Disinhibition: its effects on appetite and weight regulation

Bryant, Eleanor J.[1], King, Neil A.[2], Blundell, John E.[1]

[1]Institute of Psychological Sciences, University of Leeds, UK.

[2] Institute of Health and Biomedical Innovation, Human Movement Studies, Queensland University of Technology, Brisbane, Australia.

Key words: Disinhibition, TFEQ, weight loss, eating behaviour, food intake.

Corresponding author: Eleanor Bryant, e-mail: e.j.bryant@bradford.ac.uk, telephone: + 44 (0)1274 235541, fax: +44 (0)1274

Acknowledgements

This review was supported by the European commission quality of life project QLKI-CT-2000-00515. We acknowledge the contributions of Dr Kirsty Brown/MRC grant.

Abstract

Over the past 30 years, the understanding of eating behaviour has been dominated by the concept of dietary restraint. However, the development of the Three Factor Eating Questionnaire (TFEQ), introduced two other factors; Disinhibition and Hunger, which have not received as much recognition in the literature. The objective of this review is to explore the relationship of the Disinhibition factor to weight regulation, food choice and eating disorders, and to consider its etiology. The review indicates that Disinhibition is an important eating behaviour trait. It is not only associated with a higher BMI and obesity, but also with mediating variables, such as less healthful food choices, which contribute to overweight/obesity and poorer health. Disinhibition is also implicated in eating disorders and contributes to eating disorder severity. It has been demonstrated that Disinhibition is predictive of poorer success at weight loss, and of weight regain during weight loss regimes and is associated with lower self-esteem, low physical activity and poor psychological health. Disinhibition therefore, emerges as an important and dynamic trait, with influences that go beyond eating behaviour and incorporate other behaviours which contribute to weight regulation and obesity. The characteristics of Disinhibition itself therefore reflect many components representative of a thrifty type of physiology. We propose that the trait of Disinhibition could be more appropriately renamed as 'Opportunistic Eating' or 'Thrifty Behaviour'.

Introduction

It is over 30 years since the most widely used psychometric tool in the field of eating and obesity was developed by Stunkard and Messick (1). Following the emergence of the

Restraint Scale (2) and the development of the Three Factor Eating Questionnaire (TFEQ) (1), Restraint is the factor which has received most of the attention in the literature in relation to eating disorders, weight gain, success at weight loss and dieting. However, over this time the Disinhibition scale has gradually come to prominence due to the widespread use of the TFEQ, with over 100 published studies addressing the TFEQ factors and eating and health behaviours.

The TFEQ, measures three factors; Restraint, Disinhibition and Hunger. Restraint refers to concern over weight control and strategies which are adopted to achieve this. For instance, avoiding fattening foods, eating small portions, and stopping eating before reaching satiation are all strategies typically used to limit food intake. Disinhibition reflects a tendency towards overeating and eating opportunistically in an obesigenic environment. Examples include eating in response to negative affect, overeating when others are eating, not being able to resist temptations to eat, and overeating in response to the palatability of food. The factor of Hunger is concerned with the extent to which hunger feelings are perceived and the extent to which such feelings evoke food intake. For example, feeling so hungry an individual eats more than three times per day, or feeling so hungry that their stomach feels like a bottomless pit.

More recently, Bond et al. (3) carried out a factor analysis on the three TFEQ factors. This analysis revealed that the three factors could be further broken down into subscales, although only a few authors have used these subscales in their research to date, therefore an in-depth consideration of the subscales is not included here.

The factor of Disinhibition can be regarded as a trait that confers susceptibility to weight gain. In the development of theory in psychology, there are precedents for assigning the title of trait to the factors emerging from factor analysis of questionnaires. That is, Disinhibition represents a set of endurable characteristics which influence an individual's eating behaviour, food choice and eating pathology. These characteristics are also associated with an individual's psychometric profile and behavioural lifestyle. Therefore, we propose that Disinhibition represents a general, rather than a specific trait, which influences several aspects of an individual's life. It is important to note here the difference between trait Disinhibition (reviewed here) and the use of the term 'disinhibition effect', to describe transient episodes of overeating, resulting from the inhibition of Restraint. This review is not concerned with the inhibition of Restraint; it is concerned with the durable trait of Disinhibition, serving as a disposition for opportunistic eating.

The aim of this literature review is therefore to explore the relationship of Disinhibition with weight, food choice, eating disorders and weight management. Such a comprehensive review of Disinhibition is novel and is intended to disclose how Disinhibition contributes to the understanding of eating behaviour, in normal and overweight/obese individuals, and non-clinical and eating disordered people. The review is based on a large number of articles and the data in these studies have been assembled into tables. These tables are too voluminous to be included in this written report, but can be accessed at http//www.ADDRESS TO BE INSERTED. It is also important to note

here the importance of the longitudinal studies documenting the influence of Disinhibition over time. Both cross-sectional and longitudinal studies are included in the accompanying tables.

Disinhibition and Eating Behaviour

Relation of Disinhibition to BMI and Obesity

One of the most convincing features of studies in the literature is that Disinhibition is positively associated with BMI and obesity. Studies using a cross-sectional design show a positive association between Disinhibition and BMI across different socio-economic gradients (4), and in groups of individuals of differing dieting status (5). Also in individuals with varying weight histories, in which formerly overweight/obese individuals retained a higher Disinhibition score, despite a lower current weight (6). In addition, it is widely reported that obese and overweight individuals have higher Disinhibition scores compared to normal weight individuals (7;8). These studies show how Disinhibition is related to a higher weight across all BMI categories; however this raises the question of whether Disinhibition increases with increments in weight or whether weight increases with increments in Disinhibition?

Similarly to the cross-sectional studies, one prospective study (9) showed how Disinhibition predicted weight gain over several years and predicted current BMI. However, it should be noted that Drapeau et al., (10) found that although changes in eating behaviour traits were associated with changes in weight over 6-years, surprisingly

those with a high Disinhibition score experienced little change in their weight over this time period. This study raises the important issue that Disinhibition may not act in isolation, but frequently exerts its effect in interaction with the level of the TFEQ factors Restraint and Hunger. Hays et al., (9) suggested that when high Disinhibition is combined with a high level of Restraint, the relationship between Disinhibition and weight is weakened. Further evidence for this suggestion comes from the cross-sectional studies which revealed that those individuals with the highest weight had a high Disinhibition and low Restraint, while those individuals who expressed a high Disinhibition and high Restraint had a somewhat lower BMI (4;9;11). Similarly, when a high Disinhibition score is concurrent with a high Hunger score, a higher BMI is observed (4;7;8). Knowledge about the level of all the three TFEQ factors is therefore relevant in predicting the precise influence of Disinhibition upon body weight.

Accordingly, the association of Disinhibition with increased weight and obesity is clear, but it must be kept in mind that the strength of the other TFEQ factors can modulate this relationship. The following sections examine evidence relevant to the interaction between Disinhibition and body weight.

Disinhibition and Overeating

Since Disinhibition provides an indication of a person's higher-responsiveness to cues to eat, it would be expected that Disinhibition would be related to overeating in both normal weight and overweight samples. Studies have employed a variety of techniques to

investigate this relationship including using preload experiments, stress interventions and exercise regimes.

The preload studies suggest that Disinhibition is the best predictor of food consumption (12-14). In these studies individuals with a high Disinhibition score consumed more food (ice cream or cookies) irrespective of their Restraint score. These data encourage the view that Disinhibition and Restraint scales are independent constructs, as Disinhibition can exert its effects independently of Restraint.

Stress manipulation studies have shown that Disinhibition is an important predictor of the eating response to stress in the short term (15), particularly for those women (16), who were found to eat in response to general and specific stressors. Oliver et al., (17) also found that for women, Disinhibition was associated with an increased consumption, especially of sweet foods, under periods of interpersonal stress. In addition, Haynes et al., (15) showed how the relationship between Disinhibition and Restraint modulated the eating response to stress. Here high Disinhibition, low Restraint (HDLR) women consumed the most food; high Disinhibition, high Restraint (HDHR) and low Disinhibition, low Restraint (LDLR) women increased their food consumption in response to stress while the low Disinhibition, high Restraint (LDHR) women's intake remained unaffected by stress.

A similar pattern of eating response of the various combinations of Disinhibition and Restraint scores is also reflected in studies by Yeomans et al., (18), in which a palatability

manipulation was used. Similarly to Haynes et al., (15), Yeomans et al., found that the HDLR women were more responsive to the palatability of the food, as they consumed more food in the palatable than the bland condition. The HDHR and LDLR groups also increased their intake in the palatable condition, but LDHR did not increase intake in response to palatability. In relation to the responsivity to palatability of foods, Blundell et al., (19) provided data suggesting those individuals who are susceptible to weight gain in response to a high-fat diet are characterized by a high Disinhibition. In addition to showing a high Disinhibition score, these susceptible individuals also had a preference for high-fat foods, a strong hedonic attraction to palatable foods and a weak satiety response to high-fat foods. Together, these studies have demonstrated a complex but understandable relationship of Disinhibition to overeating.

An overeating response has also been revealed in exercise studies. In a 4 month intervention (of diet followed by exercise) those women who overate in response to exercise were characterized by high Disinhibition (20). Similarly, Visona and George (21), using an acute exercise intervention, found that women with a high Restraint and high Disinhibition score who identified themselves as dieters increased their intake 12h after exercising. However, it has also been reported that an acute exercise intervention exerted a positive influence on food intake regulation, producing a reduced motivation to eat, reduced lethargy and increased positive mood (22). The difference between these studies is probably due the use of lean versus obese participants. The influence of Disinhibition on eating (and overeating) is likely to be modulated by weight status, and possibly by other factors associated with weight status. This is particularly highlighted by

a study in which obese individual's who reported that their eating episodes were unrelated to fluctuations in hunger and satiety (fullness), were characterized by a high Disinhibition (23)**.**

Taken together these studies indicate that Disinhibition is almost inevitably linked to an increased tendency to eat when people are subjected to various challenges or interventions that threaten to disturb energy balance. These effects could be considered as the appropriate response for a thrifty biological system.

Disinhibition and Food Choice

The association between Disinhibition and food choice has been examined by prospective and cross sectional studies, both of which found that Disinhibition was associated with making less healthful food choices, consistent with data on overeating.

Cross-sectional studies have highlighted how individuals with a high Disinhibition are more likely to choose high-fat foods, high-fat and high-salt foods, processed meat, sweet fruits and vegetables, and sweet, carbonated drinks (24), and to report a higher intake of sweet foods, ice cream, butter and coffee (25). A greater 'wanting' of high-fat foods has also been experimentally demonstrated recently (26). Liking of all foods was also positively correlated with Disinhibition scores (25), a finding we have also confirmed experimentally (26). Moreover, Disinhibition has been found to be related to the amount of chocolate 'chocolate addicts' consume (27) and to overall energy intake in obese women (28). This demonstrates how high Disinhibition scores can be associated with a

lower quality diet, which could be related to the development of weight management and health problems.

Furthermore, in a study of mother's food intake and that of their child; the mother's Disinhibition scores were not only related to their own intake, but to that of their children as well. Here mothers' Disinhibition scores were positively correlated with their children's overall intake of calories, fat, cholesterol, protein and sodium and correlated negatively with intake of some vegetables (24). This clearly raises issues for childhood weight management.

In a prospective study following men over a 31 month weight loss programme, Disinhibition scores were found to be negatively related to high-fibre bread intake and fruit intake (29). This demonstrates how high Disinhibition scores are related to poorer food choice in men and women. In addition, high Disinhibition has been found to be associated with a higher rate of alcohol consumption in women (24;30) and men (29).

In summary, high Disinhibition scores are related to a higher liking for and consumption of high fat foods, sweet foods and alcohol, and negatively related to the consumption of vegetables, fruit and high-fibre bread. This suggests that this less healthful food choice could not only contribute to overweight and obesity, but also contribute to a poorer general health.

Disinhibition and Biological Factors

As described above, high Disinhibition individuals have a higher liking for foods, particularly sweet and high fat foods, and this raises the question of whether there is some biological reason for these food preferences.

The eating patterns exhibited by individuals with a high Disinhibition could be explained, in part, by the fact that hedonic reward of food is closely linked to the sensory perceptions of food in the mouth, and for many individuals this is the main drive to eat (33). DelParigi et al., (34) have demonstrated in a group of obese individuals, for whom Disinhibition was strongly correlated to their obesity, that an abnormal brain response to the sensory experience of a liquid meal following a prolonged fast was apparent. An increased activity in the insular cortex (a sensory area) was observed, and the author concluded that the greater insular response in obese participants compared to their lean counterparts, suggested a greater central sensitivity to the fat content and/or texture of the liquid formula meal. It is important to note here, that what is deemed an abnormal brain response, could in fact be a normal brain response for individuals exhibiting a higher Disinhibition score. This brain response could provide at least a partial explanation for the preference for sweet and high fat foods, shown in high Disinhibition individuals. In terms of taster status, two studies found there was no significant relationship between PROP (6-*n*-propylthiouracil) taster status and Disinhibition (31;32).

It is also plausible that the dysregulated eating patterns displayed by high Disinhibition women could be influenced by differing levels of hunger or satiety related peptides.

However, St-Pierre et al., (35) showed that ghrelin levels were not correlated with Disinhibition scores in a sample of lean women. It is now well known that ghrelin levels are lower in obese than lean people and there is a negative relationship between BMI and ghrelin concentrations within an obese group (36). This relationship has been confirmed (37). Moreover, there was a positive relationship between leptin concentrations and Disinhibition in obese women, even after BMI had been controlled. The significant relationship between Disinhibition score and two peptides (ghrelin and leptin) known to play significant roles in energy homeostasis (38), suggests that Disinhibition is intimately linked to the physiological basis of energy homeostasis. However, it should be noted that Conus et al., (39), failed to find an association between Disinhibition and satiety related peptides in normal weight women who were metabolically obese.

Disinhibition and Heritability

The majority of studies support the view that Disinhibition is a strongly heritable form of eating behaviour. However, others propose that Disinhibition is sustained largely by environmental influences. One clear message from the evidence is that Disinhibition is transmitted through families, but the extent to which this depends on genetic and/or shared environmental factors, has yet to be clarified.

Estimates of the genetic heritability of the TFEQ factors show large variation with ranges for Disinhibition varying from 40%, to 45%, 17.5% and 0% (40-43) respectively. Reported estimates for the heritability of Restraint range between 28%, 0%, 5.5%, and 24% (40-43) respectively and 9% to 8%, 28.4% and 44% for Hunger (40-43)

respectively. Therefore it is difficult to obtain a consensus about the true genetic influence on the measured level of these factors.

Compelling evidence comes from those studies that have identified specific chromosomal locations for the particular eating behaviour traits. For example, Bouchard et al., (44) found four trait loci for Disinhibition and Hunger, including associations with neuromedin B. Steinle et al., (40) also proposed four regions of linkage for Disinhibition and Hunger in proximity to leptin and plasminogen activator inhibitor genes. Since, these areas code for specific genes related to appetite control and/or metabolic regulation, it is plausible that the Disinhibition and Hunger traits are found to be associated with these genetic loci. Caution must be used when interpreting the findings of such studies, and replications are needed before such specific chromosomal location associations can be confirmed.

Other authors however have suggested a primarily environmental role for the transmission of Disinhibition (43;45). This is supported by experimental studies involving children of varying ages, where it is argued that Disinhibition can be transferred to children through modeling of parents (46-50), a mechanism which is particularly potent between mothers and daughters. Of course the observation that Disinhibition can be amplified through family interactions does not negate the existence of a genetic predisposition.

Disinhibition and Eating Disorders

Studies examining TFEQ variables and eating disorders, have generally focused on Binge Eating Disorder (BED) and measures of body dissatisfaction and maladaptive eating behaviours. Disinhibition seems to play a key role in determining the severity of eating disturbances.

Several authors have found a positive association between BED and Disinhibition (51-59), and higher Disinhibition scores were also found to be associated with bulimia nervosa (BN) (53;60). Interestingly Disinhibition was related to binge eating severity (56;57;59) and a higher eating disorder pathology in BN patients (60). Furthermore, in eating disordered patients, Disinhibition was also found to be associated with a lower psychological health; with higher neuroticism, lower extroversion, lower self-esteem (60), depression, anxiety and body dissatisfaction (55). These associations suggest that treatment for eating disorders could be directed to the management of the factor of Disinhibition. As these data refer to the association between Disinhibition and eating disorders, it is unclear whether Disinhibition is simply related to disordered eating, or actually plays a causal role in the development of eating disorders. The fact that TFEQ scores do not change much during recovery, suggests that they represent the expression of core processes underlying eating disorders.

Furthermore, in samples of non-clinical individuals, Disinhibition was again associated with higher eating disturbances (61) and a lower psychological health (62;63). A key finding arising from these eating disorder studies, is that the severity of eating pathology

is related to the association of high Disinhibition with a (very) high Restraint score. The effect of this combination is also apparent, but to a lesser extent, even in normal weight women. The conflict created by high Disinhibition and the presence of high Restraint appears to dysregulate the control of eating. It can be suggested that high Disinhibition (particularly when coupled with high Restraint) could be used as a predictor to identify those at risk of eating disorders, particularly since Disinhibition and Restraint have been shown to increase over time in women, in line with their disturbed eating symptomatology (64)**.**

Disinhibition therefore emerges as an important factor in eating disorders and the severity of eating pathology (as measured, for example, by EAT-26). The reduction of Disinhibition in eating disorders has the potential to improve symptomatology and possibly reduce relapse. The identification of individuals at risk of eating disorders also becomes possible, since high Disinhibition (plus high Restraint) is related to disturbed eating patterns in non-clinical, normal weight women, and this combination therefore becomes a psychological marker for future development of dysregulated eating.

Disinhibition and Personality Factors

Studies have also been carried out to assess which particular personality variables are associated with Disinhibition. Gendall et al., (65) found Disinhibition to be associated with personality factors which are counterproductive to the maintenance of good control over food intake. For instance, high Disinhibition was associated with novelty seeking, impulsiveness, and negatively related to self directedness (an inability to resist external

cues), which is related to the high Disinhibition individuals' tendency towards overeating. In two further studies by Carmody et al., (66;69), Disinhibition was positively related to dietary helplessness, while Restraint was inversely related. Women were found to have higher Disinhibition, Restraint and dietary helplessness compared to men. Participants with a history of weight cycling also had a higher Disinhibition and dietary helplessness.

Therefore, Disinhibition is associated with personality traits which encourage overeating and promote adverse psychological health. This has implications for weight loss strategies and implies that coping techniques could be beneficial for individuals with a high Disinhibition.

Disinhibition and Health

Reports in the literature strongly imply that Disinhibition is associated with lower physical health in normal weight, obese and diabetic individuals. The increased risk of physical ailments could be due to the dietary patterns exhibited by high Disinhibition individuals, reporting less healthful food choices (24;25;29). People with high Disinhibition scores are also more susceptible to adverse psychological symptoms (15;59). More specifically, Provencher et al (68) found that Disinhibition (particularly habitual susceptibility to Disinhibition) was negatively associated with psychological wellbeing in a sample of post-menopausal women, irrespective of their weight status.

Cross sectional studies have identified that Disinhibition is associated with negative physical health as well. For example, Hainer et al., (69) found that Disinhibition and Restraint were significant predictors of diseases which characterize the metabolic syndrome, and Hays et al., (9) found that Disinhibition was related to ill health (e.g. back pain, constipation) even when BMI was accounted for. In addition Marchesini et al., (70) reported an association between high Disinhibition and low Restraint and a lower health related quality of life. This is also reflected in work by Straub et al., (71) who found that Type II diabetics with a high Disinhibition score, were prone to poorer glycaemic control. Therefore, the potential for high Disinhibition to exert a negative impact on health can be independent of weight status. This suggests that in addition to adiposity, the characteristic behaviour of high Disinhibition individuals, puts them at a higher risk of poorer health. This raises the question of whether the influence of Disinhibition upon health is direct or indirect. Is it the case that the behavioural characteristics of high Disinhibition, such as their poorer diet, cause high Disinhibition individuals to have poorer health or does the possession of a high Disinhibition score lead directly to poor health outcome? Disinhibition is thus a trait which exerts its influence beyond just eating behaviour, and affects the person as a whole.

Disinhibition and Smoking

As well as having a negative impact on general health, high Disinhibition is also associated with behaviours which can damage health, for instance having higher Disinhibition (and Restraint) score is associated with a greater likelihood of smoking (72;73). This is possibly because high Disinhibition is associated with greater appetite

and weight control expectancies from smoking (72;74). Importantly, Disinhibition has been found to be associated with an increased food intake during smoking abstinence (75) which could explain why high Disinhibition is the best predictor of weight gain during smoking cessation (76;77). Sanchez-Johnsen et al., (78) also found that high Disinhibition scores were associated with more pathological eating behaviours during smoking cessation. Smoking cessation programs should therefore include guidance on how to cope with high Disinhibition to avoid excess weight gain and reduce pathological eating behaviours, while giving-up.

Disinhibition and Weight Loss

There is a large body of evidence detailing the relationship of Disinhibition with weight loss in weight loss treatment programs. Seven studies investigated the effect of a very low calorie diet (VLCD) on weight loss and eating behaviour scores as measured by the TFEQ (79-85). These revealed some variability on the pattern of change in the TFEQ factors over the course of treatment depending upon the success at weight loss. For example, Pekkarinen et al., (82) found that Disinhibition and Hunger scores decreased and Restraint increased only in those individuals who maintained a >10% weight loss from baseline. Those who did not achieve this weight loss, saw a return of their TFEQ scores to pretreatment levels two years following the treatment program. This raises the question of whether a change in weight leads to a lower Disinhibition score, or whether a reduction in level of Disinhibition promotes weight loss? Two studies have reported that change in Disinhibition score was independent of weight change (79;85), suggesting a level of complexity between weight loss and change in TFEQ factors.

Further studies have explored diet plus exercise regimes on weight loss and its relationship with TFEQ factors (86-92). Again, there was a unanimous finding that following treatment, which ranged from ten-weeks to one-year, Disinhibition and Hunger scores decreased and Restraint scores increased. However, Karlsson et al., (81) showed that weight regain during follow-up was strongly associated with Disinhibition level, perhaps suggesting that Disinhibition may not be a very potent factor during weight loss, yet it exerts its most influence during weight maintenance. This is reflected in work by McGuire et al., (93) who found that those who manage to maintain weight loss, are characterized by a lower Disinhibition and Hunger score; where an initial high Disinhibition score is predictive of weight regain (93).

Most of these studies also included behavioural therapy in their protocol, aimed at regulating eating behaviour (86-90). The aim of this behaviour therapy varied between the studies, with most authors targeting the increase of Restraint as their goal, one group however included decreasing Disinhibition in their therapy. Cuntz et al., (90) found that Disinhibition could be reduced by enhancing self-control without restrained eating and by reducing the dysfunctional emotional influences on eating behaviour, thus advocating the use of behavioural therapy in obesity treatment, particularly for those with high Disinhibition scores. Also, these outcomes suggest that Disinhibition can be successfully diminished through application of behavioural therapy, a concept which has not been adopted in the literature to date. Two findings have emerged from studies of weight loss. First, a high Disinhibition score works against the maintenance of body weight and tends to favour weight regain. Second, Disinhibition scores tend to decrease as weight is lost

during a treatment programme. However, it is known that ghrelin and leptin also tend to change with weight loss (94-98) and since these peptides are linked to Disinhibition, it would be expected that Disinhibition would also change.

A number of studies have investigated eating behaviour scores (TFEQ) pre and post gastric surgery (either gastric banding or gastric by-pass). Again a consensus in findings was apparent, such that following surgery and weight loss, Disinhibition and Hunger decreased and Restraint increased. (36;99-107).

Three studies have also examined the association between pharmacologically-induced weight loss and the TFEQ factors (109-111). Each study used a different drug, but consistently observed a decrease in Disinhibition and Hunger scores and an increase in Restraint scores. The drugs examined were pegylated recombinant leptin (PEG-OB) (111), Zonisamide (109) and sibutramine (110).

Disinhibition and physical activity

To date, there is not a large body of data surrounding the relationship between Disinhibition and physical activity. However, the data which are available highlight some important issues. As alluded to in the *Disinhibition and overeating* section, women who showed a high Disinhibition and who exercised, responded by increasing their energy intake (20, 21). This raises the issue of overcompensation for the energy expended during exercise, and therefore increases the likelihood of these individuals being in a positive energy balance. This coupled with evidence suggesting that women with a higher

Disinhibition score, generally exhibit more sedentary behaviour (112), poses a challenge to any weight control efforts. However, some data do suggest that exercise can exert a positive influence on women with a high Disinhibition score. For example an acute bout of exercise was shown to decrease motivation to eat (22) and increase preference for low fat foods (26) in lean women. A clearer picture of the influence exercise exerts on individuals with a high Disinhibition, will emerge with further research.

Conclusions

Disinhibition appears to be a potent and important eating behaviour trait, being associated not only with higher BMI and weight, but also with intermediaries, such as less healthful food choices, which contribute to overweight and obesity and poorer health. Disinhibition is also related to eating disorders, particularly BED, BN and weight cycling and contributes to eating disorder severity. Furthermore higher Disinhibition scores are associated with adverse psychological symptoms in eating disordered samples and normal weight samples, which could exacerbate dysregulated eating in these individuals. Disinhibition is also related to the adoption of unhealthy behaviours, such as smoking as a weight control method, and also predicts the weight gained during smoking cessation. Disinhibition has been cited as a very strong predictor of weight regain during weight loss regimes, whether based on diet, diet plus exercise, behaviour therapy, or gastric surgery. Also Disinhibition is predictive of success at weight loss, with higher Disinhibition scores predicting less weight loss.

The variations in scores on the Disinhibition factor of the TFEQ and their relationship with disordered eating and tendency to weight gain, suggest that this factor reflects the existence of a psychological entity that can be termed a trait. There are clear indications that this trait has biological associations and the strength of Disinhibition may be reflected in regional brain activity. On the other hand, Disinhibition scores can clearly be influenced by behavioural control treatments and by weight loss itself; an effect that can be amplified by certain pharmaceutical or surgical interventions.

Disinhibition and the expression of the thrifty genotype

This review has indicated that individuals with a high Disinhibition are characterized by a strong propensity to overeat, gain weight easily, have a higher liking of food and preference for high-fat foods, and a lower energy expenditure due to sedentary behaviour. These data can be considered in relation to the thrifty genotype concept (113)**.** This hypothesis proposes that evolution has favoured a genetic adaptation to allow humans to survive in the face of food scarcity and famine, by selective pressure on physiology. Although Neel's (113) original view was directed to the functional aspects of insulin resistance, the adaptive potential of a physiology protective against periodic food scarcity, can be extended to a behavioural dimension, that favours high fat foods, allows food gorging, promotes fat deposition and encourages energy conservation, through sedentary behaviour. A recent exposition of the theory by Prentice (114;115) and Bouchard (116) has drawn attention to the phenotypic expression of the thrifty genotype.

However, as we now live in an obesigenic environment, this once favoured 'thrifty' physiology has become counterproductive and promotes a weight gaining phenotype. Where the once thrifty genotype led to survival, it now serves to encourage overweight and obesity, along with several comorbidities which accompany excess adiposity (117). High Disinhibition individuals appear to show the characteristics of the thrifty genotype. A good example could be bingeing or gorging behaviour. It is reasonable to suppose that a capacity to gorge on available food would be a beneficial trait if the food supply was likely to be interrupted. Hence gorging (or bingeing) would be functional in that type of environment (still apparent today in certain parts of the world). However, this tendency to gorge (and binge) would be defined as inappropriate or even pathological in a culture in which food was plentiful and overconsumption was stigmatized.

Although the thrifty genotype can be expressed through adipose tissue processes, metabolism and muscle physiology, a behavioural dimension reflected by potent food seeking, opportunistic eating, gorging when food is available and positive responses to food has strong face validity. The trait of Disinhibition is characterized by all of these factors, to varying degrees, and therefore appears to qualify as a mediator of the thrifty genotype. It can be surmised that this trait could be advantageous in times of food scarcity but is counterproductive in an environment replete with food.

Circumstances and situations in which susceptibility to weight gain (or regain) have been observed, suggests that the trait of Disinhibition is a pervasive factor that operates in most, possibly all, circumstances where weight gain is observed. This hypothesis does

not in any way undermine arguments about the potency of an obesigenic environment in the promotion of obesity. However, in such an environment not all individuals become obese; some are susceptible and some resistant to a potentially weight inducing dietary environment. Significantly, these individuals identified as susceptible to weight gain are characterized by high Disinhibition scores.

Disinhibition (like the thrifty genotype) is dysfunctional in the current obesigenic environment. The behavioural expression is contrary to prevailing health messages and therefore produces a conflict. This is an unavoidable conflict between a biological disposition and an environmental force that leads to degrading of psychological wellbeing, loss of self-esteem and other indicators of poor health. In this context, Disinhibition seems to play a significant mediating role between the 'person' and the 'environment', and has physiological and psychological components.

Disinhibition: time for a name change?

In writing this review we have adhered faithfully to the term Disinhibition which is the label given to factor two by the original authors of the TFEQ. However, we have continually questioned whether or not Disinhibition is the correct description for this factor, since it implies an inhibition of inhibition. This leads to confusion with the notion of inhibition of Restraint which is a separate phenomenon. In addition, others have questioned the strict existence of Factor 2, separate from Factor 3 (Hunger) and have proposed one super-factor (118; 119). We are aware that other researchers also feel uneasy with the term Disinhibition since it does not seem to accurately describe the

mechanisms or consequences of the factor. We therefore propose that this factor should be renamed and our favoured term is 'Opportunistic Eating'. This term is readily related to a number of questions included in Factor 2. However, a number of possibilities could be considered including 'Readiness to Eat' and 'Thrifty Behaviour', according to the weight given to various aspects of the trait. It can often be difficult to find the appropriate label to characterize a factor that emerges from factor analysis of a questionnaire. These factors operate on 'theoretical constructs' (120) and can have potent explanatory value. Whatever the most appropriate name for Factor 2 of the TFEQ, we feel the factor (although heavily overlapping with Factor 3) has important implications for understanding a person's relationship with the obesigenic environment and the degree of control (or lack of control) that can be asserted.

Reference List

(1) Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. J Psychosom Res 1985;29(1):71-83.

(2) Herman CP, Mack D. Restrained and unrestrained eating. Journal of Personality 1975;43:647-60.

(3) Bond MJ, McDowell AJ, Wilkinson JY. The measurement of dietary restraint, disinhibition and hunger: an examination of the factor structure of the Three Factor Eating Questionnaire (TFEQ). Int J Obes Relat Metab Disord 2001; 25(6):900-6.

(4) Dykes J, Brunner EJ, Martikainen PT, Wardle J. Socioeconomic gradient in body size and obesity among women: the role of dietary restraint, disinhibition and hunger in the Whitehall II study. Int J Obes Relat Metab Disord 2004;28(2):262-8.

(5) Provencher V, Drapeau V, Tremblay A, Despres JP, Bouchard C, Lemieux S, Quebec Family. Eating behaviours, dietary profile and body composition according to dieting history in men and women of the Quebec Family Study. Brit J Nutr 91(6):997-1004, 2004.

(6) Bellisle F, Clement K, Le Barzic M, Le Gall A, Guy-Grand B, Basdevant A. The Eating Inventory and Body Adiposity from Leanness to Massive Obesity: a Study of 2509 Adults. Obes Res 2004; 12(12):2023-30.

(7) Provencher V, Drapeau V, Tremblay A, Despres JP, Lemieux S. Eating behaviors and indexes of body composition in men and women from the Quebec family study. Obes Res 11(6):783-92, 2003.

(8) Boschi V, Iorio D, Margiotta N, D'Orsi P, Falconi C. The three-factor eating questionnaire in the evaluation of eating behaviour in subjects seeking participation in a dietotherapy programme. Ann Nutr Metab 2001;45:72-7.

(9) Hays NP, Bathalon GP, McCrory MA, Roubenoff R, Lipman R, Roberts SB. Eating behavior correlates of adult weight gain and obesity in healthy women aged 55-65 y. Am J Clin Nutr 75(3):476-83, 2002.

(10) Drapeau V, Provencher V, Lemieux S, Despres J-P, Bouchard C, Tremblay A. Do 6-y changes in eating behaviors predict changes in body weight? Results from the Quebec Family Study. Int J Obes 2003;27(7):-814.

(11) Williamson DA, Lawson OJ, Brooks ER, Wozniak PJ, Ryan DH, Bray GA, Duchmann EG. Association of body mass with dietary restraint and disinhibition. Appetite Vol 25(1), 1995;:31-41.

(12) Westenhoefer J, Broeckmann P, Munch A, Pudel V. Cognitive Control of Eating Behavior and the Disinhibition Effect. Appetite 1994;23(1):27-41.

(13) van Strien T, Cleven A, Schippers G. Restraint, tendency toward overeating and ice cream consumption. Int J Eat Dis 28(3):333-8, 2000.

(14) Ouwens MA, van Strien T, van der Staak CP. Absence of a disinhibition effect of alcohol on food consumption. Eat Behav 2003;4(4):323-32.

(15) Haynes C, Lee MD, Yeomans MR. Interactive effects of stress, dietary restraint, and disinhibition on appetite. Eat Behav 2003;4(4):369-83.

(16) Weinstein SE, Shide DJ, Rolls BJ. Changes in food intake in response to stress in men and women: Psychological factors. Appetite 1997; 28(1):-18.

(17) Oliver KG, Huon GF. Eating-related thought suppression in high and low disinhibitors. Int J Eat Dis 30(3):329-37, 2001.

(18) Yeomans MR, Tovey HM, Tinley EM, Haynes CJ. Effects of manipulated palatability on appetite depend on restraint and disinhibition scores from the Three-Factor Eating Questionnaire. Int J Obes Relat Metab Disord 2004; 28(1):144-51.

(19) Blundell J, Stubbs RJ, Golding C, Croden F, Alam R, Whybrow S, Le Noury J, Lawton CL. Resistance and susceptibility to weight gain: Individual variability in response to a high-fat diet. Physiol & Beh 2005;86(5):614-22.

(20) Keim NL, Canty DJ, Barbieri TF, Wu M. Effect of Exercise and Dietary Restraint on Energy Intake of Reduced-Obese Women. Appetite 1996;26(1):55-70.

(21) Visona C, George VA. Impact of dieting status and dietary restraint on postexercise energy intake in overweight women. Obes Res 10(12):1251-8, 2002.

(22) Bryant E, King N, Blundell J. Effect of exercise on appetite control in women with high trait Disinhibition. Appetite 2005;45:363.

(23) Barkeling B, King N, Naslund E, Blundell J. Characterization of obese individuals who claim to detect no relationship between their eating pattern and sensations of hunger or fullness. Int J Ob 2007; 31(3):435-9.

(24) Contento IR, Zybert P, Williams SS. Relationship of cognitive restraint of eating and disinhibition to the quality of food choices of Latina women and their young children. Preventive Medicine 2005;40(3):-336.

(25) Lahteenmaki L, Tuorila H. Three-factor eating questionnaire and the use and liking of sweet and fat among dieters. Physiol Behav 1995;57(1):81-8.

(26) Bryant E, Finlayson G, King N, Blundell J. The influence of acute exercise on liking and preference for food on high trait Disinhibition women. Obes Rev 2006;7(2):343.

(27) Hetherington MM, Macdiarmid JI. "Chocolate addiction": A preliminary study of its description and its relationship to problem eating. Appetite 1993;21(3):-246.

(28) Lindroos AK, Lissner L, Mathiassen ME, Karlsson J, Sullivan M, Bengtsson C, Sjostrom L. Dietary intake in relation to restrained eating, disinhibition, and hunger in obese and nonobese Swedish women. Obes Res 5(3):175-82, 1997.

(29) Borg P, Fogelholm M, Kukkonen-Harjula K. Food selection and eating behaviour during weight maintenance intervention and 2-y follow-up in obese men 48. Int J Obes Relat Metab Disord 2004;28(12):1548-54.

(30) Higgs S, Eskenazi T. Dietary restraint and disinhibition are associated with increased alcohol use behaviours and thoughts in young women social drinkers. Eating Behav 2007;8:236-43.

(31) Tepper BJ, Ullrich NV. Influence of genetic taste sensitivity to 6-n-propylthiouracil (PROP), dietary restraint and disinhibition on body mass index in middle-aged women. Physiol & Beh 2002;75(3):-312.

(32) Goldstein GL, Daun H, Tepper BJ. Adiposity in middle-aged women is associated with genetic taste blindness to 6-n-propylthiouracil. Obes Res 13(6):1017-23, 2005.

(33) Drenowski A. Tast preferences and food intake. Ann Rev Nutr 1997;17:237-53.

(34) Delparigi A, Chen K, Salbe AD, Reiman EM, Tataranni PA. Sensory experience of food and obesity: a positron emission tomography study of the brain regions affected by tasting a liquid meal after a prolonged fast. Neuroimage 24(2):436-43, 2005.

(35) St Pierre DH, Karelis AD, Cianflone K, Conus F, Mignault D, Rabasa-Lhoret R, St Onge M, Tremblay-Lebeau A, Poehlman ET. Relationship between ghrelin and energy expenditure in healthy young women. J Clin Endocrin & Metab 89(12):5993-7, 2004.

(36) Schindler K, Prager G, Ballaban T, Kretschmer S, Riener R, Buranyi B, Maier C, Luger A, Ludvik B. Impact of laparoscopic adjustable gastric banding on plasma ghrelin, eating behaviour and body weight. Euro J Clin Investigation 34(8):549-54, 2004.

(37) Levin F, King N, Barkeling B, Gustafsson T, Hellstrom P, Blundell J, Naslund E. Ghrelin, leptin and adiponectin are biomarkers associated with the trait disinhibition. 2004. Ref Type: Unpublished Work

(38) Klok MD, Jakobsdottir S, Drent ML. The role of leptin and ghrelin in the regulation of food intake and body weight in humans: a review. Obes Rev 2007;8(1):21-34.

(39) Conus F, Allison DB, Rabasa-Lhoret R, St Onge M, St Pierre DH, Tremblay-Lebeau A, Poehlman ET. Metabolic and behavioral characteristics of metabolically obese but normal-weight women. J Clin Endocrin & Metab 89(10):5013-20, 2004.

(40) Steinle NI, Hsueh WC, Snitker S, Pollin TI, Sakul H, St Jean PL, Bell CJ, Mitchell BD, Shuldiner AR. Eating behavior in the Old Order Amish: heritability analysis and a genome-wide linkage analysis. Am J Clin Nutr 2002;75(6):1098-106.

(41) Neale BM, Mazzeo SE, Bulik CM. A Twin Study of Dietary Restraint, Disinhibition and Hunger: An Examination of the Eating Inventory (Three Factor Eating Questionnaire). Twin Res 2003;6(6):-478.

(42) Provencher V, Perusse L, Bouchard L, Drapeau V, Bouchard C, Rice T, Rao DC, Tremblay A, Despres J, Lemieux S. Familial Resemblance in Eating Behaviors in Men and Women from the Quebec Family Study. Obes Res 2005;13(9):1624-9.

(43) de Castro JM, Lilenfeld LR. Influence of heredity on dietary restraint, disinhibition, and perceived hunger in humans. Nutrition 21(4):446-55, 2005.

(44) Bouchard L, Drapeau V, Provencher V, Lemieux S, Chagnon Y, Rice T, Rao DC, Vohl MC, Tremblay A, Bouchard C, Perusse L. Neuromedin beta: a strong candidate gene linking eating behaviors and susceptibility to obesity. Am J Clin Nutr 2004;80(6):1478-86.

(45) de Castro JM. When identical twins differ: an analysis of intrapair differences in the spontaneous eating behavior and attitudes of free-living monozygotic twins. Physiol & Behav 82(4):733-9, 2004.

(46) Cutting TM, Fisher JO, Grimm-Thomas K, Birch LL. Like mother, like daughter: familial patterns of overweight are mediated by mothers' dietary disinhibition. Am J Clin Nutr 69(4):608-13, 1999.

(47) Hood MY, Moore LL, Sundarajan-Ramamurti A, Singer M, Cupples LA, Ellison RC. Parental eating attitudes and the development of obesity in children. The Framingham Children's Study. Int J Ob & Rel Metab Dis; 24(10):1319-25, 2000.

(48) Johnson SL. Improving Preschoolers' self-regulation of energy intake. Pediatrics 106(6):1429-35, 2000.

(49) Saelens BE, Ernst MM, Epstein LH. Maternal child feeding practices and obesity: A discordant sibling analysis. Int J Eating Dis 2000;27(4):-463.

(50) Whitaker RC, Deeks CM, Baughcum AE, Specker BL. The relationship of childhood adiposity to parent body mass index and eating behavior. Obes Res 8(3):234-40, 2000.

(51) Adami GF, Gandolfo P, Bauer B, Scopinaro N. Binge eating in massively obese patients undergoing bariatric surgery. Int J Eating Dis Vol 17(1), 1995:45-50.

(52) Adami GF, Campostano A, Marinari GM, Ravera G, Scopinaro N. Night eating in obesity: a descriptive study. Nutrition 18(7-8):587-9, 2002.

(53) Ardovini C, Caputo G, Todisco P, Grave R. Binge eating and restraint model: Psychometric analysis in binge eating disorder and normal weight bulimia. Euro Eating Dis Rev 1999;7(4):-299.

(54) Crow S, Kendall D, Praus B, Thuras P. Binge eating and other psychopathology in patients with Type II diabetes mellitus. Int J Eating Dis 2001;30(2):-226.

(55) Dalle Grave R, Todisco P, Oliosi M, Marchi S. Binge eating disorder and weight cycling in obese women. Eating Disorders: The J Treatment & Prevention 1996;4(1):-73.

(56) d'Amore A, Massignan C, Montera P, Moles A, De Lorenzo A, Scucchi S. Relationship between dietary restraint, binge eating, and leptin in obese women. Int J Obes & Rel Metab Dis; 25(3):373-7, 2001.

(57) de Zwaan M, Mitchell JE, Howell L, Monson N, Swan-Kremeier L, Crosby RD, Seim HC. Characteristics of morbidly obese patients before gastric bypass surgery. Comprehensive Psychiatry 2003;44(5):-434.

(58) Goldfein JA, Walsh B, LaChaussee JL, Kissileff HR. Eating behavior in binge eating disorder. Int J Eating Dis 1993;14(4):-431.

(59) Kensinger GJ, Murtaugh MA, Reichmann SK, Tangney CC. Psychological symptoms are greater among weight cycling women with severe binge eating behavior. J Am Dietetic Assoc; 98(8):863-8, 1998.

(60) Brown K, Bryant E, Naslund E, King N, Blundell J. Traits that promote weight gain in obesity, bulimia nervosa and EDNOS. Obes Rev 2006;7(2):330.

(61) Boerner LM, Spillane NS, Anderson KG, Smith GT. Similarities and differences between women and men on eating disorder risk factors and symptom measures. Eating Beh; 2004;5(3):-222.

(62) Ghiz L, Chrisler JC. Compulsive eating, obsessive thoughts of food, and their relation to assertiveness and depression in women. J Clin Psyc 1995;51(4):-499.

(63) Lawson OJ, Williamson DA, Champagne CM, DeLany JP, Brooks ER, Howat PM, Wozniak PJ, Bray GA, Ryan DH. The association of body weight, dietary intake, and energy expenditure with dietary restraint and disinhibition. Obes Res 1995;3(2):153-61.

(64) Rizvi SL, Stice E, Stewart A. Natural History of Disordered Eating Attitudes and Behaviour over a 6-Year Period. Int J Eating Dis 1999;26:406-13.

(65) Gendall KA, Joyce PR, Sullivan PF, Bulik CM. Personality and dimensions of dietary restraint. Int J Eating Dis 24(4):371-9, 1998.

(66) Carmody TP, Brunner RL, St Jeor ST. Dietary helplessness and disinhibition in weight cyclers and maintainers. Int J Eating Dis 1995;18:247-56.

(67) Carmody TP, Brunner RL, St.Jeor ST. Hostility, dieting, and nutrition attitudes in overweight and weight-cycling men and women. Int J Eating Dis 1999;26(1):-42.

(68) Provencher V, Begin C, Piche ME, Bergeron J, Corneau L, Weisnagel SJ, Nadeau A, Lemieux S. Disinhibition, as assessed by the Three-Factor Eating Questionnaire, is inversely related to psychological well-being in postmenopausal women. Int J Obes 2007 June 6;31(2):315-20.

(69) Hainer V, Kunesova M, Bellisle F, Parizkova J, Braunerova R, Wagenknecht M, Lajka J, Hill M, Stunkard A. The Eating Inventory, body adiposity and prevalence of diseases in a quota sample of Czech adults. Int J Obes 2006; 30(5):830-6.

(70) Marchesini G, Solaroli E, Baraldi L, Natale S, Migliorini S, Visani F, Forlani G, Melchionda N. Health-related quality of life in obesity: the role of eating behaviour. Diabetes, Nutr & Metab - Clin & Exper 13(3):156-64, 2000.

(71) Straub RH, Lamparter-Lang R, Palitzsch KD, Scholmerich J. Association between eating behaviour and current glycaemic control, body mass or autonomic nervous function in long-term type I and type II diabetic patients. Euro J Clin Investig 26(7):564-8, 1996.

(72) Copeland AL, Carney CE. Smoking expectancies as mediators between dietary restraint and disinhibition and smoking in college women. Experimental and Clin Psychopharm 2003;11(3):-251.

(73) Bryant E. Understanding Disinhibition and its influences on eating behaviour and appetite 2006. PhD Thesis; University of Leeds.

(74) Pomerleau CS, Ehrlich E, Tate J, Marks JL. The female weight-control smoker: A profile. Journal of Substance Abuse 1993;5(4):-400.

(75) Duffy J, Hall SM. Smoking abstinence, eating style, and food intake. J Consulting and Clin Psyc 1988;56(3):-421.

(76) Hall SM, Ginsberg D, Jones RT. Smoking cessation and weight gain. J Consulting and Clin Psyc 1986;54(3):-346.

(77) Hudmon K, Gritz ER, Clayton S, Nisenbaum R. Eating orientation, postcessation weight gain, and continued abstinence among female smokers receiving an unsolicited smoking cessation intervention. Health Psyc 1999;18(1):-36.

(78) Sanchez-Johnsen L, Fitzgibbon ML, Ahluwalia JS, Spring BJ. Eating pathology among Black and White smokers. Eating Behav 2005;6(2):-136.

(79) Foster GD, Wadden TA, Swain RM, Stunkard AJ, Platte P, Vogt RA. The Eating Inventory in obese women: clinical correlates and relationship to weight loss. Int J Obes & Rel Metab Dis; 22(8):778-85, 1998.

(80) Westerterp-Plantenga MS, Kempen KP, Saris WH. Determinants of weight maintenance in women after diet-induced weight reduction. Int J Obes & Rel Metab Dis 22(1):1-6, 1998 January.

(81) Karlsson J, Hallgren P, Kral J, Lindroos A-K, Sjostrom L, Sullivan M. Predictors and effects of long-term dieting on mental well-being and weight loss in obese women. Appetite 23(1), 1994:15-26.

(82) Pekkarinen T, Takala I, Mustajoki P. Two year maintenance of weight loss after a VLCD and behavioural therapy for obesity: correlation to the scores of questionnaires measuring eating behaviour. Int J Obes & Rel Metab Dis 1996;20(4):332-7.

(83) Foster GD, Wadden TA, Kendall PC, Stunkard AJ. Psychological effects of weight loss and regain: A prospective evaluation. J Consulting and Clin Psyc 1996;64(4):-757.

(84) LaPorte DJ, Stunkard AJ. Predicting attrition and adherence to a very low calorie diet: A prospective investigation of the eating inventory. Int J Obes 14(3), 1990:197-206.

(85) Wadden TA, Foster GD, Sarwer DB, Anderson DA, Gladis M, Sanderson RS, Letchak RV, Berkowitz RI, Phelan S. Dieting and the development of eating disorders in obese women: results of a randomized controlled trial. Am J Clin Nutr 80(3):560-8, 2004.

(86) Bjorvell B, Aly A, Langius A, Nordstrom G. Indicators of changes in weight and eating behaviour in severely obese patients treated in a nursing behavioural program. Int J Obes 18, 1994:521-5.

(87) Clark MM, Marcus BH, Pera V, Niaura RS. Changes in Eating Inventory scores following obesity treatment. Int J Eating Dis 1994;15(4):-405.

(88) Richman RM, Webster P, Salgo AR, Mira M, Steinbeck KS, Caterson ID. A shared care approach in obesity management: the general practitioner and a hospital based service. Int J Obes & Rel Metab Dis 1996;20(5):413-9.

(89) Fogelholm M, Kukkonen-Harjula K, Oja P. Eating control and physical activity as determinants of short-term weight maintenance after a very-low-calorie diet among obese women. Int J Obes & Rel Metab Dis 23(2):203-10, 1999.

(90) Cuntz U, Leibbrand R, Ehrig C, Shaw R, Fichter MM. Predictors of post-treatment weight reduction after in-patient behavioral therapy. Int J Obes & Rel Metab Dis 25 Suppl 1:S99-S101, 2001.

(91) Kiernan M, King AC, Stefanick ML, Killen JD. Men gain additional psychological benefits by adding exercise to a weight-loss program. Obes Res 9(12):770-7, 2001.

(92) Chaput J, Drapeau V, Hetherington M, Lemieux S, Provencher V, Tremblay A. Psychobiological impact of a progressive weight loss program in obese men. Physiol & Behav 2005;86(1-2):224-32.

(93) McGuire MT, Wing RR, Klem ML, Lang W, Hill JO. What predicts weight regain in a group of successful weight losers? J Consulting and Clin Psyc 1999;67(2):-185.

(94) Doucet E, St-Pierre S, Almeras N, Depres J-P, Bouchard C, Tremblay A. Evidence for the existence of adaptive thermogenesis during weight loss. Brit J Nutr 2001;85:715-23.

(95) Wadden T, Berkowitz R, Womble L, Sarwer D, Phelan S, Cato R, Hesson L, Osei S, Kaplan R, Stunkard A. Randomized trial of lifestyle modification and pharmacotherapy for obesity. New Eng J Med 2005;353(20):21111-20.

(96) Doucet E, Imbeult P, St-Pierre S, Almeras N, Maurege P, Depres J-P, Bouchard C, Tremblay A. Greater than predicted decrease in energy expenditure during exercise after body weight loss in obese men. Clin Sci 2003;105:89-95.

(97) Rosenbaum M, Goldsmith R, Bloomfield D, Magnano A, Weimer L, Heymsfield S, Gallagher D, Mayer L, Murphy E, Leibel R. Low-dose leptin reverses skeletal muscle, autonomic and neuroendocrine adaptations to maintenance of reduced weight. J Clin Investig 2005;115(2):3579-86.

(98) Verdich C, Toubro S, Buemann B, Holst JJ, Bulow J, Simonsen L, Sondergaard SB, Christensen NJ, Astrup A. Leptin Levels Are Associated with Fat Oxidation and Dietary-Induced Weight Loss in Obesity. Obes Res 2001 August 1;9(8):452-61.

(99) Guisado JA, Vaz FJ, Lopez-Ibor JJ, Jr., Rubio MA. Eating behavior in morbidly obese patients undergoing gastric surgery: differences between obese people with and without psychiatric disorders. Obes Surgery 11(5):576-80, 2001.

(100) Lang T, Hauser R, Buddeberg C, Klaghofer R. Impact of gastric banding on eating behavior and weight. Obes Surgery 12(1):100-7, 2002.

(101) Macias J, Leal F. Psychopathological differences between morbidly obese binge eaters and non-binge eaters after bariatric surgery. Eating and Weight Dis 2003;8(4):-318.

(102) Burgmer R, Grigutsch K, Zipfel S, Wolf AM, de Zwaan M, Husemann B, Albus C, Senf W, Herpertz S. The influence of eating behavior and eating pathology on weight loss after gastric restriction operations. Obes Surgery 15(5):684-91, 2005.

(103) Nickel C, Widermann C, Harms D, Leiberich PL, Tritt K, Kettler C, Lahmann C, Rother WK, Loew TH, Nickel MK. Patients With Extreme Obesity: Change In Mental Symptoms Three Years After Gastric Banding. Int J Psychiatry in Med 2005;35(2):-122.

(104) Hsu L, Betancourt S, Sullivan S. Eating disturbances before and after vertical banded gastroplasty: a pilot study. Int J Eating Dis 1996;19:23-34.

(105) Adami GF, Gandolfo P, Dapueto R, Jurich D. Eating behavior following biliopancreatic diversion for obesity: Study with a three-factor eating questionnaire. Int J Eating Dis 1993;14(1):-86.

(106) Karlsson J, Sjostrom L, Sullivan M. Swedish obese subjects (SOS)--an intervention study of obesity. Two-year follow-up of health-related quality of life (HRQL) and eating behavior after gastric surgery for severe obesity. International Int J Obes & Rel Metab Dis 22(2):113-26, 1998.

(107) Kalarchian MA, Wilson GT, Brolin RE, Bradley L. Effects of bariatric surgery on binge eating and related psychopathology. Eating & Weight Dis: 4(1):1-5, 1999 March.

(108) Lejeune MP, Hukshorn C, Saris WH, Westerterp-Plantenga MS. Effect of dietary restrain during and following pegylated recombinant leptin (PEG-OB) treatment of overweight men. Int J Obes 2003;27:1494-9.

(109) McElroy SL, Kotwal R, Hudson JI, Nelson EB, Keck PJr. Zonisamide in the Treatment of Binge-Eating Disorder: An Open-Label, Prospective Trial. J Clin Psychiatry 2004;65(1):-56.

(110) Hainer V, Kunesova M, Bellisle F, Hill M, Braunerova R, Wagenknecht M. Psychobehavioral and nutritional predictors of weight loss in obese women treated with sibutramine. 2004;29(2):208-16.

(111) Lejeune MP, Hukshorn C, Saris WH, Westerterp-Plantenga MS. Effect of dietary restraint during and following pegylated recombinant leptin (PEG-OB) treatment of overweight men. Int J Obes 2003;27:1494-9.

(112) Bryant, EJ (2006). Understanding Disinhibtion and its influences on eating behaviour and appetite. PhD thesis. University of Leeds, UK.

(113) Neel J. Diabetes mellitus: A "thrifty" genotype rendered detrimental by progress? Am J Hum Genetics 1962;14:353-62.

(114) Prentice A. Early influences on human energy regulation: Thrifty genotypes and thrifty phenotypes. Physiol & Behav 2005;86:640-5.

(115) Prentice A. New thoughts on the thrifty genotype hypothesis. Obes Rev 2006;7(Suppl. 2):21-2.

(116) Bouchard C. The biological predisposition to obesity: beyond the thrifty genotype scenario. Int J Obes 2007 March 13.

(117) Chakravarthy MV, Booth FW. Eating, exercise, and "thrifty" genotypes: connecting the dots toward an evolutionary understanding of modern chronic diseases. J Appl Physiol 2004;96(1):3-10.

(118) Hyland, M., Irvine, S., Thacker, C., Dann, P., & Dennis, I. Psychometric analysis of the Stunkard-Messick Eating Questionnaire (SMEQ) and comparison with the

Dutch Eating Behaviour Questionnaire (DEBQ). Current Psychological Research Review, 1989; 8, 228-233.

(119) Karlsson, J., Persson, L. O., Sjostrom, L., & 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, 24, 1715-1725.

(120) Royce, JR. Factors as theoretical constructs. Am. Psychologist, 1963; 18 (8), 522 – 528.