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Elsenburg, Leonie K.; Smidt, Nynke; Liefbroer, Aart C.

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The Longitudinal Relation Between Accumulation of Adverse Life Events and Body Mass Index From Early Adolescence to Young Adulthood

Leonie K. Elsenburg, MSc, Nynke Smidt, PhD, and Aart C. Liefbroer, PhD

ABSTRACT

Objective: Stressors, such as adverse life events, can cause weight changes through behavioral and biological mechanisms. Whether the accumulation of adverse life events is related to body mass index (BMI) across multiple time points from early adolescence to young adulthood has not been investigated to date.

Methods: Data are from 2188 children participating at T1 (10–12 years), T3 (14–18 years), and/or T5 (21–23 years) of the prospective Tracking Adolescents' Individual Lives Survey cohort study. Adverse events before T1 and between T1, T3, and T5 were measured with a parent interview at T1 and a semistructured interview (Event History Calendar) with the adolescent at T3 and T5. An adverse events score was calculated per wave. Body mass index z-scores were determined from objectively measured height and weight using the LMS (skewness, median, and coefficient of variation) reference curves of the International Obesity Task Force for children 18 years or younger. Data were analyzed using a modified bivariate autoregressive cross-lagged structural equation model.

Results: Adverse events before T1 and between T3 and T5 were related to BMI at T5 ($\beta = 0.06$, $p = .001$ and $\beta = -0.04$, $p = .04$, respectively). Specifically, health events before T1 were associated with a higher BMI at T5, and events related to relationships and victimhood events between T3 and T5 were associated with a lower BMI at T5.

Conclusions: Adverse relationship and victimhood events in their recent past were related to a lower BMI in young adults, whereas adverse health events during childhood were related to a higher BMI in young adults. No relationships were found between adverse life events with BMI in children and adolescents.

Key words: life events, body mass index, children, adolescents, young adults.

INTRODUCTION

Over the past 30 years, there has been an alarming global increase in rates of childhood overweight and obesity (1). Children with a higher body mass index (BMI) have an elevated risk of long-term morbidity and mortality from diseases such as coronary heart disease and type 2 diabetes (1–4). Identifying determinants of childhood overweight and obesity is crucial to prevent these conditions and associated disorders.

Recent studies into possible determinants of childhood overweight and obesity have focused on the potentially crucial role of stressors (5,6). Stressors are suggested to influence weight via 2 mechanisms (7–9). In response to stressors, children may change their health behaviors, for example, by reducing their physical activity levels, increasing sedentary behavior, or increasing (unhealthy) food intake. Second, stressors can cause biological changes in the body (10). Hormones released in response to stress can influence fat deposition in the body or influence health behaviors related to weight change (11,12). Two recent reviews on the

BMI = body mass index, **CFI** = comparative fit index, **EHC** = Event History Calendar, **IOTF** = International Obesity Task Force, **ISCO** = International Standard Classification of Occupations, **RMSEA** = root mean square error of approximation, **SD** = standard deviation, **SES** = socioeconomic status, **TLI** = Tucker-Lewis Index, **TRAILS** = Tracking Adolescents' Individual Lives Survey

SDC Supplemental Content

From the Department of Epidemiology (Elsenburg, Smidt, Liefbroer), University of Groningen, University Medical Center Groningen, Groningen, the Netherlands; Netherlands Interdisciplinary Demographic Institute (NIDI-KNAW) (Elsenburg, Liefbroer), The Hague, the Netherlands; and Department of Sociology, Vrije Universiteit Amsterdam (Liefbroer), Amsterdam, the Netherlands.

Address correspondence and reprint requests to Leonie K. Elsenburg, MSc, Department of Epidemiology, University Medical Center Groningen, Netherlands Interdisciplinary Demographic Institute, Lange Houtstraat 19, PO Box 11650, 2502 AR, The Hague, the Netherlands. E-mail: elsenburg@nidi.nl

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relationship between stress(ors) and overweight concluded that although a relationship seems to exist, the literature is inconsistent and incomplete (5,6). An important and rather easily identifiable set of stressors that might affect children's weight status are adverse life events, such as parental divorce, illness or death of a family member, and the experience of being bullied. Studying the effect of isolated adverse events on medical conditions is, however, suggested to overestimate the effect of that event due to its potential interrelatedness with other childhood adversities (13–15). Therefore, it is important to study the effect of the accumulation of adverse childhood events on BMI. However, most studies on this topic have been cross-sectional in nature (16–27). No studies have yet examined whether accumulation of adverse events is related to BMI across multiple time points between early adolescence and young adulthood.

Longitudinal studies to date did not focus specifically on the relation between accumulation of adverse events and BMI but instead examined the relation between BMI and cumulative risk, a composite measure that combines information on adverse events with information on housing problems, behavioral problems, and/or living in a single-parent household. No relationship between cumulative risk and BMI was found in children (28,29). One previous longitudinal study in adolescents did find a positive relation between trajectories of cumulative risk and trajectories of BMI (15). Adolescence is a period marked by major transitions in children's lives, socially, emotionally as well as physically, making adolescence a particularly interesting period to study factors associated with changes in BMI (30). In the current study, the effect of accumulation of adverse events on BMI from early adolescence to young adulthood is studied. This is the first study to examine this relationship across multiple time points in this age group. The possible bidirectionality of the relation between adverse events and BMI is taken into account (31). These relationships are examined, adjusting for household socioeconomic status (SES) (32). Whether specific types of events account for the relationship between accumulation of adverse events and BMI is also studied.

METHODS

Subjects were participants in the Tracking Adolescents' Individual Lives Survey (TRAILS) (33,34). Tracking Adolescents' Individual Lives Survey is a prospective cohort study following Dutch adolescents aged 10 to 12 into young adulthood. At the start of the study, children born between either October 1, 1989, and September 30, 1990, or between October 1, 1990, and September 30, 1991, were identified in 5 municipalities in the North of the Netherlands (n = 3483). These children were approached if their primary school agreed to participate (n = 3145). Exclusion criteria of the study were no parental or child consent, severe physical illness or mental retardation, and no Dutch-, Turkish- or Moroccan-speaking parent or parent surrogate available (n = 210). A total of 2230 children were included in the study at T1 (76.0% of eligible children in participating schools). Five waves of data collection were conducted, each 2 to 3 years apart. In the current study, data from T1, T3, and T5 are used. Data were collected from March 2001 to July 2002 (T1), September 2005 to December 2007 (T3), and April 2012 to November 2013 (T5). Mean ages of the children at these waves were 11.1, 16.3, and 22.4 years.

Adverse Events Measure

Adverse event occurrence from birth to T1 was measured using a parental interview at T1. The occurrence of adverse events between T1 and T3 and between T3 and T5 was measured with a semistructured interview, the Event History Calendar (EHC), with the adolescent at T3 and T5 (35).

Adverse events measured at T1 were hospital admission of the child; physical or mental illness of father or mother; death of a family member, friend, or loved one; parental divorce; and out-of-home placement. At T3, additionally measured events were suspension; running away from home; physical or mental illness of a family member, friend, or loved one; addiction of a family member, friend, or loved one; loss of friendships; fights with friends or family members; breaking up with a boyfriend or girlfriend; being a victim of bullying, gossiping, sexual intimidation or violence; and having had contact with the police. Due to the older age of the participants, certain events were no longer measured at T5, but several new adverse events were added to the interview, such as miscarriage; abortion; unemployment; disappointment in a partner, family member, friend, or colleague; financial setback; and being a victim of theft (for a complete list of assessed events, see Table S1, Supplemental Digital Content 1, http://links.lww.com/PSYMED/A327).

An adverse events score per wave was calculated by summing up the number of adverse events reported at T1, T3, and T5, respectively. Identical events that occurred more than once before T1, in between T1 and T3, or in between T3 and T5 were counted 3 times at maximum.

Body Mass Index

Children's weight and height were measured using calibrated scales (Seca 770, Hamburg, Germany at T1 and T3; Seca 876, Hamburg, Germany, and Besthome EB813-SL at T5) and stadiometers/measuring tapes (Seca 214, Hamburg, Germany at T1 and T3; Seca 201/222, Hamburg, Germany at T5). Body mass index was calculated by dividing weight by height squared (kg/m^2). Body mass index scores were standardized. For adolescents 18 years or younger, these standardized scores were determined based on the age- and sex-specific skewness, median, and coefficient of variation (LMS) reference curves of the International Obesity Task Force due to the dependency of BMI on age and sex in childhood (36). In addition, weight status of the respondents was determined. For adolescents, weight status was divided into thinness, normal weight, overweight, and obesity using the SD cutoffs of the International Obesity Task Force corresponding to the adult cutoffs of less than 18.5 kg/m^2, 18.5 to less than 25 kg/m^2, 25 to less than 30 kg/m^2, and 30 kg/m^2 or greater, respectively (36). Young adults were classified as underweight, normal weight, overweight, or obese using the adult cutoffs (37).

Covariates

Information on children's age, sex, SES, and ethnicity was obtained from parents at T1. Socioeconomic status was measured by taking the mean of the standardized scores for maternal and paternal education (in 5 categories from elementary to university education), maternal and paternal occupation (using the International Standard Classification of Occupations (38)) and household income (39). As most of the participants were Dutch, ethnic background was measured by a categorical variable indicating whether or not both parents were born in the Netherlands.

Pubertal status at T1 was assessed by asking parents to identify the stage of their child's pubertal development using schematic drawings of the 5 Tanner stages of pubertal development (40,41). At T3, adolescents rated their own level of physical development using the Physical Development

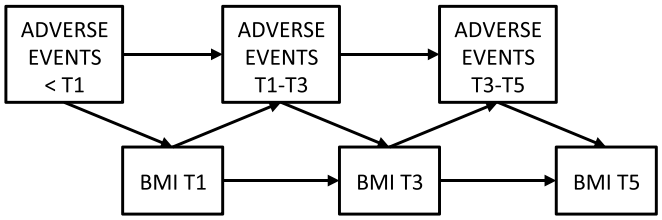

FIGURE 1. Modified bivariate autoregressive cross-lagged model of the hypothesized relation between adverse events and BMI.

Scale questionnaire (42). Scores on this questionnaire were converted to Tanner stages (43).

Statistical Analysis

A modified bivariate autoregressive cross-lagged structural equation model was estimated to test the hypothesized relation between adverse events and BMI (Fig. 1). To adjust for possible confounding by age, sex, SES, and ethnicity, paths from these variables to all adverse events and BMI scores were added. Additionally, BMI scores at T1 and T3 were adjusted for pubertal status at T1 and T3. Paths were also allowed between age at T1, T3, and T5 and between age and pubertal status at both T1 and T3. Differences in the path coefficients of the final model between boys and girls were tested using Wald tests. Furthermore, the model was tested using 3 specific types of accumulated events. These 3 types were (1) health events, (2) relationship events, and (3) victimhood events (for the division of events into types, see Table S1, Supplemental Digital Content 1, http://links.lww.com/PSYMED/A327).

Three sensitivity analyses were performed. In the first 2 sensitivity analyses, the cutoff point for the maximum number of occurrences of the same event in between waves was set to 5 and 1, respectively, instead of 3.

TABLE 1. Descriptive Statistics of the Study Sample at the Different Study Waves

	T1	T3	T5
n[a]	2188	1633	1455
Age, M (SD), years	11.11 (0.55)	16.25 (0.67)	22.44 (0.60)
Sex			
Girls, %	51.0	52.7	55.0
SES z-score,[b] M (SD)	−.05 (0.80)	0.04 (0.79)	0.11 (0.76)
Ethnicity			
Dutch, %	86.6	88.4	89.5
Non-Dutch, %	13.4	11.6	10.5
Pubertal status (Tanner stage), M (SD)	1.87 (0.76)	2.77 (0.67)	Not measured
Adverse events score,[c] M (SD)	2.38 (1.82)	6.02 (3.71)	5.99 (3.75)
0 event, %	13.8	2.2	1.3
1 event, %	21.8	5.5	4.9
2 events, %	23.6	8.1	9.3
3 events, %	18.0	12.5	11.0
≥4 events, %	22.8	71.6	73.5
Relationship events, M (SD)	0.26 (0.54)	2.80 (2.43)	3.21 (2.57)
Health events, M (SD)	2.13 (1.71)	1.71 (1.49)	1.79 (1.58)
Victimhood events, M (SD)	not measured	1.22 (1.36)	0.44 (0.71)
BMI z-score,[d] M (SD)	0.12 (1.04)	0.28 (0.95)	0.00 (0.99)
BMI categories[e]			
Thinness/Underweight, %	13.7	7.4	3.4
Normal weight, %	71.7	78.1	68.7
Overweight, %	12.1	11.7	20.8
Obesity, %	2.4	2.9	7.1

SES = socioeconomic status, BMI = body mass index.

[a] Sample size per variable ranged between 2114 and 2188, 1494 and 1633, and 1424 and 1455 at T1, T3, and T5, respectively. [b] Socioeconomic status z-score is the mean of z-scores for maternal and paternal education, maternal and paternal occupation (38), and household income (39). [c] Adverse events were measured differently at T1 compared to T3 and T5; and therefore, mean and standard deviation values of T1 are lower. [d] Body mass index z-scores at T1 and T3 were determined based on external reference curves, whereas BMI z-scores at T5 were derived by standardization; therefore, only the BMI z-scores at T5 have a mean of 0 and standard deviation of 1. [e] Differences in the adequacy of the international (SD) cutoffs of the BMI categories for Dutch children (36) and young adults (37) can result in different percentages of children in the different BMI categories between the waves.

In the third sensitivity analysis, BMI z-scores were replaced by BMI categories (thinness/underweight, normal weight, overweight, and obesity).

All analyses were performed in MPlus (version 7.3). Models were run using maximum likelihood estimation with robust standard errors. Model fit was considered good if the comparative fit index (CFI) and the Tucker-Lewis Index (TLI) were both 0.95 or greater and the root mean square error of approximation (RMSEA) was 0.06 or less (44). The χ^2 statistic was evaluated, but due to the relatively large sample size of this study, the statistical significance of χ^2 was not used as a definitive indicator of model fit. Full information maximum likelihood was used to handle missing data. As level of significance, $p < .05$ was used.

RESULTS

Descriptive data of the sample (n = 2188 at T1) are presented in Table 1. The sample consisted of nearly as many boys as girls (49% vs 51% at T1). On average, children experienced 2.4 adverse events before T1 and 6.0 adverse events between T1 and T3 and between T3 and T5. At all waves, most children had normal weight (69–78%). At T1 and T3, approximately 12% was overweight and 3% was obese. At T5, these numbers were 21% and 7%. The percentage of thin/underweight participants at the waves was 14%, 7%, and 3%, respectively.

The model fit of the initial model examining the autoregressive and cross-lagged associations between adverse events with BMI, adjusted for age, sex, SES, ethnicity, and pubertal status, was not good (RMSEA = 0.050, CFI = 0.947, and TLI = 0.899). To establish good model fit, 3 paths were added to this model: (1) adverse events score before T1 to adverse events score between T3 and T5, (2) BMI at T1 to BMI at T5, and (3) adverse events score before T1 to BMI at T5 (Fig. 2). In addition, ethnicity and pubertal status were removed from the model. Removal of pubertal status from the model resulted in improvement of the model fit above the pre-established criteria for good model fit. Exclusion hardly changed the path coefficients of the core model. Model fit of the final model was good: RMSEA = 0.019, CFI = 0.996, and TLI = 0.990. The χ^2 statistic was significant ($\chi^2(19) = 33.432$; $p = .021$), but this is probably due to sensitivity of the χ^2 test to large samples.

The bivariate correlations between adverse events with BMI ranged from 0.015 ($p > .05$) to 0.089 ($p < .01$) (Table S2 in Supplemental Digital Content 1, http://links.lww.com/PSYMED/A327). We identified significant autoregressive paths of both adverse events and BMI from T1 to T3 and T5 and from T3 to T5. We identified a positive relation between adverse events before T1 and BMI at T5 and a negative relation between adverse events between T3 and T5 and BMI at T5 (Fig. 2, Table 2). There were no significant indirect effects, except for the indirect effect of adverse events between T1 and T3 via adverse events between T3 and T5 on BMI at T5 ($\beta = -0.011$, $p = .041$). Running the model stratified for sex revealed a significant difference between the model for boys and the model for girls ($p < .001$). Testing equality of all path coefficients of the core model separately revealed only one significant difference. The autoregressive path of BMI from T1 to T3 was stronger for boys than for girls ($p = .004$), although the difference was small ($\beta_{boys} = 0.788$, $p < .001$; and $\beta_{girls} = 0.751$, $p < .001$).

Models in which the total adverse events score was replaced by different types of accumulated events showed that health events before T1 ($\beta = .058$, $p = .001$) and relationship and victimhood events between T3 and T5 were related to BMI at wave 5 ($\beta = -.054$, $p = .003$; and $\beta = -.045$, $p = .009$). Model results and bivariate correlation matrices of these models are found in Tables S3 to S8 in Supplemental Digital Content 1, http://links.lww.com/PSYMED/A327.

Sensitivity Analyses

Sensitivity analyses with the cutoff for the maximum number of included occurrences of the same event in between waves set to 1 and 5 rather than 3 revealed no differences, except that adverse events between T3 and T5 and BMI at T5 were no longer related when the cutoff was set to 1 ($\beta = -.027$, $p = .155$). In this model, the indirect effect of adverse events between T1 and T3 via adverse events between T3 and T5 was also nonsignificant. Using BMI categories as the outcome instead of BMI z-scores revealed no

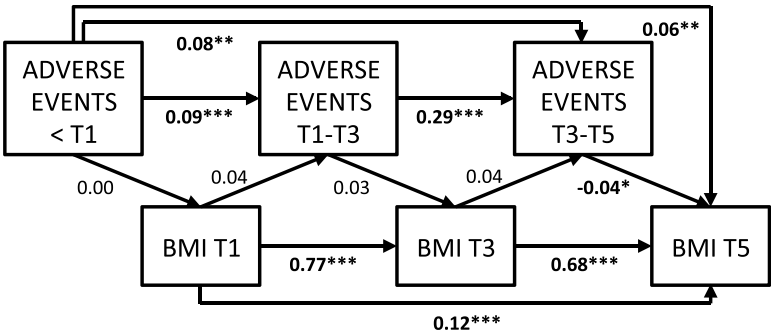

FIGURE 2. Paths and standardized path coefficients of the final adjusted bivariate autoregressive cross-lagged model of the relation between adverse events and BMI (n = 2188). The model is adjusted for age, sex, and SES. Significant paths are shown in bold, *$p < .05$, **$p < .01$, ***$p < .001$. Model fit indices: RMSEA = 0.019, CFI = 0.996, TLI = 0.990.

TABLE 2. Autoregressive and Cross-lagged Associations Between Accumulated Adverse Events and BMI from Childhood to Adolescence and Early Adulthood (n = 2188[a])

Relationship	Path Coefficient (β)	Standard Error	p Value
Adverse events score before T1 to			
adverse events score T1-T3	0.090	0.025	**<.001**
adverse events score T3-T5	0.079	0.027	**.004**
BMI z-score T1	0.002	0.023	.932
BMI z-score T5	0.059	0.017	**.001**
Adverse events score T1-T3 to			
adverse events score T3-T5	0.288	0.028	**<.001**
BMI z-score T3	0.026	0.018	.138
Adverse events score T3-T5 to			
BMI z-score T5	−0.039	0.019	**.042**
BMI z-score T1 to			
BMI z-score T3	0.766	0.011	**<.001**
BMI z-score T5	0.117	0.028	**<.001**
adverse events score T1-T3	0.036	0.026	.158
BMI z-score T3 to			
BMI z-score T5	0.676	0.027	**<.001**
adverse events score T3-T5	0.038	0.028	.176
Age T1 to			
adverse events score before T1	0.039	0.021	.067
BMI z-score T1	−0.010	0.021	.619
age T3	0.492	0.020	**<.001**
age T5	0.356	0.020	**<.001**
Age T3 to			
adverse events score T1-T3	0.188	0.027	**<.001**
BMI z-score T3	0.003	0.017	.883
age T5	0.483	0.022	**<.001**
Age T5 to			
adverse events score T3-T5	0.063	0.026	**.015**
BMI z-score T5	0.040	0.018	**.029**
SES to			
adverse events score before T1	−0.128	0.021	**<.001**
adverse events score T1-T3	−0.183	0.028	**<.001**
adverse events score T3-T5	−0.031	0.027	.255
BMI z-score T1	−0.122	0.021	**<.001**
BMI z-score T3	−0.049	0.017	**.003**
BMI z-score T5	−0.041	0.017	**.019**
Sex (girls = 0, boys = 1) to			
adverse events score before T1	0.037	0.021	.079
adverse events score T1-T3	−0.044	0.024	.068
adverse events score T3-T5	−0.071	0.024	**.004**
BMI z-score T1	−0.060	0.021	**.005**
BMI z-score T3	−0.054	0.016	**.001**
BMI z-score T5	0.030	0.017	.066

BMI = body mass index, SES = socioeconomic status.

[a] Of the 2230 participants, 42 have been excluded due to missing data on SES.

The table shows standardized path coefficients, standard errors, and p values of the final adjusted bivariate autoregressive cross-lagged model (Fig. 2). Significant p values are shown in bold.

differences in the identified relationships. In Tables S9 to S11 in Supplemental Digital Content 1, http://links.lww.com/PSYMED/A327, we have listed how many participants changed from a specific BMI category at T1 or T3 to another BMI category at T3 or T5 and how many participants were in the same BMI category at both waves. Furthermore, we provided the mean number of adverse events the participants in each of these groups experienced, and we provided odds ratios of being in any of these groups versus being in the normal weight group at both waves after experiencing one more adverse event.

DISCUSSION

In the current study, the effect of accumulation of adverse events on BMI across multiple time points from early adolescence to young adulthood was studied. Evidence was found for a small negative relationship between adverse events from 16 to 22 years and BMI at 22 years. Adverse events from 11 to 16 years were also negatively related to BMI at 22 years via adverse events from 16 and 22 years. Furthermore, a small positive relationship between adverse events before age 11 and BMI at age 22 was found. The relationship between adverse events before age 11 and BMI at age 22 could be attributed to health events, whereas adverse relationship events and victimhood events explained the relation between adverse events from 16 to 22 years and BMI at 22 years.

Comparison to Previous Research

No longitudinal relationships between adverse events and BMI in children were found in previous studies (28,29). Our findings corroborated this, as we observed no relationship between adverse events and BMI in 11-year-olds. We found no relationship in adolescents either, which contrasts with an earlier longitudinal study in adolescents that found a positive association between cumulative risk, that is, adverse events together with housing problems, and BMI trajectories (15).

We found effects of adverse events on BMI in early young adults though. Most remarkably, we observed a delayed positive effect of events before age 11 on BMI at 22 years. This effect is in accordance with results from a study on childhood sexual abuse and obesity from childhood to young adulthood (45). The rate of obesity between abused and nonabused females in that study was not significantly different until young adulthood. Another study found that higher childhood stress was associated with a more rapid gain in BMI in adult women (46). Together, these studies indicate that events or stressors in childhood can affect weight changes later in life. This might be due to children not always coping well with events in early childhood. Since children have little autonomy over their health behaviors, such as eating and physical activity, the expression could be delayed to a life stage characterized by increased autonomy over these behaviors, such as young adulthood (30,47). If so, children who experienced high adversity may benefit from learning about methods to cope with adverse experiences at a relatively early age (28,48).

We also found a negative relation between adverse events and BMI in young adulthood. This result was unexpected, as a cross-sectional study in adolescents and young adults found a positive association (26). The difference in findings could be explained by the fact that the participants in this previous study, who had a family history of cardiovascular disease and a high average BMI, may have been prone to react to adversity by increasing their (unhealthy) food intake, whereas young adults in general might be more inclined to eat less. Accumulation of adverse events has previously been related to both binge eating as well as to extreme weight control behaviors in older adolescents and young adults (49). This suggests that accumulation of adverse events can indeed result in both increased and decreased food intake and as such in overweight as well as underweight. The idea that the negative relation might be caused by an overfitted structural equation model seems to be unfounded, as a linear regression analysis with BMI at T5 as dependent variable and adverse events between T3 and T5 as independent variable, adjusted for age at T5, sex, SES, ethnicity, and BMI at T3, generates the same result.

The positive relationship between adverse events before 11 years and BMI at age 22 was attributable to adverse health events. An earlier study found a relation between adverse family health events before 11 years and overweight at age 15, while finding no relationship between adverse family relationship events and overweight (18). Although the relation was detected earlier in life than in the current study, the relation with the different types of events was the same. The negative relation between adverse events between 16 and 22 years and BMI at 22 years in this study was explained by victimhood and relationship events. Mental and physical abuses have previously been related to overweight as well as underweight, dieting, skipping meals, and vomiting in adolescents and young adults (50–53). The effect of relationship events contrasts with studies finding a positive relation between family conflict and BMI in children and with studies finding a higher likelihood of a meaningful increase in BMI after exposure to greater negative aspects of social relationships in adults (54–56). However, studies on the longitudinal relation between adverse relationship events and BMI in early young adults have not been performed to our knowledge.

Strengths and Limitations

Strengths of the current research are its longitudinal design, the analytical procedure, the large sample size, and the

assessment of exposure and outcome. The exposure was measured using a semistructured interview asking participants to recall a large number of adverse events since the last interview. This approach allowed incorporating a more complete set of events than included in most questionnaires (57). In addition, the relatively short recall period reduced the risk of recall bias. The objective assessment of BMI minimized risk of information bias.

A limitation of the current research is that very heterogeneous events were assessed. We decided not to correct for this heterogeneity by applying weights because the same event can affect different children very differently, and applying weights does not correct for this heterogeneity. We did, however, provide separate results for groups of events that are expected to have a similar impact. Another limitation of the current research is that the assessment of events relied on self-report of past events, and this could lead to under- as well as over-reporting of events. This is, however, inevitable in this type of research, and we believe that the method used to measure events minimizes the risk of both reporting and recall bias.

Mechanisms and Future Research

Stressors are suggested to influence weight via changes in health behaviors or via biological reactions in the body (7–9). An important question that remains unanswered in the current research is how accumulation of adverse events causes weight gain or weight loss. Physical activity, sedentary time, sweet food consumption, and cortisol have been shown to moderate the relation between stress and BMI in children (26,28), but whether these factors also serve as mediators, or whether other factors mediate this relation, has not been investigated.

CONCLUSION

In the current study, no longitudinal relation was found between accumulation of adverse events and BMI in children and adolescents, but a relationship was found between events in early childhood and late adolescence and BMI in young adults. These findings suggest that weight changes in response to adverse childhood events only take place at the end of adolescence or beginning of young adulthood rather than earlier in children's lives. It further suggests that the effect of adverse childhood events on physical health may take some time to develop. The exact implications of these findings for the timing and content of obesity prevention programs is an area for further research.

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