Children’s psychosocial stress and emotional eating: a role for leptin?

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Abstract

Objective Psychosocial stress can be a health threat by stimulating unhealthier eating behaviours. We aim to test the role of the hormone leptin in the association between stress and diet/emotional eating as detected in primary school children.

Method In a two-wave longitudinal study with 308 Belgian children (5-12y) in 2010-2012, the association of fasting serum leptin with reported stress (negative events and emotional problems), measured stress by salivary cortisol (overall cortisol output and awakening response), emotional eating and food consumption frequency was examined. Analyses were split by sex. Mediation and moderation by leptin change were tested.

Results One stress marker (overall cortisol output) was significantly correlated with high leptin levels, but only in girls and cross-sectionally. Only in boys, leptin was associated with low emotional eating. Leptin was not a significant predictor of unhealthy food consumption. Leptin change was not a mediator but an enhancing moderator in the link between stress (high cortisol output and emotional problems) and emotional eating in girls: high reports of emotional eating in 2012 were present in the case of combined high 2-year leptin increase and high stress at baseline.

Discussion Stress (represented by emotional problems and high daily cortisol) seems to lead to hyperleptinemia in girls; and the combination of high stress and hyperleptinemia might make girls more vulnerable to stress-induced eating. No functional data on leptin sensitivity were present, but results might suggest that stress induces lower sensitivity to the anorexigenic leptin activity.
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Chronic psychosocial stress is an important public health threat since in literature it has regularly been associated with a higher energy intake likely due to emotional or reward-based eating of comfort food rich in sugar and fat (1-3) and consequently with obesity (4). Also at young age these stress associations have been found e.g. in our primary school children cohort (5, 6). Nevertheless, stress-induced eating has not been detected in all people (depending on personality) and all situations (depending on type of stressor/emotion) (2).

Insight in biological pathways underlying these findings is pivotal. The stress-diet mechanisms are only starting to be elucidated in humans. Changes in leptin levels might be a relevant explanation for this stress-induced eating since leptin is an anorexigenic hormone that decreases appetite and reward-related eating behaviours (7, 8). Stress-induced cortisol elevations might increase leptin levels, possibly creating leptin resistance (i.e. less sensitivity) with increased comfort food intake as a result (3, 9). In several studies, psychosocial stress has been associated with higher leptin levels e.g. for self-reported stress (10), depression (11, 12), trauma (13) and peer/emotional problems (14), as was the case for chronically-elevated stress hormone cortisol levels (15). Nevertheless, sex-differences (11, 14, 16, 17) and inconsistent results (18) or non-significant studies (19-21) have also been published. In experimental studies, cortisol induced leptin resistance (22). Leptin resistance can be due to down-regulation of the cellular response to leptin and/or decreased transport through the blood-brain barrier and it is reflected by high leptin levels (23, 24). Finally, this leptin resistance stimulates food intake and hunger and as a result also overweight (23).

Several limitations exist in this research field: (a) a focus on only adult populations; (b) a limited use of stress biomarkers like cortisol patterns reflecting long-term stress exposure; (c) a lack of adjustment for relevant confounders like body fat%; and most important (d) the use of bivariate analyses on stress-leptin associations without considering their association, interaction or mediation in emotional eating. Therefore, we aimed to reveal leptin’s role in the link that we have previously reported between children’s stress and increased emotional eating. The longitudinal association
between stress, leptin and emotional eating was tested using both subjective and objective measures of stress, while also testing for mediation or moderation. We hypothesized (a) that stress reports and cortisol at baseline are positively associated with leptin at baseline and leptin change; (b) that leptin levels are associated with emotional eating and unhealthier food consumption; and (c) that leptin change is a mediator in the link between stress at baseline and emotional eating or food consumption at follow-up; or as an alternative that leptin change is a moderator in that link.

**METHODS**

**Design**

Participants were Belgian children recruited for the longitudinal ChiBS study (year 2010 and 2012). Children were between 5 and 10 years old at baseline and between 7 and 12 years old at follow-up. More details on the ChiBS study and its measurements have been described elsewhere (25). In 2010, 308 children (48.7% boys) had information on all variables for the current analyses: leptin, stress, cortisol, food consumption and body fat%. None of children received chronic psychotherapy or had Cushing or Addison disease but 5 children using chronic corticosteroids and 3 using psychostimulantia were excluded. In 2012, 174 of these children (45.4% boys) also had information on all variables: leptin, stress, emotional eating and food consumption. Children with follow-up data in 2012 were not different compared to those with only data at baseline in 2010 on stress reports, salivary cortisol levels, leptin levels (median 1286pg/ml vs 1241pg/ml) and food consumption. When testing the same for boys and girls separately, boys with follow-up had lower leptin than those without follow-up (median 1079pg/ml vs 978pg/ml), while this was not the case for girls (median 1577pg/ml vs 1591 pg/ml).

On the examination day, fasting blood withdrawal was executed (for leptin level determination) and the parent and child had to fill in questionnaires on stress, food consumption and emotional eating. Children had to collect salivary samples to measure salivary cortisol as stress biomarker; to prevent any effect of the blood sampling as stressor, salivary sample was not performed on the examination day itself, but in the two days following the examination in 2010. The study was
conducted according to the guidelines laid down in the Declaration of Helsinki and the project protocol was approved by the Ethics Committee of the Ghent University Hospital. A written informed consent was obtained from the parents and a verbal assent from the children.

**Fasting leptin (2010 and 2012)**

Leptin was measured using a venous blood sample after overnight fasting (sampling between 8 and 10 AM). The venipuncture was done at the level of the antecubital vein in the left arm. Serum tubes were stored at room temperature for 30 minutes to allow clotting. Processing of the blood samples was done within four hours after collection. All blood samples were centrifuged at 2500g for ten minutes and were stored at -80°C until further analyses of the extracted serum. Leptin serum concentrations were measured in 2010 using a Meso Scale Discovery sandwich electrochemiluminescence immunoassay (inter-assay CV 2.4% for low, 3.9% for high controls; intra-assay CV 2.7% for low, 5.1% for high controls) and in 2012 using a Millipore radioimmunoassay in a certified laboratory (inter-assay CV 3.0% for low, 6.2% for high controls; intra-assay CV 3.4% for low, 8.3% for high controls). Hyperleptinemia was not used as a categorical variable since no clinical cut-offs exist to define hyperleptinemia in children. Just for descriptive purposes, we used recently published sex- and age-specific serum leptin reference values for non-obese European children (26). When using the published 90th percentile as cut-off, 21 girls and 17 boys had hyperleptinemia. When using the published 97th percentile as cut-off, 18 girls and 10 boys had hyperleptinemia.

**Reported stress (2010 and 2012)**

Stress arises when the demands of a situation exceed an individual’s ability to cope and resolve the problematic persistent situation, resulting in emotional and behavioural disturbances (27). Because of this adaptive and dynamic state, stress is for research purposes not only operationalized on the event level (i.e. the stressor) but also on the symptom level by measuring emotions. Perceived stress was not directly asked to the children since our experience has taught us that is the word ‘stress’ is too difficult to understand for this age-group. Children were individually interviewed by a trained researcher on their negative events and parents filled in a questionnaire on children’s emotions.
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**Negative events as stressor exposure**

The Coddington Life Events Scale for children (CLES-C) is a validated and well-established 36-item questionnaire (test-retest $r=0.69$, parent-child agreement $ICC=0.45$ in 12-14y olds) (28). It assesses the prevalence, frequency and timing of stressful life events relevant for this age group during the last year filled in by the child. By measuring significant life events in terms of Life Change Units depending on timing, frequency and severity, the CLES-C can provide insight into recent events that may affect the child’s health. For the current analyses, only negative events were considered.

**Emotional problems as stress symptom**

Parents were asked to complete the standardized ‘Strengths and Difficulties Questionnaire’ (SDQ) (29) (Cronbach’s alpha=0.53-0.76, test-retest stability $r=0.88$, concurrent validity $r=0.7-0.87$). The subscale on emotional problems over the past 6 months (proxy-report) with 5 items was used.

**Salivary cortisol as biological stress reactivity (2010)**

Salivary cortisol was analysed since stress reports are not always associated with chronically changed levels of stress hormones. Previously, our group found significant associations of these children’s cortisol with negative events, emotional problems and peer problems, mainly supporting a chronic hypercortisolism due to stress (30).

Saliva was collected at home via Salivette synthetic swabs (Sarstedt, Germany) immediately after wake up (T0), 30 minutes after wake up (T30), 60 minutes after wake up (T60) and in the evening (Tev). Participants were given detailed instructions and restrictions to avoid any contamination (25): the children were asked to sample when healthy and on a normal day; to strictly respect the time points; not to eat, drink or brush their teeth in the hour before collection; to avoid physical activity two hours before sampling; and to avoid caffeine-rich drinks and minimize medication on the sampling days. Morning samples collected more than 5 minutes different from the requested time point and evening samples not collected between 7 and 9 PM were excluded. Moreover, samples of 5 children using corticosteroids were excluded. Laboratory analyses were done with a competitive electrochemiluminescence immunoassay (Roche Diagnostics,
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Mannheim, Germany; measuring range 0.49-1748.95 nmol/L; inter-assay CV=3.9%; intra-assay CV=1.9%). Summary variables have been calculated to represent chronic stress by two independent cortisol patterns over time: the cortisol awakening response and the diurnal pattern (31). To represent the cortisol awakening response, the area under the curve with respect to the ground (AUCg) was calculated between the T0 and T60 sample. The diurnal pattern was investigated using the total cortisol output (AUCg over the whole day).

**Emotional eating (2012)**

Children filled in the Dutch Eating Behaviour Questionnaire (DEBQ) on their usual eating behaviour (32). The 13 items of the ‘emotional eating’ subscale were used i.e. eating in response to negative emotions. Answer possibilities ranged between 1/never and 5/very often. The mean of all scale items was used. The subscales revealed a stable factor structure, satisfying internal consistency (Cronbach’s alpha=0.77-0.91; even for the youngest age group) and good test–retest reliability (r=0.87-0.90).

**Food Frequency Questionnaire for food consumption (2010 and 2012)**

Parents reported on their child’s food consumption during the last 4 weeks by completing a food frequency questionnaire. Test-retest reproducibility testing resulted in r=0.32-0.76 for separate items. To identify healthy and unhealthy patterns in food consumption, four indices were computed by summing up the consumption frequency of separate food items. A food index for (1) ‘sweet foods’ (i.e. sweet drinks, jam, honey, sweet breakfast cereals, sweet snacks), (2) ‘fatty foods’ (i.e. fried potatoes, chocolate- or nut-based spreads, high fat dairy, mayonnaise and mayonnaise-based products, fat meat preparations, butter, high fat snacks), (3) ‘snacks’ (including sweet and/or fat snacks), and (4) a healthy food index for ‘fruit and vegetables’ (i.e. fruit, freshly squeezed fruit juice, vegetables) was calculated. Multivariate techniques to gain an overall diet pattern have not been implemented since this revealed rather incoherent patterns with our questionnaire which are not of interest in our stress-diet hypothesis. After all, the idea is that stress mainly stimulates the consumption of certain food
items, so the identification of specific subtypes (i.e. the sweet and fatty foods; in-between meal snacks) in our current approach is a benefit.

**Background variables**

Sex, age, pubertal status, parental education level and body fat% were considered as potential confounding factors, based on previous research (33). The children’s sex and birth date were reported by the parent. To represent socio-economic status, parental education level was assessed by questionnaire according to the International Standard Classification of Education. Pubertal status was assessed in 2012 using Tanner classification based on staging of pubic hair distribution and genital development for boys and pubic hair distribution and breast development for girls. Due to the limited percentage of children in the different Tanner stages higher than stage I, these data were recoded into two groups (‘no sign of puberty’ versus ‘signs of puberty’). Body fat% was reliably measured using air-displacement plethysmography (BOD POD®) in both 2010 and 2012. Children were measured twice in tight-fitting bathing suit with swim cap to rule out air trapped in clothes or hair and child-specific conversion factors were applied.

**Statistics**

Statistical analyses were performed using SPSS 22 (SPSS Inc, Chicago, IL) and all p-values <0.05 were considered significant. In the descriptive data, sex differences were tested and the success rate difference was calculated as effect size based on the Mann Whitney U-test as follows: \(2U/(n*m)-1\) with \(n\) and \(m\) being the sample size in boys and girls, respectively.

Hypothesis testing was first done by zero-order Spearman correlations. Second, regression analyses were implemented. Standardized regression coefficients were presented. Since sex interaction terms (i.e. sex*X-variable) were often significant in our sample, analyses were split by sex. To test whether age, pubertal status and body fat% are confounders, associations with the parameters of interest (stress, leptin and diet) were tested. Leptin was highly correlated with body fat% (\(\rho=0.345\) in boys, 0.600 in girls) and to a lower extent with age (\(\rho=0.179\)) but not with pubertal status. Body fat%, age and pubertal status were not associated with stress or diet parameters. As a result, these
parameters where not included in the regression. Nevertheless, results remained the same with or without their inclusion in the regression.

For the first hypothesis, stress (2010; cortisol and reports) was tested as predictor of leptin (2010) and leptin change (2012-2010).

For the second hypothesis, leptin was tested as predictor of emotional eating and food consumption. This was done for leptin (2010) with food consumption (2010) and food consumption change (2012-2010). Since emotional eating was only available in 2012, only the cross-sectional association between leptin 2012 (x-variable) and emotional eating 2012 (y-variable) was tested.

For the third hypothesis, mediation and moderation by leptin in the association of stress reports with food indices and emotional eating were tested longitudinally (34). Mediation is defined as a variable carrying the influence of a predictor to a given outcome (an intermediate pathway), and thus accounting for the observed relationship. For this, the tested mediator should be related to predictor and outcome; and the effect of predictor on outcome should be significantly reduced after adjusting for the tested mediator. A moderator is a third variable affecting the direction and/or strength of the relationship between a predictor and outcome variable. For this, the interaction term between the predictor (baseline) and the tested moderator (change over time) was added into the regression, next to the raw effect of predictor and moderator separately. Visual representation was done by plotting predicted outcome values for moderator tertiles.

RESULTS

Descriptive characteristics

Descriptive values of some background variables, the leptin levels, stress reports, salivary cortisol patterns, emotional eating and food indices can be found in Table 1. Overweight prevalence was 6% at baseline, while 7% at follow-up. Some sex differences were detected with girls having higher leptin, body fat% and stress reports but less sweet food consumption. Zero-order correlations can be found in Table 2.

Associations of leptin with stress
Associations of cortisol (2010) and stress reports (2010) with fasting leptin (2010) and change in leptin (2012-2010) are also shown in Table 2. The only significant finding was in girls i.e. a significant positive association between total cortisol output (2010) and leptin (2010) ($\rho=0.175$).

**Associations of leptin with emotional eating and food consumption**

As can be seen in Table 2, leptin levels (2012) were cross-sectionally associated with less emotional eating (2012) in boys ($\rho=-0.278$) but not in girls ($\rho=-0.067$). As shown in supplementary material, no associations were detected between leptin (2010) and the four food indices (2010 or change 2012-2010).

**Mediation by leptin change**

Mediation by leptin change in the association of stress (2010) with food consumption (2012) or emotional eating (2012) was not present since stress (2010) was not associated with leptin change, in neither girls nor boys.

**Moderation by leptin change**

Leptin change was a significant moderator in the association between 2 stress parameters (total cortisol output and emotional problems 2010) and emotional eating (2012). Figure 1 depicts the interaction effect in boys ($\beta=0.133; p=0.751$) and girls ($\beta=0.329; p=0.029$) between total cortisol and leptin in their effect on emotional eating. The same was found for emotional problems: a significant stress-leptin interaction in girls ($\beta=0.291; p=0.029$), but not in boys ($\beta=0.096; p=0.370$). In girls, leptin had opposite patterns in stressed compared to non-stressed children, with only a positive stress – emotional eating relation in those with high leptin increases between 2010 and 2012. When stress/cortisol was low, emotional eating was highest in those with low leptin. When stress/cortisol was high, emotional eating was highest in those with high leptin. In boys, the stress – emotional eating relation was not significant and did not depend on the leptin change value.

**DISCUSSION**
In a group of Belgian primary school children, we aimed to examine the role of leptin in the stress-diet association. We hypothesized (a) that stress reports and cortisol are positively associated with leptin; (b) that high leptin levels are associated with emotional eating and unhealthier food consumption; and (c) that leptin change is a mediator in the longitudinal link between stress at baseline and emotional eating or food consumption at follow-up, or alternatively a moderator when leptin change is not associated with stress variables at baseline. The first hypothesis was only confirmed in girls and for cortisol as stress marker: higher daily cortisol output was associated with higher leptin levels. For the second hypothesis: leptin was associated with low emotional eating in boys but leptin was not related to an unhealthier food consumption. The third hypothesis was partially confirmed: leptin was not a mediator but an enhancing moderator in the link between stress (i.e. emotional problems and high daily cortisol output) and emotional eating in girls since leptin was a vulnerability factor in stress-eating. Overall, our results indicated that only in girls leptin might play a harmful role in the stress-diet association.

**Leptin and stress**

In an increasing amount of studies, psychosocial stress has been associated with higher leptin levels in adults (10-13) and in children only for peer/emotional problems (14). Nevertheless, depression has also been associated with lower leptin levels (18, 35) and some studies found no significant associations with adult depression (36) or child trauma (19). Biological markers of stress (i.e. cortisol) will increase our understanding of the physiological mechanism underlying the stress-diet relation. In lab-studies, administered cortisol increased leptin levels in both adults (17) and children (37). In epidemiological settings, the cortisol-leptin relation remains largely unexplored as subjective stress measurements have commonly been applied in this type of stress research. Serum cortisol has been associated with higher leptin levels in adolescent girls (16) but not in obese children/adolescents (20). In our study, significances were only found in girls: leptin levels were higher with higher daily cortisol output. Indeed, sex-differences have previously been mentioned in the associations (11, 14, 16, 17) with significant results in women only. Moreover, sex differences
have been revealed in leptin levels, due to testosterone-induced leptin downregulation (38) but also in prepubertal child populations (39). Sex differences are also visible in changes during follow-up with again more pronounced increases for girls, in agreement with recently published reference curves (26).

**Leptin and emotional eating**

In testing the second hypothesis, leptin was associated with less emotional eating in boys. This is in agreement with the general knowledge of leptin’s physiology as an anorexigenic and reward-lowering adipokine (7, 8). Also, leptin and leptin receptor polymorphisms have been found in emotional eaters (40). In girls, this association was probably not detected due to the stress-associated leptin increases and the hypothesized resulting leptin sensitivity drop. Indeed, the stress hormone cortisol might induce leptin resistance (22). Previous studies have not yet tested the trait ‘emotional eating’ with leptin levels but in children with loss-of-control eating, only girls had higher leptin levels, probably reflecting leptin resistance (41). In an experimental setting, high leptin levels have been associated with less stress-induced snacking during a laboratory stress test but not with general snack intake (42), although another study could not reveal effects of fasting leptin on stress-induced eating during laboratory stress.

**Leptin and food consumption**

Since mice studies indicated leptin as a stimulator for increased taste nerve responses to the sweet taste (43) and since emotional eating is mostly focused on comfort food, we also expected effects of leptin on food indices. Yet, no such significant associations were found in our study. As our group has previously demonstrated (6), child-reported emotional eating could not be linked with unhealthier food consumption because the used DEBQ questionnaire focused on intentions but intentions will not always be carried out because of parental supervision. As a result, leptin effects in children would be mainly expected on intentions and not necessarily on actual food consumption.
Mediation and moderation

Testing the final hypothesis, leptin was not a mediator but an enhancing moderator in the effects of stress (i.e. emotional problems and high daily cortisol output) on emotional eating in girls. Leptin showed opposite patterns in stressed versus non-stressed girls. When stress was low, emotional eating was highest in those with low leptin. When stress was high, emotional eating was highest in those with high leptin. When combined with the findings of the first hypothesis and with literature (3, 22), this reflects the idea that stress in girls increases leptin levels, possibly creating decreased leptin sensitivity with increased emotional eating intake as a result since these stressed girls are not sensitive anymore to the appetite-lowering effect of high leptin levels (3, 9). This moderating effect of leptin has not yet been tested directly in other observational studies but findings are in agreement with literature hypotheses and lab-based findings (3, 22). In boys, the physiological action of leptin seemed intact as emotional eating was highest in those with low leptin. A possible explanation why girls appeared the most vulnerable, is that their sex-hormones and their higher body fat% are stimulating factors for leptin and leptin resistance (33, 39). Further research on the role of leptin should certainly be stimulated as some studies suggested that also the stress-reactivity of leptin should be considered e.g. only women that had stress-induced leptin elevations ate less unhealthy foods during stress (44). It should be mentioned that only emotional problems, and not the negative events, were significantly associated with emotional eating after moderation. This might be due to an inherent difference between potential stressful events and what happens under the skin e.g. because of personal differences in the experience of events as stressful.

Strengths and limitations

Compared to existing research, our study had several strengths. First of all, we used salivary cortisol as a biological marker of stress to verify one of the hypotheses. In comparing leptin with cortisol, we were the first to test salivary cortisol patterns by using multiple sampling. As such, we were more able to represent chronic stress compared to other studies with only single cortisol salivary or serum measures. Interestingly, only the total cortisol output seems to matter, not the morning
awakening response. Secondly, sex differences were tested in the associations of leptin with stress and emotional eating as some literature already suggested these differences. Thirdly, longitudinal mediation and moderation analyses on the stress-leptin-eating associations were realizable by our follow-up design.

Of course, our study is not without limitations. It should be mentioned that this is a rather an explorative, hypothesis-generating study. We hypothesized that the high leptin levels might reflect a lower leptin sensitivity based on the findings and on literature, but no formal test on sensitivity was performed in our study (not possible in observational human studies). Although the stress reports were sampled longitudinally, the cortisol and emotional eating variables were only collected cross-sectionally. This fact limits conclusions on the cortisol-leptin directionality e.g. leptin might decrease cortisol (45) and leptin can act as an anti-depressant (18), or a triglyceride-rich diet might induce leptin resistance due to less blood-brain barrier leptin transport (46). In any way, observational research is not able to state causal relations. In the longitudinal analyses, we have the limitation that the leptin analysis kit was different in 2010 and 2012 but a previous study showed that different leptin analysis kits yielded almost indistinguishable concentrations (47). Another limitation is the at-home collection of saliva. We only used a subjective measure of time compliance in the salivary cortisol sampling, therefore we stressed the importance of timing in a manual and the implemented exclusion of self-reported non-compliers can already improve the accuracy (48). Nevertheless, it is probable that non-compliant people are the most likely to report their timing incorrectly, which could lead to missing a part of the morning increase. Finally, recent research focused on the dynamics of leptin i.e. the stress-reactivity of leptin (44) and leptin-cortisol diurnal antagonism (49). In the future, this type of research may add more insight in the underlying physiology.
CONCLUSION

Our results suggest that leptin plays a modest adverse role in the association of stress with emotional eating but only in girls. Psychosocial stress, as represented by high daily cortisol output, was cross-sectionally related to increased leptin levels in girls. In addition, leptin was an enhancing moderator in the longitudinal association between stress (emotional problems and high daily cortisol output) and emotional eating in girls; the interaction showed that high leptin levels make stressed girls more vulnerable to emotional eating. In boys, leptin was consistently associated with less emotional eating.

To explain this newly found moderation in our explorative study, we speculate, based on existing literature, that stress-induced leptin increase is accompanied by decreased leptin sensitivity since the ability of leptin to reduce reward-related feeding appears to be lost in stressed girls. An important message is that stress-induced eating can be due to physiological changes (e.g. leptin activity) and not only due to personal weaknesses. Further insight in these physiological pathways by directly testing leptin sensitivity and certainly verification in other population samples might help in pharmaceutical and behavioural control of stress-related emotional eating. In the meantime, it seems pivotal to minimize stress exposure and learn children adaptive coping skills to decrease perceived stress levels and as such to prevent stress-induced leptin changes.
References


Table 1: Descriptive statistics of the population

<table>
<thead>
<tr>
<th></th>
<th>Year 2010 (5-10 years old) (N=308)</th>
<th>Year 2012 (7-12 years old) (N=174)</th>
<th>Change (2012-2010)</th>
<th>Paired t-test</th>
<th>Sex Differences SRD (p-value)</th>
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<tr>
<td></td>
<td>Median [IQR]</td>
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<td>Sex Differences SRD (p-value)</td>
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<td><strong>Descriptive statistics</strong></td>
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<td>Body fat%</td>
<td>Boys (N=150) 17.8[14.5;20.6]</td>
<td>Girls (N=158) 20.9[18.0;27.3]</td>
<td>-0.41(&lt;0.001)</td>
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<td>Age</td>
<td>Boys (N=79) 8.4[7.4;9.4]</td>
<td>Girls (N=95) 8.4[7.6;9.2]</td>
<td>-0.02(0.755)</td>
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<td>Parental educational level</td>
<td>Boys (N=79) 78.2%</td>
<td>Girls (N=95) 67.4%</td>
<td>-0.07(0.211)</td>
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<td>Pubertal stage</td>
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<td>Leptin (pg/ml)</td>
<td>Boys (N=79) 16.6[13.1;20.7]</td>
<td>Girls (N=95) 23.3[17.5;27.8]</td>
<td>-0.46(&lt;0.001)</td>
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<td>0.556 0.08 (0.658)</td>
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<td>Salivary cortisol (nmol/l)</td>
<td>Boys (N=79) 30%</td>
<td>Girls (N=95) 35%</td>
<td>-0.03(0.679)</td>
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<td>Total cortisol output (*1000)</td>
<td>Boys (N=79) 311[257;404]</td>
<td>Girls (N=95) 335[258;420]</td>
<td>-0.09(0.350)</td>
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<td>AUCg morning</td>
<td>Boys (N=79) 22[18;27]</td>
<td>Girls (N=95) 22[18;28]</td>
<td>-0.02(0.979)</td>
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<td>Stress reports</td>
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<td>Negative event score (0-2282)</td>
<td>Boys (N=79) 32[0;69]</td>
<td>Girls (N=95) 46[10;78]</td>
<td>-0.13(0.011)</td>
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<td>Emotional problems (0-10)</td>
<td>Boys (N=79) 2[1;4]</td>
<td>Girls (N=95) 2[1;5]</td>
<td>-0.03(0.027)</td>
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<td>Emotional eating (1-5)</td>
<td>Boys (N=79) 1.62[1.08;2.15]</td>
<td>Girls (N=95) 1.62[1.23;2.23]</td>
<td>-0.04(0.427)</td>
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<td>Food consumption</td>
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<tr>
<td>Sweet food (times/week)</td>
<td>Boys (N=79) 31[24;40]</td>
<td>Girls (N=95) 29[19;37]</td>
<td>-0.16(0.037)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fatty food (times/week)</td>
<td>Boys (N=79) 26[20;35]</td>
<td>Girls (N=95) 25[19;33]</td>
<td>-0.09(0.498)</td>
<td></td>
<td></td>
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<tr>
<td>Snacks (times/week)</td>
<td>Boys (N=79) 9[6;14]</td>
<td>Girls (N=95) 9[6;13]</td>
<td>-0.01(0.599)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fruit &amp; vegetables</td>
<td>Boys (N=79) 16[11;18]</td>
<td>Girls (N=95) 14[11;19]</td>
<td>-0.01(0.532)</td>
<td></td>
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</tbody>
</table>

IQR= interquartile range; AUCg= area under the curve with respect to the ground; SRD=success rate difference based on the Mann-Whitney U statistic. Paired t-test show whether there was a significant within-individual change between 2010 and 2012.
Table 2: Zero-order Spearman correlations for boys (upper panel) and girls (lower panel) separately

<table>
<thead>
<tr>
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<tbody>
<tr>
<td>Leptin (2010)</td>
<td>rho</td>
<td>.220</td>
<td>-.294*</td>
<td>-.019</td>
<td>.103</td>
<td>.036</td>
<td>.086</td>
<td>.039</td>
</tr>
<tr>
<td>Leptin (2012)</td>
<td>rho</td>
<td>.455***</td>
<td>.783***</td>
<td>.063</td>
<td>.068</td>
<td>.086</td>
<td>.117</td>
<td>-.278*</td>
</tr>
<tr>
<td>Leptin (2012-2010)</td>
<td>rho</td>
<td>-.321**</td>
<td>.563***</td>
<td>.036</td>
<td>.040</td>
<td>.072</td>
<td>-.088</td>
<td>-.284*</td>
</tr>
<tr>
<td>AUCg morning (2010)</td>
<td>rho</td>
<td>.070</td>
<td>.014</td>
<td>-.112</td>
<td>.631***</td>
<td>.083</td>
<td>.038</td>
<td>-.249*</td>
</tr>
<tr>
<td>Total cortisol output (2010)</td>
<td>rho</td>
<td>.175**</td>
<td>.006</td>
<td>-.117</td>
<td>.654***</td>
<td>.074</td>
<td>.025</td>
<td>-.208</td>
</tr>
<tr>
<td>Negative events (2010)</td>
<td>rho</td>
<td>-.059</td>
<td>.058</td>
<td>-.091</td>
<td>.062</td>
<td>.078</td>
<td>.142</td>
<td>-.136</td>
</tr>
<tr>
<td>Emotional problems (2010)</td>
<td>rho</td>
<td>-.069</td>
<td>.050</td>
<td>.131</td>
<td>-.068</td>
<td>-.029</td>
<td>.184*</td>
<td>.094</td>
</tr>
<tr>
<td>Emotional eating (2012)</td>
<td>rho</td>
<td>-.034</td>
<td>-.067</td>
<td>-.026</td>
<td>-.100</td>
<td>-.171</td>
<td>-.130</td>
<td>.046</td>
</tr>
</tbody>
</table>

*p<0.05; **p<0.01, ***p<0.001
FIGURE LEGENDS

Figure 1: Interaction effect between leptin increase (2012-2010) and cortisol (2010) on emotional eating (2012)

Moderation by leptin change was tested by including an interaction term between the stress variable (2010) and the continuous leptin change variable. Visual representation was done by plotting predicted outcome values for moderator tertiles.
ACKNOWLEDGEMENTS

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