The Cortisol Stress Response in Youth with Overweight and Obesity: Influence of Psychosocial Variables

Ine Verbiest[a*], Sandra Verbeken[a], Taaike Debeuf[a], Stefaan De Henauw[b], Nathalie Michels[a], Caroline Braet[a]

[a] Department of Developmental, Personality and Social Psychology, Ghent University, H. Dunantlaan 2, 9000 Ghent, Belgium

[b] Department of Public Health and Primary Care, Faculty of Medicine and Health Sciences, Ghent University, C. Heymanslaan 10, 9000 Ghent, Belgium

[*] corresponding author: Ine Verbiest, Henri Dunantlaan 2, B-9000 Ghent, Belgium. Tel.: +329 264 94 27. E-mail: Ine.Verbiest@UGent.be

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Abstract

Background

Despite previous research pointing out a bifurcation in cortisol stress reactivity, it is not yet clear if all variables explaining inter-individual differences in stress responses are captured.

Objectives

To explore which (psychosocial and demographic) variables predict the cortisol response after a standardized stress- and affective state (SAS)-induction in youth with overweight and obesity.

Methods

As part of a randomized control trial (SRCTN83822934) investigating the effects of an emotion regulation (ER)-training on top of a ten-month inpatient multidisciplinary obesity treatment, 79 children and adolescents (9-15 years) with moderate obesity (*M* adjusted BMI=154.35%, *SD*=24.57) completed a SAS-induction before leaving the clinic.

Results

Those whose cortisol levels decreased (*N*=59.5%) from baseline to reactivity showed higher levels of alexithymia than increasers (p=.049). Attachment avoidance was a significant positive predictor of relative cortisol decrease after SAS-induction (p=.001). Age was significantly related to less cortisol decrease (p=.006). No significant effect of ER-intervention group on relative cortisol change was found.

Conclusions

The current study provides evidence for a bifurcation in cortisol stress reactivity in youth with obesity. Our data further suggested that psychosocial variables (alexithymia and attachment avoidance) influence the cortisol stress response. Future research should further explore whether the attenuators are a more vulnerable group.

Abbreviations

HPA: hypothalamic–pituitary–adrenal

(%)BMI: (Adjusted) Body Mass Index

SAS: stress- and affective state

ER: emotion regulation

MOT: multidisciplinary obesity treatment

VIF: Variance Inflation Factor

1. Introduction

1.1. Obesity and stress

To date, childhood obesity has reached epidemic levels and represents a major public health concern [1]. Besides experiencing short- and long-term physical health problems, youth with obesity are at increased risk for many different psychosocial stressors (e.g., weight stigma, parent-child attachment difficulties,…) [2-6]. Given the fact that youth with obesity also display less emotion regulation skills to cope with stress [7,8], they form an at-risk group for experiencing psychopathology and enduring chronic stress [8,9]. Long-lasting stress is seen as harmful for all adolescents [10], but for those with overweight, this could lead to several worse outcomes that complicate or even hinder weight loss [11]. More specific, stress can lead to and/or maintain overweight through different pathways whereby the most studied mechanism is the role of emotional eating as a way of coping with stress, ultimately leading to enhanced overweight [2,12,13]. However, not all pathways are fully captured. One of the less studied pathways is the physiological pathway, assumed to be induced by cortisol release, a hormone produced under control of the hypothalamic–pituitary–adrenal (HPA) axis and related to feelings of enhanced stress. Cortisol is linked with obesity via an increased appetite, hedonic hunger and promotion of fat deposition in the visceral region [2,14]. Since obesity and stress are bidirectionally connected, it is complicated to unravel and break this complex, self-sustaining cycle. Studying

this pathway also in younger age groups will therefore be the aim of present study, designed to shed new light on the assumed physiological pathway.

1.2. Cortisol stress response

Besides regulating our metabolism, cortisol is a well-known marker of stress as it mediates the stress response [15]. It is known that acute stress results in an enhanced secretion of cortisol which can be seen as an adaptive physiological response [16]. When a stressor becomes chronic, the HPA axis responds initially with sustained cortisol elevation, but this chronic elevation is often followed by hypocortisolism [17,18]. Interestingly, during chronic stress, an acute stressor does not significantly increases cortisol levels [19]. Indeed, previous research in children and adults that focuses on cortisol response after an induced stressor found both a heightened (accentuated) and blunted (attenuated) reaction [20-22]. This bifurcation, an increase or decrease from baseline to reactivity, shows the importance of inter-individual differences in cortisol responses that could explain why one and the same stressor could increase cortisol in some people but not in others (i.e., those who show a more blunted reaction). Based on their relationships with both obesity and cortisol, below we describe the variables of interest for the current study: demographics (age and sex), chronic psychosocial stress correlates (parent-child attachment, sleep quality and perceived stress) and emotion regulation difficulties (emotional awareness).

1.2.1. Demographics.

When studying psychosocial variables that are related to obesity for understanding variability in cortisol reactivity, **age and sex** should be taken into account. Baseline cortisol levels increase with age across adolescence, for both boys and girls [23]. However, while adolescent girls have specifically higher baseline cortisol levels [24], adolescent boys show higher cortisol reactivity from baseline to stressor [25]. The influence of sex on cortisol reactivity is less unequivocal and needs further clearance .

1.2.2. Chronic psychosocial stress correlates.

In youth, the family context can be very stressful, while a secure **parent-child attachment** is negatively related to stress and negative affect [26]. This may be especially the case for children with obesity as an insecure parent-child attachment is positively related to Body Mass Index (BMI)[3]. Moreover, high degrees of insecure attachment are linked with deviated cortisol values and dysregulation in HPA axis responses [27]. However, research in young populations with overweight and obesity is scarce and conclusions depend on the format of insecure attachment (i.e., attachment anxiety or avoidance) [28,29]. Also the direction of the association is inconsistent: insecure parent-child attachment is positively associated with both accentuated and attenuated cortisol stress responses in community adolescents [20].

Sleep quality is another correlate of chronic stress that is both linked with obesity and HPA activity. A meta-analysis in youth found that poorer self-reported sleep quality was related with higher odds of being overweight or obese [30]. Sleep problems are further associated with cortisol hyperreactivity to stress in adolescents [31]. A review concluded that both objective and subjective measures of low sleep quality potentiate the reactivity of the HPA axis [32]. However, the sleep-cortisol-obesity association has not been ample studied in youth with obesity.

In psychology, most stress-studies rely on self-reports. As the term **perceived stress** indicates, it reflects the subjective stress experience which is seen as the most reliable source when internal states should be estimated and, as expected, the association with obesity has been proven [2]. However, an association with cortisol is expected as well but this is less clear. A review on the association between the Perceived Stress Scale (PSS) [33], a commonly used perceived (chronic) stress assessment instrument, and baseline salivary cortisol found inconsistent results [34]. More than half of the studies did not find a significant relationship.

1.2.3. Emotion regulation difficulties.

Given that youth with obesity display less skills to cope with stress related emotions [8,12,13], **emotional awareness** is a variable of interest. Emotional awareness can be seen as a facet of alexithymia (i.e., difficulties in identifying and describing emotions) and is recognized as the pre-stage of emotion regulation processes [35]. In adolescents with obesity there is a high occurrence of alexithymia (22%), with alexithymia scores being positively associated with obesity severity [36]. Difficulties in differentiating and distinguishing feelings is associated with a hyperresponsive HPA system [37]. However, other studies support physiological hypoarousal when individuals with high levels of alexithymia are confronted with an emotional challenge [38]. Since these results are not unequivocal, more research is indicated.

As mentioned above, different studies reported that chronic stress may result in HPA hypoactivity, seen in an attenuated cortisol response, after initially leading to an increased cortisol activity [17,18]. This suggests that the attenuated group acts as an exhausted group. This brings us to the question why some youngsters 'move' to hypoactivity and whether they show more psychosocial difficulties.

1.3. Current study

Despite previous research pointing out a bifurcation in cortisol stress reactivity, it is not yet clear if all variables explaining inter-individual differences in stress response are captured [20]. Especially research examining psychosocial predictors of cortisol stress response in youth with obesity is lacking. Therefore, the current study focuses on cortisol responses after a standardized stress- and affective state (SAS)-induction in youth with obesity.

The first aim of the study is to explore the physiological cortisol reaction after SAS-induction. Two distinct groups are expected: those whose cortisol levels increase (accentuated response) and those whose cortisol levels decrease (attenuated response) from baseline to reactivity.

Our second aim is to explore whether these two groups can be distinguished based on the demographics age and sex, psychosocial measures of chronic stress correlates (insecure attachment, sleep quality and perceived stress), and the emotion regulation competence emotional awareness. We hypothesize that attenuators will be older, have lower emotional awareness, and higher perceived stress and insecure attachment avoidance scores than increasers. In addition, we expect that increasers will consist of more boys and have lower sleep quality and higher insecure attachment anxiety scores compared to attenuators. Next, the additional hypothesis will be explored whether the psychosocial variables predict the strength of the cortisol response after SAS-induction.

Third, since participants were part of an emotion regulation (ER) randomized control trial (SRCTN83822934), a potential intervention effect will be explored. We hypothesize that those who received an ER-training will show smaller relative changes after SAS-induction than those who did not receive the training.

2. Method

2.1. Participants

Participants were 79 children and adolescents from 9 to 15 years old (*M*=12.33, *SD*=1.67, 50.6% boys; adjusted(%) BMI[1] *Range*=119-229; *M*=154.4, *SD*=24.6) who enrolled for a ten-month inpatient multidisciplinary obesity treatment (MOT) during 2018-2020. At time of current study participants were already 6 months in the clinic. They all participated in a randomized control trial (SRCTN83822934) investigating the effects of an ER-training on top of the MOT [39]. Of current participants, 44 patients received an ER-training, while 35 patients were assigned to the MOT-only control group. Patients without cortisol values (no sample or concentration cortisol too low) on both time-points (before and after SAS-induction) (*N*=22) and two outliers (>3SD±mean baseline cortisol) were already excluded. Patients with

[1] %BMI is the BMI adjusted for age and sex resulting in the percentage of overweight

comorbid medical disorders that cause (a part of) the weight gain (i.e. serious thyroid problems) and youngsters with medical problems where obesity is secondary were not recruited for the study. Of all participants 26.6% used at least one prescription drug (e.g., inhaler, Ritalin). Those participant did not differ significantly from the other participant with regard to the variables used in current study. For more information about the data-collection see Debeuf and colleagues [39]. The study was performed according to the Declaration of Helsinki and approved by the ethics committee of Ghent University Hospital.

2.2. Procedure

Figure 1 gives an overview of the different time-points and collected data used in this study. Stable (trait) data (insecure attachment and perceived chronic stress) was measured at month 3. Sleep, alexithymia and %BMI (i.e., BMI adjusted for age and sex resulting in the percentage of overweight) were measured at time of SAS-induction (Month 6). More details can be found in Debeuf et al. [39].

- Insert here Figure 1 -

2.3. Measures

Adjusted BMI (%BMI) was calculated by adapting measured BMI (weight in kg/squared height in m²) for age and sex [(actual BMI/Percentile 50 of BMI for age and sex) x 100]. Percentile 50 of BMI for age and sex was based on normative data [40]. An %BMI score ≤85% is considered underweight, an %BMI score ≥120% as overweight and an %BMI score ≥140% as obesity.

Salivary cortisol was analyzed by an enzyme linked immune-sorbent assay (ELISA) with inter-assay CV 7.2%, intra-assay CV 6% and range 50–3200 pg/ml (Arbor Assays®)[41].

Cortisol samples were obtained via salivette synthetic swabs (Sarstedt, Germany), specifically designed for salivary cortisol analysis. Swabs were centrifuged for 4 minutes at 3000 rpm (Jouan CR412 centrifuge), and filtrates were stored at -80°C . For a more detailed description, see van Aken and colleagues [42]. Assessment hour of the study (i.e., time of the day when cortisol levels were measured) was also registered.

The Experiences in Close Relationships Scale-Revised Child version (ECR-RC)[43] is a self-report scale assessing two forms of insecure attachment: Attachment Anxiety and Attachment Avoidance. The 36 items ranged from 1 (=strongly disagree) to 7 (=strongly agree). The ECR-RC has good reliability and validity [43]. In the present sample, attachment to the mother was used and internal consistency was good for both subscales (α=.89 and α=.91).

The Chronic Sleep Reduction Questionnaire (CSRQ)[44] measures sleep reduction with 20 items that are answered on a three-point Likert scale (with 0=no, 1=sometimes, and 2=yes). The CSRQ assesses shortage of sleep, irritation, loss of energy, and sleepiness. The questionnaire has good psychometric qualities [44]. The total score was used, which showed good internal consistency (α=0.90)

The Perceived Stress Scale (PSS)[33] is a commonly used perceived (chronic) stress assessment instrument. The ten items ask about stress feelings and thoughts during the last month and range from 0 (=never) to 4 (=very often). The PSS has adequate validity and reliability [33]. In the present sample, internal consistency was acceptable (α=.68).

The Toronto Alexithymia Scale-II (TAS-20)[45] consists of 20 items to measure alexithymia, with 3 subscales: difficulties in identifying emotions, difficulties in describing emotions to others, and an externally oriented thinking style. The items are scored on a three-point Likert scale ranging from 0 (=not correct for me) to 2 (=correct for me). The TAS-20 uses cutoff scoring, with scores of 21-24 indicating possible alexithymia (subclinical range), and scores of 25-40 indicating alexithymia (clinical range). The TAS-20 has good internal

consistency and validity [45]. In current study the total score as well as the subscale 'difficulties in identifying emotions' was used. Both had a good internal consistency (α=.86 and α=.84).

The stress- and affective state (SAS)-induction consisted of a combination of three different stressors each lasting 5-10 minutes and presented in a fixed order: a mood induction using fragments from "The Champ" or "The Lion King" (affective stressor), the Leeds Food Preference Questionnaire [46](food related stressor) and finally a cognitive stressful memory task in which participants had to answer open questions regarding details of the fragment they saw (cognitive stressor); see also Debeuf and colleagues [39]. The SAS-induction was done twice, once before and once after ER-training, with three months in between. For the current study, data from SAS-induction after ER-training was used. Before, during and after SAS-induction six cortisol samples were taken of which two were analyzed (Figure 1). Due to the known time-lag of cortisol peak [16], the cortisol sample at minute 23 was used as baseline cortisol sample. Approximately 40 minutes later, 20-25 minutes after the last stressor (memory task), the cortisol sample representing 'after SAS-induction' was taken.

The *MOT* took place at a treatment center in Belgium where the patients were following a ten-month inpatient treatment to obtain a healthy body weight [47]. The intervention consists of learning about healthy food behavior, physical activity, cognitive behavioral techniques and involvement of the parents. On top of this, 44 patients also received an *ER-training* called "EuREKA"[48] consisting of 12 sessions in which different ER competences were trained.

2.4. Data-analysis

Analyses were performed using IBM®SPSS® Statistics 26. For the first aim of the study, cortisol at baseline of SAS-induction was compared to cortisol after SAS-induction using Repeated Measures ANOVA, both in the total group and, if indicated, in two subgroups separately. Assessment hour was added as covariate. To compare the increasers- attenuators as well as the two intervention groups (ER-control group), independent samples T-tests were used

for continuous variables and chi-square tests for categorical variables. Multiple regression analysis was executed with relative cortisol increase/decrease after SAS-induction as dependent variable and the psychosocial variables as independent variables, in the increasers and attenuators group separately. Regression results were robust to the effect of multicollinearity, as Variance Inflation Factor (VIF) values were smaller (highest value: 4.074) than 10 .

3. Results

3.1. Descriptive data

Table 1 shows the descriptive data of the participants. It also shows Spearman rank-order correlation coefficients on the relationship between cortisol at baseline SAS-induction on the one hand and assessment hour, age, sex, %BMI and the different psychosocial variables on the other hand. Only the negative correlation with assessment hour was significant.

- Insert here Table 1 -

3.2. Time-effects for the total group and subgroups increasers and attenuators

Regarding aim 1, based on a Repeated Measures ANOVA (Table 2) with assessment hour as covariate, there was no significant change for the total group between baseline cortisol (*M*=516.2, *SD*=418.9) and cortisol after SAS-induction (*M*=500.3, *SD*=419.3), $F(1,77)=.700$, $p=.405$.

As expected, two distinct groups could be distinguished. Those whose cortisol levels increased (*N*=32; *range*=2%-556% relative increase, *M*=99.3%) and those whose cortisol levels decreased after SAS-induction (*N*=47; *range*=1%-79% relative decrease, *M*=37.7%). When repeating above analysis for the increasers group, a significant time-effect ($F(1,30)=8.501$, $p=.007$) between baseline cortisol (*M*=358.8, *SD*=241.6) and cortisol after

SAS-induction (*M*=659.3, *SD*=492.6) was found, i.e. cortisol levels increased significantly. There was also a significant interaction between time and the covariate assessment hour (*F*(1,30)=4.406, *p*=.044), the earlier the SAS-induction, the stronger the increase. In the attenuators group, no significant time-effect (*F*(1,45)=2.554, *p*=.117) was found between cortisol levels before (*M*=623.4, *SD*=478.8) and after (*M*=392.0, *SD*=324.1) SAS-induction, although visual inspection suggests reduced cortisol levels.

- Insert here Table 2 -

For testing aim 2, increasers and attenuators were compared on all variables: significant differences were found regarding alexithymia scores, and trend significantly regarding sex, age, %BMI and chronic sleep reduction (Table 3). Those whose cortisol levels decreased after SAS-induction were older, had a higher %BMI and had higher chronic sleep reduction as well as higher alexithymia scores than increasers. There were also more girls among attenuators compared to increasers.

- Insert here Table 3 -

In the increasers group, a multiple linear regression to predict relative cortisol increase based on all psychosocial variables explained 15.9% of the variance but was not significant (*F*(10,21)=.398, *p*=.933). There were no significant predictors in this model (Table 4). In the attenuators group, the multiple linear regression to predict relative cortisol decrease explained 45% of the variance and was significant (*F*(10,36)=2.944, *p*=.008). Attachment avoidance was a significant positive predictor of relative cortisol decrease after SAS-induction (*p*=.001). Age was negatively related to cortisol decrease (*p*=.006)(Table 4).

- *Insert here Table 4* -

3.3. Comparing subgroups for intervention effects[2]

For testing aim 3, a Pearson Chi-Square was performed to determine whether the proportion of increasers/attenuators was equal between ER-group (N=44, 57% attenuators) and control group (N=35, 63% attenuators). There was no significant difference, $X^2(1, N=79)=.295$, $p=.587$.

When further exploring the influence of condition on the (time-)effect of SAS-induction on cortisol levels, condition was added as between factor to the Repeated Measures ANOVA's (see 3.2.). Conclusions remained the same and there were no significant time x condition interactions in the total group ($F(1,76)=.573$, $p=.451$), increasers group ($F(1,29)=.058$, $p=.812$) or attenuators group ($F(1,44)=.342$, $p=.561$).

When comparing ER- and control group regarding relative change, visual inspection suggests a smaller change for ER-group compared to control group (Figure 2). However, in the attenuators group, based on an independent samples t-test, relative decrease after SAS-induction was not significantly smaller for ER-group ($t=1.479$, $p=.147$) Also in the increasers group, based on a Mann-Whitney U test, there was no significant difference between ER (N=19) and control group (N=13) regarding relative increase (U=90, $p=.199$).

- *Insert here Figure 2* -

4. Discussion

[2]The ER- and control group did not differ significantly regarding %BMI ($t=.169$, $p=.867$), attachment avoidance ($t=.517$, $p=.606$), attachment anxiety ($t=-.297$, $p=.767$), perceived stress ($t=1.080$, $p=.284$), chronic sleep reduction ($t=1.435$, $p=.156$), alexithymia ($t=.586$, $p=.560$) and difficulties in identifying emotions ($t=.756$, $p=.452$) at the start of the training. They did further not differ regarding delta BMI (change in %BMI during training)($t=-.125$, $p=.901$). Concerning cortisol reactivity at start of the training, there were no significant differences between ER- and control group in the total ($t=1.731$, $p=.088$), increasers ($t=.782$, $p=.442$) and attenuatorsgroup ($t=.579$, $p=.566$).

When studying the obesity-stress relation, not all pathways are fully captured. The present study researched the role of salivary cortisol. Previous research in children and adults that focuses on cortisol response after an induced stressor found both a heightened and blunted reaction, which plead to explore inter-individual differences that could explain this bifurcation. More specific, the aim of the present study was to explore (1) the physiological cortisol reaction of youth with obesity after an induced stressor, and whether the expected two groups (increasers and attenuators) also exist in this sample (2) whether the groups can be distinguished based on demographics and psychosocial variables, and which variables predict the strength of cortisol response and (3) whether an ER-training influences cortisol response after SAS-induction.

As hypothesized in aim one, two distinct groups were found: those whose cortisol levels increased and those whose cortisol levels decreased from baseline to reactivity. This is consistent with research in healthy adolescents that described this bifurcation [20,22]. It should be noted that in the attenuators group the decrease was not significant, although the effect size was small to medium. Post hoc analyses using Gpower [49] revealed that the statistical power for the Repeated Measures ANOVA was .62, .95 and .99 for the total group, increasers and attenuators, respectively. A lack of power is thus not the reason for the non-significant decrease. A possible explanation could be the high variance in cortisol levels, which can be seen in the large standard deviation and wide range. Further, the nature of the SAS-induction might also have had an influence. Via visual analogue scales participants rated their mood before and after SAS-induction. The induction increased their sadness and decreased their happiness levels, but did not significantly influence their anxiety and stress levels. Since cortisol is influenced by stress, the induction might not have been strong enough to observe clear changes in cortisol levels in all participants [16]. Increasers and attenuators did not differ significantly regarding baseline mood, mood after SAS-induction and mood change after SAS-

induction. They did however significantly differ regarding baseline cortisol, with attenuators having higher baseline levels. This is in line with a previous study that concluded that the decreased change in the cortisol from pre to post stressor was due to elevated baseline cortisol levels [19].

Concerning the second aim, increasers and attenuators differed significantly regarding alexithymia, with attenuators showing higher levels than increasers. This is in line with a recent review [38] supporting the presence of hyporeactivity to emotional challenges in alexithymia. A potential explanation could be that alexithymia is associated with chronic stress [50]. Chronically elevated levels of stress are known to alter HPA function, i.e., chronic stress may result in HPA hypoactivity, after initially leading to an increased cortisol activity [17,18]. Attenuators can thus be seen as an exhausted group that looks emotionally 'immune'. The positive relationship between alexithymia and chronic stress is confirmed in the current data (r=.236, p=.037).

Regarding age, sex, %BMI and sleep quality, there were no significant differences between increasers and attenuators (.05 < p's <.085). This might be influenced by the small sample sizes of both groups (N=32, N=47). Indeed, post hoc analyses revealed that the statistical power was very poor (approximately 0.50). Given that current literature regarding sleep quality is not unequivocal [32,51,52], more research examining the effects of both subjective and objective sleep quality on HPA-reactivity is needed, especially in youth with obesity.

There were further no significant differences between increasers and attenuators regarding insecure attachment and perceived chronic stress. Regarding insecure attachment, higher levels of attachment anxiety were expected in increasers, as was found in adult females without obesity [29]. That study did also not find a relation with attachment avoidance. Cameron and colleagues [20] did find a positive association of insecure parent-child attachment with cortisol stress responses in a sample of adolescents. It would be interesting to know if there is a difference between attachment anxiety and avoidance. More research in youth with

overweight and obesity is thus indicated. Regarding perceived stress, we hypothesized that attenuators would report higher levels of perceived stress than increasers. Our data did not support this. A potential explanation is the chronology in which experiencing stress initially leads to hyperreactivity and only later on to hyporeactivity due to sensitization [17,18]. It is thus possible that both groups contain highly stressed individuals and that common psychosocial variables underlie the stress experiences.

We further explored the relative contribution of the different (psychosocial) variables in relative cortisol change. In the attenuators group, attachment avoidance was a significant positive predictor of relative cortisol decrease after SAS-induction, such that youngsters with higher reported attachment avoidance showed larger cortisol reductions. This finding offers support for the association between attachment quality and HPA-(re)activity. Conversely, age was significantly related to less cortisol decrease. The older the participant, the smaller their cortisol decrease after SAS-induction. There were no significant predictors in the increasers group regarding relative cortisol increase. The statistical power for the multiple regression analysis on relative cortisol increase and decrease were respectively .24 and .98.

Lastly, in aim three, a potential intervention effect of the ER-training on cortisol reactivity was explored. We did not find an effect of intervention group on relative cortisol change. The small sample sizes in each group (*Nrange*=13-25) might have impacted the power to detect a significant difference (power was <.50 for all analyses). A visual inspection (Figure 2) does suggest smaller cortisol changes after SAS-induction for those that received an ER-training. In the attenuators group, the control group shows a mean decrease of 43% from baseline to reactivity, while the ER-group declines 33%. In the increasers group, the control- and ER-group increase with 128% and 80% from baseline to after SAS-induction, respectively. This suggests that the ER-group might react less strongly to a stressor compared to the control group. However, the current study did not find significant differences so we cannot draw any

conclusions about an intervention effect. Because of potential clinical implications, future research should investigate this in a larger sample.

The present study has several strengths and limitations that have to be acknowledged. Strengths are the specific well-defined group of youth with obesity living in the standardized setting of a residential MOT (e.g., same food, same physical activity, daily rhythm), inclusion of both sexes and taking different psychosocial factors into account. Limitations include the small sample size and specific nature of the sample which may limit generalizability. Regarding the measurement of the psychosocial variables, self-report scales are recommended when internal states should be assessed but entail the risk for self-report bias that is common in subjective assessments [53]. Future research could include hair cortisol as an objective measure of chronic stress next to the subjective measure of the PSS. Further, despite including a measure of parent-child attachment, the present study did not take the participants' history of childhood adversities into account. Also the influence of the environment on stress in youth with obesity and the direction of causality (stress <-> weight status) should be studied in future studies. Regarding cortisol, the experiment took place at different times during the day. Although we controlled for assessment hour, future research could schedule all participants around the same time (e.g., 3:30-6:30 pm, when there is low variability in baseline cortisol) to prevent daily fluctuations in cortisol [20]. Further, as mentioned above, a more anxiety-provoking or frustration-provoking stressor could be a better choice to influence stress and cortisol levels of participants[54].

Despite abovementioned limitations, the current study expands the actual knowledge about cortisol reactivity in youth with obesity and may provide important clinical implications. When the influence of alexithymia and attachment avoidance can be replicated in future research, it could expose relevant treatment targets for youth with obesity. Regarding alexithymia, a specific (group)training consisting of psychoeducation, emotional awareness,

emotional coping skills and experiential approaches might be of value [55]. Since alexithymia is associated with chronic stress, such training might also positively impact their perceived stress. For individuals with high levels of attachment avoidance, extra psychological help could be indicated, e.g., family meetings or psychotherapy. In psychology, it is also well-known that children who cannot trust their parents for emotional support have less opportunities to learn adequate skills from them to cope with stress and negative emotions [56]. This plead even more for an ER-training.

In conclusion, the current study provides evidence for a bifurcation in cortisol stress reactivity in youth with obesity. Our data further suggests that increasers and attenuators differ significantly regarding alexithymia, with attenuators showing higher levels. Also attachment avoidance should be taken into account in future research since it is related to stronger decreases of cortisol after an SAS-induction. Future research should further investigate whether the attenuators are a more vulnerable group and explore the influence of ER-training on cortisol reactivity in youth with obesity as it may lead to important intervention approaches.

Conflicts of interest statement

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper. Prof. Dr. Braet and Prof. Dr. De Henauw did receive a grant from Ghent University.

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Author contributions

CB and SD conceived the study and coordinated and supervised data collection together with SV. TD and NM collected data. IV conducted the statistical analysis, interpreted the results and drafted the manuscript. All authors critically reviewed the manuscript for important intellectual content. All authors have substantially contributed to, seen and approved the final version of the manuscript and qualify for authorship.

Ethical statement

The study was approved by the Ethics Committee of the Ghent University Hospital and in accordance with the Helsinki Declaration (EC UZG 2018/0101). Written informed consent was obtained from parents and adolescents (≥12y). Children below the age of 12 provided a verbal assent to participate.

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Table 1.

Descriptive statistics and Spearman's rho correlations with cortisol

Variable	*M*	*SD*	Range	r_s	*p*
Baseline cortisol (pg/mL)	516.21	418.92	17.7-2311.7	-	-
Assessment hour	13.86	2.71	9.26-18.57	-.438	<.01
Age (years)	12.33	1.67	9-15	.09	.429
Sex (% boys)	50.6	-	-	-	.937
Adjusted Body Mass Index (%)	154.35	24.57	119-229	.194	.086
Experiences in Close Relationships Scale					
Attachment Anxiety [1-7]	2.46	.99	1-5.28	-.029	.800
Attachment Avoidance [1-7]	2.92	1.21	1-5.78	-.020	.860
Chronic Sleep Reduction Questionnaire [0-40]	17.25	8.72	0-35	.157	.168
Perceived Stress Scale [0-40]	21.49	5.82	9-35	-.038	.741
Toronto Alexithymia Scale-II [0-40]	18.85	7.84	0-40	-.015	.894
Difficulties in identifying emotions [0-14]	5.61	3.72	0-14	.042	.710

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Table 2

Results of the Repeated Measures ANOVA's for cortisol

Main effect of time	F (df)	*p*	Effect size (η_p^2)
Total group	.700 (1,77)	.405	.009
Increasers	8.501 (1,30)	**.007**	.221
Attenuators	2.554 (1,45)	.117	.054

Notes. N is equal to 79, 32 and 47 respectively

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Table 3.

Sample descriptives and T-test of the increasers versus attenuators *group*

	Increasers		Attenuators			
	M	*SD*	*M*	*SD*	*t*-test	*p*
Age	11.91	1.44	12.62	1.76	1.888	.063
Sex (% boys)	62.5	-	42.6	-	-	.082
Adjusted Body Mass Index	148.63	16.92	158.25	28.14	1.892	.062
Assessment hour	13.79	2.40	13.90	2.92	.187	.852
Experiences in Close Relationships Scale						
Attachment Anxiety	2.63	1.02	2.34	0.96	-1.268	.209
Attachment Avoidance	2.94	1.21	2.90	1.23	-.119	.906
Chronic Sleep Reduction Questionnaire	15.16	9.02	18.68	8.30	1.788	.078
Perceived Stress Scale	21.41	5.59	21.55	6.03	.109	.913
Toronto Alexithymia Scale-II	16.75	9.63	20.28	6.05	2.001	**.049**
Difficulties in identifying emotions	4.91	4.14	6.09	3.36	1.393	.168

Notes. N is respectively 32 and 47. For sex, a chi-square was used to calculate the *p*-value. Increasers and attenuators did not differ significantly regarding delta BMI (change in %BMI during training).

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Table 4.

Cross-sectional multiple regression analysis on relative cortisol increase/decrease after SAS-induction (Enter Method).

	Increasers			Attenuators		
	B	*t*	*VIF*	*B*	*t*	*VIF*
Constant	.807	.192		.615	1.586	
Assessment hour	.031	.221	1.471	.013	1.129	1.265
Age	-.216	-1.016	1.252	-.067	**-2.937***	1.917
Sex (boys as reference)	-.008	-.011	1.603	-.116	-1.552	1.682
Adjusted Body Mass Index	-.003	-.202	1.067	.001	.863	1.327
Experiences in Close Relationships Scale						
Attachment Anxiety	.193	.549	1.708	-.052	-1.332	1.670
Attachment Avoidance	.277	.926	1.741	.116	**3.464***	2.028
Chronic Sleep Reduction Questionnaire	.013	.322	1.895	.001	.276	2.144
Perceived Stress Scale	.004	.061	1.727	.003	.535	1.359
Toronto Alexithymia Scale-II	.000	.004	3.429	.001	.149	3.237
Difficulties in identifying emotions	-.099	-.737	4.074	.019	1.210	3.501

Notes. N is respectively 32 and 47; Relative cortisol increase values were natural log transformed; **p*<.05

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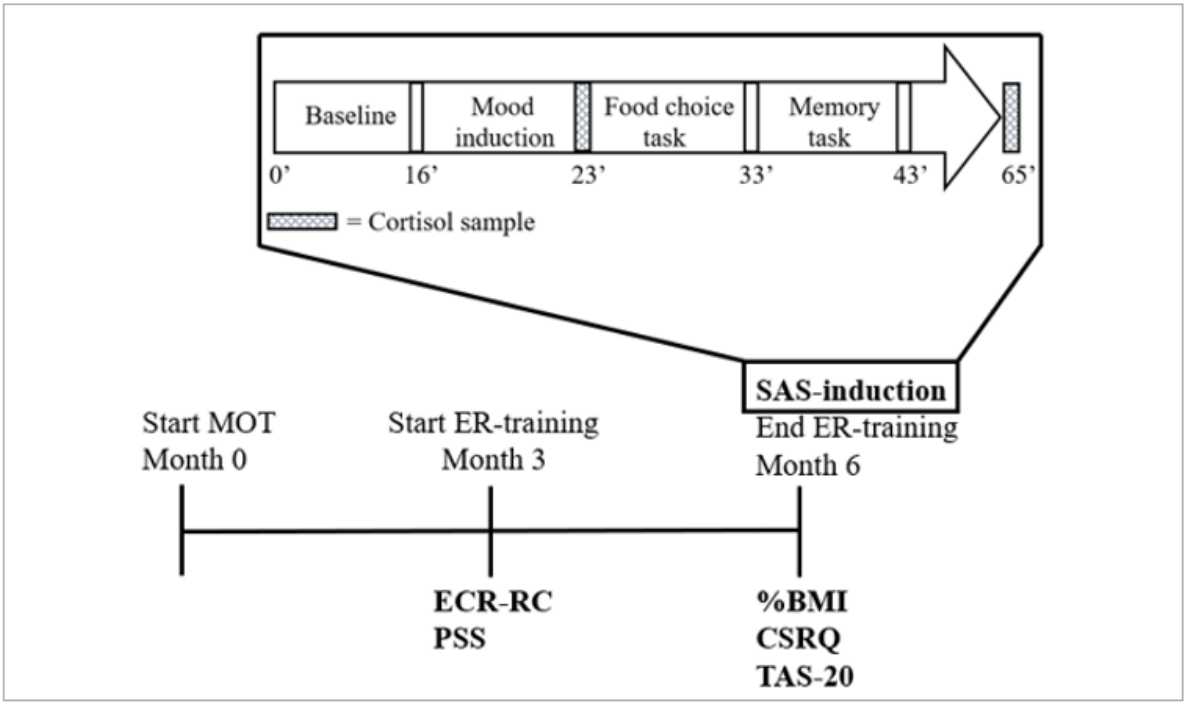
Figure 1

Timeline study design



Notes. MOT=Multidisciplinary Obesity Treatment; ECR-RC=measure insecure attachment; PSS=measure perceived stress; %BMI=Adjusted BMI; CSRQ=measure sleep quality; TAS-20=measure alexithymia

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Figure 2.

Relative cortisol change after SAS-induction in the control and emotion regulation group

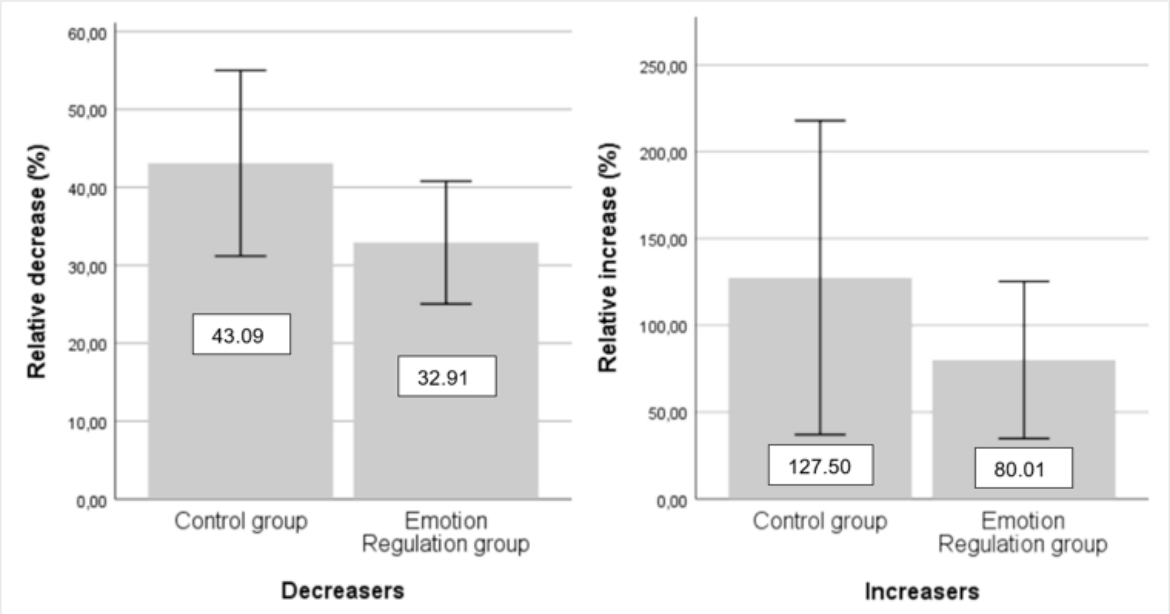

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Table and Figure Legends

Table 1: Descriptive statistics and Spearman's rho correlations with cortisol

Table 2: Results of the Repeated Measures ANOVA's for cortisol

Table 3: Sample descriptives and T-test of the increasers versus attenuators group

Table 4: Cross-sectional multiple regression analysis on relative cortisol increase/decrease after SAS-induction (Enter Method).

Figure 1: Timeline study design

Overview of the different time-points and collected data used in this study.

Figure 2: Relative cortisol change after SAS-induction in the control and emotion regulation group

Mean relative cortisol change [(|baseline cortisol - cortisol after induction|/baseline cortisol) x 100] of the control and emotion regulation group, in the increasers and attenuators group separately.