Child Psychiatry Hum Dev
DOI 10.1007/s10578-012-0299-9

ORIGINAL ARTICLE

Child and Adolescent Affective and Behavioral Distress and Elevated Adult Body Mass Index

Heather H. McClure · J. Mark Eddy · Jean M. Kjellstrand · J. Josh Snodgrass · Charles R. Martinez Jr.

Abstract Obesity rates throughout the world have risen rapidly in recent decades, and are now a leading cause of morbidity and mortality. Several studies indicate that behavioral and affective distress in childhood may be linked to elevated adult body mass index (BMI). The present study utilizes data from a 20-year longitudinal study to examine the relations between symptoms of conduct disorder, attention-deficit/hyperactivity disorder, and depression during late childhood and mid-adolescence and BMI during emerging adulthood. Data were analyzed using multiple regression. Results suggest that childhood and adolescent problems may influence adult BMI through direct impacts on adolescent overweight, a condition which then persists into adulthood.

Keywords Adolescent · Depression · ADHD · Conduct disorder · Body mass index

Introduction

Obesity rates in the United States and throughout the world have risen rapidly in recent decades, and are a leading cause of morbidity and mortality internationally [1]. Researchers increasingly emphasize obesity as a developmental condition, and seek to

H. H. McClure (✉) · C. R. Martinez Jr.
Center for Equity Promotion, College of Education, University of Oregon, 1571 Alder Street, Eugene, OR 97403, USA
e-mail: hmcclure@uoregon.edu

H. H. McClure · J. M. Eddy · J. J. Snodgrass · C. R. Martinez Jr.
Oregon Social Learning Center, Eugene, OR, USA

H. H. McClure · J. J. Snodgrass
Department of Anthropology, University of Oregon, Eugene, OR, USA

J. M. Eddy
Partners for Our Children, School of Social Work, University of Washington, Seattle, WA, USA

J. M. Kjellstrand
School of Social Work, Columbia University, New York, NY, USA

Published online: 27 March 2012

identify childhood correlates of adult body mass index (BMI), a measure indicating height in proportion to weight (kg/m^2), that is a common indicator of overweight and obesity [2]. Several recent studies indicate that behavioral distress in childhood may be linked to elevated BMI in adulthood [3, 4]. Cross-sectional and short-term studies have found significant associations between elevated weight during emerging adulthood [5], and child and adolescent mental health problems, specifically depressive symptoms and major depression in childhood and adolescence [2, 6], conduct disorder (CD) and anxiety disorder in early adolescence [7], and attention-deficit/hyperactivity disorder (ADHD) symptoms throughout adolescence [8].

Prospective longitudinal studies investigating these links, however, show mixed findings. Some of these studies provide evidence that persistent behavioral problems beginning in childhood and continuing through adolescence predict young adult BMI and obesity [3, 4]. Other studies, however, show no significant longitudinal associations between adult obesity and adolescent behavioral problems, such as depression and externalizing behaviors [9, 10].

Prior studies of child and adolescent predictors of adult BMI have tended to include a single indicator of behavioral distress (e.g., depression), or a general appraisal of child emotional and behavioral problems [e.g., scale scores from Achenbach's Child Behavior Checklist (CBCL)]. At present, little is known about interactions among distinct disorders as they relate to overweight and obesity over the life course. Pine et al. [11] examined both child depression and conduct disorder in relation to adult BMI and found that the relationship between depression and emerging adult BMI was no longer significant when controlling for CD. Because of the complex interrelationships between social and biological determinants and psychopathology [8, 12, 13], the current study seeks to map out potential direct and indirect relationships among these factors.

Specifically, the study examines interrelationships among CD, ADHD, and depression symptoms along with key covariates such as child overweight, sleep disruption, and family contextual factors, and their effects on emerging adult BMI. As the current study is concerned with early influences on later risk for obesity, and as puberty can influence children's experiences of and vulnerability to behavioral and affective distress, the present study focuses on two distinct developmental time points—ages 10 and 14 years old—typically related to puberty. Our investigation utilizes data from a 20-year longitudinal study to explore potential effects of predictors in pre-adolescence and adolescence on participant BMI at approximately 24 years of age.

Research Questions

In keeping with the literature to date, we conducted exploratory analyses guided by two questions:

1. What are the relations among the three behavioral disorders appraised when children were 10 and 14 years old, childhood and adolescent weight at each age, and adult BMI?
2. What is the relation between childhood depression and adult BMI when controlling for other behavioral disorders (e.g., CD and ADHD)?

Methods

The Linking the Interests of Families and Teachers (LIFT) study is a National Institutes of Health-funded longitudinal randomized controlled trial that began in 1991. The main focus of the study was to examine the impact of a multimodal preventive intervention, the LIFT prevention program, on the developmental trajectory of children, with a particular focus on antisocial and related deviant behaviors [14, 15]. The intervention, which occurred over a 3 month period at the beginning of the study, included parent management training, child social and problem solving skills training, and a recess-based "good behavior" incentive program. Intervention impacts at various points along the life course have been reported elsewhere [14, 16–19].

Recruitment

Twelve elementary schools located in neighborhood school catchment areas with the highest juvenile police detainment rates (i.e., the top 50 %) within a moderate sized city in the Pacific Northwest were included in a pool for random assignment. During each of 3 years, schools were randomly selected to participate either as a services as usual control school or as a LIFT prevention program intervention school. Through a second random draw, either all first grade or all fifth grade classrooms within each school were invited to participate. Of 762 full-time public school students within these classrooms, 95 % (723) of families agreed to a home visit, and 93 % (671) of families who participated in a home visit consented to enroll in the study. At the outset of the study, parent participants provided written consent for the entire family. From the age of 18 years on, original child participants provided their own written consent. Participants have been interviewed regularly (usually once per year) since enrollment. The Institutional Review Board at the Oregon Social Learning Center (OSLC) approved the research protocols and monitored human participant protections. At each assessment, all respondents received compensation for their time spent in completing assessments.

Participants

Of the original 671 participating children, sixteen of the first grade cohort discontinued participation before they were 10 years old, and were excluded from analyses. Of the remaining 655 participating children, 51 % were girls ($n = 334$ girls; 321 boys). When the children were in the fifth grade, 54 % of youth lived in two-biological parent households, 22 % in single-parent households, and 23 % in stepfamilies. Approximately 28 % of the families received some type of financial assistance. Most participating parents self-identified as Caucasian, with 15 % self-identifying as members of an ethnic or racial minority group (compared with 8 % for the metropolitan area in 1990). Assessments were conducted nearly every year between 1991 and 2010 with focal children, parents, and teachers (if the child was enrolled in school). Retention was fairly high for the LIFT sample, with 575 families with 14-year-old children remaining engaged throughout the study. Fathers were actively involved in the study. For the current study, families with 10-year-old children included 396 participating fathers, and families with 14-year-old children included 321 participating fathers. If there was no participating father, we relied on the mother's data.

Predictors for the present study were measured when participants were pre-adolescents [average age 10.2 years (SD = 0.5)] and adolescents [average age 13.9 years (SD = 0.5)].

At the time of the data collection for the final time point in these analyses, 510 of the original 655 10-year-old participants remained active in the study. These participants were emerging adults who ranged in age from an average of 22 years (first graders at baseline) to 26 years (fifth graders at baseline) or an average of 24.3 (SD = 2.1) years of age.

Measures and Constructs

Outcome

The outcome of interest is *emerging adult body mass index*. BMI was calculated from self-reported height and weight [weight in kilograms divided by the square of height in meters; (i.e., kg/m^2)], and was used as a continuous variable in all analyses.

Predictor Variables

All predictor variables were computed for 10 and 14 year olds.

Child depression was assessed from youngsters' reports of their own feelings or ideas in the past 2 weeks in response to the 27-item Child Depression Inventory [20]. Examples of questions included whether the child feels sad, is good at doing things, has fun, or feels alone. All items were re-coded from a range of 1–3 to match standardized scoring values (0, 1, 2). Several items were reversed so that higher scores reflected higher depression. Final scores were computed by summing all of the items. The CDI scale demonstrated adequate internal consistency; Cronbach's alphas were 0.85 for 10 year olds, and 0.86 for 14 year olds.

Conduct disorder was evaluated through parent report on the CBCL [21] and Overt/Covert Antisocial Questionnaire (OCA) [22]. The CBCL asks about specific child and youth behaviors within the past 12 months (0 = not true of the participating child now or in the last 12 months; 1 = somewhat or sometimes true; 2 = very true or often true). Possible OCA responses are identical to those of the CBCL (but numbered from 1 to 3), though observations are not limited to a specific time frame. Examples of questions asked of parents include whether their child has deliberately engaged in fire-setting; broken into someone else's house, building, or car; initiated physical fights; or been physically cruel to animals or people. A scale encompassing 13 behaviors described by the DSM as indicative of conduct disordered children and youth was created from items drawn from the CBCL and OCA. Cronbach's alphas were run separately for mothers and fathers and indicated adequate internal consistency; alphas for mothers' scores ranged from 0.73 to 0.75, and fathers' scores ranged from 0.62 to 0.72. As mother and father reports were significantly correlated ($r = .33$; $p < .001$ for 10 year olds; $r = .20$; $p < .05$ for 14 year olds), mean scores reflecting parent report were computed for each of ages 10 and 14. Parent variables were then recoded as dichotomous to reflect whether or not the participant child engaged in the behaviors described above. Dichotomous scales were then summed.

Attention-deficit/hyperactivity disorder was assessed through parent report on the OCA and the CBCL. A mean OCA score was computed from seven items relating to ADHD behaviors (e.g., child does not follow through on instructions; often shifts activities; interrupts, intrudes on others; has trouble remaining seated). A mean CBCL score was created from four items (e.g., child cannot concentrate; is impulsive or acts without thinking; talks too much). Cronbach's alphas for mothers' scores ranged from 0.86 to 0.91, and fathers' scores ranged from 0.84 to 0.87. Scales were computed by summing participant responses on OCA and CBCL items. As father and mother scores were

significantly correlated ($r = .48$; $p < .001$ for 10 year olds; $r = .59$; $p < .001$ for 14 year olds), scores were averaged to reflect parent report of ADHD behaviors at each of ages 10 and 14.

Covariates

An established literature shows the following variables to be strongly related to adolescent and adult BMI, and particularly to obesity, across the lifespan: child and adolescent overweight and obesity [23, 24]; maternal depression [25]; parental smoking [26]; sleep disruption [27–29]; and family income, as an important component of socioeconomic position [30]. Additional important covariates include nutrition, exercise, family eating together, family communication, and frequency of television viewing [3, 31]. A critical covariate with adult obesity is the number of biological children a woman has borne [32]. As all variables listed above, with the exception of nutrition, were available in the LIFT data set, we included them in analyses as potential covariates.

Child overweight was based on mother, father, and teacher report on the CBCL of whether the participating child was overweight. As the Cronbach's alpha of 0.84 indicated internal consistency among the three reports, a single mean score reflecting youngster overweight was calculated.

Sleep disruption was assessed through parent evaluation on the CBCL of whether their child has nightmares, is overtired, sleeps less than most children, or has trouble sleeping. As the Cronbach's alpha for eight items (four for each of mother and father report) demonstrated adequate internal consistency at 0.68, a sleep disruption variable was created reflecting the mean score across all items.

Family household income relied on self-reported data from the OSLC Parent Interview (1990) gathered when the youth were in 5th grade and included 10 categories ranging from 1 (less than $5,000) to 10 ($60,000 or more) [33].

Parent smoking was assessed through parent response to a single item about frequency of current nicotine use on the OSLC Physical Health Inventory [34]. Potential responses ranged from 1 (not at all) to 7 (several times a day). Responses were recoded to a three-part variable reflecting 1 (not at all), 2 (1 time per day or less), or 3 (several times per day).

Maternal depression was measured with the Center for Epidemiological Studies Depression Scale (CESD), a well-established validated and standardized measure of depressive symptoms [35]. In the current study, Cronbach's alpha for the CESD maternal depression scale was 0.89.

Self-report youth variables drawn from the Family Activities List (OSLC 1990) included *exercise* (8 items; e.g., "in the last week did you walk, participate in an outdoor activity with your mother and/or father?"), *family eating together* (1 item; "in the last week, did you eat at home with the whole family there?"), and *family communication* (1 item; "in the last week, did you talk with your mother and/or father for 10 min or more about your activities?") [36]. Cronbach's alpha for the youth exercise variable was 0.81 and a mean score was computed. A single item for *television viewing* ("on a school day, how many hours do you usually watch TV?") was based on youth report on the Child Interview (OSLC 1990) [37]. Each of the following emerging adult variables relied on a single self-report item taken from the Child Interview (redesigned for emerging adults; OSLC 2004) [37]: *income* (total income in the past 12 months), *exercise* (average number of days per week), *sleep duration* (average number of hours per night), *television viewing* (average hours per day in the past week), and *number of biological children*.

Analytic Strategy

Analyses were conducted in multiple steps to examine relations among the variables, in accordance with the two research questions. Statistical analyses were performed using SPSS 14.0. All data were checked for univariate normality. Descriptive summary statistics were calculated for all variables of interest. During this process, one adult BMI outlier (>2 SD above the mean) was identified. Because of the potential of this outlier biasing the analyses, the outlier was recoded to the next highest value for adult BMI plus one [38]. All predictor variables were centered around the mean (within each respective school cohort) to control for cohort effects in the analyses. Pearson's correlations were calculated to assess associations among adult BMI, child depression, CD, ADHD at ages 10 and 14, and key covariates with adolescent overweight (Table 1) and obesity (Table 2).

The study used multiple regression analysis to model relationships among the variables of interest. We examined two regression models, with the first including the outcome variable of overweight at age 14, and the three potential predictor variables of childhood depression, CD, and ADHD, along with covariates of adolescent overweight. A second regression model with the outcome variable of adult BMI included independent variables of adolescent depression, CD, and ADHD appraised when participants were 14 years old, as well as key covariates of adult BMI. A dummy variable reflecting whether participants participated in the LIFT intervention or not (0 = control; 1 = intervention) was included in each model to control for potential intervention effects.

Results

Descriptives

Prevalence rates for overweight and obesity for the sample were comparable to or lower than national averages (based on National Health and Nutrition Examination Survey [NHANES, 2007–2008] data for 20–39 year olds) [39]. Average BMI was 26.0 (SD = 4.9) and 26.5 (SD = 6.9), for young adult men and women, respectively. Among men, 35.7 % were overweight (defined as a BMI of 25.0–29.9) and 16.6 % were obese (BMI of 30.0 or higher). Among women, 24.0 % were overweight and 25.5 % were obese. Prevalence rates for behavioral symptoms in the sample were equal to or higher than national statistics (for the time period in which the LIFT participants' behavioral symptoms were appraised). About five percent (5.1 %) of 10 year olds (M = 6.2; SD = 5.9) and 5.0 % of 14 year olds (M = 6.6; SD = 5.9) in the sample tested positive on the CDI for depression (scores > 2 SD above the mean), compared with nationwide data indicating that between 5.2 and 7.3 % of children and youth were diagnosed with major depression [40], and 10–15 % of children and adolescents were diagnosed with depressive symptoms [41]. In regards to ADHD, 6.6 % of 10 year olds and 5.5 % of 14 year olds in the sample had six symptoms or more (per DSM-IV criteria [42]), compared with 3 % of youth diagnosed with ADHD nationwide [43]. Seventeen percent (17.4 %) of 10 year olds and 7.0 % of 14 year olds in the sample had three or more symptoms indicating conduct disorder (per DSM-IV criteria [42]). These are significantly higher rates than those reported for the US as a whole, at 3–4 %, [44] a finding that was expected due to the sampling strategy used in the study.

Table 1 Correlation matrix for overweight at age 14, depression, conduct disorder and ADHD at ages 10 and 14, and key covariates[a]

Variables	Overweight 14	Depression 14	Depression 10	CD 14	CD 10	ADHD 14	ADHD 10	Overweight 10
Overweight 14	1	0.10*	0.13**	0.14***	0.08†	0.13**	0.11**	0.68***
Depression 14		1	0.36***	0.28***	0.15***	0.23***	0.16***	0.08†
Depression 10			1	0.24***	0.30***	0.21***	0.28***	0.03
CD 14				1	0.63***	0.57***	0.44***	0.07
CD 10					1	0.51***	0.58***	0.04
ADHD 14						1	0.72***	0.05
ADHD 10							1	0.06
Overweight 10								1
Exercise 14								
Sleep 14								
TV 14								
Family communication 14								
Family eat together 14								
Family income								
Parental smoking								
Maternal depression								

Variables	Exercise 14	Sleep 14	TV 14	Family communication 14	Family eat together 14	Family income	Parental smoking	Maternal depression
Overweight 14	−0.02	0.18***	0.13**	−0.08†	−0.10*	−0.15***	0.09*	0.09*
Depression 14	−0.14**	0.30***	0.06	−0.13**	−0.17***	−0.12**	0.09*	0.18***
Depression 10	−0.06	0.23***	0.03	−0.13**	−0.04	−0.12**	0.02	0.20***
CD 14	0.06	0.40***	0.09*	−0.11**	−0.04	−0.16***	0.10*	0.26***
CD 10	0.07	0.29***	0.07	−0.14**	−0.03	−0.18***	0.06	0.25***
ADHD 14	0.06	0.38***	0.05	−0.09*	−0.07	−0.11**	0.06	0.22***
ADHD 10	0.10*	0.32***	0.09*	−0.13**	−0.10*	−0.13**	0.08†	0.21***

Table 1 continued

Variables	Exercise 14	Sleep 14	TV 14	Family communication 14	Family eat together 14	Family income	Parental smoking	Maternal depression
Overweight 10	−0.02	0.12**	0.14***	−0.08†	−0.07	−0.08*	0.13**	0.06
Exercise 14	1	0.05	0.08†	0.15**	0.11*	0.02	0.09†	−0.04
Sleep 14		1	0.07	−0.07	−0.08†	−0.12**	0.06	0.33***
TV 14			1	−0.06	−0.02	−0.16***	0.08†	−0.01
Family communication 14				1	0.15***	0.04	−0.03	−0.03
Family eat together 14					1	−0.01	−0.01	−0.07
Family income						1	−0.14***	−0.26***
Parental smoking							1	0.14***
Maternal depression								1

BMI body mass index; *CD* conduct disorder; *ADHD* attention-deficit/hyperactivity disorder

[a] Correlations are statistically significant at † $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

Table 2 Correlations between adult BMI, depression, conduct disorder and ADHD at ages 10 and 14, and key covariates[a]

Variables	BMI
BMI	1
Depression 14	0.09†
Depression 10	0.11*
CD 14	0.07
CD 10	0.05
ADHD 14	0.03
ADHD 10	0.05
Biological children[b]	0.14**
Adult income	0.01
Adult exercise	−0.003
Adult sleep	0.03
Adult TV viewing	0.06
Overweight 14	0.59***
Overweight 10	0.50***
Exercise 14	0.06
Sleep 14	0.05
TV 14	0.06
Family communication 14	−0.12**
Family eat together 14	−0.04
Family income	−0.08*
Parental smoking	0.16***
Maternal depression	0.07

BMI body mass index; *CD* conduct disorder; *ADHD* attention-deficit/hyperactivity disorder

[a] Correlations are statistically significant at † $p < .10$; * $p < .05$; ** $p < .01$; *** $p < .001$

[b] Number of biological children

Correlations

The Pearson's correlations revealed several significant associations among key variables of interest (Tables 1, 2). Bivariate correlations indicated that ADHD, CD, and depressive symptoms at age 10 were not significantly related to overweight at the same age. However, overweight at age 14 strongly and positively correlated with age 10 and 14 measures of depression ($p < .01$ and $p < .05$, respectively), ADHD ($p < .01$), CD ($p = .06$, and $p = .001$, respectively) (Table 1). As would be expected, behavioral problems at age 10 strongly predicted future behavioral problems at age 14 ($p < .001$ for all; Table 1). When relationships were examined between emerging adult BMI and the three behavioral variables at ages 10 and 14, only depression strongly and positively related to BMI ($p < .05$ and $p = .06$, respectively) (Table 2; to conserve space, only correlations between BMI and independent variables are shown).

Significant correlations were found between overweight at age 14 and several covariates. Higher family income ($p < .001$) and eating together with the whole family in the past week ($p < .05$) significantly related to lower incidence of overweight at age 14, with more family communication approaching significance ($p = .06$). Parental smoking ($p < .05$), maternal depression ($p < .05$), more adolescent TV viewing ($p < .01$), greater sleep disruption ($p < .001$), and overweight at age 10 ($p < .001$) all were associated with greater prevalence of age 14 overweight. Higher adult BMI significantly correlated with lower family income ($p < .05$), parental smoking ($p < .001$), overweight at ages 10 and 14 ($p < .001$ for each) and having more biological children ($p < .01$), a relationship that, upon

closer scrutiny, held true for both sexes [adult women's BMI significantly correlated with the number of biological children they reported having ($r = .14$; $p < .05$) and a trend relationship was detected for men ($r = .11$; $p < .10$)]. Adolescents who reported talking with one or both parents about their activities in the past week (family communication) had significantly lower adult BMI ($p < .01$). Potential adult covariates of television viewing, annual income, sleep duration, and exercise did not significantly relate to BMI, nor did the adolescent variables for sleep disruption, television viewing, family eating together, or exercise. Consequently, these variables were not entered into regression analyses. Finally, while there were no significant associations between the LIFT prevention program and adolescent overweight or adult BMI ($p > .10$ for each), the study condition was maintained in the models to control for any potential biases.

Regressions

Regression results are shown with coefficients in Tables 3 and 4. When relations among variables were examined with multiple regression models, analyses revealed a complex set of both direct and indirect relationships among the behavioral symptoms at 10 and 14 years and later adult BMI. We designed both models to test for potential mediating effects on depression of conduct disorder and ADHD. While Table 4 contains the full model to illustrate the mediation of depression on adult BMI, as we detected no mediating effects in the model focused on age 14 overweight, Table 3 reflects only the final step of the

Table 3 Multiple regression model for prediction of overweight at age 14

Measure and variables	Regression coefficient ($b \pm$ SE)	B[a]	Overall r^2
Overweight 14			0.53***
Constant	0.01 (0.02)		
Family income	−0.01 (0.01)	−0.05	
Parental smoking	−0.01 (0.02)	−0.01	
Maternal depression	0.00 (0.00)	−0.003	
Family communication 14	−0.02 (0.04)	−0.01	
TV 14	0.00 (0.01)	0.02	
Family eat together 14	−0.05 (0.04)	−0.04	
Sleep 14	0.05 (0.07)	0.03	
Intervention	−0.02 (0.03)	−0.02	
Depression 14	−0.00 (0.00)	−0.02	
Depression 10	0.01 (0.00)	0.06†	
CD 14	0.11 (0.06)	0.08†	
CD 10	−0.09 (0.06)	−0.06	
ADHD 14	0.02 (0.01)	0.07	
ADHD 10	−0.01 (0.01)	−0.05	
Overweight 10	0.82 (0.04)	0.70***	

BMI body mass index; *CD* conduct disorder; *ADHD* attention-deficit/hyperactivity disorder

[a] Coefficients are statistically significant at: † $p \leq .10$; * $p < .05$; ** $p < .01$

Table 4 Multiple regression model for prediction of emerging adult BMI

Measure and variables	Regression coefficient ($b \pm$ SE)	B^a	Overall r^2
BMI			0.42***
Step 1			
Constant	25.88 (0.46)		
Biological children[b]	0.91 (0.40)	0.11*	
Family income	−0.12 (0.13)	−0.05	
Parental smoking	1.08 (0.35)	0.15**	
Family communication 14	−1.85 (0.70)	−0.12**	
Intervention	0.20 (0.59)	0.02	
Step 2			
Constant	25.90 (0.46)		
Biological children[b]	0.91 (0.40)	0.11*	
Family income	−0.10 (0.13)	−0.04	
Parental smoking	1.11 (0.35)	0.15***	
Family communication 14	−1.72 (0.70)	−0.12**	
Intervention	0.20 (0.59)	0.02	
Depression 14	−0.01 (0.05)	−0.01	
Depression 10	0.09 (0.05)	0.09[†]	
Step 3			
Constant	25.89 (0.46)		
Biological children[b]	0.90 (0.41)	0.11*	
Family income	−0.10 (0.13)	−0.04	
Parental smoking	1.10 (0.35)	0.15**	
Family communication 14	−1.72 (0.71)	−0.12*	
Intervention	0.18 (0.59)	0.01	
Depression 14	−0.01 (0.05)	−0.01	
Depression 10	0.10 (0.06)	0.09[†]	
CD 14	1.05 (1.24)	0.06	
CD 10	−1.09 (1.22)	−0.06	
ADHD 14	−0.17 (0.25)	−0.05	
ADHD 10	0.12 (0.23)	0.04	
Step 4			
Constant	25.76 (0.36)		
Biological children[b]	1.22 (0.32)	0.14***	
Family income	0.08 (0.11)	0.03	
Parental smoking	0.56 (0.28)	0.08*	
Family communication 14	−1.00 (0.56)	−0.07[†]	
Intervention	0.17 (0.47)	0.01	
Depression 14	−0.04 (0.04)	−0.04	
Depression 10	0.06 (0.04)	0.05	
CD 14	0.12 (0.99)	0.01	
CD 10	−0.05 (0.96)	−0.00	
ADHD 14	−0.21 (0.19)	−0.06	
ADHD 10	0.09 (0.18)	0.03	

Table 4 continued

Measure and variables	Regression coefficient ($b \pm$ SE)	B^a	Overall r^2
Overweight 14	6.55 (0.73)	0.48***	
Overweight 10	2.74 (0.83)	0.17***	

BMI body mass index; *CD* conduct disorder; *ADHD* attention-deficit/hyperactivity disorder

[a] Coefficients are statistically significant at: † $p \leq .10$; * $p < .05$; ** $p < .01$

[b] Number of biological children

regression. Both models were significant ($p < .001$), with predictors accounting for 53 % of the variance in overweight at age 14, and 42 % of the variance in emerging adult BMI.

Child depression and adolescent conduct disorder approached significance in predicting greater overweight at age 14 ($p = .09$ and $p = .08$, respectively). When we investigated behavioral disorders in relation to adult BMI, after controlling for key covariates with the exception of CD, ADHD, and overweight at ages 10 and 14 (Table 4, step 2), higher child depression approached significance in predicting elevated adult BMI ($p = .09$). Even after adding in step 3 potential mediators of CD and ADHD at ages 10 and 14, the effect of child depression on adult BMI did not change ($p = .09$). It was not until step 4 that the effect of child depression on adult BMI was fully mediated ($p = .21$) with the addition of overweight at ages 10 and 14 ($p < .001$). In sum, behavioral symptoms in childhood and adolescence did not appear to directly predict adult BMI, rather they indirectly predicted elevated adult BMI through overweight at 14 years old. In addition, more biological children ($p < .001$) and parental smoking ($p < .05$) significantly predicted elevated adult BMI, with poorer family communication approaching significance ($p = .08$).

Discussion

Using data from the 20-year LIFT study, we found evidence that symptoms of depression and conduct disorder early in life may indirectly contribute to elevated BMI in emerging adulthood through their influence on adolescent overweight. These associations remained after adjustment for potential covariates, including family income, TV viewing, and other family environmental factors. Contrary to Pine et al. [11] findings, our model found that symptoms of childhood depression were a tentative predictor (i.e., trend finding) of elevated adult BMI even after controlling for symptoms of CD. Instead of CD, however, adolescent overweight mediated the relationship between depression and BMI. Our study contributes to knowledge in the field by outlining a pathway through which childhood and adolescent behavioral problems may influence young adult BMI, namely through direct impacts on adolescent overweight, which then persists into adulthood. In addition, findings suggest the importance of early family environmental factors, specifically parental smoking and family communication, as contributors to elevated adult BMI.

Little is known about the complex and potentially numerous pathways linking child and adolescent behavioral distress with overweight and obesity across the lifespan. Existing research points to several mechanisms, including neurobiological links, physiological changes, unhealthy environments, and social stigma [3, 45]. Recent studies indicate that dysregulation in the dopaminergic system may play a role in the neurobiology of ADHD and CD and contribute to impulsive eating and related weight gain [46–49]. Studies on the

biological underpinnings of depression point to low serotonin levels that may cause increased consumption of food high in carbohydrates [50]. Behavioral disorders (e.g., depression) also can cause physiological changes in endocrine and immunological systems [51–53], which may influence appetite control. Mamun et al. [3] note that behavioral changes among depressed children (e.g., having difficulty adhering to fitness regiments and overeating), may play a role in weight gain that can extend into adulthood, and similar observations might be made of children with CD and/or ADHD. Relatedly, researchers of social stigma point to child and adolescent obesity as a contributor to and consequence of depression among young people [55], with obesity throughout childhood and adolescence serving as a key predictor of adult overweight and obesity [24, 54].

Sleep disruption or shortened sleep duration, another potential behavioral change related to depression and other conditions, also has been shown to relate to elevated risk of overweight and obesity [27]. We detected significant correlations in this study between behavioral disorders at ages 10 and 14 and sleep disruption at age 14 ($p < .001$ for all; Table 1). The pathways between poor childhood sleep and adult overweight, however, remain unclear. Potential explanations include reduced ghrelin (an appetite-stimulating hormone), or decreased levels of leptin (an appetite-suppressing hormone), or through tiredness, which can reduce physical activity and affect dietary habits (people who feel tired may seek fast-release, high-energy foods to compensate for perceived low energy levels) [28, 29].

Family factors, such as abuse and neglect, can play a role, with research showing that childhood adversity can contribute to behavioral problems throughout childhood and adolescence [56, 57], and trigger changes in the hypothalamic–pituitary–adrenal (HPA) axis [12]. Chronic psychosocial stress and related long-term dysregulation of the HPA axis [58] can have downstream negative health consequences including increased visceral adiposity and obesity [59, 60]. Even less severe parenting behaviors, such as when parents of children with behavior problems use unhealthy foods or television as rewards [7], can intensify the risks for children of an already obesogenic environment [47].

In the current study, family communication, which we identified as a potential influence on young adult BMI, may serve as a proxy for more complex family processes that increasingly are linked to greater risk for obesity. Similarly, given participants' relative youth (average age of 24 years) at the final time point for analyses, our finding that the number of biological children predicted elevated adult BMI also points to the influence of early childbearing, a potential consequence of early adversity and difficult family environments [61, 62]. Though early childbearing itself may not link to elevated obesity, parents who have their children early and have several children in quick succession may face higher risk of obesity at younger ages (e.g., Khlat and colleagues found that women with more than three children were 2 times more likely to be obese compared with women with one child) [61]. Finally, our finding that parental smoking predicts adult BMI builds on evidence from several studies that parental smoking is an independent risk factor for childhood obesity [63–65]. Importantly, findings to date indicate that the effects of childbearing and parental smoking on obesity risk are independent of family income [61, 64].

Limitations

The current study has several limitations. First, in terms of measurement, the LIFT study focused on childhood predictors of adolescent and young adult antisocial behavior, and thus key

variables of interest to the present study were either not available or relied on only a few items drawn from assessments of youth or parent behavioral adjustment or parenting. As measured height and weight were not collected in the early phases of the LIFT study, the present study relied on the assessment of youth overweight based on multi-agent report on a single item in the parent and teacher versions of the Child Behavior Checklist. That child and youth overweight accounted for such a large proportion of the variance in adult BMI is testament both to the verisimilitude of parent and teacher reports (at ages 10 and 14), and to the role of youth overweight as a critical predictor of adult BMI. However, as has been shown in prior studies, the assessment of BMI based on self-reported, as opposed to measured, height and weight is prone to error and bias related to self-perception [66, 67].

Important covariates of adult BMI not included in current analyses include parent BMI and family and adult nutrition. Variables reflecting nutrition, and parent BMI, overweight or obesity unfortunately were not tracked in the LIFT study. In addition, though we did include variables for physical activity at all ages, given the lack of robust measurement it was unsurprising that correlations revealed no significant relationships with our overweight or BMI outcome variables. Finally, given associations between race/ethnicity and BMI [68], the low ethnic diversity in the LIFT sample due to the geographic location of the study may affect the generalizability of the findings.

Conclusion

Many questions remain regarding relationships among different forms of affective and behavioral distress in childhood and adolescence and weight gain over the life course. Future studies would benefit the field through investigation of multiple potential pathways using a range of methodologies (e.g., stress biomarkers, genetic approaches, measured variables, as well as parent and youth self-report). Available evidence suggests that mental health programs for youngsters would do well to monitor weight in children and adolescents presenting with behavior problems, and consider appropriate interventions, in order to prevent potential deleterious health outcomes in emerging adulthood, especially among those children with other risk factors for obesity and obesity-related diseases (e.g., cardiovascular disease, diabetes). Specifically, clinicians working with children with depression and adolescents with conduct disorder, and interventionists engaged in prevention activities targeting youth problem behaviors (e.g., mentoring, school-based social and problem-solving skills programs, parent training) may wish to address the potential relation between depression and conduct problems and overweight in adolescence and adulthood.

Obesity prevention efforts with adolescents may also benefit from a focus on reduced parent smoking, and on positive parental engagement with an emphasis on family communication and parenting skills related to behavioral and mental health. In addition, smoking parents and young adults with several biological children could be targets of overweight prevention and intervention efforts. Parents who smoke should be warned that not only is their own health at risk, but their children face greater likelihood of overweight and related chronic diseases. In short, these results, like those of other recent longitudinal studies, point to the importance of considering the whole person and his or her family relations over the lifespan when launching intervention and prevention efforts, rather than simply focusing on one symptom or set of symptoms currently present in the individual.

Summary

Rising obesity prevalence rates and related increases in morbidity and mortality have become an international concern. Researchers increasingly regard obesity as a developmental condition, with several studies showing that childhood and adolescent behavioral and affective distress are related to elevated adult body mass index (BMI). The present study examines links among symptoms of conduct disorder, attention-deficit/hyperactivity disorder, and depression in late childhood and mid-adolescence, and BMI in emerging adulthood. Using data from the 20-year Linking the Interests of Families and Teachers (LIFT) study ($N = 671$), examined with multiple regression analyses, we found evidence that symptoms of depression and conduct disorder early in life may indirectly contribute to elevated BMI in emerging adulthood through their influence on adolescent overweight. Parental smoking and family communication were also contributors to elevated adult BMI. Given these findings, health practitioners working with children and adolescents with behavior problems may wish to be mindful of youngsters' weight (particularly overweight or obesity), and consider strategies not only for preventing future mental health problems, but also elevated BMI in early adulthood. Child and adolescent obesity prevention efforts may benefit from a focus on reduced parent smoking, and on positive parental engagement with an emphasis on family communication and parenting skills related to behavioral and mental health.

Acknowledgments The authors thank the study participants and their parents in the Bethel, Eugene 4J, and Springfield Public School districts, and the principals and teachers in the participating schools. Special thanks to the many OSLC scientists and staff members who contributed to the LIFT study over the years, and particularly to John B. Reid, for his leadership and mentoring; to Alice Holmes, Theresa Mayne, and Becky Fetrow, for their exemplary work on project coordination over the years; to Kathy Jordan, for her work on data management and analyses; and to Paulina Nowicka and D. Molloy Wilson, for their valuable input on the research topic and analyses. Work on this project was supported by a research and development award from the Oregon Social Learning Center Scientist Council and award numbers R01 MH054248 from the National Institute of Mental Health, National Institutes of Health (NIH), and awards R01 HD054880 and ARRA R01 HD054880 S1 from the Eunice Kennedy Shriver National Institute of Child Health and Human Development, NIH.

References

1. World Health Organization, Food and Agriculture Organization of the United Nations, (WHO/FAO) (2004) Diet, nutrition, and the prevention of chronic diseases: scientific background papers of the joint WHO/FAO expert consultation. Public Health Nutr 7:245–250
2. Pine DS, Goldstein RB, Wolk S, Weissman MM (2001) The association between childhood depression and adulthood body mass index. Pediatrics 107:1049–1056
3. Mamun AA, O'Callaghan MJ, Cramb SM, Najman JM, Williams GM, Bor W (2009) Childhood behavioral problems predict young adults' BMI and obesity: evidence from a birth cohort study. Obesity 17:761–766
4. Richardson LP, Davis R, Poulton R, McCauley E, Moffitt TE, Caspi A, Connell F (2003) A longitudinal evaluation of adolescent depresesion and adult obesity. Arch Pediatr Adolesc Med 157:739–745
5. Arnett JJ (2000) Emerging adulthood: a theory of development from the late teens through the twenties. Am Psychol 55:469–480
6. Hasler G, Pine DS, Kleinbaum DG, Gamma A, Luckenbaugh D, Ajdacic A, Eich D, Rossler W, Angst J (2005) Depressive symptoms during childhood and adult obesity: the Zurich cohort study. Mol Psychiatry 10:842–850
7. Anderson SE, Cohen P, Naumova EN, Must A (2006) Relationship of childhood behavior disorders to weight gain from childhood into adulthood. Ambul Pediatr 6:297–301

8. Fuemmeler BF, Ostbye T, Yang C, McClernon FJ, Kollins SH (2010) Association between attention-deficit/hyperactivity disorder symptoms and obesity and hypertension in early adulthood: a population-based study. Int J Obes 35:852–862
9. Bardone AM, Moffitt TE, Caspi A, Dickson N, Stanton WR, Silva PA (1998) Adult physical health outcomes of adolescent girls with conduct disorder, depression, and anxiety. Child Adolesc Psychiatr 37:594–601
10. Lumeng JC, Gannon K, Cabral HJ, Frank DA, Zuckerman B (2003) Association between clinically meaningful behavior problems and obesity in children. Pediatrics 122:1138–1145
11. Pine D, Cohen P, Brook J, Coplan J (1997) Psychiatric symptoms in adolescence as predictors of obesity in early adulthood: a longitudinal study. Am J Public Health 87:1303–1310
12. Bruce J, Fisher PA, Pears KC, Levine S (2009) Morning cortisol levels in preschool-aged foster children: differential effects of maltreatment type. Dev Psychobiol 51:14–23
13. Gunnar MR, Fisher PA, The Early Experience Stress and Prevention Network (2006) Bringing basic research on early experience and stress neurobiology to bear on preventive interventions for neglected and maltreated children. Dev Psychopathol 18:651–677
14. Reid JB, Eddy JM, Fetrow RA, Stoolmiller M (1999) Description and immediate impacts of a preventive intervention for conduct problems. Am J Community Psychol 27:483–517
15. Eddy JM, Reid JB, Fetrow RA (2000) An elementary-school based prevention program targeting modifiable antecedents of youth delinquency and violence: linking the interests of families and teachers (LIFT). J Emot Behav Disord 8:165–176
16. Stoolmiller M, Eddy JM, Reid JB (2000) Detecting and describing preventative intervention effects in a universal school-based randomized trial targeting delinquent and violent behavior. J Consult Clin Psychol 68:296–306
17. Reid JB, Eddy JM (2002) Preventive efforts during the elementary school years: the linking the interests of families and teachers project. In: Reid JB, Patterson GR, Snyder JJ (eds) Antisocial behavior in children and adolescents: a developmental analysis and model for intervention. American Psychological Association, Washington, DC, pp 219–233
18. Eddy JM, Reid JB, Stoolmiller M, Fetrow RA (2003) Outcomes during middle school for an elementary school-based preventive intervention for conduct problems: follow-up results from a randomized trial. Behav Ther 34:535–552
19. DeGarmo DS, Eddy JM, Reid JB, Fetrow RA (2009) Evaluating mediators of the impact of the linking the interests of families and teachers (LIFT) multimodal preventive intervention on substance use initiation and growth across adolescence. Prev Sci 10:208–220
20. Kovacs M (1985) The children's depression inventory (CDI). Psychopharmacol Bull 21:995–998
21. Achenbach TM (1992) Revised Child Behavior Checklist. University of Vermont, Burlington
22. Oregon Social Learning Center (OSLC) (1990) Overt/Covert Antisocial Questionnaire. Eugene, Oregon
23. Deckelbaum RJ, Williams CL (2001) Childhood obesity: the health issue. Obes Res 9:239S–243S
24. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH (1997) Predicting obesity in young adulthood from childhood and parental obesity. N Engl J Med 337:869–873
25. Davis M, Young L, Davis SP, Moll G (2008) Parental depression, family functioning and obesity among African American children. J Cult Divers 13:61–65
26. Molarius A, Seidell JC, Kuulasmaa K, Dobson AJ, Sans S (1997) Smoking and relative body weight: an International perspective from the WHO MONICA project. J Epidemiol Commun Health 51:252–260
27. Ievers-Landis CE, Storfer-Isser A, Rosen C, Johnson NL, Redline S (2008) Relationship of sleep parameters, child psychological functioning, and parenting stress to obesity status among preadolescent children. J Dev Behav Pediatr 29:243–252
28. Iglowstein I, Jenni OG, Molinari L, Largo RH (2003) Sleep duration from infancy to adolescence: reference values and generational trends. Pediatrics 111:302–307
29. Landhuis CE, Poulton R, Welch D, Hancox RJ (2008) Childhood sleep time and long-term risk for obesity: a 32-year prospective birth cohort study. Pediatrics 122:955–960
30. Kestila L, Rahkonen O, Martelin T, Lahti-Koski M, Koskinen S (2009) Do childhood social circumstances affect overweight and obesity in early adulthood? Scand J Public Health 37:206–219
31. Hancox RJ, Milne BJ, Poulton R (2004) Association between child and adolescent television viewing and adult health: a longitudinal birth cohort study. Lancet 364:257–262
32. Weng HH, Bastion LA, Taylor DH, Moser BK, Ostbye T (2004) Number of children associated with obesity in middle-aged women and men: results from the health and retirement study. J Womens Health 13:85–91
33. Oregon Social Learning Center (OSLC) (1990) Parent interview. Eugene, Oregon
34. Oregon Social Learning Center (OSLC) (1990) Physical health inventory. Eugene, Oregon

35. Radloff LS (1977) The CES-D scale: a self-report depression scale for research in the general population. Appl Psychol Meas 1:385–401
36. Oregon Social Learning Center (OSLC) (1990) Family activities list. Eugene, Oregon
37. Oregon Social Learning Center (OSLC) (1990, 2004) Child interview. Eugene, Oregon
38. Field A (2005) Discovering statistics using SPSS, 2nd edn. Sage, London
39. Flegal KM, Carroll MD, Ogden CL, Curtin LR (2010) Prevalence and trends in obesity among US adults, 1999–2008. JAMA 303:235–241
40. Kazdin A (1989) Childhood depression. In: Mash E, Barkley R (eds) Treatment of childhood disorders. Guilford, New York, NY, pp 135–166
41. U.S. Department of Health and Human Services (1999) Mental health: a report of the surgeon general. U.S. Department of Health and Human Services, Substance Abuse and Mental Health Services Administration, Center for Mental Health Services, National Institutes of Health, National Institute of Mental Health, Rockville, MD
42. American Psychiatric Association (2000) Diagnostic and statistical manual of mental disorders, 4th edn. American Psychiatric Association, Washington, DC
43. Barkley R (1989) Attention deficit-hyperactivity disorder. In: Mash E, Barkley R (eds) Treatment of childhood disorders. Guilford, New York, NY, pp 39–72
44. McMahon R, Wells K (1989) Conduct disorders. In: Mash E, Barkley R (eds) Treatment of childhood disorders. Guilford, New York, NY, pp 73–132
45. Holtkamp K, Konrad K, Müller B, Heussen N, Herpertz S, Herpertz-Dahlman B, Hebebrand J (2004) Overweight and obesity in children with attention-deficit/hyperactivity disorder. Int J Obes 28:685–689
46. Biederman J, Faraone SV (2002) Current concepts on the neurobiology of attention-deficit/hyperactivity. J Atten Disord 6:7–16
47. Campbell BC, Eisenberg D (2007) Obesity, attention deficit-hyperactivity disorder and the dopaminergic reward system. Coll Antropol 31:33–38
48. Levitan RD, Masellis M, Lam RW, Muglia P, Basile VS, Jain U, Kaplan AS, Tharmalingam S, Kennedy SH, Kennedy JL (2004) Childhood inattention and dysphoria and adult obesity associated with the dopamine D4 receptor gene in overeating women with Seasonal Affective Disorder. Neuropsychopharmacology 29:179–186
49. Salamone JD, Correa M, Mingote S, Weber SM (2003) Nucleus accumbens dopamine and the regulation of effort in foodseeking behavior: implications for studies of natural motivation, psychiatry, and drug abuse. J Pharmacol Exp Ther 305:1–8
50. Wurtman JJ (1993) Depression and weight gain: the serotonin connection. J Affect Disord 29:183–192
51. Ludwig DD (2002) The glycemic index: physiological mechanisms relating to obesity, diabetes, and cardiovascular disease. JAMA 287:2414–2423
52. Sabban E, Kvetnansky R (2001) Stress-triggered activation of gene expression in catecholaminergic systems: dynamics of transcriptional events. Trends Neurosci 24:91–98
53. Tafet G, Bernardini R (2003) Psychoneuroendocrinological links between chronic stress and depression. Prog Neuropsychopharmacol Biol Psychiatry 27:893–903
54. Young-Hyman D, Tanofsky-Kraff M, Yanovski SZ, Keil M, Cohen ML, Peyrot M, Yanovski JA (2006) Psychological status and weight-related distress in overweight or at-risk-for-overweight children. Obesity 14:2249–2258
55. Puhl R, Brownell KD (2001) Bias, discrimination, and obesity. Obes Res 9:788–805
56. Eddy JM, Reid JB, Curry V (2002) The etiology of youth antisocial behavior, delinquency, and violence and a public health approach to prevention. In: Shinn M, Walker H, Stoner G (eds) Interventions for academic and behavior problems II: preventive and remedial approaches. National Association of School Psychologists, Bethesda, MD, pp 27–51
57. Reid JB, Patterson GR, Snyder J (eds) (2002) The Oregon model: understanding and altering the delinquency trajectory. American Psychological Association, Washington, DC
58. Sapolsky RM, Romero LM, Munck AU (2000) How do glucocorticoids influence stress responses? Integrating permissive, suppressive, stimulatory, and preparative actions. Endocr Rev 21:55–89
59. McEwen BS, Wingfield J (2003) The concept of allostatis in biology and biomedicine. Horm Behav 43:2–15
60. Rosmond R (2003) Stress induced disturbances of the HPA axis: a pathway to Type 2 diabetes? Med Sci Monit 9:RA35–RA39
61. Khlat M, Jusot F, Ville I (2009) Social origins, early hardship, and obesity: a strong association in women, but not men? Soc Sci Med 68:1692–1699
62. Pungello E, Kainz K, Burchinal M, Wasik B, Sparling J, Ramey C, Campbell F (2010) Early educational intervention, early cumulative risk, and the early home environment as predictors of young adult outcomes within a high-risk sample. Child Dev 81:410–426

63. Danielzik S, Czerwinski-Mast M, Langnase K, Dilba B, Muller M (2004) Parental overweight, socioeconomic status and high birth weight are the major determinants of overweight and obesity in 5–7 year old children: baseline data of the Kiel Obesity Prevention Study (KOPS). Int J Obes Relat Metab Disord 28:1494–1502
64. Huerta M, Bibi H, Haviv J, Gdalevich M (2006) Parental smoking and education as determinants of overweight in Israeli children. Prev Chronic Dis 3:1–9
65. Hui L, Nelson E, Yu L, Li A, Fok T (2003) Risk factors for childhood overweight in 6- to 7-year old Hong Kong children. Int J Obes Relat Metab Disord 27:1411–1418
66. Gorber SC, Tremblay M, Moher D, Gorber B (2007) A comparison of direct vs. self-report measures for assessing height, weight, and body mass index: a systematic review. Obes Rev 8:307–326
67. Mamun AA, Cramb S, McDermott BM, O'Callaghan MJ, Najman JM, Williams GM (2007) Adolescents' perceived weight associated with depression in young adulthood: a longitudinal study. Obesity 15:3097–3105
68. Freedman D, Khan L, Serdula M, Ogden C, Dietz W (2006) Racial and ethnic differences in secular trends for childhood BMI, weight, and height. Obesity 14:301–308