has since changed. The prevailing opinion among most researchers is that, if psychological disturbances occur in obesity, these are a consequence and not a cause of the obesity (62, 63, 64). A cross sectional study which examined the relationships between obesity and a number of psychosocial variables studied 800 women aged 38–54 years found no significant relationship between obesity and present or previous mental illness (current disability rating, depth of depression, frequency of anxiety attacks, phobia rating, current use of psychotropic drugs, maximum disability rating to date,history of psychotropic drug use or contact with a psychiatrist) (65). A longitudinal study of the same cohort showed that depression at baseline predicted weight gain six years afterwards (66).

Binge Eating Disorder (BED)

BED is characterized by recurrent episodes of binge eating at least two days a week, for a six month period, but without the compensatory behaviour that characterises Bulimia nervosa: vomiting and laxative abuse (67). The prevalence of BED in the general population is 1.5% to 2% (68, 69). Individuals with BED typically present for treatment with the multiple problems of binge eating, eating disorders, psychiatric symptoms and overweightness (70). Binge eating was first described by Stunkard in 1959 as an obese eating pattern (71). BED was introduced as an eating disorder in the DSM-IV (72). Most patients with BED are overweight or obese. Studies indicate a BED prevalence of around 30% among obese adults who seek treatment (73). BED is associated with increased psychopathology, including depression and personality disorders and might be a negative predictor of weight loss (74). In a comprehensive literature search Nicholls et al. concluded that negative mood, sadness, tension and emotional instability were antecedents of BED (75).

Cognitive behavioural therapy (CBT) is the most well-established psychotherapeutic treatment for BED (76, 77). Agras et al. reported a weight gain of 3.6 kg in subjects who continued binging at the 1-year follow-up, while those who stopped binging were 4 kg below their baseline weight (78). Cognitive behavioural therapy appears to be successful in reducing binge eating in the short term, and studies show that obese binge eaters, more often than other obesity patients, discontinue the treatment when they take part in conventional weight reduction programmes (79, 80).

Weight cycling

Weight cycling (weight loss followed by weight regain) may lead to a metabolic slowdown that makes the body efficient at fat utilization and storage over time, but this suggestion is not generally accepted (81). However, weight cycling might be linked to increased psychopathology, lower life satisfaction and a more disturbed eating pattern (82). Many epidemiological studies also show a positive relationship between weight cycling and mortality, in that repeated periods of weight cycling are thought to impose a strain on the cardiovascular system that can lead to cardiovascular morbidity (82, 83).

1.4 Obesity as a risk factor for physical diseases

Weight loss benefit

Obesity develops after a long period of imbalance between energy intake and energy consumption. An average energy surplus of ca. 50 kilocalories (kcal) per day is sufficient for obesity to develop over a five-year period if this energy surplus is not compensated for by physical activity (14). The obesity treatment principles are mainly the same today as 30 years ago (84). Obesity is associated with several major health problems: diabetes, high blood pressure, certain forms of cancer, sleep apnoea, heart failure and stroke (85, 86). It has been increasingly clear that modest weight loss is associated with significant health benefits. A five-to-ten percent reduction in weight is accompanied by clinically important improvements in cholesterol and other health indices (87).

The Institute of Medicine publication Weighing the Options: Criteria for Evaluating Weight-Management Programmes define treatment success as a 5-10% weight loss maintained for at least one year (88). Similarly, a report from the UK Royal College of Physicians likewise defines “successful” weight loss as a loss of more than 5% of initial weight (89). However, these criteria are not in line with the more conventional objectives of achieving greater weight loss and new approaches to the prevention of weight regain (90).

Mortality

Most studies show a relationship between high BMI and mortality. The high mortality at higher BMI is mainly associated with cardiovascular disease. All prospective studies with more than 20,000 participants and virtually all cohort studies with more than 7000 participants have shown a two-fold increase in total mortality in individuals with morbid obesity (91, 92).

Cardiovascular disease

Obesity is associated with cardiovascular risk factors, abnormal blood lipid concentrations and non-insulin-dependent diabetes mellitus – a combination referred to as the metabolic syndrome. The risks of heart failure and sudden death increase, as do the risks of other cardiovascular diseases, such as stroke, effort angina and myocardial infarction. A number of studies have shown that the relative risk of death from cardiovascular disease in obesity is

lower for elderly people than for young and middle aged people, while a study involving 17,000 women aged 25–64 showed that mortality was similar for all BMI values (93). A strong link has been demonstrated between myocardial infarction and abdominal obesity in both women and men (91), while later studies have shown that mortality is higher in older people with obesity only if they also suffer from diabetes and cardiovascular disease (94, 95). However, in 2016 researchers at Umeå University have taken things a step further and shown in a study with twins that it is genetic inheritance that controls diseases such as myocardial infarction and stroke, and not obesity itself (96).

Diabetes mellitus (type 2)

A large number of clinical studies and cohort analyses have observed a strong association between obesity and type 2 diabetes (96, 97, 98). A man with a BMI over 35 has around a 40-times greater risk of developing type 2 diabetes than a man of normal weight and a woman with a BMI over 35 has nearly 100 times the risk that a woman of normal weight has (97, 98).

Type 2 diabetes is thought to occur because of reduced sensitivity to insulin in the body's tissues and because of reduced insulin production. The serious complications that can occur in diabetes are retinal changes, which can lead to blindness, and renal failure, which will require dialysis or a kidney transplant. Weight reduction and increased physical activity can improve sensitivity to insulin and result in more normal blood sugar levels.

Metabolic syndrome (Mets)

The official definitions of MetS from the USA and WHO are mainly based on criteria for obesity, disrupted sugar metabolism, dyslipidaemia and high blood pressure. Type 2 diabetes, high blood pressure, myocardial infarction, stroke and dyslipidaemia often occur together with obesity. These illnesses are thought to have a common background and are included under the term “metabolic syndrome” in which insulin resistance and increased amounts of abdominal fat are important components (15). Other factors that are known to activate stress reactions in the brain, such as high alcohol consumption, smoking, depression and anxiety related to psychosocial factors, can also contribute to the development of this syndrome. Therefore metabolic syndrome appears to be influenced by both lifestyle factors and psychosocial conditions (99). In a review, Blair and Brodney ask the question, what is the greatest risk factor for mortality: being overweight or being physically inactive? After examining 24 prospective observational studies, it was concluded that physical activity not only reduces health risks associated with being overweight or obese but also reduces morbidity and mortality in individuals who are overweight or obese to below the levels seen in people of normal weight who have sedentary lifestyles (100).

Abdominal obesity is the characteristic form of obesity in MetS, with a clinical increase in abdominal girth. Atherogenic dyslipidaemia is characterized by high triglyceride levels and low HDL-cholesterol levels. High blood pressure is strongly associated with being

overweight and with insulin resistance. Almost all patients with MetS have insulin resistance. Drug treatment can contribute to insulin resistance and metabolic syndrome, as with high doses of beta blockers or thiazide diuretics; British therapeutic recommendations do warn about this. Patients who are treated for schizophrenia with neuroleptics often develop metabolic syndrome (101).

Cancer

Cancer is the second biggest cause of death in Sweden. Obesity and being overweight are thought to increase the risk of various forms of cancer. This mainly concerns hormone-based diseases such as uterine and breast cancer in women and prostate cancer in men, as well as cancers of the kidney, gall bladder and gastrointestinal tract. In a study which merged data from obese patients admitted to hospital under the Swedish Cancer Registry, the authors conclude that obesity is associated with more forms of cancer than had previously been thought. Obesity has increased the total incidence of cancer by 25% in men and 37% in women (102).

Sleep apnoea

Sleep apnoea is very prevalent (75.9 – 86.9 %) amongst all BMI groups who seek bariatric surgery (103). According to Mohsenin there is new evidence that obstructive sleep apnoea increases the risk of stroke, independently of traditional risk factors. Obstructive sleep apnoea occurs amongst the majority of stroke patients (104). The fact that overweight individuals are more likely to suffer from sleep apnoea is probably due to the accumulation of fat under the tongue, in the palate and in the throat, which makes it more difficult for air to pass through. Another cause is the abdomen pressing up towards the chest and respiratory organs. Untreated sleep apnoea can increase the risk of high blood pressure, heart arrhythmias, heart attack, heart failure, stroke, obesity, and diabetes (105). The tiredness and sleepiness caused by untreated sleep apnoea often result in patients feeling that they are unable to exercise in order to burn off the extra calories.

Other diseases

Non-insulin-dependent diabetes, gallbladder disease, reflux oesophagitis, hiatus hernia and osteo-arthritis in hip and knee joints are common complications of obesity (106).

1.5 Psychosocial consequences

The negative effects of obesity on psychosocial functioning are significant and these disturbances are associated with poor mental wellbeing (107). In the SOS (Swedish obese subjects) subjects reported anxiety/depression to a greater extent than the controls and their reported quality of life was worse than that of individuals with chronic pain and cancer survivors. The obese women also had an even worse quality of life than the obese men (108). Obesity in itself often causes fewer problems for patients than do the psychological problems that can arise as a result of reactions and comments they may encounter within their social

environment. Numerous studies have demonstrated a relationship between obesity/overweightness in women and low socioeconomic status as measured by educational attainment, social class, income, employment status and occupational position (109, 110), which can mean discrimination in both employment and rates of pay for people who are obese (111). Therefore weight reduction in obese individuals often results in improvements in psychosocial functioning as well as a sense of mental wellbeing stemming from their progress in maintaining a lower weight (112).

2 BACKGROUND

2.1 Non-psychological obesity treatment studies

Conservative treatment programmes

Diet and physical exercise are the cornerstones of obesity treatment. Programmes including diet and exercise often recommend a 0.5 kg weight loss over one week. People often lose weight while they are participating in a programme and are being given guidance. But they often regain weight after the end of the programme. The aim of obesity treatment is to reduce the total energy intake. A practical approach is to reduce the intake of dietary fat (113).

A general recommendation is that 30% should came from fat, which is substantially lower than the average intake in adults (114). Other studies have indicated that energy density rather than macronutrient composition per se affects total energy intake (115). Exercise is an important target in obesity treatment. A review of randomised studies has shown that a combination of dietary awareness and exercise can produce a 20% greater weight loss both by the end of the treatment and for up to one year thereafter, when compared with dietary awareness alone. However, one year after the end of the programme nearly half the participants regained the weight they had lost, despite using the combined method (116). A controlled study in postmenopausal women showed positive effects from a combination of diet and aerobic exercise, other than just weight loss. The diet + exercise group showed an increase in metabolism when compared with the control group, as well as a fall in systolic blood pressure and a reduction in waist-to-hip ratio (117).

However, studies of the importance of increased activity in obesity are associated with a number of methodological problems. Controls were not used in the assessment of either food intake or level of activity as the data was based on self-reporting. In general one can conclude that physical activity increases weight loss, which produces significant secondary benefits (118). Tate and Wing believe that the best results with regard to weight loss are achieved with treatment programmes which offer a longer period of contact within the treatment programme, have more structured methods with regard to diet and have higher targets for physical activity (119).

Very low calorie diets (VLCD)

VLCD is a collective term for products with very low calorie content. The diets all provide an energy intake which does not exceed 800 kcal/day. The products are manufactured from high-quality protein sources, with the addition of essential fatty acids, vitamins, minerals and trace elements. VLCD is used as an individual treatment or as an introduction to a programme in which diet and exercise are combined. The currently accepted treatment period is 12 weeks, or sometimes 16 weeks. Treatment with highly nutritious products can, according to Livsmedelsverket (the National Food Agency), be conducted without medical supervision for up to two weeks. Especially rapid and large weight losses, as achieved by VLCD, are often followed by rapid and large weight gains in the follow-up period or even

during treatment, and sometimes result in smaller cumulative weight reductions than those achieved with less restrictive treatments (120). Wadden reported an RCT study in which 40 patients were monitored over one year. The VLCD group (420 kcal/day over 16 weeks) showed a significantly greater weight loss after six weeks when compared with a group which received only behavioural therapy and a low calorie diet. After one year the difference was not significant, 12.2 kg compared with 10.9 kg (121).

Drugs

The drug that is currently approved for the treatment of obesity in Sweden is Orlistat. Orlistat blocks the action of enzymes which break down neutral fat (triglycerides) in food to fatty acids. This reduces the uptake of fatty acids, cholesterol and fat-soluble vitamins (A, D, E and K) from the gastrointestinal tract. Side effects such as diarrhoea are caused by an increase in the amount of fat in the intestines, but are reduced when the food contains less fat. Orlistat was approved within the EU in 1999 (14).

A number of studies show that after one year the average weight reduction with Orlistat was ca. 3 kg more than with placebo treatment (122, 123). One review shows weight losses of 1.3 and 2.8 kg at 12 and 57 weeks of follow-up, resespectively after using Orlistat. A 4-year double blind prospective study showed increased weight loss in those who received Orlistat rather than placebo: 3.6 vs. 1.4 kg. Even though the weight reduction was only moderate the studies showed an improvement in quality of life and better control of glucose metabolism in diabetes patients treated with Orlistat (124, 125).

From and including 2010 sibutramine was no longer approved for sale in Sweden because of its side effects. Sibutramine was tested as an agent for treating depression but was shown to be ineffective. On the other hand, one of its side effects was weight loss. Dry mouth, sleep problems and constipation are very common side effects (>10 %) of sibutramine. Its common side effects include rapid pulse, palpitations, an increase in blood pressure, sweating and numbness. According to the study “Sibutramine Trial of Obesity Reduction and Maintenance” weight loss was positively affected by sibutramine, with a large initial weight loss when combined with physical activity (walking and cycling) and reduced TV watching (126). 605 obese patients took part in a combination programme involving sibutramine and a low calorie diet (max. 600 kcal/day). The programme achieved weight loss in 77% of obese patients and sustained weight loss in most patients who continued with therapy for 2 years (127).

Surgery

Bariatric surgery can be seen as a radical way of imposing a change in eating behaviour in obese individuals, with the aim of bringing about weight loss. Three surgical methods are currently used in Sweden and the rest of the world: gastric banding, vertical banded gastroplasty and gastric bypass. Nowadays it is primarily gastric bypass that is used, above all because this method gives greater weight loss than the other methods (128).

4047 obese individuals took part in the prospective controlled Swedish Obese Subjects (SOS) study. 2010 of them were treated with bariatric surgery and 2027 controls received traditional treatment at health centres. Follow-up data from up to10 years after the operation indicated that bariatric surgery gave substantial weight loss immediately after the operation, with a 16% weight loss being observed at follow-ups up to 10 years later. The large weight loss also resulted in improved health. Two years after the operation patients had improved values for blood pressure, blood lipids and other risk factors. At both 2 and 10 years after the operation, the patients had a lower prevalence of diabetes than the control group. A total of 5 of the 2010 patients who underwent surgery died as a result of the operation, which gave a mortality rate of 0.25 %. The study observed that bariatric surgery for severe obesity was associated with long-term weight loss and decreased overall mortality (129, 130,131). A majority of researchers are critical of bariatric surgery (132, 133). Koanders et al. believe that bariatric surgery only treats the symptom and not the disease and say: “this is like treating an alcoholic by surgically removing his bladder” (133). Colles et al. stress that bariatric surgery does not treat underlying problems such as emotional eating (134), while other studies show that, despite the surgery, it is possible for small amounts of food to be consumed over long periods of time, so-called grazing, which works against the maintenance of weight loss (135).

2.2 Psychological obesity therapies

Behavioural therapy

In the 1960s traditional treatment programmes for obesity were complemented with contributions from behavioural therapy. Behavioural therapy is based on the basic principles of learning. The goal is behavioural modification; to increase the number of desirable behavioural traits and to reduce the frequency of undesirable forms of behaviour (136).

Behavioural therapy including very low calorie diets has been established as an effective treatment method for weight loss. Behavioural treatment was developed in the 1960s and has be en refined over the years (137). A typical behavioural programme, incorporating 1200 kcal/day, produces a weight loss of about 10 percent of initial body weight among those patients who complete the treatment (about 80 percent) (138). Like most other therapeutic modalities for obesity, behavioural therapy does not lead to long-term maintenance of weight loss. Participants generally lose weight and maintain the weight loss while they are still engaged in behavioural therapy programmes, but, when they withdraw from treatment, more often than not they gradually regain the weight they had lost (139). About 40 percent is regained over the first year following treatment and much of the rest over the following three years (140).

Thus non–surgical evidence–based treatments for obesity produce, on average, about ten percent weight loss among treatment completers, with the weight lost being regained once treatment is stopped (90, 141). Several other authors have come to the same conclusion: Behavioural programmes are commonly associated with a pattern of weight regain after treatments end (142), but Papp and colleagues showed in a one-year follow-up study that the majority of participants (75.8%) did keep their minimum weight loss of 5% (136).

Cognitive behavioural therapy

At the end of the 1990s the concept of cognitive behavioural therapy (CBT) began to be used in the treatment of obesity. As with behavioural therapy, the goal of CBT is to changenegative behaviour, but CBT is based on the assumption that individuals' perception of themselves and their interpretation of a particular situation affect their feelings and behaviour. Therefore the treatment involves strategies which aim to increase patients' awareness of their own negative thought processes which lead to negative feelings and negative behaviour, so that through self-understanding they are able to alter their adverse behaviour (90, 141).

Sbrocco and colleagues compared in a study (N=24) a traditional behavioural weight-management programme with a cognitive–behavioural (CBT) programme (13 weeks, 1.5 hour group treatment sessions). In the follow-up 1 year after the end of the programme the CBT group showed a 10 kg decrease in weight, whereas the BT group showed a 4.3 kg increase (143). Spahn et al. reviewed 87 articles and concluded that there was strong evidence to support CBT in facilitating the targeting of dietary habits, weight and cardiovascular and diabetes risk factors (144). Van Dorsten also shows in a review that cognitive interventions in the treatment of obesity not only produce weight loss but also significantly contribute to preventing health risks (145).

A randomised controlled trial compared M-CBT with standard cognitive-behavioural therapy (S-CBT). The M-CBT programme used behavioural and cognitive principles but alsoincorporated elements from psycho-education. Both programmes were successful at improving modest weight loss, as well as reducing distress, improving dietary quality and reducing cardiovascular disease risk factors. The mean weight loss by the end of treatment was 3.9 kg in the S-CBT group and 1.3 kg in the M-CBT group. By the 52-week follow-up, mean weight loss in the S-CBT group had reduced to 3.6 kg and increased slightly in the M-CBT group to 2.0 kg (146).

The positive weight loss effects of the combination of CBT, diet and exercise have been confirmed in a number of studies. Shaw el al. observed in a review that CBT, when combined with diet and exercise intervention, gave greater weight loss than diet and exercise alone (147). Painot et al. drew the same conclusions from a study with a 3-month follow-up which showed weight loss in the combined treatment group, but weight gain for the group treated with CBT only (148). Although the content of CBT programmes in obesity treatmentmay appear different, the review does provide support for CBT in the treatment of obesity (149), even though in a prospective study in primary care a more easily accessible CBT programme is called for, for use in primary care (150).

In their review of studies of the treatment of obesity focusing on cognitive and behavioural approaches, Van Dorsten and Lindley highlight the fact that a number of researchers emphasise the need for clearer cognitive components in cognitive behavioural therapy in order to improve both short- and long-term results with obesity, particularly in the treatment of patients who have experienced previous treatment failures (145). In a review, Fabricatore questions whether there is any difference between behavioural therapy and CBT, as both therapies aim to alter patients' eating patterns in order to achieve weight loss (151). Cooper, on the other hand, makes a clear distinction between cognitive behavioural therapy, the aim

of which is to alter behaviour by changing thought patterns, and behavioural therapy, which does not consider that cognitive changes are necessary to alter behaviour (90).

The question as to whether occupational groups other than certified CBT therapists can treat patients with obesity has not been much discussed but has been brought more into the spotlight because of the global growth in obesity. As early as 1976 Paulsen et al. wrote in a study: "To reach large groups of persons that are overweight or obese, cognitive programs have been sought that can be used by professional groups other than the traditional ones within psychiatry. There is good reason to believe that dietitians and registered nurses with special training and support could successfully use CBT in persons who are overweight. For instance, CBT could be used within a program that also includespsychological goals such as mood changes, self-esteem and a better understanding of stress." (152). Liao also expresses the opinion that it should be possible to use cognitive programmes with exercise in the treatment of obesity by occupational groups (dietitians, nurses) other than the traditional ones within psychiatry (141).

Van Dorsten observed that, despite increased awareness and provision of treatment, the global incidence of obesity is continuing to grow. He therefore thinks that, besides Paulsen's and Liaos' suggestions of increased training in cognitive behavioural therapy for non-traditional psychiatric personnel and weight specialists, the development of web-based obesity treatment programmes is important (145).

2.3 Eating behaviour

Theory

The background to eating behaviour in obesity has been explained via three theories: Psychosomatic theory (153), Externality theory (154), and Restrained Eating (155, 156).

The observation that feelings trigger eating in people who are overweight forms the basis of the psychosomatic model of obesity. As early as 1957 Kaplan and Kaplan concluded that the ultimate cause of obesity was psychologically induced hyperphagia (153). Their anxiety reduction model showed that eating reduced anxiety in overweight individuals, which led to the assertion that it was this association which developed and maintained hyperphagia (153). The supposition that feelings have a powerful influence on eating, hereinafter referred to as emotional eating, has generated major interest in clinical and experimental studies. The study shows evidence of an association between negative feelings and overeating, mainly in overweight women, but with less consistent results in overweight men (163). Externality theory is based on the concept of external eating which alludes to eating as a response to food-related stimuli, regardless of hunger or satiety, whereas restrained eating can involve behaviour from both emotional eating and external eating (155, 156). However, Van Strien expressed reservations over these conclusions. In a study about the interrelationships between the three eating behaviour scales, restrained eating was found not to be related to the other two eating behaviour components (157).

Hamburger described the following four eating patterns in obesity: 1. Overeating as a response to non-specific stressors (reported in 54% of patients). Where patients were

lonely, bored or worried, an interim goal could help them feel better, even if they had recently eaten. 2. Overeating as compensation for an unacceptable life pattern (39%). 3. Overeating as a symptom of depression (44%). 4. Overeating as an expression of desire (craving) for food (44%) (158).

In the study of eating behaviour three self rating questionnaires have been used: the Restraint Scale (156), the Dutch Eating Behaviour Questionnaire (159), and the Three-Factor Eating Questionnaire (TFEQ)(160). Karlson et al. (2000) developed a revised version of TFEQ, including 18 items containing three scales corresponding to Emotional Eating (EE), Uncontrolled Eating (UE) and Cognitive Restraint (CR) in order to evaluate their applicability to overweight men and women (161). Emotional Eating (EE) is explained by an inability to resist emotional signals, Uncontrolled Eating (UE) by a tendency to eat more than normal because of loss of control, and Cognitive Restraint (CR) by a conscious restriction of food intake to control body weight or promote weight loss (161).

Emotional eating (EE)

Addressing the "obesity problem" is generally considered to involve changing lifestyle factors. However, Koenders et al. do not support this idea. In a study involving 1562 employees which investigated the association between emotional eating, lifestyle factors (diet, exercise, smoking, alcohol) and changes in BMI, it was instead concluded that it is feelings which influence the eating patterns of people who are overweight and that " If we want to cure the disease, psychological treatment strategies have to be developed"(162).

A study which used the Three-Factor Eating Questionnaire (TFEQ) examined eating patterns in 4377 obese women and men showed emotional eating in half the participants, with significantly greater prevalence amongst the women (163). Feelings associated with grief or disappointment, experiences of being abused or misunderstood, thoughts of worthlessness and shame can all lead to worry and anxiety. If the causes of worry and anxiety are not revealed and worked on (and the question is asked whether there is good reason for this worry and anxiety), a strategy of living with these feelings could lead to a negative eating pattern, that is to say, emotional eating. Emotional eating can therefore be seen as a form of coping emotionally by minimising, controlling and preventing emotional stress, in the hope of alleviating negative feelings (164). According to Blandine de Lauzon et al, emotional eating is defined as "an inability to resist emotional signals" and according to Spoor et al. as "an increase in food intake in response to negative emotions"(163, 164). In a review Ganley (1989) concludes that emotional eating, in association with anger, melancholy, stress, depression and loneliness, can be found in ¾ of mildly-to-morbidly overweight patients who seek treatment for their obesity (165). With regard to the association between weight gain and emotional eating, Keskitalo raises the question of whether the weight gain leads to emotional eating or whether people with a tendency towards emotional eating gain weight more easily than do others (27). Another study, aimed at comparing weight regainers with women of a healthy weight, found that psychological factors such as self-esteem, dichotomous thinking and a tendency to regulate one's mood by eating were contributory causes of weight gain (166).

Similar conclusions were arrived at in a study which compared 147 obese and normal-weight individuals and found that, whereas 98% of obese patients reported that anxiety and other negative feelings provoked a need to eat, only 43% of non-obese patients experienced this association. (167). Emotional eating is prevalent in morbidly obese people across socioeconomic levels, but in studies of Leon and of Spitzer there is speculation that emotional and psychological factors are associated with obesity in middle- and upper-class women because of the social stigma attached to obesity (165, 168, 169).

Buchanan studied individuals who had undergone group treatment for their obesity and found that their eating patterns were often related to feelings such as loneliness, anger or depression (170). Buchanan hypothesised that overeating was an expression of anger (170). Edelman (171) estimated that 84% of overeating by overweight women was triggered by various emotional states. Craddock emphasised both psychological and social factors as causes of overeating and considered that 81% of patients use eating as a compensation for specific disappointments and unhappiness in their lives (172). Ganley thinks that, even though the studies in his review do not focus on social causes of overeating, they do nevertheless point to obesity and emotional eating being linked to roles and interactions, while Rand and Bruch believe that emotional eating tends to occur episodically in response to psychological stimuli (173, 174, 175, 176). Van Strien and colleagues ask whether individuals with a high score on a scale for eating in response to negative emotions also show high food intake in response to positive emotions when compared with those who were low on the emotional eating scale (177). The results showed that whereas low emotional eaters ate comparable amounts when they felt sad or happy, high emotional eaters ate significantly more when feeling sad than when feeling happy (177). One study showed that personality traits contributed to eating patterns (178).

Many studies emphasise that difficulties in dealing with negative feelings, and feelings of dejection or anxiety, contribute to overeating and obesity – as do difficulties in managing everyday demands. Food, therefore, becomes a refuge in which unpleasant feelings are eased and which contributes to overeating, often termed “comfort eating”. Certainly, overeating can provide short-term solace when someone has feelings of dissatisfaction, grief or anger – but this does not last. Eating can also be a reaction to strong positive feelings. It is therefore not a question of overeating being used as consolation but rather being an expression of an individual's inability to accommodate strong feelings. According to Heatherton overeating can instead be regarded as a refuge from everyday negative stimuli which threaten one's self-esteem by switching the focus onto more pleasurable stimuli such as food (179). Spoor offers the same interpretation and sees overeating as an emotion-focused form of coping aimed at emotional stress. An individual thereby compensates for negative feelings such as anxiety and worry by eating: compensatory eating behaviour (180). This coping can therefore function as a temporary escape and compensation, but it does not offer any lasting relief. Instead, this escapist behaviour can give rise to more worry and anxiety, often associated with feelings of failure. A study has shown that individuals first stop emotional eating when this gives rise to negative feelings (181).

Emotional eating and food preference

Chronic life stress seems to be associated with a greater preference for energy- and nutrient-dense foods, namely those that are high in sugar and fat (182). A predilection for sweet and fatty foods in emotional eating and emotional stress have been observed in a Finnish study of twins (27). Levine et al. consider that this predilection may be due to sweet foods relieving the stress because of the rewarding effects of the ingestion of sugars (183). Many overweight people regard themselves as sugar-dependent and compare themselves with alcoholics. Their craving involves an intense desire to consume their favourite foods and is not the same as normal hunger. Foods with high sugar contents, such as chocolate, lead to sugar cravings through an interaction of glucose with the opioid system in the brain. Significant positive relationships between BMI and food cravings and between BMI and cravings for sweets, high-fat foods, carbohydrates and fast foods that are high in fats, were observed in one study, while another study noted an association between cravings and emotional eaters as well as individuals diagnosed with BED (184, 185). This association is not surprising, as Hill was able to show that food cravers have higher ratings of boredom and anxiety than non-cravers (186). Dysphoric mood was prominent prior to the cravings themselves. Hill observed that food cravings are closely associated with mood both as a antecedent to craving and also as a consequence of craving (186). Food craving may therefore be an anticipation of the rewarding effects that eating provides; a brief freedom from negative feelings.

Emotional eating and stress

People who suffer from being overweight/obese, or alternatively from stress-related illnesses such as burnout, often feel worried and anxious during their everyday lives. Both illnesses have similarities with regard to what triggers them. In overweight/obese individuals feelings of anxiety and worry can lead to escapist behaviour: compensatory eating behaviour. With stress, anxiety can give rise to escapist behaviour which expresses itself as work addiction –or its opposite, resignation and helplessness. Both compensatory eating behaviour andwork addiction can be seen as escapist behaviour aimed at overcoming a reality which involves emotional strains. The reverse side of escapist behaviour is that the briefsatisfaction it provides gives rise to those very feelings of worry and anxiety that the behaviour was intended to ease – something which was also confirmed by Bennet et al (181).

Psychological stress is a common cause of emotional eating and this has been confirmed by a number of studies (182, 187); in a meta-analysis Wardle concludes that psychosocial stress is certainly a risk factor for weight gain but the effects are very small (188). Stress resulting from personal or occupational pressure is seen as a risk factor for becoming overweight or obese through an alteration in behavioural patterns such as eating and physical activity (190).

Psychological characteristics associated with eating were identified in 192 undergraduates. Coping was characterized by greater BMI in emotion-triggered eating and perceived stress reactivity in females and by anger/frustration–eating in males (191). With regard to the relationship between stress and eating behaviour, it has been observed that recurring stress is associated with a major preference for food containing sugar and fat, particularly in men (182). An Australian study involving 2781 women and 2377 men, aimed at examining the association between stress and BMI, showed that psychosocial stress, both experienced stress

and stressful life events, were associated with weight gain over a 5-year period (192). Different types of stress also seemed to affect women and men differently. Whereas stress outside of work, such as in family relationships and other social relationships, was associated with weight gain in women but not in men. Work-related stress appeared to affect men more than women. The authors concluded that future treatment of overweightness and obesity should also consider psychosocial factors that can influence weight gain (192). Bennet et al. came to the same conclusion, finding that overeating did not generally reduce emotional suffering, but rather reinforced it through the guilt that was felt over eating (181). Qualitative analysis of gender differences with respect to the causes of emotional eating, revealed that women in particular identified stress as a cause, often because of feelings of guilt. Instead, men saw unpleasant feelings such as melancholy and anxiety as triggering factors and did not experience guilt as often after episodes of emotional eating. What men and women had in common was that during episodes of emotional eating they chose unhealthy foods. According to Bennet et al. the results point to a need for interdisciplinary scientific interventions, focusing on emotions, stress management and dietary knowledge, in order to reduce the risk of weight gain associated with emotional eating (181). Other authors (182, 187, 193) also support the idea that emotional eating is used as an effective coping strategy for dealing with negative influences. Research shows that emotional eating is common amongst individuals who suffer from bulimia nervosa. The causal chain is not thought to have been finally established, but certain types of behaviour are considered to be precursors to eating disorders – or to be a consequence of them (193). At the same time studies with twins have shown a genetic correlation between emotional eating and a liking for sweet and fatty foods, as well as a craving for sweet foods during emotional stress (27).

Uncontrolled Eating (UE)

Uncontrolled eating is defined as a tendency to eat more than normal because of a lack of control over how much one eats, often combined with subjective feelings of hunger (195). Loss of control is often expressed through habitual eating, something which has been confirmed by van Riet et al. in their observation that a number of studies show that habit is one of the most powerful predictors of eating behaviour yet few interventions that are based on habit theory have been tested in a food context (196). Habits are established by performing particular actions on a regular basis. If an action is carried out regularly it becomes more or less automatic and, with stimulus recognition, this can be sufficient for the brain to remember the action. When the stimulus disappears, the habit also tends to disappear (197, 198, 199).

A study examined relationships between eating patterns and attention/executive function testperformance (194). Using a Three-Factor Eating Questionnaire (161) they found that obesity is associated with reduced cognitive functioning, particularly attention and executive function, as well as maladaptive eating behaviour such as uncontrolled eating (194). To some extent the same factors lie behind loss of control over eating (uncontrolled eating) in general and behind eating triggered by emotional situations (emotional eating) but, whereas emotional eaters prefer foods that are sweet and have a low fat content, uncontrolled eaters instead prefer salty and high fat foods (27).

In my clinical experience many patients who eat uncontrollably and who therefore are not defined as "emotional eaters", have said that they do not think about stuffing food into their mouths now and again – they do it habitually and do not note that they are doing it. Habitual eating, which is generally uncontrolled, is often associated with recurring everyday situations: at the end of work, taking children to and from school, driving the car, watching TV, or when one is bored.

Cognitive Restraint (CR)

Restraint eating means a conscious restriction of food intake in order to control body weight or produce weight loss (195). Rideout demonstrated that it was precisely this form of moderated eating, rather than dieting, that could contribute to successful weight control (200). Restraint eaters appear to experience a negative reaction to eating, namely guilt, and Mann et al. do not consider that studies show clear indications that dieting results in successful weight loss (201).

"Normal dieters" can be said to differ from restrained eaters in that they follow a diet for shorter or longer periods whereas restrained eaters take note of the calorie content of food over time. What both groups have in common is a desire to control body weight (200). Just such a "strict diet" can serve as a gateway to obesity (202). Similarly, it appears that emotional eating triggered by stress and "negative moods" can trigger binge eating behaviour (203), particularly if overweight individuals eat less than the body physiologically requires (204). However, Klesges et al., who examined the association between dietary restraint and body weight over 2.5 years found no association between dietary restraint and weight gain over time (205). The result was confirmed by Rideout who, by comparing dieting and restrained eating among women, found that long-term efforts such as restricting dietary intake are more effective for achieving and maintaining a lower body weight than is time-limited dieting (200).

Orthorexia nervosa involves fixating on a healthy lifestyle which, though characterised by wholesome eating, often also involves such excessive amounts of exercise that behaviour becomes almost compulsive (207). The individual has good awareness of the nutritional content of food and uses this knowledge to "control" food intake. The concept of orthorexia nervosa is used in those cases where diet becomes an escape from reality and every day is dominated by planning, buying and preparations for "approved" meals. Orthorexia nervosa, which is not a psychiatric diagnosis, is therefore similar to the eating disorder Anorexia nervosa, but is generally seen as an exaggerated expression of our society's values and trends (207).

2.4 Theoretical background for Cognitive Behavioural Therapy Targeting Eating Behaviour (CBT-TEB)

The programme is a form of educational treatment with theoretical roots in cognitive psychology. The programme has been developed on the basis of this theory as well as knowledge derived from cognitive psychotherapy and psychoeducational methods, combined with its own clinical experience in obesity treatment over many years.

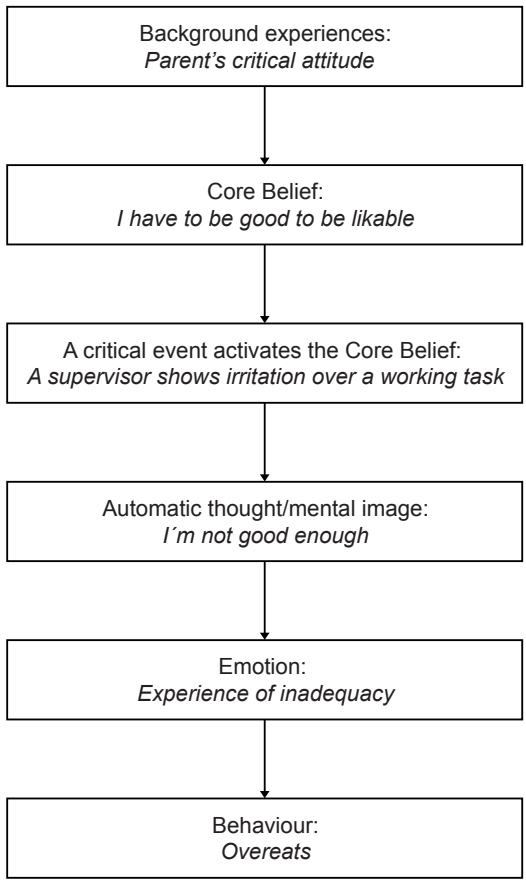
Cognitive psychology

Cognitive psychology is defined as the science of thinking. Its practical application in clinical situations is via cognitive psychotherapy. Cognitive psychology deals with human information processes: how we gather, process and use external information (208). The process is subjective, which means that interpretation and processing of the information is also subjective. How we interpret previous events and situations in life dictates how we approach and interpret present and future events and situations. In making these interpretations we tend to selectively focus on information which confirms our own previous conceptions. A person with a negative self-image therefore tends to select information which confirms this negative self-image. With the help of cognitive distortions/thought patterns, information which contradicts any negative corroborations (209, 210, 211, 212) is excluded. These distortions mean that experiences of situations are wrongly interpreted and falsified in such a way that our self-schema is retained and sometimes reinforced. Seligman discovered the concept of "learned helplessness" (210). According to this, an individual's experience of lack of control over one's actions and external events leads to an expectation that present and future events are also uncontrollable, and this can generate general passivity and lead to anxiety and depression. Aaron T. Beck, the father of cognitive therapy (the clinical application of cognitive psychology), found in his work with patients that affective and cognitive processes are related to the self-schema and to models which develop as we grow up (214). According to Beck we gradually create, through interaction with others, stable and general rules/life rules for what is right or wrong, which lay the ground for our self-schema (209, 215). If the mental construction is integrated with far too much criticism or rejection, dysfunctional assumptions about oneself can arise: "If I am not liked by others, I am of no value".

The Schema Theory

The Schema Theory forms the backbone of cognitive psychology. The Schema Theory is an explanatory model which relates a patient's problems to their background history. Our inner schemas are constructed from basic life rules and complex thought patterns which determine how situations and experiences in everyday life are categorised. These schema lie behind negative automatic thoughts and cognitive distortions (216). Figure 1 illustrates how overeating can start according to Beck`s general Schema Theory (209

Figure 1. Application of Beck`s general Schema Theory



Cognitive psychotherapy

Cognitive psychotherapy is the cognitive psychologist's clinical tool. In the therapeutic process the patient learns to:

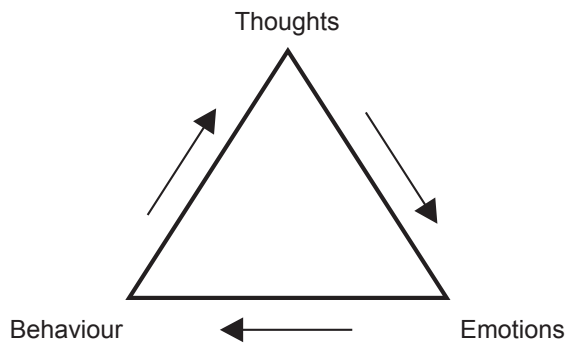
1. become aware of his/her own automatic thoughts,
2. become aware of cognition, emotions and behaviour,
3. examine the evidence for and against automatic thoughts,
4. alter distorted thoughts so that they become more in line with reality,
5. dentify and alter inappropriate and incorrect schema which predispose to the distorting of experiences (215).

Cognitive Therapy (CT) versus Cognitive Behavioural Therapy (CBT

Over the years cognitive therapy has provided methods that are used in behavioural therapy and the concept of cognitive behavioural therapy has developed into a form of therapy itself. The concept of cognitive behavioural therapy, or CBT, has often come to mean the same thing as cognitive therapy, CT. One difference is that CBT focuses more on altering behaviour by learning and education, whereas CT places greater emphasis on gaining insight. For both CT and CBT the models are problem focused and the observable is the point of departure. In problem situations CT proceeds from the association between thoughts, feelings and actions (Figure 2). Together with important life events, this triad comprises the basis for conclusions about the basic assumptions and life rules which direct and influence the individual.

In CBT one proceeds from final element of the cognitive triad: action and its consequences in problem situations. The work of implementing change focuses on increasing or decreasing the behaviour that brings about the desired improvement. The model consists of a contextual analysis, with work focusing outwards rather inwards as in CT. However, the term CBT has gradually developed into an umbrella expression which covers several treatment methods.

Figure 2. The cognitive triad



Psychoeduction

Psychoeducational/psychopedagogic treatment involves treating psychological problems with the help of pedagogic principles. The treatment addresses both patients and relatives, is normally group-based and comprises 12-18 sessions. The programme is often presented as "patient instruction" or "psychologist instruction". Psychoeducational treatment is mainly used in the treatment of schizophrenia, but individual programmes have been developed for patients with bipolar disorder (217). The aim of psychopedagogic treatment is to increase patients' knowledge of their psychological illness, in order to thereby promote cognisant problem solving strategies (218). The instruction is structured through a prearranged pedagogic programme. The programme includes training in accordance with behavioural

therapeutic principles where it concerns the management of different everyday situations in a constructive manner. Assessments of the programme also showed that patients felt they were participating in the treatment and that the distance between the therapist and the patient had decreased, perhaps because therapists often encourage patients themselves to search for relevant knowledge about their problems, either individually or in a group together with other individuals with similar problems to their own (219).

A few studies have reported the treatment of obesity with psychoeducational methods. A study with female college students showed preliminary results over four months which indicated that psychoeducational intervention could be of benefit in combatting eating disorders and reducing overweightness amongst students (220).

3 OBJECTIVE

3.1 General purpose

To describe the CBT-TEB programme and evaluate its medium-to-long term weight reducing efficacy and effectiveness in overweight and obese people.

3.2 Specific aims/research questions

1. To describe the efficacy of CBT-TEB in obese waiting-list patients in an academic obesity clinic (Paper I)
2. Is the efficacy of CBT-TEB the same for obese patients with and without BED? (Paper I).
3. To describe the efficacy of CBT-TEB in non-patient working women with obesity where controls receive another active intervention (Paper II).
4. To what extent do CBT-TEB and the control programme convey different kinds of knowledge? (Paper II).
5. To describe the effectiveness of CBT-TEB in an observational study of primary care patients with abdominal obesity where ordinary staff members deliver the therapy after a short training (Paper III).
6. To describe patients´ expectations before CBT-TEB and experiences reported after therapy (Paper III).
7. To describe staff members´ new experiences and attitudes to the CBT-TEB programme after acting as group leaders (Paper III).
8. Is it feasible to implement CBT-TEB in primary care? (Paper III)
9. To identify possible specific changes in eating behaviour during CBT-TEB (Paper III).
10. To identify changes in obesity-related problems during CBT-TEB (Paper III).

4 METHODS

4.1 Study groups

The assessment was carried out with a sample of three patients with obesity with the aim of determining whether the treatment programme might be applied more generally. Participants in Study I were waiting-list patients from the Obesity Unit, Karolinska University Hospital, Huddinge. Participants in Study II were workers from a child care centre in the Huddinge community in Stockholm, and participants in Study III were patients from five primary care centres in Hisingen Gothenburg. The participants in study I and study II were all women. 74 Women and 9 men participated in study III.

The number of participants in the CBT-TEB groups varied between the studies. Study I comprised six CBT-TEB groups of 8-12 patients, as well as three control groups of 14-15 patients. Study 2 comprised two CBT-TEB groups of 5 and 11 participants, respectively, and in the control programme there were two treatment groups of 12 and 14 participants, respectively. Study III comprised 14 CBT-TEB groups, each with 4 -11 participants. The group composition was heterogeneous. The ambition was to have both younger and older individuals in each group – this was to allow the programme's discussions to be enriched by the experiences and viewpoints of people of different ages. Another ambition was to divide medical diagnoses between the groups to avoid creating subgroups from diagnostic identification. Table 3 describes the demographic factors of participants in the three studies.

Table 3. Description of the study samples

	Study I		**Study II**		**Study III**
	CBT-TEB N(%)	Control N(%)	CBT-TEB N(%)	Control N(%)	CBT-TEB N(%)
Working full-time or part-time	46(74)	26(60)	16(100)	26(100)	57(48)
Unemployed					17(14)
Sick leave				10(8)	
Sick pension or retirement pension					28(30)
Born outside Scandinavia				10(12)	
Primary school	20(32)	17(39)	2(13)	3(12)	18(22)
Secondary school	12(19)	10(23)	3(19)	4(15)	38(46)
Vocational training	10(16)	10(22)	10(63)	9(35)	
Higher education	20(32)	7(16)	1(60)	9(35)	27(32)
Mean age (years):	45.4	45.2	50.1	47.0	52.8
BMI	40.4	39.2	35.5	34.1	34.6

4.2 Procedure

Overall procedure

All patients were weighed when included in the treatment, at the end of treatment, 6 months after the end of treatment (studies 1, 2, 3), 12 months after the end of treatment (studies 1-2), and 18 months after the end of treatment (studies 1-2). Weighing of the controls took place at times corresponding to the starts of the treatments, 6 months (study 1-3), 12 months (studies 1–2) and 18 months (studies 1–2) after the end of treatment. All weighings were checked by staff.

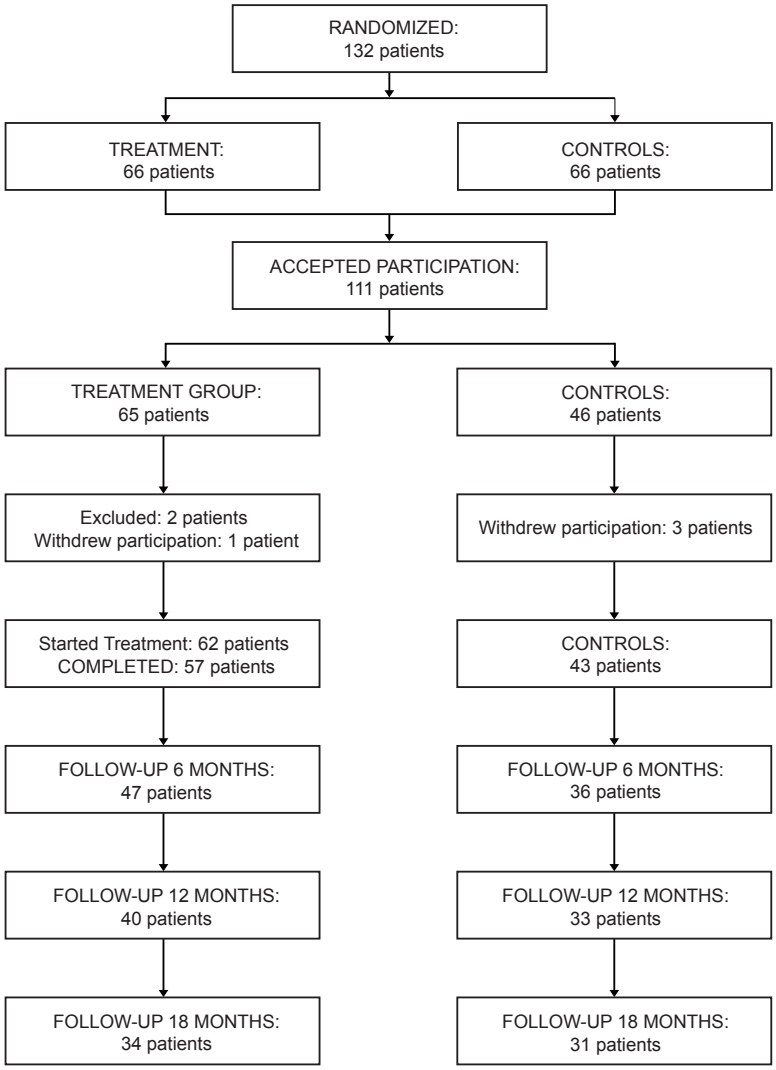
Study specific procedures

Study I

Inclusion criteria: a BMI ≥30 kg/m, Female, Age: 18 – 60 years. Exclusion criteria: Signs of active tumour disease, Pregnancy, Symptoms of psychosis

One hundred and thirty-two patients were asked to participate in the study. Sixty-six of thosewere randomised to the CBT-TEB programme and the other 66 served as controls. After randomisation all patients were asked to respond to a written query about their participation in the study. One hundred and eleven of the patients agreed to participate in the trial; 65 in the CBT-TEB group and 46 in the control group. Of the participants in the CBT-TEB group two fulfilled one of the exclusion criteria. One patient that completed the treatment and three of the control patients withdrew their consent to participate any further. These four individuals are for ethical reasons not included in the data analysis of this paper. Therefore, this is a report on 62 patients who began treatment and 43 controls. Of the patients who began treatment 57 completed it. Of the five who did not complete the treatment three said this was because of the long distance from the city to the treatment location and difficulties in getting off work; two patients said they did not agree with the treatment method. (Figure 3).

Figure 3. Flow Chart (Paper 1).



Ethical approval. All participants received both oral and written information about the study. The study was approved by the Ethics Committee of Karolinska Institutet.

Study II

Inclusion criteria: BMI ≥30 kg/m, Female, Age: 21– 61 years. No exclusion criteria were imposed.

94 individuals, employed at children care centres in Huddinge community, were asked to participate in a randomized controlled trial involving two weight-reducing programmes. Of the 94 eligible women, 54 voluntarily agreed to participate in this study. Twenty-seven women were randomiaed to the CBT-TEB programme and 27 to a weight-reducing programme resembling those often used at occupational health care centres in Sweden. The participants in the latter group served as controls. Each of the programmes consisted of two therapy groups. Both programmes started at the same time. Taking into account those who withdrew or dropped out before treatment, the number of participants in the CBT-TEB programme was 16 (divided into two subgroups of 5 and 11, respectively), whereas in the control programme the number of participants was 26 (divided into two subgroups of 12 and 14, respectively) (Figure 4). In the CBT-TEB program three withdrawals before the start of therapy were due to change of employment. One person was excluded after she missed attending the first therapy session. Seven participants dropped out before therapy started: 3 because their job situation hindered them from attending, 3 because they had lost their motivation, and 1 because her doctor had advised her not to participate because of medical reasons. (Figure 4).

The control programme

The control programme consisted of lectures, group discussions and practical demonstrations. The participants received both theoretical information and practical training related to the contents of the programme. The instructors for the control programme were nurses, occupational therapists who were local government employees, and external assessors. The following topics were included:

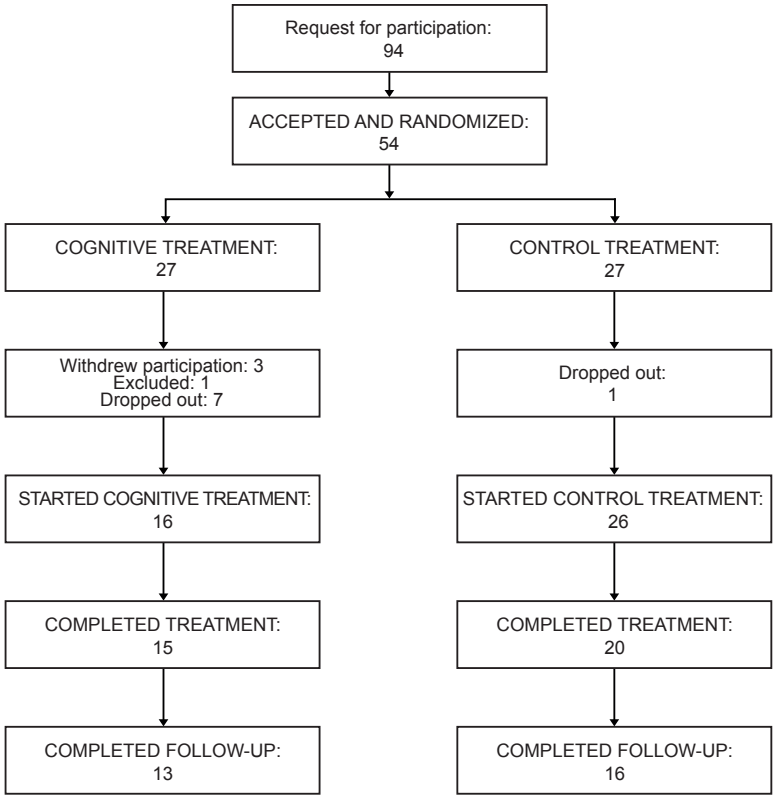
- Women's health and lifestyle. How weight loss can be achieved. Information on dieting.
- The principles of dieting. Applied knowledge about food; practical training.
- Computers as an aid to eating well and achieving nutritional goals; practical training.
- The importance of knowing your body. Relaxation techniques and how to handle stress.
- The importance of physical exercise. Strength training. Instructions and practical exercises.

Knowledge test

To examine the differences in their levels of knowledge all the participants were sent a questionnaire via the control programme and the CBT-TEB programme one month after the

end of the programmes. The questions, which were knowledge-specific for each programme, were formulated by the programme instructors. Replies to the questions were anonymous.

Figure 4. Flow Chart (Paper II).



Ethical approval. All participants received both oral and written information about the study. The study was approved by the Ethics Committee of Karolinska Institutet.

Study III

Inclusion criteria: 18-69 years of age and the combination of (1) waist circumference (WC) >88 cm females or 102 cm (males) or WHR ≥ 0.88 (females) or ≥ 1.0 males, and, (2) BMI ≥25kg/m^2.

Exclusion criteria: previous anorexia nervosa; endocrine or metabolic disease with or without treatment, except for diabetics who were only on a diet, and diabetic drug-treated patients who were well known at the primary care centre and who checked their blood sugar regularly; apparent cognitive reduction such as mental retardation, dementia or other cerebral lesions; difficulties in understanding Swedish; current alcohol or drug misuse or dependency; current or previous psychosis; giving birth in the last six months or pregnancy; treatment with

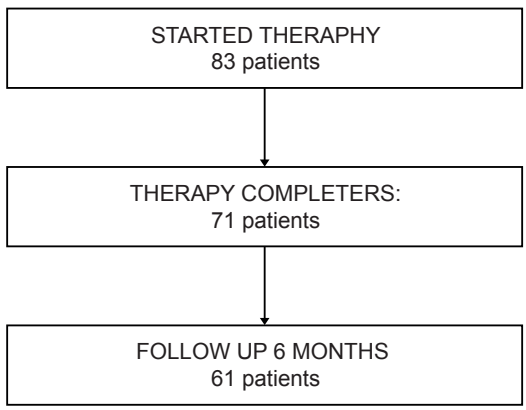
weight-reducing drugs during the last six months; malignant disease such as cancer, lymphoma, and leukaemia during the last five years.

All patients attended primary care for health problems other than obesity. All patients were invited to answer anonymously nine questions about lifestyle, genetic predisposition to cardiovascular disease, and level of motivation towards lifestyle change. Thereafter, these individuals were offered a health profile to work through at their own pace and a nonphysical examination including measurement of blood pressure and blood sugar. The participants were invited to an interview based on the results from their health profile. Height and weight were measured. Participants in different programmes targeting lifestyle change were invited. CBT TEB was one of these programmes and addressed patients who were overweight or obese. Patients who were interested in participating in the CBT-TEB programme were invited to a second interview (221).

Eighty-three patients (74 females and nine males) started in 14 CBT-TEB groups (Figure 5). All fulfilled criteria for abdominal obesity based on WC and 75 (90%) fulfilled WHR-based criteria. Baseline BMI was 25.0-29 in 12% of cases, 30.0-34.9 in 45%, 35.0-39.9 in 33%, 40.0-44.9 in eight percent and >45.0 in two percent of cases. Seventy-one (86%) completed the CBT-TEB programme (63 females and eight males). Completers were older than drop-outs (mean difference 7.4 years; $p<0.05$).

Medical and social data were gathered from records and by interview. Primary outcome variables (WC, WHR, Weight and BMI) were recorded before and after therapy and at six months after the end of therapy. Measuring was supervised and recording was performed by study coordinators. Patients were weighed at the treatment sessions. Secondary outcome variables were measured before and after therapy.

Figure 5. Flow Chart (Paper III).



Ethical approval. The study was conducted with the understanding and the consent of the participants. The study was approved by the regional ethics committee in Gothenburg

4.3 The CBT-TEB programme

The programme´s structure

The programme includes an introductory meeting and 10 sessions. Each session lasts for 2-3 hours and takes place once a week. The programme is conducted within a group and is a learning-based treatment.

The programme`s objective

The programme's objective is to provide the participants with knowledge about the causes of their own dysfunctional eating behaviour and give tools to address these causes.

It is expected that this will be achieved by the participants learning:

- To identify situations and social relationships in everyday life which give rise to negative thought patterns and feelings.
- To examine thoughts and subject them to reality testing.
- To identify the feelings generated by these thoughts.
- To examine the effect of these feelings on eating behaviour.
- To identify the causality between thoughts, feelings and eating behaviour.
- To alter the causes of negative thoughts and feelings. To enable the patients to acquire knowledge about the amount of food they can eat daily to achieve and maintain weight loss.

4.4 How the CBT-TEB programme was conducted

The CBT-TEB programme was included in all three studies. The programme is described in two comprehensive manuals for group leaders and patients. The two books match each other's content (222, 223). The programme description in both books begins with the theoretical background adapted for each target group: the group leaders and the patients. The patient's handbook contains practical examples and questions to work with at home, based on the programme's themes. The participants are made aware of the importance of preparing at home before each session by acquainting themselves with the content of the next session.

Small differences arose between the studies in the CBT-TEB programme methodology. In study I CBT-TEB was conducted as a three-hour session once a week for 10 weeks. In studies II and III CBT-TEB was conducted as a two-hour session once a week for 10 weeks. In study III CBT-TEB was carried out by instructors (nurses, district nurses and health pedagogues) employed at health centres. The instructors received 12 hours of training for the programme, with a 4-hour follow-up.

Preperatory interview

Programme participation is preceded by a personal interview. As the instructors do not meet the participants on an individual basis during the programme, the interview gives the instructors an opportunity to ask questions that can have significance for the treatment results, for example, participant motivation. The interview also gives participants the opportunity to ask the instructors questions. Examples of interview questions: Why are you seeking help for your overweightness/obesity at this particular moment in time? How many children do you have? When did your weight problem start? How has it developed over the years? How often do you find yourself losing control over your eating? Do you sometimes make yourself vomit after meals? What weight loss programmes have you taken part in previously? Why do you think the previous weight loss programme failed to achieved the results you had hoped for? Why do you believe that this time you will be able to permanently alter your eating patterns? If the solution to your weight problem involves you making changes to your life – how would you regard that?

The Key questions

“The Key Questions” are the fundamental tool for helping the participants to acquire knowledge of their own individual causes of overeating. Figure six shows The Key Questions, of which there are five. The participants ask themselves these questions every evening, between sessions, write down the answers, and take these with them to the next session. The instructors begin each session by asking the patients The Key Questions and then they replace the concept “today” with “during the week”. As both instructors and participants record the participants' weight each week and the instructors are therefore aware of each participant's weight pattern, there may be an opportunity during programme time to specifically direct The Key Questions at participants who have a negative weight pattern. This is in order to determine whether there is anything in the participant's working day which at that point in time is making weight loss more difficult, or whether the participant has possibly not understood The Key Questions (Figure 6).

Figure 6. The Key Questions

1. What has happened today/during the week?
2. Describe events/situations at home or at your work which have engaged and affected you in various ways.
   A. What have you thought about what happened?
   B. What feelings have arisen in you as a result of what happened?
3. How do you think the events/situations have affected your eating behaviour today?
4. If you think through what occurred, do you believe that your thoughts about what happened, and your reactions to it, were realistic?
5. How would another interpretation of, or reaction to, the events/situations have affected your eating behaviour today?

Agenda (Content of the Sessions)

All the sessions have a common framework, the agenda, which comprises four different parts: A, B, C and D.

- A. Weighing. Going through answers to The Key Questions.
- B. Discussion of homework for the previous session.
- C. Psychological theme.
- D. New homework

Part A. Weighing. Looking through answers to The Key Questions

The participants weigh themselves when they enter the session room. Participants and group leaders both sign each weighing. During the first three sessions the instructors "go round the room" asking each participant The Key Questions, but thereafter the questions are posed generally. The group leaders begin each session by ascertaining whether each participant has understood the thematic content of the previous session as well as what thoughts and ideas the themes provoked in the participants, with regard to their own causes of overeating. Throughout the entire programme the instructors encourage the participants to actively discuss and communicate both positive and negative experiences in their use of The Key Questions. This activity is crucial – the participants' understanding of the content of the sessions' themes, combined with their answers to The Key Questions, represents the knowledge which enables participants to achieving lasting weight loss after the end of the programme.

Part B. Previous session's homework.

Discussion about the participants' experience of carrying out their homework and what they feel about the homework itself

Part C. Theme

Whereas The Key Questions give the patients knowledge of issues in everyday life that can trigger overeating, the programme's themes provide tools for addressing these issues. The programmes' themes are based on tools developed from general schema theory (209) and from personal clinical experience. Each session represents a knowledge theme which has a specific place in the programme and makes learning cumulative. As the theme text is contained in both the instructor's handbook and the patient's handbook, the aim is not to present the contents of the theme to the participants through lectures, but rather as a topic for discussion between instructors and participants. Thus, in the first instance, the instructor's role in the theme sections concerns conversational pedagogy rather than lectures. Together with The Key Questions, it is the psychological content of the programme's theme that is crucial for the treatment achieving results. Each theme dialogue is concluded with a group discussion.

Images and individual examples

The CBT-TEB programme includes around thirty images and ten personal examples. These are all found in both instructor's and patient's handbooks. The images are created for the instructors' use on the computer. The images follow the sessions' contents and are used as a factual source and as a basis for discussions. Each session includes a personal example which, in a clear and concrete manner, explains the content of each theme. Images and personal examples are not shown here.

Part D. Homework

Each session is concluded with the allocation of homework which is related to the content of the theme. The area covered by the homework varies from one session to another. The homework gives the participants the opportunity to reflect during the week on the contents of the theme and/or training in the practical application of the theme's contents in their own everyday life.

4.5 The sessions – a short manual

For reasons of available space, the description of the sessions' themes in the following pages represents a very limited summary of the contents of the sessions. The sessions are described in detail in both the instructors' and patients' handbooks (222, 223). Figure 7 gives the theme titles for the Introduction and all the sessions.

Figure 7. Theme titles (Introduction and all sessions).

Introduction. Being overweight and being obese are not in the first instance to do with what you eat – but rather why

This section looks at the association between feelings, loss of control and overeating,and at the programme's aims, objectives and content.

Session 1. The Key Questions

This examines the aim, content and use of The Key Questions.

Sesion 2. The Dietary Programme. Target Weight

This looks at the content and application of the dietary programme and at the participants' target weights.

Session 3. Schema model 1. The significance of self-image in eating behaviour. Life rules

This examines how self-image is constructed and what affects it, and how one's own self-image might affect eating behaviour.

Session 4. Schema model 2. Thoughts, feelings and (eating) behaviour

This examines the association between thoughts, feelings and (eating) behaviour and how negative thoughts and attitudes can be replaced by more functional ones.

Session 5. Schema model 3. Automatic and conscious thoughts

This examines the difference between automatic and more conscious thoughts and how thoughts can affect (eating) behaviour.

Session 6. Schema model 4. Dysfunctional/functional thought patterns and (eating) behaviour.

This examines how the participants can replace dysfunctional thinking with more functional thinking.

Session 7. Control and helplessness – effect on (eating) behaviour

This examines the difference between experiencing control and helplessness/hopelessness in everyday life and how the participants and "acting as if".

Session 8. Stress – a part of life 1. Stress and (eating) behaviour

This looks at stress as a cause of obesity: that stress is for the most part a subjective experience and that it is therefore possible to address and deal with the causes of stress.

Session 9. Stress - a part of life 2. The importance of being able to say no and to set limits

This examines the association between saying yes, stress and overeating, as well as the causes of one's own overeating which the programme has made the participants aware of.

Session 10. Weight loss pitfalls and strategies

This looks at the participants' experiences of weight loss pitfalls as well as strategies dealing with them.

4.6 Introduction

Theme: Being overweight and being obese are not in the first instance to do with what you eat – but rather why

"Why do I eat incorrectly, when I actually know how to eat properly? And why can I not maintain my weight loss?" are questions asked by many people who are overweight. Everyone knows that eating too much food over a long period of time results in overweightness or obesity. Similarly, it is a known fact that eating less food and taking more exercise leads to weight loss. Why, therefore, do people not behave accordingly?

1 million Swedish women and men are obese, while 2.7 million women and men are overweight (5,11). The majority of overweight and obese people are emotional eaters and many are uncontrolled eaters. Studies have shown an association between negative feelings and overeating, mainly in overweight women, which is less pronounced in men (195, 163). Emotional eating associated with anger, melancholy, stress, depression and loneliness occurs in circa ¾ of overweight patients who seek treatment for their obesity (165). Most researchers are agreed that the mental and physical strains imposed by stress can cause obesity to develop, particularly the harmful form, abdominal obesity (55).

The need to put something in our mouths when we don't feel good has been with us since we were babies. If one is a smoker, a cigarette is what one longs for, but if one is overweight,one expects that something good, often sweet, is what will satisfy the craving instead. The desire for sweets foods may therefore be due to the reward effect that eating can offer; a brief respite from negative feelings (186). Feelings that are associated with grief or disappointment, experiences of having been hurt or misunderstood, thoughts of worthlessness and shame, can all lead to worry and anxiety. And both anxiety and other negative feelings have been shown to trigger a need to eat in the majority of overweight individuals (167, 170). If the causes of worry and anxiety are not ascertained and worked with – and an individual does not ask the question of whether there is a valid reason for this worry and anxiety, a strategy of living with these feelings can lead to emotional eating. Emotional eating can therefore be seen as escapist behaviour in an attempt to minimise, regulate and block emotional stress in the hope that the misuse of food will alleviate the negative feelings (164).

One of our overweight patients once said “being overweight – it is of course all about feelings”. She believed that her desire to eat too much and her craving for something nice but not good for her, often arose when she felt stressed, irritated, tired or dissatisfied. Stuffing something nice into her mouth meant negative feelings disappeared for a moment. This behaviour is supported by scientific studies (164, 195); many overweight and obese individuals simply compensate for negative feelings by eating, which momentarily alleviates the feelings. However, overeating often results in self-criticism because of the guilt which this eating gives rise to (181); feelings which in turn can be ameliorated by something new to eat. It is therefore easy to find oneself in a vicious circle which gives rise to compensatory eating behaviour.

Therefore to achieve lasting weight loss an individual needs to become aware of the causes of his/her own overeating and to acquire the knowledge with which to deal with these causes. In order to provide this knowledge the programme includes, above all, two tools: 1. The Key Questions, which help participants in the programme to become aware of what triggers overeating and what the underlying causes are. 2. The programme's cognitive themes, which give participants the knowledge with which to address these causes.

4.7 Session 1

Theme: The Key Questions

Appraisal of aims and contents of The Key Questions. Instructions from the suprrvisor on how patients should make use of the questions (these are included in both the instructors' and the patients' handbooks). Information about the answering of questions by the participants each evening during the period of the programme.

Question 1. What has happened today? Describe those events/situations which have engaged and affected you.

Here the participants identify events/situations during the day, at home and/or at work, which in various ways have triggered both cognitive and emotional reactions.

Example:

Think of when you left your home this morning. What happened up to the time you arrived at work or wherever you went? Then what happened before noon? During the afternoon? Anything positive or negative which happened at work today? Anything which made you happy or unhappy? Anything positive or negative which happened at home? Or elsewhere? Anything else which made you feel happy, pleased with yourself, and anything you accomplished or contributed to? Or, on the other hand – which made you feel unhappy, irritated, stressed or unsuccessful? Anyone who said something that made you happy? Or unhappy and irritated? Problems with cooperation at work? Differing opinions at work? Differing opinions within the family? Have you experienced stress today? What was this related to? If so, how has this stress affected your performance?

No event or situation is too small or trifling to be noted by a patient; what is important is the patient's reaction. Being reprimanded by a work colleague for not washing one's cup after a coffee break is no less important for participants to talk about than is failing to carry out a work task. For participants to be open with regard to The Key Questions, it is vital that the instructor provides a sense of security during the therapy sessions.

Question 2 A. What have you thought about what happened?

Question 2 B. What feelings have you experienced as a result of what happened?

The patient should ponder over how he/she had thought about the event/events. What thoughts and reflections arose? Where these negative or positive? What did the patient think about what happened? That it was bad for him/her to fail to complete a work task? That it actually made no difference if the work task was postponed for a day? That the carelessness with the unwashed coffee cup led to thoughts such as: typical, my usual bad luck? Or to the reflection that we all make mistakes sometimes? How did the patient feel about the event/events (upset, disappointed, irritated, undervalued, angry, satisfied, etc.)?

In order that the participants understand what is meant by situations and events, examples are given: when I felt stressed, when I did not manage to do something I had resolved to do, when I faced major demands from my employer or family, when a new task imposed demands on me that were too great, when I felt that my efforts were not appreciated, when I "took to heart" a negative comment at work, when I had difficulty "saying no", when I felt let down by my work colleagues, when I helped out a friend but received no thanks, when we disagreed about something within the family before I left for work, when the house hadn't been cleaned when I returned home from work, when I realized that my family's needs usually came before mine, when I experienced sadness, when I felt pursued by all the demands.

In terms of feelings this can mean, for example, that I experienced: worry, anxiety, happiness, sadness, grief, disappointment, irritation, dejection, hopelessness or anger.

Question 3. How do you think that events/situations have affected your eating behaviour today?

The aim of the question is to draw the patient's attention to the fact that there are often scenarios in everyday life which, via negative thoughts and feelings, can contribute to triggering overeating. And that it can therefore be very important to make changes and deal with these situations.

Question 4. If you think through these occurrences, do you believe that your thoughts about what happened and your actions were realistic?

This question will encourage patients to make a realistic assessment of their approaches towards what occurred. Are there other ways of looking at what occurred, other than the patient's own?

Question 5. How might another interpretation of or reaction to the events have affected your eating behaviour today?

The question aims to also engage other group participants and, via group discussions, to communicate to the participants the fact that their understanding of or attitude towards an event is not always "right".

Homework: The participants will answer The Key Questions each day during the week and write down their answers in the handbook. The participants' answers to the questions are discussed during the next session.

4.8 Session 2

Theme: The Dietary Programme. Target Weight

The dietary programme was compiled by a registered dietitian. The dietary programme is based on the understanding that it is above all the amount of food eaten which determines whether one's weight goes up or down. The dietary programme therefore restricts the daily

amount of the various foods that are eaten but leaves participants free to choose the composition of the food they eat, and how it is put together. This allows the participants to eat the same food as other family members, though the amount is restricted, and permits the use of most recipes in preparing their food. Therefore special recipes for losing weight are not required.

The dietary programme provides 1200 -1300 kcal/day. Even though weight loss requires a restriction in the number of kcal, the participants are encouraged not to completely refrain from sweets/cakes/confectionery but rather, when experiencing a craving for sweet foods, to identify situations and contexts where this craving arises and then to make a conscious choice to eat or to refrain. This is to avoid the emergence of anxiety and worry, feelings which studies have shown to be capable of triggering overeating (192) and which have been confirmed by patients in their clinical obesity treatment. The participants weigh the foods they have chosen for preparing their meals and thereby have control over how much they eat in total during the day. This means that the participants never need to think about how many meat balls are on their plate or whether the potatoes are too large; or whether one has eaten too much or too little fat, protein or carbohydrates. These are common issues in dieting and can spoil less well thought-through efforts to lose weight and result in thoughts such as "I'll start again in the morning instead".

Experience shows that after about three weeks most of the participants have gained a good idea of how much bread, cheese, sliced ham, cutlets, fish and chicken fillets to weigh out and are therefore good at estimating the content of their food, even when they are eating out. Nor is it the 20 grammes of food eaten now and again during meals which affect participants' weight – rather, it is what they eat between meals which has this effect.

Homework: The participants write down their own weight targets: after 10 weeks (end of programme), 6 months and 1 year. Prior to the homework the participants are informed that studies have shown that a 5 – 10 percent weight loss provides good health benefits (88, 89). And that the aim of the programmes is not extreme weight loss but to furnish participants with knowledge about the causes of their overeating as well as with tools which allow them to address these causes.

4.9 Session 3

Theme: Schema model 1. The significance of self-image in eating behaviour. Life rules

The schema model is an explanatory model in cognitive psychology for showing how our self-image is created by our thoughts and assumptions (basic assumptions) about ourselves and by how we think we should behave (life rules). Our self-image is central as it affects both thoughts and feelings and therefore also our behaviour and consequently our eating behaviour. The schema model helps us to see the causes of our actions and is an aid to understanding why we often use eating as a form of escapism or compensation when we feel stress, grief or disappointment. The schema model comprises four sessions: Session 3 (this

one) deals with self-image and life rules, Session 4 with the relationship between thoughts, feelings and eating behaviour, Session 5 with automatic and conscious thought patterns, and Session 6 with functional and dysfunctional thought patterns and (eating) behaviour. (The word “eating” is placed in parentheses to draw the participants' attention to the fact that it is not only eating behaviour that is affected, but also other behaviour).

As we grow up we learn from our parents, siblings, teachers and friends how we should behave in order to be accepted in our own social group, and what is expected of us. The schema model thereby illustrates how thoughts about ourselves and about how we should behave in order to be regarded as acceptable (life rules) form the basis for our self-schema. If my self-schema includes the idea that I must be thin in order to be accepted by close relatives, it is not difficult to see that I will carry an assumption that I will not be accepted as long as I am fat. Life rules that are tolerant, accepting and enhancing have a positive effect on one's self-image. Life rules that are full of demands and are conditional - if I eat too much people will not like me, if I am fat others will stay clear of me, if I become thinner people will like me more, if I become more presentable people at work will like me more, if I make a mistake people may change their opinion of me, if I am criticised it means I have failed - may instead contribute to creating a fragile self-image. A negative self-image leads to negative thoughts and feelings that can give rise to various forms of compensatory behaviour, for example, compensatory eating behaviour.

A positive self-image contributes to good self-esteem and to the realization that I deserve to be loved, both by myself and by others, despite my faults and shortcomings. On the contrary, I feel that as I grew up I was mostly appreciated for what I did rather than what I was. This can lead to a performance-based self-esteem and life rules which dictate that I need to be seen as capable before I can be accepted and loved. If one feels one has to perform well and show competence in order to enhance one's self esteem, this can mean surrendering to other peoples' opinions and judgements.

The participants are encouraged to think about what guiding principles and values prevailed during their upbringing. Was there an atmosphere of openness which allowed one to speak about one's shortcomings, about conflict and illnesses? Or were questions concerning weakness “swept under the carpet”? Was it okay to be sad sometimes or was one always brusquely told to, for example, “pull oneself together”? What values did you grow up with regarding eating, and with what views on people who were overweight or obese? How have these values affected your self-image and how you view your own body today? The media often presents thinness as being synonymous with success and being valued – how do the participants feel that their own self-image is affected by this? One of the programme's implicit aims is to enable participants to be proud of themselves and to turn their negative views of themselves into more positive ones.

Homework: The participants' homework involves choosing from amongst ten suggestions and trying out at last three of them during the week. The following are three examples out of the ten:

Say “I” when you mean it. Often we use the word “one” when we actually mean “I”. Use the word “I” to show that you stand by your opinions.

Accept the appreciation of others. Do not devalue your achievements with apologies: “It was nothing special”, when someone shows their appreciation.

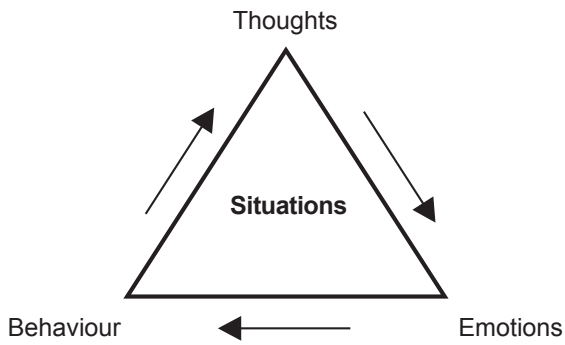
Perform an exercise in showing that you appreciate yourself: On each day during the week write down at least three things you are pleased with and to which you have contributed.

4.10 Session 4

Theme: Schema model 2. Thoughts, feelings and (eating) behaviour

Session 3 looks at the impact of life rules on our self-image and self-esteem. Both self-image and self-esteem have significance for how we think in everyday life. Session 4 therefore deals with the next step in the schema model: our thoughts and how these influence both feelings and behaviour. The cognitive triangle describes the association between thoughts, feelings and behaviour. This is a simple tool for the participants to use in everyday life, even as an alternative to The Key Questions (Figure 7).

Figure 7. The cognitive triangle



Question 1. What has happened during the week? Describe events/situations at home or at work that have affected you.

“Situation” within the triangle.

Question 2A. What have been your thoughts about the events?

Question 2B.What emotions have these events triggered in you?

The triangle's “thoughts” and “emotions”.

Question 3. How do you believe these events have affected your eating behaviour today

The triangle's “behaviour”.

Question 4. If you think through these events, do you believe that your thoughts and actions in response to what happened were realistic?

Question 5. How would a different interpretation of the events or your reactions to them have affected your eating behaviour?

From “behaviour”, proceed to “thoughts”.

Our thoughts can make us feel happiness, disappointment, irritation, sorrow, hopelessness, anger and stress. They can make us feel pleased with ourselves, or the opposite: disappointed, irritated, sad or angry – feelings which in their turn affect our behaviour, including our eating behaviour. We ourselves largely determine whether our feelings become positive or negative. Positive thoughts can make us feel happy, hopeful and pleased with ourselves. Negative thoughts, on the other hand, can make us feel disappointed, sad, irritated, stressed and unsuccessful. Self-image and self-esteem are also important here. The image we have of ourselves and our own ability affects our thoughts and attitudes with regard to what happens both at work and at home. If one's thoughts depict a future situation as hopeless, there is always a risk that negative thoughts will affect the outcome of this situation if these thoughts lead to feelings of uncertainty and worry, which in their turn could have a negative impact on one's actions. If one thinks instead “I'll make the best of this situation”, feelings will become more hopeful and positive. There is also a good chance that the situation will have a positive outcome.

This same difference in the way of thinking could also be useful in a weight reduction programme. If an overweight individual thinks it would be pointless to try once again to lose weight after several previous failed attempts, there is a risk that negative thoughts will lead to feelings of hopelessness and worry which can be alleviated by compensatory eating (172). The new failure that was anticipated has then become a self-fulfilling prophecy – not because of inability but as a result of one's own negative thoughts. A way of changing negative

thought patterns is to subject them to a realistic assessment and to ask oneself the question: how realistic are my negative thoughts? On what do I base these thoughts? If one deduces that the negative thoughts are based on unwarranted worry or are a result of incorrect conclusions, there is good reason to replace the negative thoughts by more positive ones.

If one departs from the idea that one's normal way of assessing a situation is always the correct one, it can be good to know that if many people describe an event which everyone has experienced, there are generally as many interpretations as there are people. We often think that our own interpretation of what happens is the only correct one and in the first instance we refuse to give up our own view of the situation . Therefore the discussions about the sessions' theme contents can in themselves provide the participants in the programme with new perspectives and can be just as rewarding as the programme content itself.

Homework: Use the cognitive triangle in everyday life to identify those thoughts and feelings which precede overeating.

4.11 Session 5

Theme: Schema model 3. Automatic and conscious thoughts

While session three has emphasised the importance of life rules for both self-image and self-esteem and session four has shown how self-image and self-esteem affect the association between thoughts, feelings and behaviour, the schema model now continues by pointing out the difference between what we term automatic thoughts on the one hand and more conscious thoughts on the other, so that the next session can conclude with an examination of the difference between functional and dysfunctional thoughts.

Thoughts which arise spontaneously during or just prior to a situation are normally referred to as automatic thoughts, whereas more conscious thoughts often take the form of "contemplation". It is often automatic thoughts that contribute to someone losing control over both eating and actions: at the start of the week the individual concerned notices in a food shop a special offer when buying two chocolate cakes rather than one. Automatic thoughts: "great – I'll save money, so I can buy both chocolate cakes for the weekend. These automatic thoughts activate the more conscious thoughts: "Hold on there, you know very well that you'll eat one chocolate cake when you get home and the other one later in the evening". The subsequent action is determined by whichever thoughts the individual bases his/her decision on. One choice will probably lead to the individual feeling pleased with himself/herself – the other to dissatisfaction and perhaps to compensatory eating. Uncontrolled eating and emotional eating are the commonest causes of overeating.

Every day we adopt many different attitudes. Yet we base our decisions on our thoughts, conscious or unconscious: whether we choose to work overtime, skip exercise or decide to buy an extra large portion of dessert cheese, simply because at that moment life seems boring. Our automatic thoughts can cause us to react emotionally, which rarely leads to a positive outcome. If instead we hold back for a few seconds, there is a good chance that our conscious

thoughts are activated and help us to control the situation we find ourselves in. Previous sessions have shown how one can abandon inappropriate life rules and thereby enhance one's self-image. This can similarly apply to our thoughts if we subject them to a realistic appraisal. This is examined in the next session.

Homework: To observe the effects of our automatic or conscious thoughts on both eating behaviour and other behaviour, as well as the effects on participants, in certain cicumstances, of replacing automatic thoughts with more conscious ones.

4.12 Session 6

Theme: Schema model 4. Dysfunctional/functional thought patterns and (eating) behaviour

The previous session looked at the differences between automatic thoughts and more conscious thoughts. It examined how automatic thoughts often appear suddenly, sometimes without our thinking very much about their content. Our conscious thoughts, on the other hand, are a consequence of our thinking a little further. Our conscious thoughts are therefore generally more realistic than our automatic thoughts. But there are also other differences between our thoughts; some of them are functional, whereas others are dysfunctional. These thoughts are commonly referred to as positive and negative, respectively, and even though this terminology does not properly comply with the meaning of the scientific concept, it is used during this session which also concludes by looking at the contents of our thoughts.

Positive thoughts give rise to emotions which make us feel good, whereas negative thoughts, on the other hand, generate emotions which feel negative and may involve experiences of dissatisfaction, disappointment, irritation and dejection. Whether our everyday thoughts are positive or negative depends on our own assessment of the situations we find ourselves in or of what we actually accomplish, an assessment that is coloured by our self-image and self-esteem. Will I cope with this situation or task as I or my employer would want me to? But thoughts are also associated with habits and it seems that we humans find it easier to think negative rather than positive thoughts. This is thought to be a legacy from the time when we lived among wild animals and needed to make quick decisions which sometimes involved life-or-death situations. Negative thinking means being cautious and is therefore prioritised. The dangers we encounter nowadays do not involve wild animals, yet we live in a reality which constantly exposes us to many different attitudes. We often use negative thinking as a safety measure but fail to see that this approach can prevent us from both personal development and positive experiences. Beck and colleagues identified a number of common negative thought patterns which we may all experience to a greater or lesser degree (224). The majority of our thought patterns are common to us all. When these thought patterns continually recur and have a negative effect on both feelings and patterns of behaviour, then this way of thinking needs to be observed and altered.

Some of the negative thought patterns that are given as examples in the programme:

Thinking selectively

Individuals who have major demands to cope with often attach great importance to those performance that they are not pleased with and which they often regard as examples of failure. In their thoughts these "failures" can become so exaggerated that they entirely overshadow those performances which they are pleased with. This thought pattern means that these individuals focus on their own negative actions as an expression of their negative self-image. Selective thinking can also mean people selecting a single negative detail from what for them was an otherwise positive situation and allowing the negative detail to dominate over the positive whole – this can contribute to the retention of a negative self-image.

Overgeneralised thinking**,** which is closely related to selective thinking, means that an individual, after an action which has a negative outcome, concludes that his/her actions always have similar negative outcomes – without also paying attention to any actions with positive outcomes. An example of both these thought patterns is someone who has lost weight but has failed to maintain the weight loss. This person therefore thinks in advance that a new attempt to lose weight loss would end in failure.

Drawing hasty conclusions

Hasty conclusions can reflect a poor self-image. For example, when meeting someone for the first time, I get the impression that this new person does not approve of me. This can be an expression of a hasty conclusion based on the fact that I feel insecure with this person. I may therefore have projected my feelings of uneasiness onto this person whose response may suggest uncertainty or rejection, which I interpret as disapproval – but which is actually a result of my own uncomfortable feelings towards the individual.

Catastrophic thinking

Catastrophic thinking means always expecting that the worst will happen. The aeroplane might crash; if one doesn't hear from relatives at the agreed time one concludes that there may have been an accident, etc. Negative thoughts have the power to trigger worry and anxiety. For individuals who have adopted eating as a coping strategy, it is particularly important to subject their thoughts to a reality check in order to prevent such thoughts from arising.

Labeling

People who often use the expression "I'm not good enough", "I can't cope", put a negative label on their abilities. Despite it being a destructive label, it may be used as an excuse to avoid change. Such a label can be regarded as a personal "benefit"; one has something to gain from seeing oneself as not good enough as it means that there is therefore no need to exert oneself and no need to be appreciated by others.

The Labeling and Personalisation of Thought Patterns (seeing oneself as responsible for external events) are part of what is termed learned helplessness. The next session deals with this form of behaviour. Discussion on negative thought patterns.

Homework: Identifying one's own thought patterns. Which negative thought patterns do you use? What effects do these thought patterns have on your eating behaviour?

4.13 Session 7

Theme: Control and helplessness – effects on (eating) behaviour

To allow circumstances ("everything will sort itself out"), fate or other people to direct one's life means adopting a helpless approach to the opportunities and problems of everyday life. Accepting instead the possibilities for choice in everyday life, and regarding oneself as responsible for one's life not always working out as one had expected, means an approach that focuses on "acting as if" and on positive solutions rather than on negative shortcomings.

The concept "learned helplessness" was introduced by Martin Seligman as a theory which compared experiences of a lack of control and of helplessness (210, 211, 212, 225). Seligman's theory is based on a perception that if people are repeatedly exposed to situations which they are unable to influence, they develop a negative expectation that new situations will also be out of their control and they therefore experience helplessness. The theory can be applied to the participants' own experiences of having failed many times to maintain their weight loss and of the hopelessness they often feel at the start of a programme. If, despite good intentions, one has failed to maintain weight loss, one often approaches another attempt with a sense of helplessness. One has "learned" that there is no point in trying to lose weight again. This learned helplessness affects not only the area in which one has experienced failure but can also give rise to helplessness in other areas.

Previous sessions have highlighted the importance of one's own experiences and background history (Schema theory) with regard to choices and attitudes in everyday life, as well as the importance of the conscious reality testing of these choices. These same possibilities also apply to experiences of helplessness and lack of control. Compensatory eating behaviour is an example of individuals feeling that they have lost control over their eating behaviour. The feeling of failure is so strong that belief in one's own ability disappears. One adopts an attitude of helplessness with regard to one's own capability and takes on the role of a victim. Both compensatory eating behaviour and learned helplessness are examples of escapist behaviour: one does not believe one is able to deal with the causes behind these forms of behaviour (225).

Learned helplessness can be eliminated if an individual's negative perceptions are replaced by positive experiences; for example, the achievement of weight loss. The experience of helplessness can be altered by examining one's own thought patterns in a particular situation. Are my thoughts realistic? Is my interpretation of a particular situation realistic? Is there a reason why I feel helpless in this situation? What might happen if I "acted as if" instead?

"Acting as if" may also be reinforced by visualising a situation in which one would feel concerned about something. One can experience greater confidence and control in a particular situation by creating a mental picture of the situation and, in one's thoughts, preparing oneself to resolve, in various ways, those potential problems which might concern one. Generally, the more detailed and concrete the visualisation practice, the more positive is its result.

Homework: To record when one has "acted as if" in everyday life or has found oneself adopting a more helpless approach – to examine the effects of these different behaviours on one's eating pattern.

4.14 Session 8

Theme: Stress – a part of life 1. Stress and (eating) behaviour

The previous session looked at how experiences of control and of its opposite, loss of control/helplessness, can affect self-image and eating behaviour both positively and negatively. Whereas an experience of control is mostly a positive one, an experience of helplessness is quite the opposite and can lead to stress. Therefore this session deals with stress. Stress is one of the commonest causes of obesity. It is all too easy to start snacking when one feels stressed and it is also easy to then lose control over how much one eats. The culprit in this drama is termed cortisol; a stress hormone which is excreted when we become stressed and creates a desire for carbohydrates, especially sweet ones. (182, 187). Negative stress can arise in the workplace if an employee's capabilities do not match the employer's demands. If the work is dull and monotonous and moreover, if the employer's demands dictate that there is little opportunity for the employee to influence the situation, stress reactions will occur which may be harmful to both mental and physical health (44).

Factors that can trigger stress:

- Low level of control – not being able to influence and control situations and circumstances.
- Lack of perspective – feeling that existence is meaningless and difficult to deal with.
- Conflict between goals – and with the possibility of realising them.
- A lack of social support in one's private life and work life.

However, stress research has provided us with the knowledge that stress can be positive as well as negative, and that positive stress is not as harmful to health as negative stress (43). Yet this conclusion is not unanimous amongst researchers; there is therefore reason to suppose that, particularly in the long term, positive stress can present health risks.

Chronic stress

Whereas the human body has practically remained unchanged for thousands of years, society is developing ever more rapidly – as are the demands on people. Development of the internet and mobile telephones means that most of us are accessible for most of our waking hours.

Even though what we nowadays refer to as stress has certainly been around as long as humans have been on the planet, today we are exposed to stress situations which our bodies have not evolved to deal with. For prehistoric humans it was a matter of fight or flight when danger threatened. The body prepared itself for physical exertion. When the computer is acting up or we find ourselves sitting in a traffic jam, we cannot solve the problem with physical activity, yet the same stress hormones are secreted as when we are threatened by wild animals. If the body does not use the energy that is released then fats will remain in the blood. With long-term stress it is therefore easy to find oneself in a vicious circle in which the stress results in elevated levels of cortisol which, in their turn, contribute to the storage of fats in the abdomen, so that these fats are not then used. Repeated stress therefore causes long term damage to the body and can result in cardiovascular disease.

Subjective stress

This is experienced when stress is primarily subjective. The causes vary from person to person. Whereas one person might find monotonous work stressful because there is no opportunity to use one's own initiative, another person may find work stressful because it requires the use of initiative. Stress can therefore be associated with both under– and overstimulation. It also means that we ourselves can influence the causes of stress. Whether or not stress is experienced can depend on how imminent or ongoing situations are interpreted in terms of our schema and thought patterns. It is easy for me to put myself down with thoughts such as “this will never work” or “I'll never manage this”. This will hamper my ability to perform a task in a positive manner. Or is the stress a result of my demanding too much of myself? Or a lack of proper scheduling? Or a lack of work-related information? Or an inability to set limits and say no? This will be examined in the next session.

If I cannot influence the causes of stress or am unaware of the causes, it is easy to develop coping strategies for living with the negative feelings from stress. Smoking can be such a strategy; excessive use of alcohol and excessive exercise are other examples. But above all, stress is one of the commonest causes of overeating and obesity.

Stress in women

Studies show that stress in women increases after work, whereas for men it is the other way round (43). It is thought that periodically recurring stress does not constitute a health risk provided that there is stress-free leisure time to create a balance. Without this balance the risk to health increases. Stress in women is therefore often a question of gender equality.

Homework: Self assessment of stress. The participants should acquire knowledge about stress in everyday life. A circle is drawn and divided into different segments, each of which represents recurrent activities and sleep during a normal 24-hour period. The different segments can, for example, represent: travel time, work time, taking children to and from school, cleaning, doing homework, preparing meals, spending time with children/partner,

spending time with parents, spending time with friends, time alone for oneself, sleep and so on.

When the participants have completed their circles, they mark with a pen how much of the time in each segment they find stressful. The participants reply to each of the following questions: Which of your segments is most associated with stress? How do the participants experience the demand imposed on them at work in relation to the possibilities for influencing their work tasks? How does stress affect your eating pattern? How will participants deal with the causes of their own stress situations in the future?

4.15 Session 9

Theme: Stress – a part of life 2. The importance of being able to say no and to set limits

Most of us regard stress as part of working life and not harmful, provided our private lives include counterbalances and breaks – in their homework the participants therefore look at the balance between stress and recovery in their everyday lives. Large parts of the session's theme time therefore involve discussion and examining this balance in the participants' stress analyses.

Stress in everyday life often involves an inability to say no. Saying yes may be motivated by the individual wanting to be helpful and to not be seen as uncooperative. Saying yes may thereby be seen as a means of achieving popularity. Saying yes can therefore be associated with a negative self-image – believing that by saying yes and being helpful one will be more appreciated . Being unable to say no is also a common cause of overeating in a social context. Many people who are overweight say that they want to avoid drawing attention to themselves by saying no to dishes that are offered. This attitude is so common that it has given rise to the concept of "trying too hard to please disease" (226). But there are contexts in which the word "no" can actually become an obstacle, when it concerns attitudes to change.

All changes involve the possibility of something new and also moving away from old, habitual ways. Challenging oneself and replacing old habits with something new and unknown can also contribute to strengthening one's self-esteem. What new knowledge has the programme imparted to the participants with regard to the causes of their overeating/obesity? Has the programme made the participants aware of dysfunctional thought patterns and/or their attitudes in everyday life that can also affect their eating behaviour negatively? Proceed from The Key Questions and discuss:

Key Questions 1-2: which changes have taken placed since you began your participation in the programme, in terms of being able to identify recurrent situations in everyday life which often give rise to feelings of unhappiness, irritation, anxiety, worry, etc?

Key Question 3: Can the participants see a connection between situations, thoughts and feelings today and those at the start of the treatment, with regard to their effects on eating patterns? If so, how would they use this knowledge?

Key Questions 4 – 5: Do the participants use reality checks in everyday life when they find themselves in unpleasant situations? Can one stop negative thoughts now in a different manner from when one started the programme? If so, what effect do these changes in thinking have on everyday life?

One of the programme's aims is to make the participants aware of the causes that lead to emotional or uncontrolled eating. The programme has given the participants tools with which to deal with these the causes. Has working with the programme allowed the participants to discover that there are causes in everyday life which need to be addressed in order that they can feel better and not indulge in compensatory eating? If so, do these involve relationship problems at home or at work? If so, do these problem concern the family or work colleagues? Or has one observed: that it is mainly stress that lies behind one's losing control over how much one eats; and that it is often one's own demands which contribute to the stress? If so, has the programme awakened a desire to manage oneself in a better way or to take care of oneself more? Dealing with changes almost always activates old life rules, self-image and thought patterns. If the participants have grown up with the understanding that one should not believe one is anything special or that one should not tempt fate, there is always the risk that a desire for change will be associated with worry and uncertainty where one's own capability is concerned.

Homework: The participants' homework therefore involves writing down their thoughts about changes, based on their own needs and desires, under the following headings in their handbooks: “I want to make this change with regard to myself”, “I want to make this change with regard to myself and my family”, “I want to make this change with regard to myself and my work”

Example:

Changes under the heading Myself can, for example, involve: paying more attention to the positive things I do than to the negative ones; thinking about what I say “yes” and “no” to; standing up for my own views; accepting that I make mistakes sometimes, just as others do; exercising regularly; being more kind to myself.

Under the heading Myself and my family changes can, for example, involve: letting each family member take more responsibility; no longer cleaning up after everyone; allocating food preparation days to each family member; planning for the week jointly; grumbling less – instead I should tell the family what I want.

Under the heading Myself and my work, proposals can, for example, involve the following areas: telling the workplace management that information within the company could be better; offering suggestions for workplace improvements; being clearer with work colleagues; not routinely saying yes to overtime.

4.16 Session 10

Theme: Weight loss pitfalls and strategies

Most participants have had experiences of one or more instances of weight loss and know that there are a number of circumstances or situations which can easily lead to both snacking and overeating. The final session therefore involves looking at the potential pitfalls in losing weight, of which the participants may or may not be aware, and discussing strategies for eliminating them. These pitfalls can be deceptive, which is why people are generally not aware of them - or do not want to be aware of them. Being aware of the weight loss pitfalls one might encounter can therefore make the difference between success and failure when it comes to the participants continuing with their weight loss – or maintaining it.

The Key Questions are aimed at making clear to the participants that there are mental pitfalls in everyday life which affect eating. Mental pitfalls, in the form of thoughts, attitudes and negative feelings, have taken up a large portion of the programme (The Key Questions), as has the question of how one might rectify and resolve these pitfalls (the programmes' themes/cognitive tools). The contents of the programme's themes have served as an aid, offering strategies to enable the participants to address and influence the causes of overeating. In this final session, therefore, examining weight loss pitfalls and strategies for dealing with these pitfalls offers a practical revision of the content of the cognitive section of the programme.

PLEASE NOTE: Just a few examples are given below of the pitfalls associated with weight loss and of the strategies for avoiding these. These pitfalls and strategies are both complemented during the session with examples from the participants of their own real life experiences.

Examples of common weight loss pitfalls:

1. **Emotional eating**
2. **Uncontrolled eating**
3. **Stress**
4. **Negative thinking**
5. **Difficulty saying no**
6. **The participants' own suggestions**

1. Emotional eating

Facts: Introduction, Sessions 3 and 4.

Strategies for preventing emotionally controlled eating:

● Think about the situations and circumstances in which you find yourself just now.

● Record your feelings.

● What is it in the situation that is triggering these feelings?

● Are your feelings motivated?

● Can you deal with these feelings in any way other than by stuffing something into your mouth to alleviate them – for example, by addressing the causes?

2. Uncontrolled eating.

Facts: Sessions 4, 7, 9.

Strategies for preventing uncontrolled eating:

● Check whether you really are hungry or just "craving".

● Think – how long is it until lunch?

● Do you feel impelled to stuff something into your mouth just now?

● How will you feel afterwards?

● Is the brief sense of wellbeing from the taste in your mouth worth this?

3. Stress

Facts: Sessions 6, 7, and 8.

Strategies for preventing stress:

● Ask yourself: has the stress you are experiencing just now been triggered by something?

● If you answered yes – what can you do to reduce your stress in the future?

● On what knowledge did you base your analysis of the causes of stress in everyday life which you need to address?

● What would reduce your stress?

● When do you think you will implement the changes that are needed?

4. Negative thinking

Facts: Sessions 4, 5, and 6.

Strategies for changing your negative thinking:

● What is it in the situation that is causing you to have negative thoughts just now?

● Are your negative thoughts related to old habits?

● If you think realistically – do you consider that your negative thoughts are justifiable?

● How could more positive thinking affect your actions in this situation?

5. Difficulty in saying no

Facts: Sessions 6 and 7.

Strategies for saying no:

● Is "yes" what you want to say just now?

● If not – why are you not saying "no"?

● Is it important for you to help out when a person asks?

● Are you concerned that you will not be popular if you say no?

● If this is the case – ask to be allowed to come back later with your reply.

● Say no – without motivation causes

4.17 Anthropometry

Weight (kg), (Paper I–III)

Weight was measured (light clothing without shoes) to the nearest 100 g.

Height (cm), (Paper I–III)

Height was measured to the nearest 0.5 cm.

Body Mass Index (BMI), (Paper I–III)

BMI = weight(kg)/height^2 (m^2).

Waist circumference (WC) (cm), (Paper III)

WC was measured with a tape to the nearest 0.5 cm at the maximum circumference between the last rib and the highest part of the iliac crest.

Waist–to-hip ratio (WHR) cm/cm, (Paper III)

WHR was determined by measuring waist circumference at the maximum level over clothing to the nearest 0.5 cm and dividing this with the hip circumference.

Pulse (Paper III)

The pulse was measured in the sitting position.

Systolic and diastolic blood pressure (Paper III)

Blood pressure was measured to the nearest mm in the right arm with the patient in the sitting position, usinga standard mercury sphygmomanometer.

4.18 Laboratory analysis (Paper III)

Total cholesterol

High-density lipoproteins (HDL-C)

Low-density lipoproteins (LDL-C)

Triglycerides

Blood glucose

4.19 Questionnaries

Assessment of Anorexia/Bulimia (BAB) (Paper I)

44 questions for assessing eating disorders and psychosocial problems (227).

The Three Factor Eating Questionnaire (TFEQ) (Paper III)

The Three Factor Eating Questionnaire comprises three subscales: cognitive restraint (six statements), uncontrolled eating (nine statements) and emotional eating (three statements). Internal consistency reliability, Cronbach´s alpha, varied between 0.76 and 0.85. Construct validity was satisfactory (161).

The Obesity Related Problems Scale (ORPS) (Paper III)

The Obesity Related Problems Scale measures the impact of obesity on psychosocial functioning. Subjects are asked how anxious they feel in a broad range of social situations. Higher scores indicate more psychosocial dysfunction. The reliability coefficient was high and construct validity was satisfactory (107).

Eating Disorder Patient´s Expectations (EDPEX 1) (Paper III)

EDPEX 1 was filled in at baseline. It consists of 14 statements related to the question: "What do you think would help you during treatment?" Each of the 14 items belonged to one of the three subscales: Support (5 items), Control (4 items) and Insight (5 items). Inter-item reliability for the subscales Support, Control and Insight was: 0.81, 0.66 and 0.77, respectively (161).

Eating Disorder Patient´s Experiences (EDPEX 2) (Paper III)

EDPEX 2 was filled in immediately after 10 sessions. It has the same structure as EDPEX 1 and allows the participants to answer the question: "What did help you during treatment?" Inter-item reliability for the subscales Support, Control and Insight was: 0.86, 0.83 and 0.80, respectively. Principal components analysis reflected the same structure of both EDPEX Scales (161).

Knowledge questionnaire (Paper II)

20 questions were given to the treatment participants, specifically relevant to each of the two programmes.

Staff experiences (Paper III)

Customized Questionnaire to group leaders.

This was filled in when the group leaders had gained experiences from two to four groups each. It consisted of 10 questions and statements about experiences from and attitudes to the CBT-TEB programme (Paper III).

4.20 Statistical methods

Paper I

Student's t-statistic was used to test differences between group means. Differences inproportions between groups were analysed with the Chi-square test. Statistically significant differences were assumed when $p<0.05$ (two-tailed test).

Paper II

Student´s t-test statistic was used to test differences in mean weight change between groups. The Mann-Whitney U test was applied in the analysis of the obesity knowledge test. Differences in proportions between groups were analysed with the chi-square test. Statistically significant differences were assumed when $p<0.05$ (two-tailed test).

Paper III

Mean changes from baseline were tested for significance with paired t-tests when effect measures were on interval scales. Ninety-five percent confidence intervals were calculated. The Wilcoxon matched-pairs signed ranks test was used when effect measures were on ordinal scales. Spearman rank order correlation was used. Fisher´s exact test, t-test or analysis of variance were used when between-group differences were tested for significance. The chi-square distribution test was used. Forward stepwise multiple linear regression was employed to examine the extent to which changes in primary outcome variables could be explained by changes in the different eating behaviours, and, how changes in obesity-related problems could be explained by changes in the primary outcome variables. Effect size was calculated as change divided by the standard deviation of change (standardised response mean, SRM) (228). $P<0.05$ (two-tailed test) was considered statistically significant.

5 RESULTS

5.1 Paper I

The study aims to investigate the effects of CBT–TEB in obese patients in comparison with waiting-list controls.

Mean weight changes at follow-up 18 months after end of CBT-TEB treatment. Per-Protocol analysis

Table 4 shows weight at baseline and 18 months after the end of treatment for patients and controls. Table 5 shows mean weight changes for patients and controls after treatment and 18 months later. Average BMI fell from 40.4 to 36.6 in the treatment group.

Table 4. Mean weight at baseline and 18 months after end of treatment for CBT-TEB patients and controls.

	Weight (kg) at baseline Mean (SD)	Weight (kg) at 18 months follow-up Mean (SD)	p
CBT-TEB patients (n=34)	111.0 (16.1)	100.6 (15.9)	<0.001
Controls (n=31)	111.1 (15.5)	113.4 (18.6)	

Table 5. Mean weight changes after end of treatment and at 18-month follow-up for CBT-TEB patients and controls.

	Weight change (kg) immediately after treatment Mean (SD)	Weight change (kg) at 18 month-follow-up Mean (SD)	p*
CBT-TEB patients (n=34)	-8.5 (16.1)	-10.4 (10.8)	<0.001
Controls (n=31)		+ 2.3 (7.0)	

*Sign. diff. between groups at 18 months (mean weights).

Twelve patients (35%) showed a weight loss of 10% or more after 18 months. Six of these showed a weight loss of 15%. The weight differences between the groups were significant ($p<0.001$) at all follow-ups.

Mean weight changes at 12-month follow-up. Per-Protocol analysis

40 patients (70.2%) in the treatment group participated in the follow-up 12 months after the end of treatment (Table 6)

Table 6. Mean weight changes after treatment and at follow-up 12 months after end of treatment

	Weight change (kg) immediately after treatment Mean (SD)	Weight change (kg) at follow-up treatment after 12 months Mean SD
CBT-TEB patients (n=40)	-8.3 (4.3)	-10.7 (11.0)

Of the patients (n=34) in the treatment programme that participated in the 18-month follow-up, 14 showed a weight reduction whereas 20 showed an increase in weight as compared with the 12-month follow-up. All of the patients (n=6) who took part in the 12-month follow-up but not in the 18-month follow-up showed an increase in weight at twelve months from baseline.

Binge Eating Disorder (BED). Per-Protocol analysis

Of the 105 patients (62 of whom began treatment and 43 controls) 58 (55%) were diagnosed with BED. Of these 58, 34 patients (59%) took part in the follow-up 18 months after treatment. Eighteen of these were in the treatment group and 16 were in the control group (Table 7).

Table 7. Patients with BED. Mean weight at baseline and weight change 18 months after treatment.

	Weight change (kg) at baseline Mean (SD)	Weight change (kg) after18 months Mean (SD)	p*
CBT-TEB patients (n=18)	115.5 (16.0)	-11.2 (11.6)	
			<0.001
Controls (n=16)	109.0 (13.8)	+3.0 (7.0)	

*Sign. diff. between groups at 18 months (mean weights).

Mean weight changes at 18 months after end of treatment. Intent-to-treat (ITT) analysis

The rate of drop-outs in the 18-month observation period was 40% together with the drop-outs from therapy. Therefore a Baseline-Carried-Forward Analysis was undertaken. In this the mean weight difference between the CBT-TEB group and controls was 7.3 kg at 18 months (Table 8).

Table 8. Mean weight changes at 18 months after end of treatment. Baseline-carried-forward analysis.

	Weight change (kg) After 18 months Mean (SD)
CBT-TEB patients (n=62)	-5.7 (9.5)
Controls (n=43)	+1.6 (6.0)

The difference between the groups was significant (p-<0.05).

Of the 34 treated patients who participated in the follow-up at 18 months after the end of treatment 23(67%) showed a weight loss of 5% of more, whereas only 3(10%) of the control patients showed a weight loss of 5% or more.

5.2 Paper II

The aim of this study was to describe the efficacy of CBT-TEB in non-patient working women with obesity where controls received another active intervention.

Per-Protocol analysis

Thirteen (87%) of the women who completed the CBT-TEB programme and 16 (80%) of the women who completed the control programme participated in the follow-up 18 months after treatment terminated (Table 9).

Table 9. Mean weight at baseline, weight after the end of treatment and 18 months after the end of treatment (Per-Protocol analysis).

	Weight (kg) at baseline Mean (SD)	Weight (kg) after treatment Mean (SD)	Weight (kg) after 18 months Mean (SD)
CBT-TEB patients (n=13)	95.1 (11.5)	86.5 (10.4)	89.2 (12.7)
Controls (n=16)	91.5 (14.1)	90.8 (14.5)	91.8 (14.0)

The weight change differences between the cognitive group and controls groups were significant ($p<0.01–0.001$) at all follow-up weighings.

Table 10 shows weight changes for CBT-TEB patients and controls after treatment and at the 18-month follow-up.

Table 10. Weight changes for CBT-TEB patients and controls after treatment and at the 18-month follow-up.

	Weight change (kg) after treatment Mean (SD)	Weight change (kg) at 18-month follow-up Mean (SD)
CBT-TEB patients (n=13)	-8.6 (2.9)	-5.9 (5.4)
Controls (n=16)	-0.7 (1.2)	+0.3 (4.3)

The weight change differences between the cognitive group and controls groups were significant ($p<0.01–0.001$) at all follow-up weighings. The effect size (standardized response mean) for the CBT-TEB group at 18 months was 1.1.

Intention-to-treat (ITT) analysis

All participants who started the cognitive (n=16) or control (n=26) programmes were included here (Table 11).

Table 11. All weight changes for patients and controls after treatment and at the 18-month follow-up.

	Weight change (kg) after treatment Mean (SD)	Weight change (kg) at 18-month follow-up Mean (SD)
CBT-TEB patients (n=16)	-7.7 (3.8)	-5.5 (5.5)
Controls (n=26)	-1.4 (1.6)	-0.6 (5.5)

The weight change differences between groups were highly significant ($p<0.001$) at 18 months after the end of treatment. Effect size at 18 months was 1.0 for the cognitive programme.

Knowledge examination

The cognitive group scored much higher than did the control group on knowledge relevant to cognitive treatment of obesity but somewhat lower on knowledge of nutrition and physical activity (Table 12).

Table 12. The Results of the Obesity Knowledge Questions and Statements.

	Cognitive Group (n=14) Mean score	Control Group (n=13) Mean score	p
Knowledge relevant to cognitive programme	17.5	4.0	<0.001
Knowledge relevant to control programme	16.0	18.0	0.025

The results indicate that the two treatment programmes convey different types of information.

5.3 Paper III

The study aims to investigate the effectiveness of CBT–TEB in primary care patients in an observational study.

Intent-to-treat (ITT) analysis

In Table 13 baseline data and mean changes in the primary outcome measures after therapy and at six months post therapy are presented. The proportions of patients who had lost at least five percent of the baseline values at the 6-month follow-up were: WC 41%, WHR 28%, Weight 39% and BMI 39%. Forty-seven percent had lost 5 cm or more of baseline WC.

Table 13. Primary outcome measures at baseline, change after therapy and at 6 months after end of therapy. ITT analysis (N=83).

Outcome measure	Baseline Mean (sd)	Change after therapy Mean (sd)	P*	Change baseline – 6 months after end of therapy Mean (sd)	95% Cl	P*)	SRM**)
WC (cm)	111.9 (10.3)	-4.9 (6.7)	<0.0000	-4.8 (7.0)	6.3, -3.3	<0.0000	0.69
WHR (cm/cm)	0.95 (0.07)	-0.022 (0.04)	<0.000>	-0.027 (0.04)	0.037, 0.018	<0.0000	0.68
Weight (kg)	96.7 (13.7)	-4.5 (3.9)	<0.0000>	-4.4 (4.9)	5.5, -3.4	<0.0000	0.90
BMI (kg/m^2)	34.6 (4.3)	1.6 (1.4)	<0.0000>	-1.6 (1.8)	2.0, -1.2	<0.0000	0.89

*) Paired t-test, **) Standardized response mean (228).

Patients who completed therapy (n=71) were included in Table 14. Baseline data and changes in primary outcome measures after therapy and six months after the end of therapy are shown in the table. The proportion of patients who had lost five percent or more at the 6-month follow up were: WC 48%, WHR 32%, Weight 44% and BMI 43%.

Completers analysis

Table 14. Completers analysis (N=71). Primary outcome measures at baseline, change after therapy and at 6 months after end of therapy. Last observation carried forward.

Outcome measure	Baseline Mean (sd)	Change after therapy Mean (sd)	P*	Change baseline – 6 months after end of therapy Mean (sd)	5% Cl	P*)	SRM**)
WC (cm)	112.0 (10.0)	-5.8 (7.0)	<0.0000	-5.5 (7.3)	-7.3, -3.8	<0.0000	0.76
WHR (cm/cm)	0.95 (0.06)	-0.026 (0.05)	<0.0001	-0.031 (0.05)	-0.042, -0.020	<0.0000	0.68
Weight (kg)	96.5 (13.5)	-5.0 (3.8)	<0.0000	-5.0 (5.0)	-6.1, -3.8	<0.0000	1.00
BMI (kg/m^2)	34.5 (4.3)	-1.8 (1.3)	<0.0000	-1.8 (1.8)	-2.2, -1.3	<0.0000	0.98

*) Paired t-test, **) Standardized response mean (228).

Changes of eating behaviour and obesity related psychosocial problems (ITT)

Eating behaviours changed significantly during therapy in a salutogenic direction according to TSEQ. Also obesity related problems (ORPS) decreased significantly during therapy (Table 15)

Table 15. Eating behaviour (TSEQ) and obesity related psychosocial problems (ORPS) at baseline and change after therapy. Excluded: 7 patients where baseline data were lacking.

Outcome	Baseline		Change after therapy			
measure	Mean	(sd)	Mean	(sd)	P*)	SRM**)
TSEQ	41.1	(17.5)	+27.1	(20.6)	<0.0000	1.32
Cognitive restraint						
TSEQ	48.1	(22.1)	-15.8	(18.9)	<0.0000	0.84
Uncontrolled eating						
TFEQ	59.3	(23.9)	-20.6	(21.7)	<0.0000	0.95
Emotional eating						
ORPS	52.7	(27.2)	-14.1	(23.4)	<0.0000	0.60

*) Wilcoxon matched-pairs signed ranks test, **) Standardized response mean (228).

Patients expectations and experiences of the CBT-TEB programme before and after participation

Table 16 shows patient expectations before participating in the programme and their experiences of the programme, given after participating

Table 16. Mean (SD) scores of EDPEX 1 subscales (expectations) before therapy and mean (SD) differences between EDPEX 1 and EDPEX 2 (experiences). Attrition: 7 patients. N=64.

EDPEX	Expectations at baseline		Difference between expectations and experiences of therapy		
Subscale	Mean	(SD)	Mean	(SD)	p*)
Control (4 items)	17.9	(4.1)	+1.1	(4.7)	ns
Insight (5 items)	19.2	(6.5)	+2.2	(6.7)	<0.01
Support (5 items)	20.2	(6.3)	+3.9	(7.2)	<0.0001

*) Wilcoxon matched-pairs signed ranks test.

Group leaders experiences of the CBT-TEB programme

The group leaders experiences of the programme are shown in Table 17.

Table 17. Group leaders´ (N=10) experiences (absolute numbers): "How well do these statements agree with your experiences?"

Statement	Disagree totally	Disagree partly	Agree partly	Agree totally	p*
My competence increased	-	2	1	7	<0.01
My stress increased	7	2	1	-	<0.01
I had interesting experiences	-	-	2	8	<0.001
My self-confidence increased	1	4	2	2	ns
My interest in obesity treatment increased	-	4	2	4	ns

*) Chi-square distribution test (null-hypothesis: uniform distribution).

6 DISCUSSION

6.1 Methodological aspects

The CBT-TEB programme

In three studies the intention was, in the short and medium terms, to evaluate a new treatment programme for obesity. The programme is short (Introduction +10 sessions), takes place within a group and is manualised. The programme is an educational treatment, the contents of which deviate from the majority of treatments for obesity. Theoretically, by combining CBT and psychopedagogy; in practice, through anticipated weight loss achieved via the patients' knowledge of the causes of their dysfunctional eating behaviour and through being given cognitive tools that address these causes – and not through focusing on eating behaviour per se. Other differences from most obesity treatments are that the dietary programme that is part of CBT-TEB only aims to restrict the amount of food eaten per day and does not involve any form of diet, plus the fact that the programme is short. Similarities with other programmes are the group format, the fact that it is CBT-related and the inclusion of a dietary programme.

In Studies I and II the patient groups were guided by the author, whereas the groups in Study III were guided by personnel at health centres who had undertaken a short training course. The fact that all groups in Studies I and II were guided by the author may have provided an advantage over the groups in study III. The participants in Study III were guided by 10 instructors from the healthcare sector. The majority of these had not previously dealt with obesity patients, either individually or in a group. In addition, the instructors were not familiar with the patient group and its problem.

The dietary programme

The dietary programme component is a traditional dietary programme, compiled by a registered dietitian and based on normal food. The participants can therefore eat the same food as their families and follow their own favourite recipes. The amount of food eaten per day is controlled by weighing the food. What part does the dietary programme play in the results from the CBT-TEB programme? All participants in the studies, apart from one, had previously tried a variety of dietary programmes in order to lose weight. The majority of participants had previously been members of ViktVäktarna (weight watchers). ViktVäktarna uses various methods to restrict the amount of food eaten – one of them had previously just involved weighing the food. The dietary programme was the method that was most familiar to the majority of the participants. So is a dietary programme really needed in CBT-TEB? The majority of the participants, of course, already knew how to eat in order to lose weight.

A question frequently asked by overweight and obese individuals is “why do I eat badly, when I know how to eat correctly?” Via the programme, CBT-TEB aims to provide participants with the knowledge both to answer this question and to acquire mental tools that will deal with the causes of their problems. Would it therefore not be sufficient for the

participants to simply acquire this mental knowledge? Katzer et al. (2008) conducted a randomised study which answered this question. Stress had been identified as a major cause of overweight women eating too much, so the initiative was taken to set up a 10-week Stress Reduction programme to enable overweight women to lose weight. A follow-up 12 months after the end of the programme showed improved stress management but no weight loss (229). If studies have now identified stress as a contributory cause of obesity, why does an anti-stress programme without a dietary programme lead to improved stress management but not to weight loss? In my clinical experience most overweight and obese people eat too much without being aware of it. Participants who, during interviews with me, said "I always eat so little" often at the end of the programme expressed surprise over what large portions they had previously been eating, which at the time they had thought were small. How much food to consume per day is therefore an important item of knowledge for someone attempting to lose weight; without this knowledge people who are overweight will continue to eat too much and will therefore remain overweight. CBT-TEB's dietary programme gives the participants knowledge about how much food they should eat per day in order to achieve weight loss. The results from studies I – III show that the combination of dietary programme + cognitive programme can be regarded as a successful combination.

Study groups

Study groups consisted of both patients and non-patients. The purpose was to examine the programme's generalisability to different groups of participants. This means that participants in Study I were waiting-list patients from the Obesity Unit at Karolinska University Hospital, Huddinge, participants in Study II were workers from a child care centre in the Huddinge community in Stockholm, participants in Study III were patients from five primary care centres in the Hisingen primary healthcare area, Gothenburg. The participants in Study I and Study II were all women, whereas the participants in Study III consisted of 74 women and 9 men.

Only the participants in Study I had themselves taken the initiative to obtain treatment for their obesity – the participants in studies II and III had been seen to be obese by healthcare personnel and had been asked if they were interested in participating in the weight loss programme. There is reason to suppose that this difference was of significance in the study results. The majority of participants in the three studies were women, which represents a shortcoming with regard to the generalisability of the treatment. At the same it indicates that women are more interested in treatment for their obesity than men are.

Both participants and controls in Study I had higher mean BMI than the participants in the two other studies. One inclusion criterion in Paper I and II was BMI >30 kg/ m^2. In Study III one of the inclusion criteria was BMI >25 kg/m^2 and 12% of the participants had a baseline BMI of 25.0-29.9. Moreover, the participants in Paper I showed higher mean BMI as well as greater weight loss than the participants in Paper II and Paper III at the final follow-up 18 months after treatment and when compared with participants in Paper III after treatment. A

major reason for this difference might be that Study I had a larger number of participants with a BMI over 35 than did Studies II and III. It is reasonable to assume that someone with a higher BMI consumes more kcal per day than someone with a lower BMI. The dietary programme's calorie restriction can therefore result in a greater weight loss in individuals with higher BMI than in those with a lower BMI.

Effect measures

Weight loss immediately after therapy and at the follow-up 18 months after the end of therapy was the effect measure in Papers I and II. In Papers I - III the results were recorded both per-protocol and via ITT. However, in Paper III waist circumference and waist-to-hip ratio were used as the primary measures of outcome. 4 out of 6 of the secondary measures of outcome have been shown to have good validity and reliability (the scales EE, UE, RE and ORPS). The EPDEX scales have been shown to have good reliability (161).

Limitations

The small sample size in Paper II and the fact that only women served as participants, are two methodological limitations in Paper I and Paper II. Another limitation concerns the high drop-out rate in Paper II that occurred before treatment started in one of the subgroups randomised to the cognitive programme. Consequently, this subgroup became small, which could have impacted on the degree of individual focus. Moreover, there could have been a selection bias in this subgroup that could have affected the outcome. However, there was no substantial difference between the two cognitive subgroups regarding outcome at 18 months. Another limitation is the short follow-up period of Study III and the lack of a control group.

6.2 Discussion of results

Weight loss

Weight loss differences between the studies

There are very few randomised studies which have evaluated behaviour-oriented weight reduction programmes with a follow-up period longer than one year. It has been concluded that within one year of treatment, most patients regain 60-70% of the weight they lost during the programme. Earlier research indicates both weight gain or a noticeable return to baseline weight one year after the end of treatment (141, 230, 90, 112). Cooper & Fairburn argue that the weight increase often begins at the same time that treatment ends (90). However, this is not the case in Study I and Study II.

Successful weight loss defines as a loss of more than 5% of initial weight (89). It has been shown that this loss of body weight produces beneficial changes in health risk factors (231, 87). In Study I, 67% of the treated patients still fulfilled this success criterion at 18 months after the end of treatment. In the same study, only 6% of the treated patients weighed more at 18 months than at baseline, compared with 71% of the controls (per-protocol). The mean

differences between the CBT-TEB group and controls was 12.7 kg per-protocol (7.3 kg, ITT) at 18 months.

In Study II, 46% of those participating in the CBT-TEB programme 18 months after treatment ended showed a weight reduction of 5% or more, compared with only 13% of those in the control program. Thus, the long-term result of CBT-TEB can be described as successful in about half of the cases in this study. In Study III 44% (39% therapy completers analysis) of the CBT-TEB participants showed a weight reduction of 5% or more six months after the end of treatment. Overall, these results show a favourable outcome.

Motivation has been of major importance in participation and in the implementation of psychological programmes. A number of studies emphasise what is crucial in altering behaviour (232, 233 234). The results should therefore be seen within the context of the following circumstances: virtually no patient in Paper II and III had approached heathcare services with the intention of requestingweight loss therapy. Unlike in Paper I, where the participants in CBT-TEB were waiting-list patients and had requested treatment, the participants in Paper II and Paper III filled in health questionnaires and were asked by healthcare personnel if they wished to participate in a weight loss programme. It is reasonable to assume that the participants in Study I were more motivated to cooperate in the treatment programme and that is reflected in the weight loss.

The majority of CBT-TEB participants in Paper I and Paper II worked full-time or part-time. In Paper III nearly half the participants were not in the jobs market because they were on sick leave, unemployed or on an old age pension. Several CBT-TEB participants in Paper III were born outside Scandinavia. To benefit from the CBT-TEB programme a good knowledge of the Swedish language is needed. If one's understanding of the language is inadequate this can affect one's understanding of the knowledge which the programme aims to impart and can thereby also affect the outcome of the programme. These assumptions were supported by Elfhag & Rössner who, in their study of the causes of drop-out, concluded that completers had a higher educational level, were more often born in Sweden and were occupied with working or studying (235).

Weight loss in abdominal obesity

BMI is the most commonly used measure for estimating the degree of an individual's obesity. The BMI measure is limited by the fact that it does not differentiate between fat and muscles (16). The inclusion age was higher in study III than in the other studies. The risks for women and men with high BMI were highest under the age of 50 and studies have emphasized that the measurement of waist circumference should be used to a greater extent (16, 17, 18,). As this measure is well correlated with abdominal obesity, which involves an increased risk of cardiovascular disease, there was particular reason to use it to complement weight and BMI with waist circumference (WC) and waist-to-hip ratio (WHR), above all in study III, where the inclusion age was lower than in the other studies.

In Study III, primary outcome variables, apart from weight and BMI, were therefore waist circumference (WC) and waist-to-hip ratio (WHR). It is well known that a 5-10 % loss produces beneficial changes in health risk factors (89, 236). In this study the proportions of patients who had lost at least five percent of the baseline values (ITT) at the 6-month follow-up were: WC 41%, WHR 28%, weight 39% and BMI 39%. As a control group is lacking, Study III can be mainly seen as exploratory and as an hypothesis-generating investigation and not a study in which hypotheses are tested rigorously. Conclusions must therefore be drawn with caution. That said, the main results should be regarded as satisfactory. However, comparative values 6 months after treatment were not found in either study I or study II. The results must therefore been seen within the context of this limitation and the short follow-up time.

Weight loss in BED

Binge Eating Disorder (BED) was diagnosed via questionnaire (Clinton & Norring) and DSM IV (227, 67). Of the 105 patients, 58 (55%) were diagnosed with BED. Of these, 34 (59%) (18 from the treatment group and 16 from the control group) participated in the 18-month follow-up.

BED is regarded as a complicating factor in the treatment of obesity. It was therefore seen as interesting that the treated BED patients showed a greater weight reduction than other patients. This difference is not statistically significant, however. The CBT-TEB programme included the understanding that overeating is often an expression of a compensation mechanism for negative feelings, which result in a dysfunctional behavioural approach to everyday situations that quickly leads to overeating. Analyses of events that preceded loss of control in eating were therefore central to the present approaches to treatment. Perhaps this approach, and the special tools in the programme called “Key-Questions”, might explain the good treatment results for patients both with and without BED. There is reason to investigate this possible connection.

CBT is successful in reducing binge eating in the short term (72, 79). Few studies to date have given an account of 1-year follow-up results of CBT treatment of BED. One such study has shown a smaller weight increase during the follow-up period (237). Another one-year follow-up study with group CBT showed that patients who stopped binge eating during the study maintained a weight loss of 4.0 kg over the follow-up period (230).

Comparison with other similar studies

Van Dorsten established in a review of cognitive and behavioural approaches in the treatment of obesity (in which the methods and results for Studies I and II are also recorded) that it is difficult to compare behaviour-focused and cognitive studies because of their different setups, contents and follow-ups (145). A few programmes have short treatment times similar to those of CBT-TEB, as well as similar follow-up times. Other studies often lack information about the recording of weight per-protocol or via ITT analysis. Brown et al. describe weight results

from a lifestyle-related educational programme, SliM, for obesity treatment, in a 6-month non-randomised study without follow-up (239). The programme teaches participants to use cognitive techniques to help them cope with the stresses of life, instead of using food as a coping strategy. 56% of the participants completed the programme, achieving a mean weight loss of 5.5 kg.

Comparison with CBT-TEB: 92% of the participants completed the CBT-TEB programme in Study I, 87% in Study II, and 86% in Study III. Mean weight loss for patients after treatment and participated in the 18 month follow-up at the end of the program (10 weeks) was in Study 1: 8.5 kg(n=34, 60 %), in Study II 8.6 kg (n=16, 80%). In Study III the mean weight loss for patients who participated in the 6 month follow-up was: 5.0 kg (n=61, 73,5%).

Cooper et al. compared the results from CBT, BT (behaviour therapy) and GSH (guided self-help) for three years and concluded that after 3 years practically all participants had regained the weight they had lost during the programme (240). The CBT and BT programmes each lasted 44 weeks and the GSH programme 24 weeks. (GSH is based on a commonly used weight reduction programme: LEARN, which is designed to bring about change in five areas: lifestyle, exercise, attitudes, relationships and nutrition) (241). As this study does not give results for an 18-month follow-up, I have selected a 12-month follow-up for comparison with the 18-month follow-ups in Studies I and II. The results for the 12-month follow-up were: GSH (N=51) -2.3 kg, CBT (N=47) -3.3 kg, BT (N= 44) -7.7 kg.

Comparison with CBT-TEB: weight loss at 18-month follow-up: Study I: 10.4 kg (per-protocol) 5.7 kg (ITT), Study II: 5.9 (per-protocol), 5.5 kg (ITT).

The aim of another study was to evaluate in clinical practice a treatment programme for obesity, which mainly focused on changing nutrition and lifestyle (BASEL) and which included elements from psychoeducation, (242). There was a total of 122 patients with an average age of 45.2 years. The programme consisted of three groups: two treatment groups and one control group. The group treatment consisted of a total of 16 sessions of 90 min each and was standardised according to the therapy concept of the BASEL programme. The mean changes in body weight at follow-up one year after treatment in the treatment groups were -4.7% and -2.9 %, respectively.

More and more studies are recording weight loss greater than the 5% loss regarded as conferring health benefits and as defining successful weight loss (243). Wilfley et al. also observed at a one-year follow-up that ¼ of their CBT patients had lost more than 5% of their body weight (244).

Comparison with CBT-TEB: 67% of the patients treated in Study I and 46% of the patients in Study II had lost at least 5% at 18 months after treatment (per-protocol). In Study III 39% (ITT) had lost at least 5% at 6 months after treatment.

Rapaport et al. reported results from a study in which both a modified form of CBT and traditional CBT were used to treat 63 overweight women (146). Both treatment groups

involved 10 weekly 2-hour group meetings. The results following treatment showed a weight loss of 3.9 kg for the 38 individuals in the CBT group, compared with 1.3 kg for the group which used the modified CBT. The mean weight loss after the end of treatment was 3.9 kg in the S-CBT group and 1.3 kg in the M-CBT group. By the 52-week follow-up, mean weight loss in the S-CBT group had reduced slightly to 3.6 kg and in the M-CBT group had increased slightly to 2.0 kg.

Comparison with CBT-TEB: Papers I and II included no weight values at the 1-year follow-up. Weight losses at the 18-month follow-up for CBT-TEB were: Study I, 10.4 kg (per-protocol) and 5.7 kg (ITT); Study II, 5.9 kg (per-protocol) and 5.5 kg (ITT).

Did CBT-TEB provide any specific knowledge?

In recent years CBT has been developed into an umbrella diagnosis under which authors often define lifestyle-changing programmes as cognitive behaviour-oriented (245). Other authors, such as Cooper and Fairburn, maintain that the concept of cognitive behavioural therapy should be reserved for those treatments whose primary goal is cognitive change (90). According to Cooper and Fairburn cognitive behavioural therapy for obesity does not seek to change eating behaviour per se but aims to do so via the cognitive processes that maintain these forms of behaviour. This is a treatment objective which CBT-TEB strives to achieve through the programme's transfer of knowledge, not only of the thought patterns and attitudes that affect our behaviour, but also of the interpersonal factors that underlie our cognitions.

To examine the differences in content between the CBT-TEB programme and the control programme in study II, the programme participants answered a number questions which were knowledge-specific for each programme. The results showed that the cognitive programme participants scored much higher than did the control group on knowledge relevant to the cognitive treatment of obesity but somewhat lower on knowledge of nutrition and physical activity. This suggests that CBT-TEB probably gives patients knowledge which they can apply not only to the causes of their eating behaviour but also to the causes of their behaviour in general. As low self-esteem is a common cause of both negative thoughts and feelings, knowledge of the causes of poor self-esteem is imparted and tools to enhance self-esteem are provided. A number of studies emphasise stress as a common cause of overeating (47, 48, 49, 51, 53, 56, 60, 182). Knowledge of both the general and individual causes of stress is therefore a major part of the programme – knowledge which patients can of course apply to their everyday life and not only to their overeating.

Certainly, CBT therapy supplies a large part of the knowledge which CBT-TEB also provides for patients. On the other hand, the self-knowledge which the programme gives patients about the causes of their dysfunctional eating, combined with the cognitive tools which the programme provides to address these causes, should be regarded as specific knowledge for imparting via CBT-TEB, making this weight loss programme different from others.

Changes of eating behaviour after CBT-TEB

According to the TFEQ, eating behaviours changed significantly during therapy. Cognitive eating restraint (TFEQ CR) increased whereas uncontrolled eating (TFEQ UE) and emotional eating (TFEQ EE) decreased. This change was not unexpected. The increase in restrained eating was expected because the participants followed a dietary programme. The decrease in emotional eating was also expected as CBT-TEB placed a lot of emphasis on the causes behind emotional eating and the provision of cognitive tools to address these causes. Koenders thinks that emotions may drive people who are overweight or obese to overeat and believes that if we want to cure the disease, psychological treatment strategies have to be developed (133). Emotional eating is seen by Spoor et al. (164) as an emotion-focused coping and is defined in another study as an inability to resist emotional signals (195). With both uncontrolled eating and emotional eating, some of the same factors predispose an individual to loss of control over eating in general (uncontrolled eating) and to eating triggered by emotional situations (emotional eating) (27). It is therefore not surprising that even uncontrolled eating was reduced by the programme. Because of reductions in both emotional and uncontrolled eating, restrained eating increased. The dietary programme which dictates, in terms of food weight, how much a participant can eat each day and still lose weight, probably also contributed to this increase.

Changes of life quality after CBT-TEB

In agreement with several studies it was shown that obese people experience a poorer health-related quality of life (HRQL) compared to the general population (246, 247, 248, 249, 250). According to Kaplan & Ries there are two major approaches to quality of life assessment: profile and decision theory (251). The psychometric approach is used to offer a profile summarising different dimensions of quality of life. The best known example of the psychometric tradition is the Medical Outcomes Study 36-Item Short Form (SF-36) (252).

Does CBT-TEB bring about positive changes in obesity-related quality of life? Before and after the treatment the participants had to answer questions about the impact of obesity on their psychosocial functioning; the Obesity Related Problems Scale (ORPS) (107). Subjects were asked how anxious they felt in a broad range of social situations. Examples of questions: Do you feel embarrassed because of your weight or body shape when you: travel (?), try and buy clothes (?), have sexual intercourse? Higher scores indicate more psychosocial dysfunction. After CBT-TEB treatment the scores were lower than before treatment.

Low self-esteem and body shame are common consequences of obesity (253, 254). With higher self-esteem, weight reduction is maintained over a longer period of time (255). Therefore CBT-TEB also provides both advice and exercises to enhance the participants' self-image and self-esteem. The programme advises how to avoid thoughts of resignation and hopelessness/helplessness, and how to replace these with more positive thoughts as well as enthusiastic and solution-oriented actions, knowledge which may be reflected in answers to

questions in ORPS. Given that answers to these questions are limited to numbers, and that there is no control group, it could be concluded that the positive change in obesity-related quality of life can probably be achieved by CBT-TEB.

The positive changes in both eating behaviour and quality of life which came about after CBT-TEB, can be seen as contributing to future obesity treatment, but one must also look beyond the limited population at whom the questions were targeted and consider the fact that there was no control group in Paper III. However, the results can serve as an example of a short term treatment for obesity, possibly affecting variables other than weight loss, some of which, moreover, are amongst the underlying causes of obesity.

How acceptable is CBT-TEB to patients?

The number of drop-outs during an obesity treatment is generally high (256, 257, 258, 259). A randomised 2-year clinical trial without follow-up reported a drop-out rate of 26% during treatment (260). Colombo et al (2014) reported in a 6-month dietary behavioural weight-loss treatment that the drop-out rates were 21% at 1 month and 57% at 6 months, posing the question of whether drop-out from treatment is a predictable and preventable event (261).

In Paper I, 92% of the 57 patients who started CBT-TEB, completed the programme. 75% who completed treatment were still in the study at the 12-month follow-up. Of the 16 women in Paper II who started the CBT-TEB programme, 15 (94%) women completed the programme. For those 26 women in Paper II who started the control programme, 20 (77%) completed the programme. In Paper III 71 (86%) individuals completed the CBT-TEB programme. Since 86-94% of those who began CBT-TEB completed the programme, the answer to the question must be that the treatment method was very acceptable to the majority of the patients.

The group leaders' experiences of the programme

Of the ten group leaders only one had previous experiences of the CBT-TEB programme. Group leaders' experiences of the programme were mostly very positive. Nine of them were willing to be the group leader for the CBT-TEB programme in the future. Half of the group leaders perceived their training as too short – important knowledge for the future.

Nevertheless their experiences of CBT-TEB were positive and most felt that their competence increased and their interest in obesity treatment was enhanced. Also the majority of group leaders had a very positive opinion of the programme: nine out of ten were in favour of the programme. When they were asked the question: "Can you imagine yourself continuing as a CBT-TEB group leader in the future?", nine answered "Yes, absolutely". The group leaders' comments represent a positive response to the questions below.

Would it be feasible to implement CBT-TEB in primary care using primary care personnel to carry out the treatment?

CBT-TEB was developed mainly for use in primary care and obesity units (see preface) by healthcare personnel without psychotherapeutic training. This is why the entire treatment programme is manualised (222, 223). Since 2007, 272 individuals have undergone a 3-day training course in CBT-TEB. They comprise dietitians, social workers and nurses, the majority from healthcare centres around the country. As far as I know no scientific assessment of their work is available. At the same time, no studies have been published on the programme's implementation in primary care using CBT-TEB-trained healthcare personnel (personal communication).

After therapy, patients rated the components of support and insight in the CBT-TEB programmes higher than they had expected. Those features that were regarded most helpful during therapy, as reflected in the questions asked after the end of treatment (EDPEX 2), were increased control over eating behaviour, the acquisition of knowledge with which to reflect upon the issues that caused overeating, an awareness of the importance of regularmealtimes, and the fact that the patients had been treated with both care and consideration. The participants' replies show that the programme was mostly received positively by patients.

Because of its short duration, its group format and its results, CBT-TEB is regarded as a cost-effective treatment programme for obesity. The low treatment failure rates in three studies show that the programme is valued by patients, something which is also apparent in patient replies to the questionnaire (EDPEX 2) in study III, and in the instructors' assessment of the programme (study III). It is therefore concluded that CBT-TEB, by reason of the above, should have major potential for successful implementation in primary care.

6.3 What is wrong: the treatments or the obese people?

Cooper and colleagues noted that it is noticeably difficult for people with obesity to maintain their lower weight following weight loss (240). Most of those who seek treatment for obesity can lose weight but few are able to maintain the changes in behaviour that are required to prevent subsequent weight gain. After a CBT programme was modified to prevent weight gain after 44 weeks of treatment, it was observed at the 1-year follow-up that nearly all the participants (99%) had regained 50% of the weight they had lost and at the 3-year follow-up that almost all the weight lost had been regained (89.8% for those who had undergone BT and 88.6% for those who had participated in CBT). The authors therefore concluded that "obesity is resistant to psychological methods of treatment, if anything other than a short-term perspective is adopted" (240).

However, with regard to the results from the CBT-TEB studies, the authors' conclusions are seen as unconvincing. As humans we tend to adopt forms of behaviour which benefit us and we persist with this behaviour for as long as it is useful – and often for longer. The literature

clearly emphasises that the majority of overweight/obese people are emotional eaters and that it is often negative feelings which trigger overeating; as early as 1957 Kaplan and Kaplan stressed that obesity is caused by psychologically induced hyperphagia (153). The literature also clearly indicates that study after study has reported that the commonest way of helping people to lose weight is by focusing on changing eating behaviour, that is to say on the immediate causes of obesity rather than on the underlying psychological causes. This means that if there are negative feelings or a lack of control, which often trigger overeating, neither dietary programmes nor awareness of the causes of behaviour will prevent overeating in the long term until the causes of the negative feelings and the loss of control are worked through or dealt with. It also means that if overeating is used as a coping mechanism in negative situations, people who are overweight need to address the reasons why they need such a mechanism. Van Strien et al. observed that high emotional eaters are in the risk zone for becoming overweight as overconsumption appears to be more closely related to being overweight in individuals with high levels of emotional eating (262). The authors therefore concluded that it might be wise to seek an emotional explanation for the current epidemic of overweightness and to pose the implied question as to whether an individual's ultimate body weight is determined by a human tendency towards emotional eating.

However, emotional eating is not the only eating behaviour that is of importance. Eating behaviours can be divided into three types: Emotional eating, External eating and Restraint eating (153-156). The different eating behaviours are generally triggered by different stimuli and circumstances. These individual causes are important. Many people find it difficult not to eat something nice when they are stressed or feel worried. Others instead regard themselves as habitual eaters, and people in a third group control their eating so strictly that they finally give up and often entirely lose control over their eating. Nowadays we do not separate the treatment of obesity from these causative scenarios. Would the results of treatment be better if, before treatment, we identified the eating patterns of each presumptive obesity patient using scientifically designed eating behaviour questionnaires, and then adjusted the treatment accordingly? Clinical treatment studies are needed. We already have questionnaires but these need to be developed (159, 161).

Cooper observed that it was very difficult for people with obesity to maintain their weight loss and saw as an explanation for this the fact that few people are able to maintain the changes in behaviour that are needed to prevent subsequent weight gain. Obesity is an area of medicine that has traditionally been associated with the condemnation of certain types of personality. It was not until some years later that was it acknowledged that obesity could have genetic links (25, 26, 27), an association which previously had often been rejected by those administering treatment. Perhaps it is not so surprising that it is difficult to maintain weight loss if one takes into account what Hill and colleagues describe as the “Energy Gap”: changes in energy requirements that occur as a result of weight loss (263). When individuals lose body mass their total energy requirement also decreases. This results in lower energy consumption, which creates an energy gap that biases the body toward regaining lost weight. Results from studies with rats support the supposition that there is a compensatory metabolic

reaction to weight loss which induces the body to regain lost weight (264, 265). According to the authors a greater understanding is needed of how metabolic changes affect weight loss in order to devise strategies for helping patients to achieve a stable body weight following substantial weight loss.

6.4 Future directions

In clinical use, CBT-TEB should be combined with 2-hour follow-ups every 6 and12 months, as all studies show a certain amount of weight gain six months after treatment. Dorsten & Lindley observed in a review that "The call has been sounded to conceptualizeobesity as a chronic health condition requiring lifelong treatment (145)". I think there hasbeen excessive belief in and expectations of the ultimate abiding "lifetime effect" after a single treatment for obesity and I therefore support Dorsten & Lindley's understanding that the conceptualisation of cognitive behavioural therapies as a one-time treatment is outmoded. However, this presents healthcare with a financial challenge and, as the current number of obesity specialists and behaviourally trained professionals is insufficient to combat this problem, there may have to be increased emphasis upon training non-traditional weight specialists and non-behavioural community providers (150, 152, 141, 266, 267).

CBT-TEB can offer such an alternative: the programme, which uses manualised handbooks, was developed so that, after 3 days of training, community providers can be employed as non-traditional weight loss specialists amongst community providers. Innovative measures to promote behavioural changes in overweight and obese individuals should, according to Okorodudu et al., be encouraged and existing technical advances should allow treatment interventions to be delivered with the help of mobile phones, the internet and active video games (268). In a review the authors examined the results of internet-based interventions in obesity and found that, when web-based interventions were used to complement face-to-face interventions, weight loss was increased when compared with that achieved by just face-to-face treatment.

CBT-TEB has already been developed as web-based treatment programmes and it willsimple to translate these into mobile apps. There have also been positive experiences withthe use of mobile phones in healthcare as contact tools to remind patients of their treatmentschedules and also as a positive support mechanism after the completion of treatment.Video-conversation, such as with Skype, is a cost-free face-to-face tool for both computersand mobile phones and is nowadays also used successfully in psychological treatment.Using CBT-TEB as a traditional face-to-face form of care and supplementing this with web-based sessions may provide additional benefit in terms of weight loss and improved cost, and is a model for practical applications. Studies show that this combination is a promising tool and may be applicable to other and younger population as well (268).

The majority of overweight and obese people remain a treatment challenge for healthcare and there is a major potential for adopting new and innovative approaches. Treatment tools, which mean reduced personnel requirements and increased cost effectiveness, should

therefore be seen as a driving force and an investment motivation for reaching those large target groups which do not traditionally seek healthcare support for their obesity and which the healthcare sector does not have the resources to help

7 CONCLUSIONS

The conclusions are related to the specific aims/research questions of the thesis.

1. The medium/long-term (1.5 years) CBT-TEB targets for differences in weight change between patients undergoing CBT-TEB and waiting-list controls are 12.7 kg (per-protocol) and 7.7 kg (ITT), which demonstrates a satisfactory efficacy for the CBT-TEB programme. About two thirds of the therapy completers still fulfilled the widely held success criteria of having lost 5% or more of initial weight at 1.5 years after the end of therapy. The satisfactory effect size was confirmed by a high standardised response mean (1.0) at end of follow-up (Paper 1)

2. Regarding the medium/long-term efficacy of the CBT-TEB programme no significant difference was found between patients with or without BED (Paper I).

3. When the efficacy of the CBT-TEB programme was compared with another obesity treatment programme, the difference in weight change between programmes were 6.2 kg (per protocol) and 4.9 kg (ITT) from baseline to 1.5 years after the end of therapy, in favour of the CBT-TEB programme. About half of the patients in the CBT-TEB programme had still lost 5% or more of the baseline weight at 1.5 years after the end of therapy. The corresponding proportion among patients in the control programme was 13% (and 63% displayed a weight gain). ITT analysis 1.5 year after treatment showed a difference of 6.1 kg in weight, between patients in the CBT-TEB programme and the control programme (Paper II).

4. The CBT-TEB programme conveyed specific CBT-related knowledge which the control programme did not. There was no significant difference regarding knowledge of diet and physical activity among participants in the CBT-TEB and control programmes (Paper II).

5. Although lower than in the efficacy studies (Paper I-II), the short-term (6 months after the end of therapy) the effectiveness of CBT-TEB both in reducing body weight and abdominal obesity wasis regarded as satisfactory and seems to surpass most or all published CBT-based programmes for weight reduction in patients attending primary care. Effect sizes were high for weight loss (1.0 for therapy completers, 0.9 for ITT) and moderately high for reduction of waist circumference (0.8 for therapy completers, 0.7 ITT) six months after the end of therapy. The concept of effectiveness is not identical with efficacy which is determined in randomises clinical trials. The conclusions from the results in Study III must therefore be drawn with caution. In all probability, however, the CBT-TEB programme seems to cause a reduction in abdominal obesity (Paper III).

6. After having experienced the CBT-TEB programme, patients rated the programme's helpful components of support and insight more favourably than they had expected before therapy (Paper III).

7. The group leaders reported very positive experiences from the CBT-TEB programme and most felt that their competence increased and their interest in obesity treatment was enhanced. Probably future group leaders should receive a somewhat enlarged training programme (Paper III).

8. The inexpensive short-term group format, the amount of weight loss, and the limited drop-out rate, together with patients' and group leaders' positive experiences from the CBT-TEB programme, demonstrate its feasibility forimplementation in primary health care (Paper I-III).

9. After the CBT-TEB programme patients reported beneficial changes in all studied eating behaviours (EE, UE, and CR). This was expected as the programme to a great extent targeted eating behaviour. Reductions in EE and UE after therapy probably led to decreased abdominal obesity measured as WC, a potentially highly salutogenic outcome (Paper III).

10. After CBT-TEB patients reported an increased obesity-related quality of life. The weight loss during therapy makes this plausible. A reduction of abdominal obesity measured as WC was found to be particularly related to diminished obesity-related problems (Paper III).

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The ambition to change
The ability to implement
The courage to dare

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9 REFERENCES

1. WHO. Obesity: preventing and managing the global epidemic. Report of a WHO consultation on obesity. Geneva: World Health Organisation; 1998.

2. WHO. Obesity and overweight. Media centre; 2016.

3. Marie Ng et al. Global, regional and national prevalence of overweight and obesity in children and adults 1980-2013: A systematic analysis. Lancet. 2014 Aug 30;384(9945):766–781.

4. Ma GS, Li YP, Wu YF, Zhai FY, Cui ZH, Hu XQ, Luan DC, Hu YH, Yang XG. The prevalence of body overweight and obesity and its changes among Chinese people during 1992 to 200. Zhonghua Yu Fang Yi Xue Za Zhi. 2005 Sep;39(5):311-5.

5. Statistiska centralbyrån (SCB). Persson L. Var tionde svensk är fet. Välfärd 2007;(1):8-9. Örebro.

6. Sundquist K, Qvist J, Johansson SE, Sundquist J. Increasing trends of obesity in Sweden between 1996/97 and 2000/01. Int J Obes Relat Metab Disord. 2004;28(2):254-61.

7. Johansson G. Overweight and obesity in Sweden. A five year follow-up, 2004–2008. Scand J Public Health. 2010;38(8):803-9.

8. Gorber SC, Tremblay M, Moher D, Gorber B. A comparison of direct vs. self-report measures for assessing height, weight and body mass index: a systematic review. Obes Rev. 2007;8(4):307-26.

9. Berg C, Rosengren A, Aires N, Lappas G, Toren K, Thelle D, et al. Trends in overweight and obesity from 1985 to 2002 in Goteborg, West Sweden. Int J Obes Relat Metab Disord. 2005;29(8):916-24.

10. Lilja M, Eliasson M, Stegmayr B, Olsson T, Soderberg S. Trends in obesity and its distribution: data from the Northern Sweden MONICA Survey, 1986-2004. Obesity (Silver Spring). 2008 May;16(5):1120-8.

11. Statistiska centralbyrån (SCB). Undersökningarna av levnadsförhållanden (ULF/SILC). Hälsa – fler indikatorer 2014-2015.

12. Garrow JS. Treatment of morbid obesity by nonsurgical means: diet, drugs, behavior modification, exercise. Gastroenterol Clin North Am. 1987 Sep;16(3):443-9.

13. WHO. Physical status: The use and interpretation of anthropometry. WHO Technical Report Series 854: Geneva;1995.

14. SBU-The Swedish council on technology assesment in health care. Fetma - problem och åtgärder: en systematisk litteraturöversikt. Rapport nr 160. Elanders Graphc systems: Göteborg, 2002. (In Swedish).

15. Björntorp P. Abdominal fat distribution and disease: an overview of epidemiological data. Ann Med. 1992 Feb;24(1):15-8.

16. Hauner H1, Bramlage P, Lösch C, Steinhagen-Thiessen E, Schunkert H, Wasem J, Jöckel KH, Moebus S. Prevalence of obesity in primary care using different anthropometric measures-results of the German Metabolic and Cardiovascular Risk Project (GEMCAS). BMC Public Health. 2008 Aug 11;8:282.

17. Jee SH, Sull JW, Park J, Lee SY, Ohrr H, Guallar E, Samet JM. Body-mass index and mortality in Korean men and women. N Engl J Med. 2006 Aug 24;355(8):779-87. Epub 2006 Aug 22.

18. Balkau B, Deanfield JE, Després JP, Bassand JP, Fox KA, Smith SC Jr, Barter P, Tan CE, Van Gaal L, Wittchen HU, Massien C, Haffner SM. International Day for the Evaluation of Abdominal Obesity (IDEA): a study of waist circumference, cardiovascular disease, and diabetes mellitus in 168,000 primary care patients in 63 countries. Circulation. 2007 Oct 23;116(17):1942-51.

19. Lakka HM, Lakka TA, Tuomilehto J, Salonen JT. Abdominal obesity is associated with increased risk of acute coronary events in men. Eur Heart J 2002;23:706-13.

20. Lindqvist P, Andersson K, Sundh V, Lissner L, Björkelund C, Bengtsson C. Concurrent and separate effects of body mass index and waist-to-hip ratio on 24-year mortality in the Population Study of Women in Gothenburg: evidence of age-dependency. Eur J Epidemiol 2006;21:789-94.

21. Taylor RW1, Keil D, Gold EJ, Williams SM, Goulding A. Body mass index, waist girth, and waist-to-hip ratio as indexes of total and regional adiposity in women: evaluation using receiver operating characteristic curves. Am J Clin Nutr. 1998 Jan;67(1):44-9.

22. Han et al, 1997. Waist circumference and Waist-hip ratio. Report of a WHO Expert Consultation, World Health Organization. Geneva 8-11 december. 2008.

23. Waist circumference and Waist-hip ratio. Report of a WHO Expert Consultation, World Health Organization. Geneva 8-11 december. 2008.

24. Iribarren C, Darbinian JA, Lo JC, Fireman BH, Go AS. (2006). “Value of the Sagittal Abdominal Diameter in Coronary Heart Disease Risk Assessment: Cohort Study in a Large, Multiethnic Population”. American J of Epidemiology 2006;164 (12):1150-1159.

25. Stunkard AJ, Pederson NL, McClearn GE. The body-mass index of twins who have been reared apart. N Engl J Med 1990;322(21):1483–7.

26. Rissanen A, Hakala P, Lissner L, Mattlar CE, Koskenvuo M, Rönnemaa T. Acquired preference especially for dietary fat and obesity: A study of weight-discordant monozygotic twin-pairs. Int J Obes Relat Metab Disord. 2002 Jul;26(7):973-7.

27. Keskitalo K, Tuorila H, Spector TD, Cherkas LF, Knaapila A, SKaprio J, Silvertoinen K, Perola. The Three-Factor Eating Questionnaire, body mass index, and responses to sweet and salty fatty foods: a twin study of genetic and environmental associations. Am J Clin Nutr. 2008 Aug;88(2):263-71.

28. Frayling TM et al. A common variant in the FTO gene is associated with Body Mass Index and predisposes to childhood and adult obesity. Science. 2007 May 11;316(5826):889-94. Epub 2007 Apr 12.

29. Sonestedt E, Roos C, Gullberg B, Ericson U, Wirfält E, Orho-Melander M. Fat and carbohydrate intake modify the association between genetic variation in the FTO genotype and obesity. Am J Clin Nutr. 2009 Nov;90(5):1418-25.

30. Shungin D et al. New genetic loci link adipose and insulin biology to body fat distribution. Nature 518. 2015 Febr 187-196.

31. Seidell J C. Obesity in Europé: scaling an epidemic. Int J Obes Relat Metab Disord 1995 Sep;19 Suppl: S1–4.

32. Lapidus L, C Bengtsson C, Hällström T, Björntorp P. Obesity, adipose tissue distribution and health in women-results from a population study in Gothenburg, Sweden. Appetite.1989;13(1):25–35.

33. Norberg M, Lindvall K, Stenlund H, Lindahl B. The obesity epidemic slows among the middle-aged population in Sweden while the socioeconomic gap widens. Glob Health Action 2010 Dec 10;3.

34. Sundquist K, Qvist J, Johansson SE, Sundquist J. Increasing trends of obesity in Sweden between 1996/97 and 2000/01. Int J Obes Relat Metab Disord 2004;28 (2):254-61.

35. Nyholm M, Gullberg B, Haglund B, Rastam L, Lindblad U. Higher education and more physical activity limit the development of obesity in a Swedish rural population. The Skaraborg Project. Int J Obes (Lond) 2008;32(3):533-40.

36. Kark M, Rasmussen F. Growing social inequalities in the occurrence of overweight and obesity among young men in Sweden. Scand J Public Health. 2005;33(6):472-7.

37. Rössner S, Ohlin A. Pregnancy as a risk factor for obesity: lessons from the Stockholm Pregnancy and Weight Development Study. Obes Res 1995;3 Supp l2:675-758.

38. Rössner S. Pregnancy, weight cycling and weight gain in obesity. Int J Obes Relat Metab Disord 1992;16:145-7.

39. Mullins A. Overweight in pregnancy. Lancet.1960 Jan 16;1(7117):146-7.

40- Linné Y, Rössner S. Interrelationships between weight development and weight retention in subsequent pregnancies: the SPAWN study. Acta Obstet Gynecol Scand. 2003 Apr;82(4):318-25.

41. Domecq JP et al. Drugs commonly associated with weight change: a systematic review and meta-analysis. J Clin Endocrinol Metab. 2015 Feb;100(2):363-70. Epub 2015 Jan 15.

42. Szabo S, Tache Y, Somogyi A.The legacy of Hans Selye and the origins of stress research: a retrospective 75 years after his landmark brief "letter" to the editor# of nature. Stress. 2012 Sep;15(5):472-8.

43. Frankenhaeuser M. The psychophysiology of workload, stress and health: comparison between the sexes. Ann of Behav Med.1991;4:197-204. Frankenhauser & Ödman, 1997, Brombergs förlag AB, Stockholm.

44. Karasek R, Theorell T. Healthy work: Stress, productivity, and the Reconstruction of working life. New York: Basic books; 1990.

45. Travis DJ, Lizano EL, Mor Barak ME. 'I'm So Stressed!': A Longitudinal Model of Stress, Burnout and Engagement among Social Workers in Child Welfare Settings. Br J Soc Work. 2016 Jun;46(4):1076-1095. Epub 2015 Mar 4.

46. Statistiska centralbyrån (SCB). Theorell T. Utmattning och samhällsförändringar. Välfärd 2003;(3):8-10. Örebro.

47. van Strien T, Roelofs K, de Weerth C. Cortisol reactivity and distress-induced emotional eating. Psychoneuroendocrinology. 2013 May;38(5):677-84. Epub 2012 Sep 19.

48. Troop NA,Holbrey A,Treasure JL. Stress, coping and crisis support in eating disorder. Int J Eat Disord. 1998 Sep;24(2):157-66.

49. Block JP, He Y, Zaslavsky AM, Ding L, Ayanian JZ. Psychosocial stress and change in weight among US adults. Am J Epidemiol. 2009 Jul 15;170(2):181-92. Epub 2009 May 2.

50. Lallukka T, Laaksonen M, Martikainen P, Sarlio-Lähteenkorva S, Lahelma E. Psychosocial working conditions and weight gain among employees. Int J Obes (Lond). 2005 Aug;29(8):909-15.

51. Epel E, Lapidus R. McEven B, Browell K. Stress may add a bite to appetite in women: a laboratory study of stress-induced cortisol and eating behavior. Psychoneuroendocrinology 2001 Jan;26(1):37-49.

52. Overgaard D, Gyntelberg F, Heitmann BL. Psychlogical workload and body weight: is there an association? A review of the literature. Occup Med (Lond). 2004 Jan; 54(1):35-41.

53. Block JP, He Y, Zaslavsky AM, Ding L, Ayanian JZ. Psychosocial stress and change in weight among US adults. Am J Epidemiol. 2009 Jul 15;170(2);181-92. Epub 2009 May 22.

54. Rosengren A et al. Psychosocial factors and obesity in 17 high-,middle- and low-income countries: the Prospective Urban Rural Epidemiologic study. Int J Obes (Lond). 2015 Aug;39(8):1217-23.

55. Björntorp P. The regulation of adipose tissue distribution in humans. Int J Obes Rlat Metab Disord 1996;20:291-302.

56. Björntorp P, Rössner S, Uddén J. Kortisolstegringen vid stress ger leptinresistent fetma. Läkartidningen 2001;98:5458-61.

57. Erlandson-Albertsson C, Wingren A. Belöning och beroende.Västerås: ICA bokförlag; 2008.

58. Erlanson-Albertsson A 2006. Mat för hjärnan.Västerås: ICA bokförlag; 2006.

59. Pecoraro N, Reyes F, Gomez F, Bhargava A, Dallman MF. Chronic stress promotes palatable feeding, which reduces signs of stress: feedforward and feedback effects of chronic stress. Endocrinology. 2004 Aug;145(8):3754-62. Epub 2004 May 13.

60. Dallman MF, Pecoraro NC, la Fleur SE. Chronic stress and comfort foods: self-medication and abdominal obesity. Brain Behav Immun. 2005 Jul;19(4):275-80.

61. Leon GR, Roth L. Obesity: psychological causes, correlations, and speculations. Psychol Bull.1977; 84:117–39.

62. Rodin J, Schank D, Striegel-Moore R. Psychological features of obesity. Med Clin North Am. 1989 Jan;73(1):47-66.

63. Stunkard AJ, Wadden TA. Psychological aspects of severe obesity. Am J Clin Nutr. 1992 Feb;55(2 Suppl):524-32.

64. Wadden TA, Stunkard AJ. Social and psychological consequences of obesity. Ann Intern Med. 1985 Dec;103(6(Pt 2)):1062-7.

65. Hällström T, Noppa H. Obesity in women in relation to mental illnessm social factors and personality traits. J Psychosom Res. 1981;25(2):75-82.

66. Noppa H, Hällström T. Weight gain in adulthood in relation to socioeconomic factors, mental illness and personality traits: a prospective study of middle-aged women. J Psychosom Res.1981;25(2):83-9.

67. Diagnostic and Statistical Manual of Mental Disorders (DSM-IV). Fourth Edition. American Psychiatric Association. Washington D.C.1994.

68. Götestam K G, Agras WS General population-based epedemiological study of eating disorders in Norway. Int J Eat Disord.1995 Sep;18(2):119-126.

69. Kinzl JF, Traweger C, Trefalt E, Mangweth B, Biebl W. Binge eating in females: a popolation.based investigation. Int J Eat Disord. 1999 Apr;25(3):287-92

70. Wilfley DE, Schwartz, MB, Spurell E,B Fairburn CG. Using the eating disorder examination to identify the specific psychopathology of binge eating disorder. Int J Eat Disord. 2000 Apr;27(3)259-69.

71. Stunkard AJ. Eating patterns and obesity. Psychiatr Q. 1959 Apr;33:284-95.

72. De Zwaan M. Binge eating disorder and obesity. Int J Obes Relat Metab Disord. 2001 May;25 Suppl 1:51-5.

73. Spitzer RL1, Yanovski S, Wadden T, Wing R, Marcus MD, Stunkard A, Devlin M, Mitchell J, Hasin D, Horne RL. Binge eating disorder: its firther validation in a multisite study. Int J Eat Disord. 1993 Mar;13(2):137-53.

74. Bulik CM, Sullivan PF, Kendler KS. Medical and psychiatric morbidity in obese women with and without binge eating. Int J Eat Disord. 2002 Jul;32(1):72-8.

75. Nicholls W, Devonport TJ, Blake M. The association between emotions and eating behaviour in an obese population with binge eating disorder. Obes Rev. 2016 Jan;17(1):30-42. Epub 2015 Dec 8.

76. Chambless DL, Ollendick TH. Empirically supported psychological interventions controversies and evidendence. Annu Rev Psychol. 2001;52:685-716.

77. Wilson GF, Fairburn CG. Eating disorders. Treatments that work. New York: NY Oxford University Press; 2001.

78 Agras WS, Telch CF, Arnow B, Eldredge K, Wilfley, DE. Reaburn SD, Henderson, J, Marnell M. Weight loss, cognitive–behavioral, and desipramine treatments in binge eating disorder. An additive design. Behav Ther. 1994;25:225-38.

79 de Zwaan M, Mitchell JE, Howell LM, Monson N, Swan-Kremeier L, Roerig JL, Kolotkin RL, Crosby RD. Two measures of health-related quality of life in morbid obesity. Obes Res. 2002 Nov;10(11)1143-51.

80. Dingemans AE, Bruna MJ, van Furth EF. Binge eating disorder: a review. Int J Obes Relat Metab Disord.2002 Mar;26(3):299-307.

81. Blackburn GL,Wilson GT, Kanders BS, Stein LJ, Lavin PT, Adler J, Brownell KD. Weight cycling: The experience of human dieters. Am J Clin Nutr. 1989 May;49 (5 Suppl):1105-9.

82.. Brownell KD, Rodin J. Medical, metabolic and psychological effects on weight cycling. Arch Intern Med. 1994 Jun 27;154(12):1325-30.

83. Montani JP,Viecelli AK, Prévot A, Dulloo AG. Weight cycling during growth and beyond as a risk factor for later cardiovascular diseases: the 'repeated overshoot' theory. Int J Obes(Lond). 2006 Dec;30 Suppl 4:58–66.

84. Rössner S. Long-term intervention strategies in obesity treatment. Int J Obes Relat Metab Disord. 1995 Dec;19 Suppl 7:29-33.

85. Alley DE, Chang VW. The changing relationship of obesity and disability, 1988-2004. JAMA 2007 Nov;298(17):2020–7.

86. Klein S, Burke LE, Bray G, Blair S, Allison DB, Pi-Sunyer X, Hong Y, Eckel RH; American Heart Association Council on Nutrition, Physical Activity and Metabolism. Clinical implications of obesity with special focus on cardiovascular disease: a statement for professionals from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism: endorsed by the American College of Cardiology Foundation. Circulation 2004 Nov 2;110(18):2952–67. Wpub 2004 Oct 27.

87. Wing RR, Jeffery RW. Effect of modest weight loss on changes in cardiovascular risk factors: are there difference between men and women or between weight loss and maintenance? Int J Obes Relat Metad Disord. 1995 Jan;19(1):67-73.

88. Institute of Medicine. Weighing the Options: Criteria for Evaluating Weight-managements Programs.Washington, DC, National Academy Press, 1995.

89. Royal College of Physicians Clinical Management of Overweight and Obese Patients. London, The Royal College of Physicians of London, 1998.

90. Cooper Z, Fairburn CG. A new cognitive behavioural approach to the treatment of obesity. Behav Res Ther. 2001.May;39(5):499-511. Review.

91. Sjöström, LV. Morbidity of severely obese subjects. Am J Clin Nutr 1992 Feb;55(2):508-515.

92. Carmelli D, Zhang H, Swan GE.Obesity and 33 year follow-up for coronary heart disease and cancer mortality. Epidemiology.1997 jul;8(4):378-83.

93. Rissanen A, Knekt P, Heliövaara M, Aromaa A, Reunanen A, Maatela J. Weight and mortality in Finnish women. J Clin Epidemiol. 1991;44(8):787-95.

94. Wing RR, Jeffery RW. Effect of modest weight loss on changes in c ardiovascular risk factors: Are there difference between men and women

or between weight loss and maintenance? Int J Obes Relat Disord.1995 Jan;19(1):67-73.

95. Grabowski, DC, Ellis, JE, High body mass index does not predict mortality in older people:analysis of te Longitudinal Study of Aging. J Am Geriatr Soc. 2001 Jul;49(7):968-79.

96. Nordström P, Pedersen NL, Gustafson Y, Michaëlsson K, Nordström A. Risks of Myocardial Infarction, Death, and Diabetes in Idential Twin Pairs with Different Body mass Indexes. JAMA Intern Med. 2016 Aug 1. [Epub ahead of print].

97. Chan JM, Rimm EB, Colditz GA, Stampfer MJ, Willett WC. Obesity, fat distribution, and weight gain as risk factors for clinical diabetes in men. Diabetes Care. 1994 Sep;17(9):961-9.

98. Colditz GA, Willett WC, Rotnitzky A, Manson JE. Weight gain as a risk factor for clinical diabetes mellitus in women. Ann Intern Med. 1995 Apr 1;122(7):481-486.

99. Björntorp P. Visceral Obesity:A "Civilization Syndrome". Obes Res. 1993 May;1(3):206-22.

100. Blair SN, Brodney S. Effects of physical inactivity and obesity on morbidity: current evidence and reserch issues. Med Sci Sports Exerc. 1999 Nov;31(11 Suppl):646-62.

101. Fagerberg B. Metabola syndromet – dags att införa diagnosen i rutinsjukvården? Läkartidningen, 2004;101(48):3902-3911.

102. Wolk A, Gridley G, Svensson M, Nyrén O, McLaughlin, JK. Fraumeni JF, Adam HO. A prospective study of obesity and cancer risk (Sweden). Cancer Causes Control. January 2001 Jan;12(1):13-21.

103. O'Keeffe T, Patterson E. Evidence supporting routine polysomnography before bariatric surgery. Obes Surg. 2004;14(1):23-26.

104. Mohsenin V. Obstructive sleep apnea: a new preventive and therapeutic target for stroke: a new kid on the block. Am J Med. 2015 Aug;128(8):811. Epub 2015 Feb 27.

105. "Sleep Apnea: What Is Sleep Apnea?" NHLBI: Health Information for the Public. U.S. Department of Health and Human Services. May 2009. Retrieved 2010-08-05.

106. Sjöström LV. Mortality of severely obese subjects. Am J Clin Nutr 1992 Feb; 55(2 Suppl):516-23.

107. Karlsson J, Taft C, Sjöström L, Torgerson JS, Sullivan M. Psychosocial f unctioning in the obese before and after weight reduction: construct validity

and responsiveness of the Obesity-related Problems scale. Int J Obes Relat Metab Disord. 2003 May;27(5):617-30.

108. Sullivan M et al.Swedish obese subjects (SOS)–an intervention study of obesity. Baseline evaluation of health and psychosocial functioning in the first 1743 subjects ewamined. Int J Obes Relat Metad Disord.1993 Sep;17(9):503-512.

109. Lissner L, Johansson SE, Qvist J, Rössner S, Wolk A, Social mapping of the obesity epidemic in Sweden. Int J Obes Relat Metab Disord. 2000 Jun;24(6):801-5.

110. Golobardes B, Morabia A, Bernstein MS. The differential effect of education and occupation on body mass and overweight in a sample of working people of the general population. Ann Epidemiol. 2000 Nov;10(8):532-7.

111. Stunkard AJ, Wadden TA. Psychological aspects of severe obesity. Am J Clin Nutr. 1992 Feb;55(2 Suppl):524-532. Review.

112. Byrne S, Cooper Z, Fairburn C. Weight maintenance and relapse in obesity: a qualitative study. Int J Obes Relat Metab Disord. 2003 Aug;27(8):955-62.

113. Bray GA, Popkin BM. Dietary fat intake does affect obesity. Am J Clin Nutr 1998 Dec;68(6):1157-73.

114. Becker W et al. Nordic Nutrition Recommendations. Ugeskr Laeger 2006 Jan 3;168(1):76-7; author reply 77.

115. Bell EA, Castellanos VH, Pelkman CL, Thorwart ML, Rolls BJ. Energy density of foods affects energy intake in normal-weight women. Am J Clin Nutr 1998 Mar;67:412–20.

116. Curioni CC, Lourenco PM. Longterm weight loss after diet and exercise: a systematic review. Int J Obes (Lond) 2005 Oct;29(10):1168-174.

117. Svendsen OL, Hassager C, Christansen C. Effect of an energy-restrictive diet, with or without exercise, on lean tissue mass, resting metabolic rate, cardiovascular risk factors, and bone in overweight postmenopausal women. Am J Med. 1993 Aug;95(2):131-40.

118. Kirk EP, Jacobsen DJ, Gibson C, Hill JO, Donnelly JE. Time course for changes in aerobic capacity and body composition in overweight men and women on response to long-term exercise: the Midwest exercise trial. Int J Obes Relat Metad Disord 2003 Aug;27(8):912-9.

119. Tate DF, Wing RR. Endotext [Internet]. Behavior Modification for Obesity. South Dartmouth (MA): MDText.com, Inc.; 2000-2002 Mar 1.

120. Wadden, TA, Foster GD, Letizia KA. (1994). One-year behavioral treatment of obesity: comparison of moderate and severe caloric restriction and the effcts

of weight maintenance therapy. J Consult Clin Psychol. 1994 Febr;62(1):165-71.

121. Wadden TA, Barlett SJ. Very low calorie diets. An overview and appraisal. In T.A. Wadden and T.B VanIttalie (eds) Treatment of seriously obese patient. New York: The Guilford Press;1992.

122. Sjöström L, Rissanen A, Andersen T, Boldrin M, Golay A, Koppeschaar HP, Krempf M. Randomised placebo-controlled trial of orlistat for weight loss and prevention of weight regain in obese patients. European Multicentre Orlistat Study Group Lancet. 1998 Jul 18;352(9123):167-72.

123. Davidson MH, Hauptmab J, DiGirolamo M, Foreyt JP, Halsted CH, Heber D, Heimburger DC, Lucas CP, Robbins DC, Chung J, Heymsfield SB. Weight control and risk factor reduction in obese subjects treated for 2 years with Orlistat: a randomized controlled trial. JAMA 199 Jan 20;281(3):235-42.

124. Norris SL, Zhang X, Avenell A, Gregg E, Schmid CH, Lau J. Pharmacotherapy for weight loss in adults with type 2 diabetes mellitus. Cochrane Database Syst Rev. 2005 Jan 25;(1):CD004096.

125. Torgerson JS, Hauptman J, Boldrin MN, Sjöström L. XENical in the prevention of diabetes in obese subjects (XENDOS) study: a randomized study of orlistat as an adjunct to lifestyle changes for the prevention of type 2 diabetes in obese patients. Diabetes Care. 2004 Jan;27(1):155-61.

126. van Baak MA van Mil E, Astrup AV, Finer N, Van Gaal LF, Hilsted J, Kopelman PG, Rössner S, James WP, Saris WH; STORM Study Group.et al. Leisure-time activity is an important determinant of long-term weight Maintenance. Am J Clin Nutr. 2003. Aug;78(2):209-14.

127 James WP, Astrup A, Finer N, Hillsted J, Kopelman P, Rössner S, Saris Wh, Van Gaal LF. Effect of sibutramine on weight maintenance after weight loss: a randomised trial. STORM Study Group. Sibutramine Trial of Obesity Reduction and Maintenance. Lancet. 2000 Dec 23. 30;356(9248):2119-25.

128. Ramström H. Övervikt och fetma. Läkemedelsboken. 19:e upplagan. Uppsala: Läkemedelsverket: 201-219; 2014.

129. Sjöström L et al; Swedish Obese Subjects Study. Effects of bariatric surgery on mortality in Swedish obese subjects. N Engl J Med. 2007 Aug 23;357(8):741-52.

130. Sjöström L, Lindroos AK, Peltonen M, Torgerson J, Bouchard C, Carlsson B, Dahlgren,S, Larsson B, Narbro K, Sjöström CD, Sullivan M, Wedel H; Swedish Obese Subjects Study Scientific Group. Lifestyle, diabetes, and cardiovascular risk factors 10 years after bariatric surgery. N Engl J Med. 2004 Dec 23;351(26):2683-93.

131 Sjöström L. Review of the key results from the Swedish Obese Subjects (SOS) trial – a prospective controlled intervention study of bariatric surgery. J Intern Med. 2013 Mar;273(3):219-34. Epub 2013 Feb 8.

132. Hsu LK, Sullivan SP, Benotti PN. Eating disturbances and outcome of gastric bypass surgery: a pilot study. Int J Eat Disord. 1997 May; 21(4):385-90.

133. Koenders, PG, van Strien T. Emotional eating, rather than lifestyle behavior, drives weight gain in a prospective study in 1562 employees. J Occup Environ Med. 2011 Nov;53(11):1287-93.

134. Colles A, Dixon J, O´Brien P. Grazing and loss of control related to eating: two high-risk factors following bariatruc surgery. Obesity. 2008 Mar;16(3);615-22. 2008 Jan 17.

135. Dodsworth A, Warren-Forward H, Baines S. Changes in eating behavior after laparascopic adjusttable gastric banding: a systemic review of the literature. Obes Surg. 2010 Nov;20(11):1579-93.

136. Papp, Czegledi E, Udvardy-Meszaros, Vizin G, Percel Forintos D. Outcome of a one-year behavior Therapy weight loss program. Orv Hetil. 2014 jul 27;155(30):1196-202.

137. Stuart RB. Behavioral control of overeating. Behav Res Ther.1967;5:357-65.

138. Wing R. Behavioral approaches to the treatment of obesity. In: Bray GA, Bouchard C, James WPT, editors. Handbook of obesity. New York: M. Dekker; 1998. p. 855–73.

139. Williamson DA, Perrin LA. Behavioral therapy for obesity. Endocrinol Metab Clin North Am. 1996 Dec;25(4):943-54.

140. Stalonas PM, Perri MG, Kerzner AB. Do behavioral treatments of obesity last? A five–year follow–up investigation. Addict Behav.1984;9(2):175-183.

141. Liao KL.Cognitive-behavioural approaches and weight management: an overview. JR Soc Prom Health. 2000 Mar;120(1)27-30.

142. Glenny AM, O´Meara S, Melville A, Sheldon TA, Wilson C. The treatment and prevention of obesity: a systemic review of the literature. Int J Obes Relat Disord.1997 Sep; 21(9):715-37.

143. Sbrocco T1, Nedegaard RC, Stone JM, Lewis EL. Behavioral choice treatment promotes continuing weight loss: preliminary results of a cognitive-behavioral decision-based treatment for obesity. J Consult Clin Psychol. 1999 Apr;67(2):260-6.

144. Spahn JM, Reeves RS, Keim KS, Laquatra I, Kellogg M, Jortberg B, Clark NA. State of the evidence regarding behavior change theories and strategies in nutrition counseling to facilitate health and food behavior change. J Am Diet Assoc. 2010 Jun;110(6):879-91.

145. Van Dorsten B, Lindley EM. Cognitive and behavioral approaches in the t reatment of obesity. Med Clin North Am. 2011 Sep;95(5):971-88. Review.

146. L. Rapaport, M. Clark, J. Wardle. Evaluation of a modified cognitive behavioural programme for weight management. Int J Obes Relat Metab Disord. 2000 Dec;24(12):1726-37.

147. Shaw K, O'Rourke P, Del Mar C, Kenardy J Psychological interventions for overweight or obesity. Cochrane Database of Systematic Reviews 2005, I ssue 2. Art. NO.: (2005) CD003818.

148. Painot D, Jotterand S, Kammer A, Fossati M, Golay A. Simultaneous nutritional cognitive behavioural therapy in obese patients. Patient Educ Couns. 2001 Jan;42(1):47 52.

149. Shaw K1, O'Rourke P, Del Mar C, Kenardy J. Psychological interventions for overweight or obesity. Cochrane Database Syst Rev. 2005 Apr 18;(2):CD003818.

150. Eichler K, Zoller M, Steurer J, Bachmann LM. Cognitive-behavioural treatment for weight loss in primary care: a prospective study. Swiss Med Wkly. 2007 Sep 8;137(35 -36):489-95.

151. Fabricatore AN. Behavior therapy and cognitive-behavioral therapy is there a different? J Am Diet Assoc. 2007 Jan;107(1): 92-9. Review.

152. Paulsen BK, Lutz RN. McReynolds WT, Kohrs MB. Behavior therapy for weight control: long-term results of two programs with nutritionists as therapists. Am J Clin Nutr.1976 August;29(8):880-8.

153. Kaplan HI, Kaplan HS. The psychosomatic concept of obesity. J Nerv Ment Dis. 1957 Apr-Jun;125(2):181-201.

154. Schachter S, Rodin J. Obese humans and rats. Potomac, Md.,: L. Erlbaum Associates; distributed by the Halsted Press Division, Wiley, New York; 1974. ix, 182 p. p.

155. Herman, CP, Mack D. Restrained and unrestrained eating. J Pers 1975;43(4): 647-60.

156. Herman CP, Polivy J. Anxiety, restraint and eating behavior. J Abnorm Psychol 1975 Dec;84(6): 66-72.

157. Van Strien T, Frijters J, Roosen RG. Knuiman-Hijl WJ, Defares PB. Eating behaviour, personality traits and body mass in women. Addict Behav 1985 10(4):333-43.

158. Hamburger WW. Psychological aspects of obesity. Bull NY Acad Med, 195 Nov;33(11):771-82

159. Van Strien T, Frijters JER, Bergers GPA, Defares PB. The Dutch Eating Behavior Questionaire (DEBO) for assessment of restrained, emotional and external eating behavior. Int J Eat Disord 1986 5(2):295-315.

160. Stunkard AJ, Messick S. The three-factor eating questionnair measure dietary restraint, disinhibition and hunger. J Psychosom Res 1985;29:71-83.

161. Karlsson J, Persson LO, Sjöström L and Sullivan M. Psychometric properties and factor structure of the Three-Factor Eating Questionnaire (TFEQ) in obese men and women. Results from the Swedish Obese Subjects (SOS) study. Int J Obes Relat Metab Disord 2000 Dec;24(12):1715-25.

162. Koenders PG, van Strien, T. Emotional eating, rather than lifestyle behavior, drives weight gain in a prospective study in 1562 employees. J Occup Environ Med. 2011 Nov;53(11): 1287-93.

163. de Lauzon B, et al. The Three-Factor Eating Questionnaire-R18 is able to distinguish among different eating patterns in a general population. J Nutr. 2004 Sep;134(9)2372-80.

164. Spoor ST, Bekker MH, Van Strien T, van Heck GL. Relations between negative affect, coping, and emotional eating. Appetite. 2007 May;48(3):368-76. Epub 2006 Dec 4.

165. Ganley. M. Emotion and eating in obesity: A review of the literature. Int J Eat Disord. 1989 May;8(3):343-61.

166. Byrne S, Cooper Z Fairburn C. Weight maintenance and relapse in obesity: a qualitative study. Int L Obes Relat Metab Disord. 2003 Aug;27(8):955-62.

167. Rand CS. Psychodynamics of obesity. J Am Acad Psychoanal. 1978 J an;6(1):103-15.

168. Leon G. Personality and behavioral correlates of obesity. In: Wolman BB, DeBerry S, editors. Psychological aspects of obesity: a handbook. New York: Van Nostrand Reinhold; 1982. p. ix, 318 p.

169. Spitzer L, Rodin J. Human eating behavior: A critical review of studies in normal weight and overweight individuals. Appetite, 1981;2:293-329.

170. Buchanan JR. Five year psychoanalytic study of obesity. Am J Psychoanal. 1973;33(1):30-41.

171. Edelman, B. Binge eating in normal weight and overweight individuals. Psychol Rep.1981 Dec;49(3):739-46.

172. Craddock D. Obesity and its management. 3d ed. Edinburgh; New York: Churchill Livingstone; 1978. xi, 194 p. p.

173. Ganley RM. (1986). Epistemology, family patterns, and psychosomatics: the case of obesity. Fam Process. 1986 Sept;25(3):, 437-51.

174. Ganley RM. Family patterns in obesity: With consideration of emotional eating and restraint. Fam Syst Med. 1992;10(2):181-199.

175. Rand CS, Stunkard AJ. Psychoanalysis and obesity. J Am Academ Psychoanal. 1997 Oct;5: 459-97.

176. Bruch, H. Eating disorders: Obesity, anorexia nervosa and the person within. New York: Basic Books; 1973.

177. van Strien T1, Cebolla A, Etchemendy E, Gutiérrez-Maldonado J, Ferrer-García M, Botella C, Baños RC,. Emotional eating and food intake after sadness and joy. Appetite. 2013 Jul;66:20-5.

178. Elfhag K, Carlsson AM, Rössner S. Subgrouping in obesity based on Rorschach personality characteristics. Scand J Psychol. 2003 Dec;44(5):399-407.

179. Heatherton TF, Baumeister RF. Binge eating as escape from self-awareness, Psychol Bull. 1991 Jul;110(1):86-108.

180. Spoor ST, Bekker MH, Van Strien T, van Heck GL. Relations between negative affect, coping, and emotional eating. Appetite. 2007 May;48(3):368-76. Epub 2006 Dec 4.

181. Bennett J, Greene G, Schwartz-Barcott D. Perceptions of emotional eating behavior. A qualitative study of college students. Appetite. 2013 Jan;60(1):187-92.Epub 2012 Oct 6.

182. Torres SJ, Nowson CA. Relationship between stress, eating behavior, and obesity. Nutrition. 2007 Nov-Dec;23(11-12): 887–94. Epub 2007 Sep 17.

183. Levine AS, Kotz CM, Gosnell BA. Sugars: hedonic aspects, neuroregulation, and energy balance. Am J Clin Nutr. 2003 Oct;78(4):834-842.

184. Chao A, Grilo CM, White MA, Sinha R. Food cravings, food intake, and weight statu in a Community-based sample. Eat behav. 2014 Aug;15(3):478–82.

185. Ng L, Davis C. . Cravings and food consumption in Binge Eating Disorder. Eat Behav. 2013 Dec;14(4):472-5. Epub 2013 Aug.

186. Hill AJ, Weaver CF, Blundell JE. Food craving, dietary restraint and mood. Appetite. 1991 Dec; 17(3):187-97.

187. Pelletier JE, Lytle LA, Laska MN. Stress, Health Risk Behaviors, and Weight Status Among Community College Students. Health Educ Behav. 2016 Apr;43(2):139-44. Epub 2015 Aug 13.

188.. Wardle J, Chida, Gibson EL, Whitaker KL, Steptoe A. Stress and adiposity; a meta analysis of longitudinal studies. Obesity (Silver Spring) 2011 Apr;19(4):771-8. Epub 2010 Oct 14.

189. Everson-Rose SA, Lewis TT. Psychosocial factors and cardiovascular diseases. Annu Rev Public Health. 2005;26:469–500.

190. Schulz AJ, House JS, Israel BA, Mentz G, Dvonch JT, Miranda PY, Kannan S, Koch M. Relational pathways between socioeconomic position and carediovascular risk in a multiethnic urban sample: complexities and their implications for improving health in economically disadvantaged populations. J Epidemiol Community Health. 2008;62:638- 46.

191. Boggiano MM, Wenger LE, Burgess EE, Tatum MM, Sylvester MD, Morgan PR, Morse KE. Eating tast foods to cope, enhance reward, socialize or c onform: What other psychological characteristics describe each of these motivs? L Health Psychol. 2015 Aug 26. (Epub ahead of print).

192. Harding JL, Backholer K, Williams ED, Peeters A, Cameron AJ, Hare MJ, Shaw JE, Magliano DJ. Psychosocial stress is positively associated with body mass index gain over 5 years: evidence from the longitudinal AusDiab study. Obesity (Silver Spring). 2014 Jan;22(1):277-86.

193. Spence S, Courbasson C. (2012). The role of emotional dysregulation in concurrent eating disorders and substance use disorders. Eat Behav 2012 dec;13(4):382-5. Epub 2012 Jun 7.

194. Calvo D, Galioto R, Gunstad J, Spitznagel MB. Uncontrolled eating is associated with reduced executive functioning. Clin Obes. 2014 Jun;4(3):172-9. Epub 2014 May 7.

195. Blandine de Lauzon et al. The Three-Factor Questionnaire–R18 Is Able to Distinguish among Different Eating Patterns in a General Population. J Nutr. 2004.Sept;134(9):2372-80.

196. van´t Riet J, Sijtsema SJ, Dagevos H, De Bruijn GJ. The importence of habits in eating behaviour. An overview and rekommendations for future research. Appetite. 2011 Dec;57(3):585-96. Epub 2011 Jul 27.

197. Graybiel AM. Habits, rituals, and the evaluative brain. Ann Rev Neurosci. 2008;31:359-87.

198. 198. Fleig L, Lippe S, Pomp S, Schwarzer R. Intervention effects of exercise self-regulation on hysical exercise and eating fruits and vegetables: a longitudal study in orthopedic and cardiac rehabilitation. Epub 2011 Jul 14.

199. Lally P, Gardner B. Promoting habit formation. Health Psychol Rev. 2011 Jul;7(1):137-58.

200. Rideout CA, Barr SI. “Restrained Eating” vs “Trying to Lose Weight”: how are they associated with body weight and tendency to overeat among postmenopausal women? J Am Diet Assoc. 2009 May;109(5):890-3.

201. Mann T, Tomiyama AJ, Westling E, Lew AM, Samuels B, Chatman J. Medicare’s search for effective obesity treatments: Diets are not the answer. Am Psychol. 2007 Apr;62(3): 220-33.

202. Wilson GT. Relation of dieting and voluntary weight loss to psychological functioning and binge eating. Ann Intern Med. 1993 Oct;119(7 Pt2):727-30.

203. Arnow B, Kenardy J, Agras WS. The Emotional Eating Scale: the development of a measure to assess coping with negative affect by eatinng. Int J Eat Disord. 1995 Jul;18(1):79-90.

204. Nisbett RE (1972) Hunger, obesity and the ventromedial hypothalamus. Psychol Rev Nov;79(6): 433-53.

205. Klesges RC, Klem ML, Epkins CC. A longitudinal evaluation of dietary restraint and its relationshipt o change in body weight. Addict Bahav1991;116(5):363-8.

206. Potenza MN, Grilo CM. How Relevant is Food Craving to Obesity and its Treatment? Front Psychiatry. 2014 Nov 19;5:164. E-collect. 2014.

207. Janas-Kozik M1, Zejda J, Stochel M, Brozek G, Janas A, Jelonek I. Orthorexia—a new diagnosis? Psychiatr Pol. 2012 May-Jun;46(3):441-50.

208. Lund LG, Mongomery H, Waern Y. Kognitiv psykologi. Lund. Studentlitteratur:1992.

209. Beck, AT. Cognitive Therapy and The Emotional Disorders. New York. International Univerities Press; 1976.

210. Miller WR, Seligman ME. Depression and learned helplessness in man. J Abnorm Psychol. 1975 Jun;84(3):228-38. PMID: 1169264.

211. Rosellini RA, Seligman ME. Frustation and learned helplessness.J Exp Psychol Anim Behav Process. 1975 Apr;1(2):149-57. PMID: 1141820.

212. Miller WR, Seligman ME, Kurlander HM. Learned helplessness, depression, and anxiety. J Nerv Ment Dis. 1975 Nov;161(5):347-57.

213. Klein DC, Fencil-Morse E, Seligman ME. Learned helplessness, depression, and the attribution of failure. J Pers Soc Psychol. 1976 May;33(5):508-16. PMID: 1271223

214. Beck AT. The current state of cognitive therapy: a 40-year retrspective. Arch Gen Psychiatry 2005 Sep;62(9):953-9.

215. Beck AT, Rush AJ, Shaw BF, Emery G. Cognitive therapy for depression. New York Guilford Press; 1979.

216. Beck AT, Freeman, A. Cognitive Therapy of Personality Disorders. New York: Guilford Press; 1990.

217. Goldstein,MJ. Psychosocial strategies for maximizing the effects of psychotropic medications for schizophrenia and mood disorder. 1992;28(3):237-40.

218. Goldman CR. Toward a definition of psychoeducation. Hosp Community Psychiatry.1988 jun;39(6):666-8

219. Orhagen,T: Working in families in schizophrenic disorders. The practice of psychoeducational intervention. Linköping University Medical Dissertations 363, Linköping;1992.

220. Stice E, Ragan J. A preliminary controlled evaluation of an eating disturbance psychoeducational intervention for college students. Int J Eat Disord. 2002 Mar;31(2):159-71.

221. Blomstrand A, Ariai N, Baar AC, Finbom-Forsgren BM, Thorn J, Björkelund C. Implementation of a low-budget, lifestyle-improvement method in an ordinary primary healthcare setting: a stepwise intervention study. BMJ Open 2012 Aug 8;2(4). Print 2012.

222. Stahre L. Kognitiv behandling vid övervikt och fetma (Cognitive therapy for overweight and obesity). Lund: Studentlitteratur; 2002.

223. Stahre L. Övervikt handlar om känslor (Emotional eating). Lund: Studentlitteratur; 2002.

224. Beck AT. Thinking and depression. II. Theory and Therapy. Arch Gen Psychiatry. 1964 Jun;10:561-71.

225. Seligman ME. Learned helplessness. Annu Rev Med. 1972;23:407-12.

226. Braiker H. The Disease to Please: Curing the People-Pleasing Syndrome. Mc Graw-Hill Education:New York;2002.

227. Nevonen L1, Broberg AG, Clinton D, Norring C. A measure for the assessment of eating disorders: reliability and validity studies of the Rating of Anorexia and Bulimia interview-revised version (RAB-R). Scand J Psychol. 2003 Sep;44(4):303-10.

228. Katz JN, Larson MG, Phillips CB, Fossel AH, Liang MH. Comparative measurement sensitivity of short and longer health status instruments. Med Care 1992 Oct;30(10):917-25.

229. Katzer L, Bradshaw AJ, Horwath CC, Gray AR, O'Brien S, Joyce J. Evaluation of a "nondieting" stress reduction program for overweight women: a randomized trial. Am J Health Promot. 2008 Mar-Apr;22(4):264-74.

230. Agras WS1, Telch CF, Arnow B, Eldredge K, Marnell M. One-year follow-up of cognitive-behavioral therapy for obese individuals with binge eating disorder. J Consult Clin Psychol. 1997 Apr;65(2):343-7.

231. Goldstein DJ. Beneficial health effects of modest weight loss. Int J Obes Relat Metab Disord. 1992 Jun;16(6):397-415. Ed. New York: Guilford Press;2002.

233. Burke LE, Steenkiste A, Music E, Styn MA. A descriptive study of past experiences with weight-loss treatment. J Am Diet Assoc. 2008 Apr;108(4):640-7.

234. Byrne SM. Psychological aspects of weight maintenance and relapse in obesity. J Psychosom Res. 2002 Nov;53(5):1029-36.

235. Elfhag K1, Rössner S. Initial weight loss is the best predictor for success in obesity treatment and sociodemographic liabilities increase risk for drop-out. Patient Educ Couns. 2010 Jun;79(3):361-6.

236. HanTS,Richmond P, Avenell A, Lean ME. Waist circumference reduction and cardiovascular benefits during weight loss in women. Int J Obes Relat Metab Disord 1997 Febr;21(2):127-34.

237. Wilfley DE, Agras WS, Telch CF, Rossiter EM, Schneider JA, Cole AG, Sifford LA, Raeburn SD. Group cognitive-behavioral therapy and group interpersonal psychotherapy for the nonpurging bulimic individual: a controlled comparison. J. Consult Clin Psychol. 1993 Apr;6(2):296-305.

238. Fossati M, Amati F, Painot D, Reiner M, Haenni C, Golay A.Cognitive-behavioral therapy with simultaneous nutritinal and physical activity education in obese patients with binge eating disorder. Eat Weight Disord. 2004 Jun; 9(2):134-8.

239. Brown A et al. Description and preliminary results from a structured specialist behavioural weight management group intervention: Specialist Lifestyle Management (SLIM) programme. BmJ Open 2015 Apr 8;5(4).

240. Cooper, Z, Doll HA, Hawker DM, Byrne S, Bonner G, Eeley E, O'Connor ME, Fairburn CG. Testing a new cognitive behavioural treatment for obesity: A randomized controlled trial with three-year follow up. Behav Res Ther. 210 Aug;48(8):706-13.

241. Brownell KD. The Learn Program for Weight Control. Lifestyle, Exercise, Attitudes, Relationships, Nutrition. Dallas: American Health Publishing Company; 1997.

242. Munsch S, Biedert E, Keller U. Evaluation of a lifestyle change programme for the treatment of obesity in general practice. Swiss Med Wkly 2003 Mar 8;133 (9-10):148-54.

243. Clinical guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults– the Evidence Report. National Institutes of Health. Obes Res.1998 Sep; 6 Suppl 2:51-209.

244. Wilfley DE1, Welch RR, Stein RI, Spurrell EB, Cohen LR, Saelens BE, Dounchis JZ, Frank MA, Wiseman CV, Matt GE. A randomized comparison of group cognitive behavioral therapy and group interpersonal psychotherapy for the treatment of overweight individuals with binge-eating disorder. Arch Gen Psychiatry. 2002 Aug;59(8):713-21.

245. Foreyt JP, Potson WS 2nd. What is the role of cognitive-behavior therapy in patient management? Obes Res. 1998 Apr;6 Suppl 1:18-22.

246. van Nunen AM, Wouters EJ, Vingerhoets AJ, Hox JJ, Geenen R.The Health-Related Quality of Life of Obese Persons Seeking or Not Seeking Surgical or Non-surgical Treatment: a Meta-analysis. Obes Surg. 2007 Oct; 17(10): 1357-66.

247. Doll HA, Petersen SE, Stewart-Brown SL. Obesity and physical and emotional well-being: associations between body mass index, chronic illness, and the p hysical and mental components of the SF-36 questionnaire. Obes Res. 2000 Mar;8(2):160–70.

248. Marchesini G, Bellini M, Natale S et al. Psychiatric distress and health-related quality of life in obesity. Diabetes Nutr Metab. 2003 Jun;16(3):145-54.

249. Choban PS, Onyejekwe J, Burge JC, Flancbaum L. A health status assessment of the impact of weight loss following Roux-en-Y gastric bypass for clinically severe obesity. J Am Coll Surg. 1999 May;188(5): 491-7.

250. Dixon JB, Dixon ME, O'Brien PE. Quality of life after Lapband placement: influence of time, weight loss, and comorbidities. Obes Res. 2001 Nov;9(11):713-21.

251. Kaplan RM, Ries AL. Quality of life: concept and definition. COPD. 2007 Sep;4(3):263-71.

252. Ware J E, Jr., Gandek B. Overview of the SF-36 Health Survey and the International Quality of Life Assessment (IQOLA) Project. J Clin Epidemiol 1998 Nov; 51(11):903-112.

253. Iannaccone M, D'Olimpio F, Cella S, Cotrufo P. Self-esteem, body shame and eating disorder risk in obese and normal weight adolescents: A mediation model. Eat Behav. 2016 Jan 2;21:80-83.

254. Self-esteem outcomes over a summer camp for obese youth. McGregor S, McKenna J, Gately P, Hill AJ. Pediatr Obese. 2016 Feb 24. [Epub ahead of print]

255. Nir Z, Neumann L. Relationship among self-esteem, internal-external locus of control, and weight change after participation in a weight reduction program. J Clin Psychol.1995 Jul; 51(4): 482-90.

256. Andersson I, Rössner S. Weight development, drop-out pattern and changes in obesity-related risk factors after two years treatment of obese men. Int J Obes Relat Metab Disord. 1997 Mar;21(3):211-6.

257. Melchionda N, Besleghi L, Di Domizio S, Pasqui F, Nuccitelli C, Migliorini S, Baraldi L, Natale S., Manini R, Bellini M, Belsito C, Forlani G, Marchesini G. Cognitive behavioural herapy for obesity: One-year follow-up in a clinical setting. Eat Weight Disord. 2003 Sep;8(3):188-93.

258. Inelmen EM, Toffanello ED, Enzi G, Gasparini G, Miotto F, Sergi G, Busetto L,. Predictors of drop-out in overweight and obese outpatient. Int J Obes (Lond). 2005 Jan;29(1):122-8.

259. Sherwood NE, Jeffery RW, Wing RR. Binge status as a predictor of weight loss treatment outcome. Int J Obes Relat Metab Disord, 1999 May;23(5):485-93.

260.. Melin I, Karlström B, Lappalainen R, Berglund L, Mohsen R, Vessby B. A programme of behaviour modification and nutrition counselling in the treatment of obesity: a randomised 2-y clinical trial. Int J Obes Relat Metab Disord. 2003 Sep;27(9):1127-35.

261. Colombo O, Ferretti VV, Ferraris C, Trentani C, Vinai P, Villani S, Tagliabue A. Is drop-out from obesity treatment a predictable and preventable event? Nutr J. 2014 Feb 3;13:13.

262. van Strien T, Herman CP, Verheijden MW. Eating style, overeating, and overweight in a represative Dutch sample. Does external eating play a role? Appetite. 2009 Apr;52(2):380-7. Epub 2008 Nov 27.

263. Hill JO, Thompson H, Wyatt H.Weight maintenance: what's missing? Am Diet Assoc. 2005 May;105(5 Suppl 1): 63-6.

264. MacLean PS, Higgins JA, Johnson GC, Fleming-Elder BK, Donahoo WT, Melanson EL, Hill, JO. Enhanced metabolic efficiency contributes to weight regain after weight loss in obesity-prone rats. Am J Physiol Regul Integr Comp Physiol, 2004 Dec;287(6):R1306-15. Epub 2004 Aug 26.

265. MacLean PS, Higgins JA, GC Johnson, Fleming-Elder BK, Donahoo WT, Melanson EL, Hill, JO. Metabolic adjustments with the development, treatment, and recurrence ofobesity in obesity-prone rats. Am J Physiol Regul Integr Comp Physiol. 2004 Aug;287(2):288-97. Epub 2004 Mar 25.

266. Van Dorsten B. Behavior change components of obesity treatment. In: Barnett AH, Kumar S, editors. Diabetes and obesity. New York: Blackwell Publishing, in press.

267. Mauro M, Taylor V, Wharton S, Sharma AM. Barriers to obesity treatment. Eur J Intern Med. 2008 May;19:173-80. Epub 2007 Nov 19.

268. Okorodudu DE, Bosworth HB, Corsino L. Innovative interventions to promote behavioral change in overweight or obese individuals: A review of the literature. Ann Med. 2015 May;47(3):179-85. Epub 2014 Jul 10.