Cross-Lagged Associations Between Children's Stress and Adiposity: The Children's Body Composition and Stress Study

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Objective: The public health threats stress and adiposity have previously been associated with each other. Longitudinal studies are needed to reveal whether this association is bidirectional and the moderating factors. **Methods:** In the longitudinal Children's Body Composition and Stress study, 316 children (aged 5–12 years) had measures of stress (questionnaires concerning negative life events, problem behavior, and emotions) and adiposity (body mass index, waist-to-height ratio, and fat percentage) in three waves at 1-year intervals. The bidirectionality of the association between stress and adiposity was examined using cross-lagged analyses. We tested moderation by cortisol and life-style (physical activity, screen time, food consumption, eating behavior and sleep duration). **Results:** Adiposity (body mass index: $\beta = 0.48$ and fat percentage: $\beta = 0.18$; $p < .001$) were associated with subsequent increased stress levels, but stress was not directly related to subsequent increases in adiposity indices. Cortisol and life-style factors displayed a moderating effect on the association between stress and adiposity. Stress was positively associated with adiposity in children with high cortisol awakening patterns ($\beta = 0.204$; $p = .020$) and high sweet food consumption ($\beta = 0.190$; $p = .031$), whereas stress was associated with lower adiposity in the most active children ($\beta = -0.163$; $p = .022$). **Conclusions:** Stress is associated with the development of children's adiposity, but the effects depend on cortisol levels and life-style factors. This creates new perspectives for multifactorial obesity prevention programs. Our results also highlight the adverse effect of an unhealthy body composition on children's psychological well-being. **Key words:** stress, body mass index, waist, fat percentage, cortisol, life-style.

AUCg = area under the curve with respect to the ground; **AUCi** = area under the curve with respect to increase; **BMI** = body mass index; **ChiBS** = Children's Body Composition and Stress; **CFI** = comparative fit index; **RMSEA** = root mean square error of approximation; **WHtR** = waist-to-height ratio.

INTRODUCTION

Recent decades have been characterized by a global overweight epidemic. Most alarming is the rise in childhood overweight, with a prevalence ranging between 10% and 40% in European countries (1). Research has broadened its view on obesity contributing factors to extend beyond the traditional concept of an energy imbalance caused by diet and physical activity. In this context, the psychological determinants of obesity have received special attention (2,3). Recent meta-analyses in adults (4,5) and some reviews in children/adolescents (6–8) have mainly uncovered an adiposity increasing effect of stress (stressors, perceived stress, depression, anxiety, and behavioral problems), although null effects and even opposite results have been observed as well. Interestingly, this association between adiposity and stress might be bidirectional, with adiposity also causing higher stress levels due to physiological and psychological mechanisms (4,9,10).

The effects of psychosocial stress on childhood adiposity have been hypothesized to result from endocrinological and behavioral pathways. First, metabolic changes (such as increased visceral fat disposition and a stimulation of appetite) are primarily caused by stress system dysregulation and stress hormone production (cortisol and catecholamines) (11–13). Second, stress may indirectly facilitate adiposity through behavioral pathways: stress may change the energy balance by interrupting one's choice to participate in healthy behaviors such as physical activity and adequate sleep duration, while promoting unhealthy behaviors such as emotional eating of unhealthy food items and increased sedentary screen time (14–23). Up to now, little evidence on these underlying factors is available (10). In the literature mentioned above, life-style and cortisol are generally considered as mediators (i.e., explaining factors) in the relation between stress and adiposity (24–26). Other research has suggested that life-style and cortisol are rather moderators (i.e., interaction) in the stress-adiposity relation. After all, diverse stress-adiposity relations may exist because of inter-individual differences in life-style and physiology. In this concept of moderation, there might only be an effect of stress on adiposity when a high cortisol response is present (i.e., when the stress gets under the skin) or when the individual has an unhealthy life-style (e.g., physical activity as a protective factor against stress-induced effects).

Overall, the literature recommends further research with longitudinal designs, accurately measuring body composition and analyzing the endocrinological and behavioral factors (27). Moreover, there is a lack of evidence for this relation in primary school children (6,10). Because adult psychopathology (28), life-style (29–32), and adiposity (33) may have their origin in childhood, research in children is of high importance. After all, chronic alterations in stress activity may have permanent effects on the endocrine and metabolic systems (34,35).

To address this need, the first aim of the current study was to examine the bidirectional longitudinal association between stress and adiposity (body mass index [BMI], waist, and accurate fat percentage [fat %] measurements) in Belgian primary school children (5–12 years old). The second aim was to test moderation in the stress-adiposity relation by both life-style parameters (physical/sedentary activity, diet, eating behavior, and sleep duration) and cortisol levels.

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METHODS

Design

This article is based on the longitudinal Children's Body Composition and Stress (ChiBS) study (2010–2012) in Belgian children (half male, half female). The children were measured during three waves (i.e., measurement periods) with 1-year interval: 523 children in 2010 (Wave 0 [W0]), 455 children in 2011 (Wave 1 [W1]), and 330 children in 2012 (Wave 2 [W2]). At baseline, the children were between 5 and 10 years old. More details on the ChiBS study and its measurements have been described elsewhere (36). Children were measured for adiposity, physical activity, and cortisol levels. Furthermore, questionnaires were filled in on stress and life-style (physical activity, screen time, food intake, dietary behavior, and sleep duration). Data collection for most parameters was performed in all three waves, but data were only available in two waves for sleep duration, accelerometry, eating behavior, and waist-to-height ratio (WHtR). Salivary cortisol data were only available in W0.

We included the longitudinal data on reported stress and adiposity parameters from our ChiBS study. As a consequence, 316 children were included in the cross-lagged and moderation analyses. This number decreased to 305 children when using fat % as outcome. Participants with and without complete data were compared using Little's Missing Completely At Random test (37). A nonsignificant χ^2 test statistic suggests that missing data are missing completely at random and hence do not introduce any bias with regard to the central research question. No differences between the follow-up and dropout population were seen for stress, adiposity, and tested moderators, except for a higher screen time in those who dropped out ($p = .028$).

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.

Reported Stress (W0, W1, and W2)

We have used a broad definition of stress. After all, stress is an adaptive, dynamic state that is composed of several aspects. The initiating stimulus is the "stressor." This is the environmental demand, challenge, or event. When being confronted with these stressors, people evaluate whether this is a potential threat. When there is a discrepancy between what is expected and what is happening in reality, a coping response will be initiated that induces arousal. If the person is unable to handle the persistent situation, the sustained, chronic arousal can trigger emotional and behavioral disturbances that might put a person at risk for psychiatric or physical disease (38). Because of this broad definition, different aspects of the stress process were measured in the ChiBS study: negative events, emotions, and behavioral problems. A composite stress score was calculated by summing up the *z* scores of the three stress aspects per child: one for negative events, one for negative emotions, and one for behavioral problems. This composite stress score enabled a relative representation of reported stress in our study population, but should not be interpreted in an absolute way.

Children were individually interviewed by a trained researcher on negative events and negative emotions. Parents reported behavioral problems of their children.

Negative Events (Child-Reported)

The children were asked to fill out the Coddington Life Events Scale for Children. This is a validated (reliability $r = 0.69$) 36-item questionnaire assessing the frequency and timing of stressful life events relevant for this age group during the last year (39). The score of negative life events was used for the analyses.

Negative Emotions (Child-Reported)

Because it concerned a young childhood sample, a short and easy-to-understand questionnaire on emotions was chosen. Children were asked to report on how they mostly feel (not only today). Feelings of anger, anxiety, and sadness could be rated on a 0 (not at all) to 10 (very strong) Likert-scale. Validation of this questionnaire was tested in Wave 3 (W3): the negative emotions score (sum of three negative emotions) showed a Spearman correlation of $r = 0.48$ ($p < .001$), with the negative affect score of the validated Positive and Negative Effect Schedule questionnaire (40) in a subsample of 153 children of at least 9 years old (this minimum age was a requisite for the questionnaire).

Behavioral Problems (Parent-Reported)

Parents were asked to complete the standardized Strengths and Difficulties Questionnaire (41), (Cronbach α = .53–.76, test-retest stability $r = 0.88$, concurrent validity $r = 0.7$–0.87) indicating their children's behavioral problems over the past 6 months (proxy-report). The children were too young to complete the self-report version of the questionnaire. The statements were divided into three subscales (with five items each): peer problems, conduct problems, and emotional problems. Higher scores reflect difficulties or problems.

Salivary Cortisol for Stress Reactivity (Only W0)

Because stress reports are not always associated with chronically changed levels of stress hormones, levels of salivary cortisol were analyzed. Variability in cortisol response is partially attributable to the nature of the stressor (type and controllability) as well as the person facing stress (emotional response and psychiatric sequelae) (42). Previously, we found significant associations of these ChiBS children's cortisol levels with negative events, emotional problems, and peer problems, mainly supporting a chronic hypercortisolism due to stress (43).

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). Morning samples collected more than 5 minutes before or after the requested time point and evening samples not collected between 7 and 9 PM (271 of 3290 samples) were excluded. Moreover, samples of five children using corticosteroids were excluded. Based on laboratory analyses with a competitive electrochemiluminescence immunoassay (Roche Diagnostics, Mannheim, Germany), summary variables were calculated to represent two independent cortisol patterns over time: the cortisol awakening response and the daily output (44,45). To represent the cortisol awakening response, the area under the curve with respect to the ground (AUCg) and the area under the curve with respect to increase (AUCi) were calculated between the T0 and T60 sample. These two parameters can reveal different information. The AUCi measures the pure morning increase, reflecting the sensitivity of the cortisol axis, and has commonly been related to stress perception. The AUCg also measures the morning increase but takes into account the basal level, thus reflecting the total hormonal output; therefore, it has been related more to physical complaints. The daily output was represented by the area under the curve between T0 and Tev ($AUC_{diurnal}$) and the diurnal decline measured as the concentration of T0 minus Tev, divided by the number of hours between these sampling periods (with a more positive value representing a steeper decline).

Adiposity (at W0, W1, and W2 but WHtR Only at W0 and W2)

Adiposity was measured based on a) age- and sex-specific BMI *z* scores calculated according to the method and reference data of Cole et al. (46), b) fat % calculated using air-displacement plethysmography (BOD POD), and c) the WHtR. BMI and fat % reflect overall adiposity, whereas the WHtR reflects central body fat. Weight and height were measured in bare feet and light underwear with an electronic scale (TANITA BC 420 SMA) and a stadiometer (Seca 225). For fat %, 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. In addition, waist circumference was measured once with an inelastic tape (Seca 200) halfway between the top of the iliac crest and the lower coastal border (10th rib) with the subject in a standing position. To correct for growth-related changes in waist, the WHtR was calculated (47,48). The waist-to-hip ratio was not taken into account because it is highly affected by sex and pubertal changes in the hip.

Life-style Factors

Parent-Report of Physical Activity and Screen Time (W0, W1, and W2)

At each measurement wave, parents were asked about the usual physical activity and screen time of their child. The sum of these "usual" hours of physical activity outdoors and at sports clubs per week was used as a measure of physical activity, whereas the reported usual number of screen time hours

per week (e.g., television and computer time) was used as a measure of sedentary behavior.

Objective Measure of Physical Activity and Sedentary behavior (W0 and W2)

Physical activity was also measured with accelerometers worn at the hip for at least 3 days in W0 and W2. Sedentary time and moderate-to-vigorous physical activity were determined using the cutoff points of Evenson (49) and expressed in percentages to correct for wearing time. A between-day intraclass correlation of 0.79 and 0.82 was found for moderate-to-vigorous activity and sedentary time, respectively.

Food Frequency Questionnaire (Parent-Reported at W0, W1, and W2)

The parents reported their child's dietary pattern during the last 4 weeks by completing a food frequency questionnaire. This questionnaire was designed and validated for the European IDEFICS study: test-retest reproducibility testing resulted in $r = 0.32–0.76$ for separate items (50), and the questionnaire revealed a general overestimation compared to a 2-day 24-hour recall (51). To identify dietary patterns, three indices were computed by summing up the consumption frequency of separate food items: "sweet foods" (i.e., sweet drinks, jam, honey, sweet breakfast cereals, and sweet snacks), "fatty foods" (i.e., fried potatoes, chocolate- or nut-based spreads, high-fat dairy, mayonnaise and mayonnaise-based products, cheese, fat meat preparations, butter, and high-fat snacks), and "fruit and vegetables" (i.e., fruit, freshly squeezed fruit juice, and vegetables). After all, stress has been hypothesized to mainly stimulate the consumption of "comfort food," that is, food high in fat or sugar (20).

Dutch Eating Behavior Questionnaire (Child-Reported at W1 and W2)

The children filled out the 33-item Dutch Eating Behavior Questionnaire on their usual eating behavior (52). Three types of eating behavior were identified in which the appropriate self-regulating mechanism of food intake is diminished or lost: eating in response to negative emotions (emotional eating), eating in response to the sight or smell of food (external eating), and eating less than desired to lose or maintain body weight (restrained eating). The different subscales revealed a stable factor structure, satisfying internal consistency (Cronbach α = .77–.91) and good test-retest reliability ($r = 0.87$-0.90) (53,54). The Dutch Eating Behavior Questionnaire has been validated in children as young as 7 years old (55,56). In our population, good internal consistency was found: α = .906 for emotional eating, .866 for external eating, and .738 for restrained eating.

Sleep (Parent-Reported at W0 and W2)

The parents reported the typical time at which the child goes to bed and is getting up in the morning for weekdays and weekend days, from which the child's average sleep duration was calculated.

Possible Confounders

Sex, age, and socioeconomic status were considered as potential confounding factors in the stress-adiposity relation. The children's sex and birth date were reported by the parent. Parental education (to represent socioeconomic status) was assessed by one question according to the International Standard Classification of Education (ISCED) (57).

Statistics

Cross-Lagged Stress-Adiposity Relations

To determine longitudinal associations between reported stress (the composite stress score: negative events, negative emotions, and total problems) and adiposity (BMI, fat %, and WHtR), structural equation modeling was performed in Mplus (version 5.1) using maximum-likelihood estimation with robust standard errors. A significance level of $p < .05$ was applied. Moreover, a number of fit indices were used to evaluate the model (58): the χ^2 test, the comparative fit index (CFI), and the root mean square error of approximation (RMSEA). χ^2/df Ratio of 2 or less, CFI values of 0.90 or more, and RMSEA values of 0.06 or less were indicators of acceptable fit (58).

The cross-lagged models included cross-lagged paths (e.g., from stress at baseline to adiposity at follow-up), autoregressive paths (e.g., stress at Time 1 to stress at Time 2), and correlations within waves. To control for possible confounders (age, sex, and socioeconomic status), paths were allowed from each of these three variables to all the constructs included in the structural models. The results of the cross-lagged associations between W1 and W2 will be preferably interpreted because these are corrected for all the factors and associations of the previous wave (59).

Statistical multigroup comparisons were tested to investigate potential structural differences for the cross-lagged models depending on sex and age. Age was transformed into a categorical variable by creating two groups based on the median age (one group <8 years, one group ≥8 years). The effect of age and sex was analyzed by comparing the fit between the constrained model (in which the structural relations between both groups were not allowed to vary) and the unconstrained model (in which the structural relations were set free). The difference in χ^2 and CFI statistic between these two models was calculated as follows: "constrained model statistic − unconstrained model statistic." Nonequivalence between the groups was considered in the case of a significant χ^2 difference and a CFI difference higher than 0.01.

Cortisol and Life-style as Moderators

A moderator is a third variable affecting the direction and/or strength of the relationship between a predictor and an outcome variable. Moderation by life-style factors (food indices, eating behavior, and physical and sedentary behavior) and salivary cortisol (AUCg, AUCi, $AUC_{diurnal}$, and diurnal decline) in the bidirectional relation between reported stress and adiposity was tested by including an interaction term between the predictor and the possible moderator at the same wave. If the interaction term was significant, visual representation was done by plotting predicted outcome values for moderator and predictor levels 1 standard deviation below the mean (=low) and 1 standard deviation above the mean (=high). Also for moderation, cross-lagged associations between W1 and W2 were preferably tested, that is, the moderation between stress and moderator W1 on adiposity W2. Nevertheless, associations between W0 and W2 were tested for cortisol, accelerometry, and sleep because of missing data at W1.

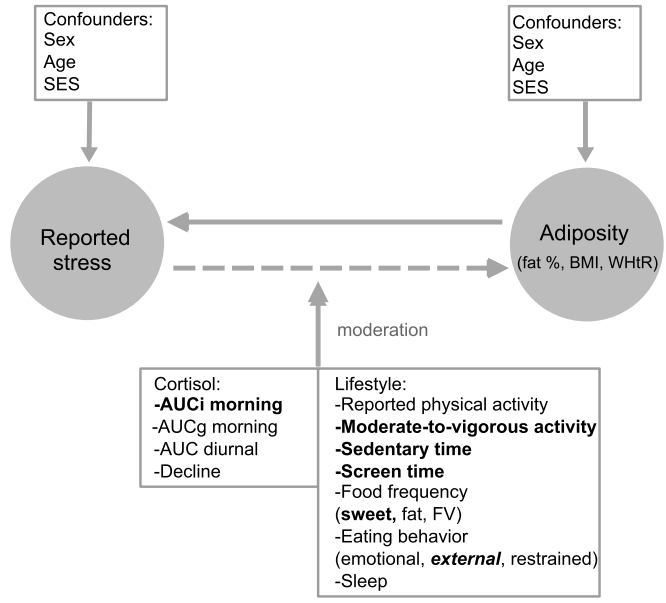

Figure 1. Hypotheses in this study. Hypothesized stress-adiposity relations testing bidirectionality and moderation by cortisol and life-style are shown. The cortisol and life-style parameters in bold were the significant moderators in the stress effects on adiposity. The life-style parameter in bold italic was the significant moderator in the adiposity effects on stress. AUC = area under the cortisol curve; AUCg = area under the curve with respect to the ground; BMI = body mass index; FV = fruit and vegetables; SES = socioeconomic status; WHtR = waist-to-height ratio; fat % = fat percentage.

RESULTS

Figure 1 depicts the tested relations: cross-lagged stress-adiposity relations and moderation by cortisol and life-style. Longitudinal data on reported stress and adiposity were available for 316 children. Table 1 shows descriptive data of the reported stress and measured adiposity parameters. A significant increase in negative events and behavioral problems and a significant decrease in negative emotions were observed between W0 and W2. Furthermore, there were changes in adiposity: increased fat %, WHtR, and BMI *z* scores. The overweight prevalence in this cohort was around 8% (following the International Obesity Task Force classification). Concerning socioeconomic status, 55% of the children had at least one parent with tertiary education (ISCED level 5 or higher).

Cross-Lagged Stress-Adiposity Relations

Cross-lagged analyses showed that adiposity (BMI and fat %, $p < .001$) could influence the overall reported stress level over time (see Fig. 2). Nevertheless, the models could not confirm the hypothesis that reported stress influences adiposity longitudinally ($p > .05$). These significances remained when only allowing unidirectional relations. To examine which specific stress aspects were responsible for this relation, the cross-lagged analyses were repeated for the three stress items separately (negative events, negative emotions, and behavioral problems): only negative emotions were found to be related to consequent adiposity. Because the effect of adiposity on stress could conceal a possible effect of stress on adiposity, the analyses were repeated for only those children who were not overweight at baseline, but this did

TABLE 1. Descriptive Data of Stress and Adiposity and Possible Moderators at the Different Study Waves

	2010 (Wave 0)				2011 (Wave 1)				2012 (Wave 2)					
	P25	P50	P75	*n*	P25	P50	P75	*n*	P25	P50	P75	*n*	p^a	*n* for Analysis[b]
Reported stress														
Negative events (score)	10	40	73	491	47	97	157	418	24	63	106	320	<.001	316
Negative emotions (0–30)	3	7	11	491	3	6	10	418	2	5	9	320	<.001	316
Behavioral problems (0–30)	2	5	8	491	4	7	11	418	4	7	10	320	<.001	31
Adiposity														
Body mass index *z* score	−0.82	−0.14	0.46	523	−0.84	−0.19	0.48	453	−0.86	−0.21	0.49	330	.016	316
Body fat percentage	14.8	18.5	23.6	497	14.8	18.6	24.0	453	15.4	19.7	24.5	330	.003	316
Waist-to-height ratio	0.42	0.44	0.46	518	Not measured				0.48	0.50	0.52	319	<.001	305
Tested moderators														
Physical activity/inactivity														
Screen time, h/wk	6.8	9.5	15.3	483	7.0	9.9	16.1	391	8.5	12.3	17.8	323	<.001	316
Reported activity, h/wk	9.5	13.3	18	488	9.4	13.2	18	397	9.5	13.5	17.5	308	.45	316
Sedentary time by accelerometer, %	47.3	51.9	56.1	366	Not measured				53.1	58.8	63.5	153	<.001	316
MVPA time by accelerometer, %	5.3	7.1	9.3	366	Not measured				4.4	5.7	7.5	153	<.001	316
Sleep														
Sleep duration, h/night	10.5	11	11.3	333	Not measured				10.3	10.6	11	258	<.001	316
Food consumption frequency, consumptions/wk														
Fatty foods	19	25	33	375	19	26	36	341	20	27	37	297	.030	316
Sweet foods	21	29	39	375	21	29	39	341	22	29	39	305	.13	316
Fruit and vegetables	11	14	18	375	11	14	19	341	11	14	21	305	.043	316
Eating behavior (Dutch Eating Behavior Questionnaire)														
Emotional eating (1–5)	Not measured				1.4	1.9	2.5	438	1.2	1.6	2.2	312	<.001	316
External eating (1–5)	Not measured				2.7	3.1	3.6	438	1.4	2.0	2.6	312	<.001	316
Restrained eating (1–5)	Not measured				1.6	2.2	2.7	438	2.5	3.0	3.5	312	.002	316
Salivary cortisol														
AUCi	−6.12	−1.66	3.05	439	Not measured				Not measured					316
AUCg	17.96	22.29	28.18	439	Not measured				Not measured					316
Diurnal decline	−0.99	−0.79	−0.61	439	Not measured				Not measured					316
Daily output	255022	320817	413127	439	Not measured				Not measured					316

MVPA = moderate-to-vigorous physical activity; AUCi = area under the morning curve with respect to increase; AUCg = area under the morning curve with respect to the ground; P25 = 25th percentile; P50 = 50th percentile; P75 = 75th percentile.

[a] Wilcoxon signed rank *p* value for individual change in 2010 to 2012.

[b] Available sample size for the current analyses.

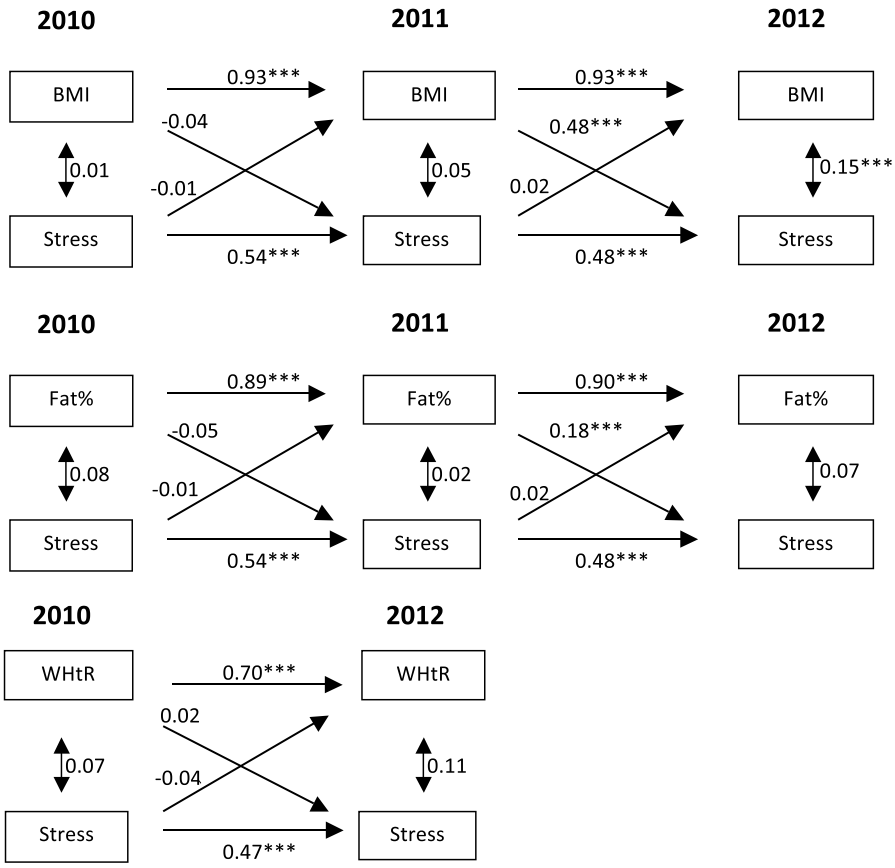

Figure 2. Longitudinal cross-lagged models for the stress-adiposity relation. Standardized coefficients are given for forward and reverse associations between adiposity and the composite stress score. The results of the cross-lagged associations between the 2011 and 2012 wave will mainly be interpreted because these are corrected for all the earlier factors and associations. Fit indices were as follows: CFI = 0.983 and RMSEA = 0.024 for body mass index, CFI = 0.978 and RMSEA = 0.026 for fat %, and CFI = 0.914 and RMSEA = 0.045 for waist-to-height ratio. The models are adjusted for age, sex, and socioeconomic status. CFI = comparative fit index; RMSEA = root mean square error of approximation. * $p < .05$, ** $p < .01$, *** $p < .001$.

not change the results. Moreover, multigroup analyses showed that the models were not different for sex and age groups (χ^2 difference $p > .05$ and CFI difference <0.01).

Moderation by Cortisol

Significant stress-cortisol interactions in predicting adiposity (=cortisol moderation) are depicted in Figure 3. Only in the case of a high AUCi in W0, there was a positive longitudinal association between reported stress in W0 and central adiposity (WHtR) in W2. To investigate which specific stress aspects were responsible for this relation, the cross-lagged analyses were separately performed for the three separate stress items: again, only the negative emotions provided significant results. For behavioral problems, the diurnal decline and the $AUC_{diurnal}$ were also significant moderators. Furthermore, cortisol was not a moderator in the opposite direction, that is, in adiposity-induced stress or a predictor for longitudinal adiposity changes.

Moderation by Life-style

Life-style factors could moderate the longitudinal stress-adiposity relation.

Figure 4 shows that screen time W1, sedentary time W0, moderate-to-vigorous activity W0, and sweet food consumption W1 were significant moderators in the effects of stress on adiposity W2. Once more, the cross-lagged analyses were repeated for the three stress items separately to explore which aspects of stress might be responsible for this effect: results were significant

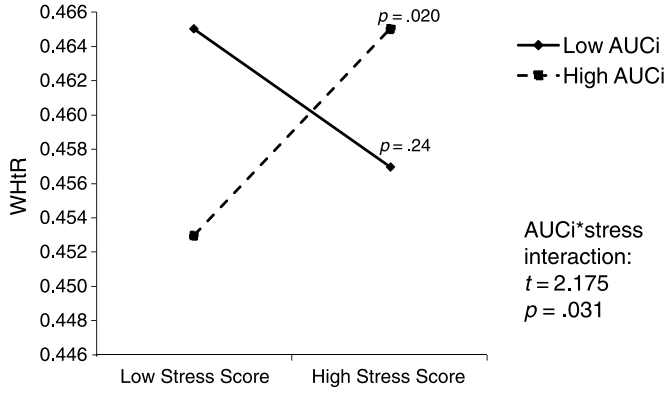

Figure 3. Cortisol as a moderator in the longitudinal stress effects on adiposity. The graph illustrates the association in which cortisol was a significant moderator. The longitudinal effect of reported stress on adiposity (only significant for WHtR) between Wave 0 and Wave 2 is visualized for moderator (at Wave 0) and reported stress levels (at Wave 0) 1 standard deviation above the mean (=high) and 1 standard deviation below the mean (=low). If the stress–body composition relation was significant in one of the two subgroups of the moderator (groups with score above or below the mean cortisol), those significance and β values are also given. AUCi = area under the curve of morning samples with respect to increase; WHtR = waist-to-height ratio.

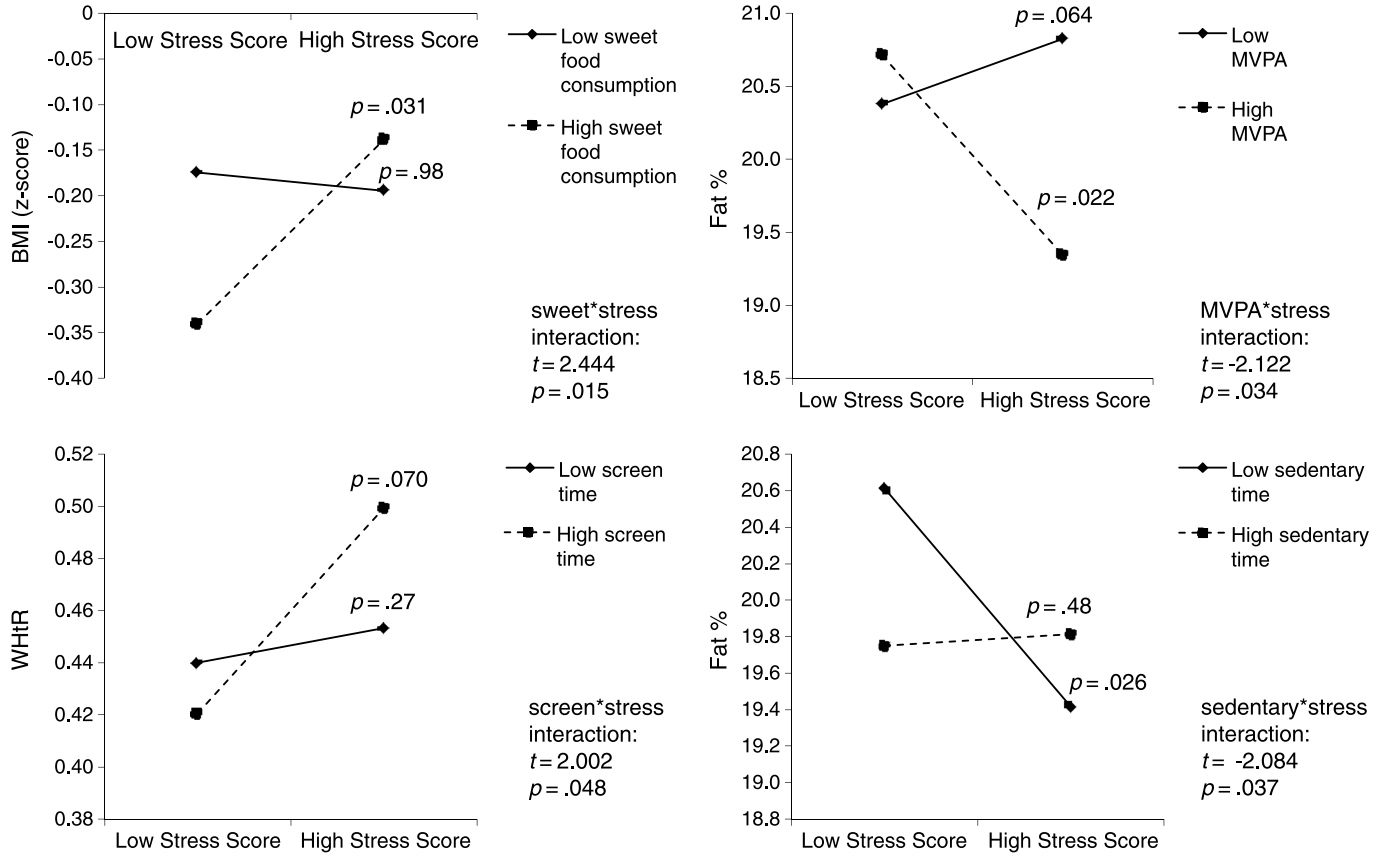

Figure 4. Life-style as moderator in the longitudinal stress effects on adiposity. Graphs illustrate the four associations in which life-style was a significant moderator as found by interaction in Mplus cross-lagged analyses: stress * sedentary time and stress * sweet food consumption at Wave 1 on adiposity Wave 2; stress * moderate-to-vigorous activity and stress * sedentary time at Wave 0 on adiposity Wave 2 (because these moderators were not available at Wave 1). The longitudinal effect of reported stress on adiposity (BMI, fat %, or WHtR) is visualized for moderator and stress levels 1 standard deviation above the mean (=high) and 1 standard deviation below the mean (=low). If the stress–body composition relation was significant in one of the two subgroups of the moderator (groups with score above or below the mean of the life-style factor), those significance and β values are also given. Note: The interaction graph concerning sedentary behavior seems to indicate that fat % will be the highest in children with low stress and low sedentary behavior. However, post hoc analyses showed that this was not significant; only the pure interaction; that is, the direction of the two lines (with given p values) should be interpreted. BMI = body mass index; fat % = fat percentage; WHtR = waist-to-height ratio; MVPA = moderate-to-vigorous physical activity.

for negative emotions (moderator sweet food, screen time, sedentary time), negative events (moderator sweet food), and behavioral problems (moderator moderate-to-vigorous activity). Moderation was not significant for the other tested life-style parameters (fatty food consumption W1, fruit and vegetables W1, eating behavior W1, self-reported physical activity W1, and sleep W0). These moderations show that reported stress was associated with higher adiposity in children with a high sweet food consumption frequency and long screen time. In contrast, low sedentary time or high moderate-to-vigorous activity lead to an adiposity-lowering effect of reported stress.

Although this was not mentioned in the main research question, a life-style moderation was also found in the relation between adiposity W1 and stress W2. Namely, adiposity seemed to cause higher levels of stress in children with frequent external eating but not in those with low external eating (see Fig. 5).

DISCUSSION

The aim of this study was to examine the bidirectional relation between stress and adiposity in approximately 300 primary school children. In addition, we tested the moderation of cortisol and life-style in this relation. Evidence was found of a direct relation in the adiposity-stress direction: adiposity was related to higher stress reports in a longitudinal way (especially for negative emotions). For this adiposity-stress relation, external eating was a moderator

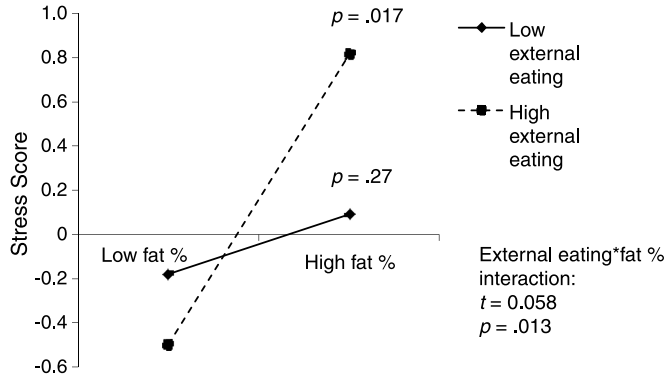

Figure 5. Life-style as a moderator in the longitudinal adiposity effects on stress. The graph illustrates the adiposity-stress association in which life-style was a significant moderator as found by interaction in Mplus cross-lagged analyses: adiposity * external eating at Wave 1 on stress Wave 2. The longitudinal effect of adiposity (fat %) on reported stress is visualized for moderator and stress levels 1 standard deviation above the mean (=high) and 1 standard deviation below the mean (=low). Fat % = fat percentage.

(enhancer). In the stress-adiposity relation, no direct effect was found, but we observed a stress effect on adiposity when considering cortisol and life-style as moderators. Reported stress was associated with increased adiposity in children with a high cortisol awakening response (i.e., high stress reactivity), high sweet food consumption, or long screen time, whereas stress was associated with lower adiposity in those with low sedentary time or high moderate-to-vigorous activity.

Adiposity Influences Stress

Even in this population with low overweight prevalence, body composition had an impact on reported stress because overall adiposity (BMI and fat %) induced stronger negative emotions. Several underlying pathways in the effect of adiposity on stress have been suggested in the literature. Physiologically, adiposity increases circulating proinflammatory cytokines and leptin levels which may stimulate cortisol secretion (9). Psychologically, obese people may face lower self-esteem and negative emotions because of external stigma, internal body image dissatisfaction, failing repeated dieting, functional impairment, and lower self-rated health (60). Indeed, mainly negative emotions were predicted by adiposity in our sample. In the older-age group of our study sample, behavioral problems could also be predicted by adiposity. The latter finding is in line with a previous study in 3- to 5-year-olds (61). In adiposity-induced stress, external eating was a significant moderator (enhancer). This habit of external eating, that is, eating in response to food may hinder the control of their body weight and could as such increase the adiposity-related psychological stress. After all, external eating has been shown to increase anxiety feelings in this population (62).

Moderation in the Influence of Stress on Adiposity

In the opposite cause-effect direction, no significant direct relationship was found, but our results revealed significant relations of stress with subsequent overall and central adiposity parameters when considering the moderation by life-style and cortisol. Of course, longitudinal effects of stress on adiposity could be thwarted if adiposity-controlling life-style and hormonal factors are differentially influenced by stress (14–23), depending on individual characteristics (sex, age, stress reactivity, behavioral preferences, and eating behavior) and/or exposure characteristics (type and severity of stressor) (17,63–65). These individual differences in stress-life-style relations were also found in our child cohort (Michels, in press). Because of this moderation, an unhealthy life-style (high sweet food consumption and screen time) may make children more prone to stress-induced adiposity or, vice versa, a healthy life-style (lower sedentary time and higher moderate-to-vigorous activity) may attenuate the pivotal stress effects on adiposity or even lower adiposity.

Until now, the evidence on intermediate and moderating factors in the stress-adiposity relation is scarce in the literature (10). In a recent review, an association between high screen time and poorer mental health was reported in children (18). Other research has also indicated that the stress impact on screen time could depend on their usual level of screen time (17) and on stress reactivity (66). Although no moderating effect of sedentarism has been described in the literature, physical activity was found to buffer (i.e., moderate) the effect of stress on adiposity and metabolic syndrome in adolescents (25,26) or even on overall health (67).

The stress-diet relation is even more theoretically grounded. Stressed people may eat increased amounts of unhealthy food rich in sugar or fat because eating is a way to cope with stress (64) and stress can influence reward and appetite pathways (20–23). In children, comfort food may be defined as mainly food with a sweet taste as was shown in cross-sectional relations with cortisol and stress in our population (68,69). Some specificity in this relation has been suggested with eating being stimulated by mostly moderate, not very intense emotions and depending on the person's characteristics, for example, their eating behavior (64). Indeed, our reported moderation reflects specificity: only children who have an unhealthy diet with high sweet food consumption are vulnerable to increased adiposity.

Moderation by cortisol (i.e., only children with elevated cortisol are susceptible to stress-induced adiposity) might be explained by the fact that stress reports are not always associated with chronically elevated cortisol patterns but only in certain stress situations and certain people (42). This moderating effect of cortisol encourages further research on determinants for chronically elevated cortisol levels to identify those children who are the most vulnerable to stress-induced adiposity. Moderation by cortisol in the overall stress-adiposity relation was only seen when using the cortisol awakening response (AUCi), the parameter that reflects the pure increase and as such the cortisol system sensitivity, which has been positively associated with general life stress (70). Nevertheless, the total amount of secreted cortisol is also important because post hoc analyses showed diurnal decline and whole-day secretion ($AUC_{diurnal}$) as significant moderators in the relation between problem behavior and adiposity. The cortisol moderation was found specifically for the outcome of central adiposity, which is theoretically grounded because stress might preferentially lead to central adiposity due to the higher density of cortisol receptors in the abdominal region (13). A study in 8- to 11-year-old girls confirmed cortisol as a moderator in the relation between stress events and abdominal fat: a higher number of school-related negative events were related to more abdominal fat for girls with a high cortisol awakening response, but such an association was not found for girls with a low cortisol awakening response (24).

Comparison With Our Cross-Sectional Results

Finally, we would like to compare these longitudinal findings with our recently published cross-sectional findings on the same population sample (71). The main results of the cross-sectional and longitudinal analyses are similar: a significant relation between stress and adiposity with moderation by physical activity, diet, and cortisol. However, there are also some differences with the present longitudinal study. First, sleep was a significant moderator only in the cross-sectional findings. Second, the specificity differs; for example, negative emotions were significant predictors in the longitudinal analyses but not in the cross-sectional analyses, whereas the positive emotion "happiness" was significant only at cross-sectional level.

Several reasons could exist for the different results when comparing cross-sectional and longitudinal analyses. First of all, a longitudinal analysis is more likely to suggest cause-effect relationships and reflect directionality. However, our longitudinal analyses suffer from a lower statistical power because the sample size decreased during follow-up. Time-effect reasons might also exist: there may only be an acute effect (i.e., no long-term effect) or the relation may be age dependent. This time-effect reason might be the case for findings on sleep because the stress-sleep relation was only significant in cross-sectional analyses. To conclude, these findings confirm the need for more longitudinal studies because such studies might reveal more real effects.

Strengths and Limitations

A central asset of this study is the longitudinal design revealing the directionality of the relation, in contrast to the literature. Moreover, this is one of the first studies examining the stress-adiposity relation in children while at the same time considering the moderating role of biological stress reactivity measures and life-style factors. Several stress concepts, life-style factors, and adiposity measures (both central and overall adiposity) have been measured.

Caution should be taken when generalizing the results to the overall population because cultural variations have been described in stressors and coping (72) and because our population showed a low overweight prevalence of 8%. Stronger associations between stress and adiposity might be encountered in more overweight populations. A considerable dropout was experienced during follow-up, although this did not introduce large bias. In addition, no information was available on parental health behavior as a contextual factor, although their behavior might influence the children's behavior or bias the report on their child's behavior. Finally, the relatively short follow-up of 2 years may be insufficient to see full effects.

CONCLUSIONS

The results of this study demonstrate the importance of moderation: stress is associated with increased adiposity when children have high cortisol awakening patterns and/or an unhealthy life-style (high sweet food consumption and low physical activity). Although stress is not always inevitable, the way people cope with stress can be targeted. This emphasizes the value of incorporating education on stress management in obesity prevention programs. Because life-style behavior can also be applied as a coping mechanism and because it moderates the stress-adiposity relation, multifactorial obesity prevention programs should be created by focusing concurrently on stress and life-style. Children and their parents should realize the effects of stress on their diet, and an activity-friendly environment should be created to minimize sedentary time.

Notably, our results also highlighted the unfavorable effect of adiposity on psychological health. This underlines the need for a transdisciplinary obesity treatment with a special focus on psychological support.

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