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Stress-Induced Release of Eating Related Hormones in Young Women Classified as Restrained Eaters

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Abstract

Eating related hormones may be strongly involved in the control of food intake under stressful conditions. The development and maintenance of eating disorders such as obesity, binge eating disorder, and bulimia nervosa might be in part explained by the altered release of insulin, ghrelin or leptin in particular in vulnerable subjects such as restrained eaters. The present investigation describes the secretion of these hormones before and after stress in a sample of 44 young women, who were selected according to a high degree of dietary restraint. Hormones were analysed from blood samples which were drawn 30 and 60 minutes after a validated stressor. Ghrelin levels significantly rise after stress induction. Insulin and Leptin both significantly fall after stress. The results are discussed with regard to biological as well as psychological causes and consequences of stress related hormone release in restrained eaters.

Keywords: Stress; Insulin; Ghrelin; Leptin; Restrained eating; Eating disorders

Introduction

The eating disorders bulimia nervosa and binge eating disorder show prevalence rates up to 5 % in community samples [1].

Patients with bulimia nervosa usually are in the normal weight range. The core symptoms of the disorder are bulimic attacks that mean the uncontrolled intake of a large amount of food (up to 10.000 calories) in a short period of time. This behaviour may take place several times a day. Most of the patients practice vomiting after their bulimic attacks, but the abuse of laxatives is also very common to compensate the excessive intake. Psychologically they are characterised by extreme concerns about weight and shape [2].

Binge eating disorder (BED) is an eating disorder characterised by the recurrent uncontrolled intake of large amounts of food. In contrast to bulimia nervosa, BED subjects do not practise compensatory behaviours such as vomiting or the use of laxatives after a binge episode. Most of these individuals with BED therefore are obese. Due to their abnormal eating behaviour both disorders show numerous biological abnormalities in endocrine and neurotransmitter systems.

For both disorders current aetiological models (overview in [3] are multifactorial, but postulate as a main vulnerability factor for the onset and maintenance repeated periods of dieting or the permanent practice of restrained eating. In particular, dieting has been linked to binge eating. This has been investigated in many laboratory studies, which have shown that overeating under stressful conditions is a stable characteristic of restrained eaters [4].

Whether stress also provokes short-term changes in hormones that regulate food intake is still unclear.

According to the reviews of [5] and [6] it can be concluded that peripheral release of Ghrelin is linked to the regulation of food intake in so far as it promotes food intake.

Fasting plasma levels of Ghrelin in patients with bulimia nervosa were significantly higher than those in controls [7]. Restrained eating was also significantly associated with higher Ghrelin levels [8] Acute psychological stress leads to a significant increase in Ghrelin levels [9].

The role of stress and dieting for insulin release has not been extensively studied in empirical investigations, but some data are available.

Unstimulated plasma Insulin levels were not different between restrained and unrestrained eaters [10].

Restrained eating was associated with lower fasting insulin plasma levels [11]. The authors did not draw any conclusions from their data with respect to the development or maintenance of eating disorders. Whether restrained eating is the cause or a consequence of the lower fasting insulin remained open. Furthermore, possible influences of stress were not investigated.

Dieting during a behavior weight loss program did not significantly change insulin release in obese subjects [12].

Insulin release was significantly lowered through calorie restriction in obese older adults [13].

This was also observed in young men after a 4 weeks dieting period in the context of Ramadan fasting [14].

A further study of [15] analysed changes in insulin and their association to weight regain after a low calorie diet. Insulin secretion and fasting insulin were significantly reduced at the end of the diet and inversely correlated to weight regain.

The relevance of metabolic control for the onset of disturbances in eating behaviour which may include binge eating is demonstrated by the prospective data of [16] in adolescent girls with Type I diabetes. An impaired metabolic control was a significant predictor for disturbances in eating behaviour after five years, although the girls did not fulfil diagnostic criteria for a clinically significant eating disorder.

4 studies reported a negative relationship between unstipulated leptin levels and cognitive restraint. The data can be interpreted that leptin signals a positive energy balance perpetuating maintenance or development of obesity, which in turn promotes the further practicing of restrained eating [17-20].

In subjects who are not characterized by habitually restrained eating leptin values increased after the TSST [21].

In older women, not characterized by high dietary restraint, a significant increase in leptin was observed after the TSST and was related to a lower intake of high fat, high sugar foods but not to other types of foods [22].

This suggests that leptin reactivity may be involved in reducing stress-induced eating behavior. It is speculated that leptin may be acting as a modulator of stress eating.

After one night of sleep deprivation leptin levels were significantly higher than before the stressor in healthy younger subjects [23].

The results of the available empirical studies on dietary restraint and its relationship to eating related hormones at present do not allow drawing clear conclusions. In particular, the role of stress has not been taken into account.

The present investigation therefore will address the question whether stress plays a role for changes in the release of eating related hormones such as insulin, ghrelin and leptin in restrained eaters.

Method

44 subjects were recruited by advertisement on the University campus. Weight had to be in the normal range (BMI 19-24). Exclusion criteria were clinical eating disorders in the past or at present and acute medical illness. With respect to these criteria, all subjects were checked by a medical doctor.

All participating subjects were selected as restrained eaters according to norm-tables of the German version of the Three Factor Eating Questionnaire (cognitive restraint scale) [25].

The mean age of the sample was 23,5 ± 2,6, the mean BMI was 21,9 ± 2,4.

Stressor was the Trier Social Stress Test (TSST [25]. The procedure combines a social stressor (public speech) with a mental stressor (arithmetics under time pressure), and is a validated tool to provoke psychobiological stress responses.

Blood sampling

All subjects were studied in the afternoon. Blood samples were drawn through an indwelling butterfly catheter in a forearm vein. Blood samples for insulin, ghrelin, and leptin were collected at time 0 (before stress) and 30 and 60 minutes after stress induction. The blood was sampled in EDTA treated tubes and immediately centrifuged to yield plasma for hormone determinations.

Body composition was estimated by bioelectrical impedance analysis [26]. Percentage of body fat was calculated.

Manipulation check

Success of stress induction was tested by using subjective ratings, provided by visual analogue scales ranging from 0 (no stress) to 10 (maximum stress). The ratings were obtained before stress induction and 15 minutes after stress induction and analysed by MANOVA for repeated measurement. There was a significant effect of stress: $F(1,44) = 62,3$, $p<.001$,

Results

Before Stress	30 min after stress	60 min after stress	Significance paired t-test 2-tailed
26,5 ± 22,1	21,8 ± 18,2	21,8 ± 21,9	.02

Values show µU/ml

Table 1: Mean insulin before stress and 30 min and 60 min after stress

The mean insulin level decreased significantly after the stressor (Table 1).

Before Stress	30 min after stress	60 min after stress	Significance paired t-test 2-tailed
860,7 ± 381,6	904,7 ± 411,8	921,7 ± 439,0	.01

Values show pg/ml

Table 2: Mean ghrelin before stress and 30 min after stress

A significant rise of ghrelin was observed after the stressor (Table 2).

Before Stress	30 min after stress	60 min after stress	Significance paired t-test 2-tailed
11,2 ± 7,7	9,9 ± 4,9	10,6 ± 6,7	.02

Values show ng/ml

Table 3: Mean leptin before stress and 30 min and 60 min after stress

Mean leptin was significantly lower 1 hour after the stressor (Table 3).

Correlation between baseline leptin and body fat (%) was .52 ($p<.001$). The correlation between post stress Leptin (60min) and body fat was .52 ($p<.001$).

Discussion

Our data show, that ghrelin levels significantly rise after stress induction. Insulin and leptin both significantly fall after stress.

Two explanations of our results for ghrelin may be considered. The increased stress induced Ghrelin release may be partly a cause of the restrained eating behaviour. High ghrelin secretion in stressful situations leads to increased food intake which in turn promotes weight gain with the danger of becoming overweight in the long-term. To prevent this, chronic or repeated dieting behaviour has to be practiced.

In obese women a stress-related elevation of ghrelin has been found [9,27]. The consequence may be a biologically determined higher motivation for food intake in stress situations which promotes further weight gain and maintenance of obesity. Therefore obese women often practice repeated dieting or permanent restrained eating which is reflected in high scores on scales measuring cognitive restraint [28].

The other interpretation is that the increased ghrelin release is a consequence of the frequent dieting behaviour, seen in people classified as restrained eaters. Some support comes from a study of [8] who reported a significant correlation between degree of restraint and circulating ghrelin in normal weight subjects. The data of [29] are also in agreement with this hypothesis. Obese women had significantly elevated ghrelin levels after a dieting period.

For leptin, the results of our study are not in accordance with the data from [30], who found a significant increase in leptin after an acute stressor. However, the stress was not a psychosocial but a physical stressor. A stress induced increase of leptin was also observed in [31], whereas support for our results comes from [32], who also found a stress-related decrease in leptin.

Brydon *et al.*, [33] found an increase in leptin after a mental stressor. As in our study they also showed a positive correlation between stress related leptin and body fat. This was interpreted for a possible role of stress related leptin for generating more body fat resources and in so far promoting central adiposity in women. If leptin falls after stress, as demonstrated in our data, this would then signal that not enough fat is stored and on a behavioral level the person might become subsequently vulnerable to overeat and develop binge eating disorder or in other cases bulimia nervosa. Empirical support for this interpretation comes from the study of [34], where lower leptin levels were significantly correlated with higher bulimic tendencies in patients with anorexia nervosa and obesity. A further study from [35] found a significant association of low leptin levels with the eating disorder bulimia nervosa.

Post stress insulin was lower compared to baseline. Three days of dieting also lead to lower insulin in women being in a normal weight range [11] and can be interpreted to act comparable to a stressor in a longer time period. A subsequent rise of insulin may be a consequence so that glucose is transported to muscles, which need energy, but blood glucose must also be restored by the intake of sugar containing nutrients, which in turn make a person vulnerable for obesity [36-41].

Conclusion

Although there are limitations of this study such as a small sample size, a specific age range, and the investigation of only women, the results support a significant involvement of eating related hormones in the stress dependent development and maintenance of disordered eating behaviour.

Ethical Approval

Compliance to ethical standards: The study was approved by the ethics committee of the University of Trier. All subjects were paid for participation.

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