

Comorbidity, Codevelopment, and Temporal Associations Between Body Mass Index and Internalizing Symptoms From Early Childhood to Adolescence

Praveetha Patalay, PhD; Charlotte A. Hardman, PhD

IMPORTANCE Obesity and internalizing mental illness begin in childhood, have common risk factors, and are leading causes of disease burden.

OBJECTIVES To examine the comorbidity, codevelopment, and temporal precedence in body mass index (BMI) and internalizing symptoms from early childhood to midadolescence and to investigate the sex differences and socioeconomic confounding in their association.

DESIGN, SETTING, AND PARTICIPANTS This longitudinal study used data on individuals from the Millennium Cohort Study, a nationally representative, prospective birth cohort study in the United Kingdom of more than 19 000 individuals born from September 1, 2000, to January 11, 2002, who were assessed to date at the ages of 9 months and 3, 5, 7, 11, and 14 years. Data from 17 215 participants (88.21% of full cohort sample) with socioeconomic information in early childhood and BMI or internalizing symptoms during at least 1 assessment from 3 to 14 years of age were included in the present study. Data analysis was performed from May to November 2018.

MAIN OUTCOMES AND MEASURES Outcomes were BMI estimated from objectively measured height and weight and internalizing symptoms assessed using parental reports. Three measures of socioeconomic position were controlled for: parent educational level, occupational status, and equivalized family income.

RESULTS Among the 17 215 participants (8394 [48.8%] female), obesity and internalizing problems were not more likely to co-occur in early childhood (odds ratio [OR] at 3 years, 1.02; 95% CI, 0.69-1.50), whereas at 11 and 14 years of age, they were more likely to co-occur (OR at 11 years, 1.68; 95% CI, 1.38-2.05; OR at 14 years, 1.49; 95% CI, 1.22-1.83). Piecewise latent growth models revealed no codevelopment of BMI and internalizing symptoms from 3 to 7 years of age ($r = 0.01$), whereas their slopes were associated between 7 and 14 years of age ($r = 0.23$). Initial level and rate of change in each domain in early childhood was not associated with rate of change in the other domain at older ages. Cross-lagged models indicated no cross-domain pathways before 7 years of age, with some weak cross-domain pathways emerging between 7 and 14 years of age. Socioeconomic position attenuated some of these associations, leaving a BMI-to-internalizing symptoms pathway in later childhood and an internalizing symptoms-to-BMI pathway in early adolescence.

CONCLUSIONS AND RELEVANCE The findings suggest that BMI and internalizing symptoms become more associated and reciprocal as children get older and that some of their temporal associations can be attributed to socioeconomic factors. The emergence of cross-domain temporal pathways in middle childhood suggests that social, physiologic, and psychological processes begin to play an increasingly important role in these health outcomes. Prevention and early intervention efforts may benefit from targeting both health outcomes in childhood.

Author Affiliations: Department of Psychological Sciences, University of Liverpool, Liverpool, United Kingdom (Patalay, Hardman); MRC Unit for Lifelong Health and Ageing and Centre for Longitudinal Studies, University College London, London, United Kingdom (Patalay).

Corresponding Author: Praveetha Patalay, PhD, MRC Unit for Lifelong Health and Ageing and Centre for Longitudinal Studies, University College London, Gower St, London WC1E 6BT, United Kingdom (p.patalay@ucl.ac.uk).

JAMA Psychiatry. 2019;76(7):721-729. doi:10.1001/jamapsychiatry.2019.0169
Published online March 20, 2019.

Obesity and common mental health disorders make large contributions to the global disease burden.^{1,2} Both have their roots in childhood,^{3,4} with substantial negative consequences for a range of health and economic outcomes through the lifecourse.⁵⁻⁸ Evidence from some studies also indicates substantial increases in childhood prevalence of obesity and internalizing symptoms in recently born generations,⁹⁻¹¹ further underscoring the importance of understanding their development, risk factors, and early intervention. Tackling these public health challenges is high on international government agendas.^{12,13}

Both cross-sectional and longitudinal evidence exists of moderate associations between obesity and internalizing symptoms in childhood¹⁴ and adulthood.^{15,16} Longitudinal evidence of both pathways exists whereby earlier obesity is associated with later internalizing symptoms¹⁷ and internalizing symptoms are associated with later obesity¹⁵; however, the possibility of bidirectional development cannot be overlooked. Longitudinal analyses aiming to understand the codevelopment and temporal precedence are mainly found in literature on the adult and the aging populations.^{18,19} To our knowledge, no studies have modeled the longitudinal codevelopment (association in rates of change) in body mass index (BMI) (calculated as weight in kilograms divided by height in meters squared) and internalizing symptoms from early childhood through adolescence. A study¹⁸ in adults found, for example, that from ages in the early 20s to early 40s, the codevelopment in depressive symptoms and BMI is moderate in females and small in males. In the pediatric literature, studies^{20,21} investigating temporal precedence are limited to childhood and suggest that there is no temporal association between these health domains in early childhood, with evidence of a BMI to internalizing pathway emerging in midchildhood. Of note, although sex differences in the longitudinal associations between BMI and internalizing symptoms are consistently reported in the adult literature,¹⁸ findings are mixed^{14,21} or not investigated in children.²⁰

Socioeconomic gradients in obesity and poor mental health are well established,^{22,23} with children from disadvantaged backgrounds more likely to experience more unhealthy BMIs and poorer mental health cross-sectionally and to longitudinally exhibit greater increases in both domains during childhood.^{17,24} In understanding the association between these 2 domains, it is essential to investigate whether the observed associations are mainly a function of shared socioeconomic disadvantage or whether they have an association beyond what is explained by socioeconomic factors. The shared risk hypothesis would be that the codevelopment and temporal pathways between these outcomes are explained by socioeconomic factors,²⁵ a hypothesis that was empirically tested in the current study.

Using a large, nationally representative sample with objective BMI measurements and consistently measured internalizing symptoms, we estimated comorbidity at different ages and codevelopment (ie, association in their slopes) from early to midchildhood (3-7 years of age) and from midchildhood to midadolescence (7-14 years of age). We then examined temporal precedence (which comes first) in the longitudinal as-

Key Points

Question How are body mass index and internalizing symptoms associated from early childhood to midadolescence?

Findings In this study of 17 215 individuals from a birth cohort study in the United Kingdom, there were no associations between body mass index and internalizing symptoms in early childhood; however, between midchildhood and adolescence, associations were observed in their codevelopment, with increased likelihood of comorbidity.

Meaning The findings suggest that obesity and internalizing problems are more likely to co-occur as children get older and that there is an association in the development of body mass index and internalizing symptoms from midchildhood.

sociation between BMI and internalizing symptoms. Sex differences and a shared socioeconomic risk hypothesis for comorbidity, codevelopment, and temporal precedence were also examined.

Methods

Participants

Participants were from the Millennium Cohort Study (MCS),²⁶ a UK birth cohort study of 19 517 individuals born at the start of the millennium (September 1, 2000, to January 11, 2002) who have been assessed at 6 waves to date: 9 months and 3, 5, 7, 11, and 14 years. The MCS cohort is sampled from all 4 countries of the United Kingdom and oversampled in areas of higher deprivation and ethnic minorities; sample weights were applied to ensure population-representative estimates.²⁷ Greater details of the study design, variables, and attrition can be found on the Centre for Longitudinal Studies website (<https://cls.ucl.ac.uk/cls-studies/millennium-cohort-study/>). For the purposes of this research, the sample consisted of 17 215 children (88.2% of full cohort; 1 per family) with BMI and internalizing symptom data available for at least 1 of the 5 waves used in this analysis (3-14 years of age) and complete childhood socioeconomic data. Analyses of response and attrition indicated that socioeconomic disadvantage, parental age, housing tenure, ethnic minority, and parent employment status determine nonresponse.²⁸ Data analysis was performed from May to November 2018. Written informed consent was received from parents or caregivers and all children, and data were deidentified. Ethics approval for each wave of the MCS was received from a national research ethics committee; no ethics approval for this specific analysis was required.

Measures

Body Mass Index

Height to the nearest millimeter and weight were recorded (with children wearing no shoes and outdoor clothing) by trained interviewers using standardized instruments, from which we estimated BMI. Similar to another study¹⁷ that used these data, outlier values (BMI <10 or >50) were removed, resulting in 4 values removed from the second wave, 0 from the

third wave, 3 from the fourth wave, 5 from the fifth wave, and 1 from the sixth wave. Obesity was estimated using thresholds of the International Obesity Taskforce, accounting for sex and age in months at the time of assessment.²⁹

Internalizing Symptoms

Internalizing symptoms were measured using the emotional problems subscale of the Strengths and Difficulties Questionnaire (SDQ)³⁰ reported by a parent or guardian (>95% of respondents were mothers). The scale consists of 5 items (eg, often seems worried), which are summed to create an overall score that can range from 0 to 10, with a score of 5 or above being considered high. Self-reported symptoms at 14 years of age were also assessed using the Short Moods and Feelings Questionnaire,³¹ which is a 13-item measure of depressive symptoms, with each item rated on a 3-point scale (true, sometimes, and not true). We present co-occurrence of BMI with these data to provide contrast with parent-reported symptoms.

Socioeconomic Position

Three measures of socioeconomic position (SEP) were used in this study. Household income is represented in Organisation for Economic Co-operation and Development UK equivalized quintiles (number [unweighted percentage] in quintile 1 [lowest income quintile], 4045 [23.5%]; in quintile 2, 3845 [22.3%]; in quintile 3, 3334 [19.4%]; in quintile 4, 3104 [18.0%]; and in quintile 5 [highest income quintile], 2887 [16.8%]). Parental educational levels are represented by the highest National Vocational Qualifications (NVQ) level in the household (number [unweighted percentage] in NVQ levels 1-5, where NVQ level 1 represents General Certificate of Secondary Education grade D-G or lower/no qualifications [2810 (unweighted 16.3%)]); level 2, General Certificate of Secondary Education grades A*-C and other equivalent [4112 (23.9%)]); level 3, A-levels and other equivalent [2826 (16.4%)]); and level 4, earning a degree [5829 (33.9%)]); and level 5, having a higher degree or diploma [1638 (9.5%)].³² Employment status and occupational class are represented using the National Statistics Socio-economic Classification 3-class coding of higher managerial and professional occupations (7746 [45.0%]), intermediate occupations (3405 [19.8%]), and routine and manual occupations (5286 [30.7%])³³ and a separate fourth category denoting a workless household (778 [4.5%]). These 3 SEP measures were moderately correlated ($r = 0.56$ - 0.62).

Statistical Analysis

Descriptive statistics and number of individuals with obesity and high rates of symptoms were estimated, followed by extent and odds of their co-occurrence at each age. All modeling was conducted in Mplus³⁴ using full information maximum likelihood and weights. Sex and ethnicity were controlled for in all models. Given known comorbidity of externalizing and internalizing symptoms³⁵ and bias in parent-reported symptoms attributable to their own mental health,³⁶ we controlled for externalizing symptoms (SDQ conduct problems scale)³⁰ and the reporting of parent's mental health (K6 scale)³⁷ on the internalizing scores at each time point. To investigate

codevelopment of BMI and internalizing symptoms, piecewise latent growth models with 2 slopes were estimated for each domain: 1 from early to midchildhood (3, 5, and 7 years of age) and 1 from midchildhood to early adolescence (7, 11, and 14 years of age), accounting for the differences in gaps between time points in the model specification for estimating the slope (ie, uneven gaps between time points are specified when estimating the slope to accurately reflect the structure of the data). This modeling approach resulted in simultaneous estimation of an intercept (representing initial levels) and 2 slope estimates for each domain for each participant. To investigate the associations between initial rate and development in each domain in early childhood (3-7 years of age) with development at later ages (7-14 years of age), regression paths determining the BMI and internalizing symptoms slopes were estimated (first without socioeconomic factors and then controlling for these). To examine sex differences, a multigroup model was estimated and differences in slope associations and regression paths were compared between boys and girls.

To further investigate temporal precedence, cross-lagged models were estimated with longitudinal pathways from BMI to internalizing symptoms and vice versa across time points, while accounting for temporal stability in each construct over time and their concurrent associations at each time point. This was first estimated for the overall sample, and then sex differences were investigated using a multigroup cross-lagged model with estimated sex differences in each cross-lagged pathway. The final model included pathways from SEP variables to BMI and internalizing symptoms at all time points. A 2-tailed $P = .05$ was considered to be statistically significant.

Results

Data from 17 215 participants (8394 [48.8%] female) were analyzed using data from assessments at 3, 5, 7, 11, and 14 years of age. Data were available for 15 369 participants at 3 years of age, 15 147 at 5 years of age, 13 764 at 7 years of age, 13 146 at 11 years of age, and 11 450 at 14 years of age. Of the 17 215 participants included in the analysis, 9170 (53.3%) had available data in all 5 waves, followed by 3105 (18.0%) in 4 waves, 2073 (12.0%) in 3 waves, 1520 (8.8%) in 2 waves, and 1347 (7.8%) in 1 wave. In the unweighted sample, 14 192 (82.4%) identified as white, 1609 (9.4%) as Asian, 636 (3.7%) as black, 525 (3.1%) as mixed ethnicity, and 253 (1.5%) as other ethnicities.

Descriptive statistics for BMI and internalizing symptoms are presented in Table 1. The BMI decreased slightly from 3 to 7 years of age and then increased with age, with girls having a higher BMI at 11 and 14 years of age.⁹ Internalizing symptoms increased with age, and a substantial sex difference in prevalence was observed at 14 years of age, with more internalizing symptoms in girls. There was co-occurrence of obesity and above-threshold symptoms at each age and increased comorbidity with age (Table 1). The correlations between BMI and internalizing symptoms were -0.03 at 3 years of age, -0.02 at 5 years of age, 0.01 at 7 years of age, 0.06 at

Table 1. Descriptive Statistics for the BMI and Internalizing Symptoms and Their Co-occurrence at Each Time Point^a

Variable	Full Sample				Boys			Girls			Obese and High Internalizing, No. (%)	
	Obese or High Internalizing, No. (%) ^a		Obese and High Internalizing, No. (%)		Mean (SD)	No.	Obese or High Internalizing, No. (%) ^a		Mean (SD)	No.		
	No.	Mean (SD)	No.	Mean (SD)			No.	Mean (SD)				
BMI												
Age, y												
3	14 317	16.85 (2.04)	650 (4.5)	30 (4.9) ^c	7240	17.00 (2.14)	332 (4.6)	15 (4.8) ^c	7077	16.70 (1.93)	318 (4.5)	15 (5.1) ^c
5	14 949	16.36 (1.90)	864 (5.8)	49 (6.0) ^c	7635	16.40 (1.91)	409 (5.4)	26 (6.7) ^c	7314	16.33 (1.88)	455 (6.2)	23 (5.3) ^c
7	13 414	16.66 (2.36)	814 (6.1)	80 (10.1) ^c	6799	16.6 (2.32)	365 (5.4)	40 (11.2) ^c	6615	16.71 (2.40)	449 (6.8)	40 (9.2) ^c
11	12 801	19.25 (3.66)	855 (6.7)	137 (17.9) ^c	6484	19.05 (3.55)	412 (6.4)	72 (19.4) ^c	6317	19.47 (3.76)	443 (7.0)	65 (16.5) ^c
14	10 767	21.47 (4.14)	814 (7.6)	137 (19.8) ^c	5478	20.95 (4.04)	403 (7.4)	57 (16.6) ^c	5289	22.00 (4.18)	411 (7.8)	80 (22.9) ^c
Internalizing Symptoms												
Age, y												
3	14 694	1.38 (1.52)	655 (4.5)	30 (5.1) ^d	7497	1.38 (1.52)	324 (4.3)	15 (5.2) ^d	7197	1.38 (1.52)	331 (4.6)	15 (4.9) ^d
5	14 694	1.40 (1.6)	823 (5.6)	49 (6.2) ^d	7514	1.37 (1.61)	417 (5.6)	26 (6.5) ^d	7180	1.43 (1.59)	406 (5.7)	23 (5.8) ^d
7	13 401	1.54 (1.78)	1024 (7.6)	80 (8.1) ^d	6819	1.51 (1.81)	543 (8.0)	40 (7.7) ^d	6582	1.56 (1.74)	481 (7.3)	40 (8.6) ^d
11	11 822	1.87 (2.00)	1316 (11.1)	137 (11.0) ^d	5943	1.81 (2.00)	643 (10.8)	72 (11.9) ^d	5879	1.93 (1.99)	673 (11.5)	65 (10.1) ^d
14	10 123	2.04 (2.14)	1369 (13.5)	137 (11.2) ^d	5065	1.75 (2.02)	545 (10.8)	57 (11.6) ^d	5058	2.33 (2.22)	824 (16.3)	80 (10.9) ^d
SR symptoms at 14 y of age	11 190	5.52 (5.85)	1731 (15.5)	166 (9.6) ^d	5539	4.03 (4.57)	456 (8.2)	47 (10.3) ^d	5551	6.99 (6.57)	1275 (22.6)	119 (10.3) ^d

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); SR, self-reported.

^a Descriptive estimates are presented for available participants at each time point without accounting for survey design weights and sample attrition in these estimates.^b The number (percentage) of obese participants is given under the BMI heading and was determined using the International Obesity Taskforce thresholds for children; the number (percentage) of participants with high internalizing symptoms is given under the Internalizing Symptoms heading, showing children above the established clinical cutoff for the Strengths and Difficulties Questionnaire.^c Of those who are classified as obese based on BMI.^d Of those with above cutoff levels of internalizing symptoms.

11 years of age, and 0.10 at 14 years of age. The numbers of individuals above clinical thresholds for both obesity and internalizing symptoms were 30 (0.2%) at 3 years of age, 49 (0.3%) at 5 years of age, 80 (0.6%) at 7 years of age, 137 (1.2%) at 11 years of age, and 137 (1.4%) at 14 years of age.

Figure 1 presents the odds of co-occurrence at each age. Controlling for SEP comorbidity was more likely at 11 and 14 years of age. Obesity and internalizing symptoms were not more likely to co-occur in early childhood (odds ratio [OR] at 3 years of age, 1.02; 95% CI, 0.69-1.50), whereas at 11 and 14 years of age, they were more likely to co-occur (OR at 11 years of age, 1.68; 95% CI, 1.38-2.05; OR at 14 years of age, 1.49; 95% CI, 1.22-1.83). Interactions terms in the analysis revealed no sex differences in the odds of co-occurrence at all ages. For contrast with self-reported symptoms, we also estimated the co-occurrence with obesity at 14 years of age and observed that similar proportions with a high rate of self-reported symptoms were obese (9.6%), compared with parent-reported symptoms (11.2%), albeit with higher proportions with above-threshold symptoms and a larger sex difference in the prevalence of above-threshold symptoms.

Table 2 gives the intercepts for both outcomes and the slopes for each in the 2 developmental periods and how these are associated. In terms of cross-domain associations in slopes (codevelopment), there was no codevelopment from the ages of 3 to 7 years ($r = 0.01$; $P = .07$), whereas there was observable codevelopment between the ages of 7 and 14 years ($r = 0.23$; $P < .001$), suggesting that from midchildhood onward there was an association in the rate of development in BMI and internalizing symptoms.

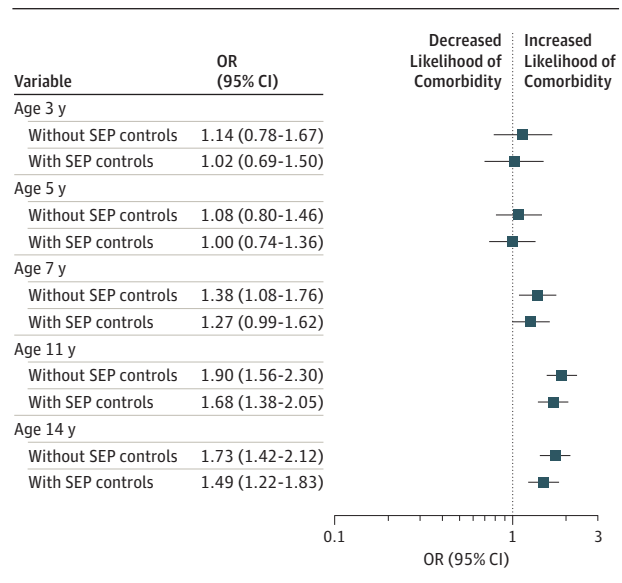
When predicting the BMI and internalizing slopes (Table 3), initial levels in each domain predicted the slopes in the same domain. However, there were no cross-domain predictions from the initial level or the rate of change (slopes) and the rate of development in the other domain. Multigroup analysis indicated no sex differences in these associations (multigroup model mean square error of approximation [RMSEA], 0.026; comparative fit index [CFI], 0.94; Tucker-Lewis index [TLI], 0.94; and standardized root mean square residual [SRMR], 0.047).

Figure 2 shows the baseline and socioeconomic shared risk-adjusted cross-lagged models, including all the pathways between the 2 health domains (controls are not shown) and the model fit indexes, which demonstrate adequate fit. There were no cross-lagged paths between the ages of 3 and 7 years, with pathways emerging between the ages of 7 and 11 years in both directions and an internalizing to BMI pathway between the ages of 11 and 14 years. A multigroup analysis indicated no significant sex differences in any of the cross-lagged pathways (multigroup model RMSEA, 0.023; CFI, 0.96; TLI, 0.95; and SRMR, 0.033). Adjusting for shared socioeconomic risk factors resulted in some attenuation of pathways, with the BMI to internalizing symptoms pathway remaining from 7 to 11 years and the internalizing symptoms to BMI pathway from 11 to 14 years.

Discussion

In a large, nationally representative cohort study with consistently measured BMI and internalizing symptoms, we inves-

Figure 1. Odds of Co-occurrence of Obesity and High Internalizing Symptoms



The odds ratio (OR) estimates (95% CIs) are controlling for sociodemographic characteristics. Interaction analyses indicated that there were no sex differences in the odds of co-occurrence at these ages. SEP indicates socioeconomic position.

tigated the co-occurrence of obesity and internalizing problems at 3, 5, 7, 11, and 14 years of age and examined the associations in the longitudinal development of BMI and internalizing symptoms using 2 advanced statistical approaches. The results suggest that these 2 health outcomes increasingly co-occur with age and that there is an emergent positive association in their codevelopment in midchildhood. However, there was no discernible temporal association between BMI development and internalizing symptoms in early childhood. In contrast, a reciprocal association between these 2 health domains emerged between 7 and 14 years of age; however, these cross-domain associations were not strong. Thus, this study provides new and detailed insights into how these domains of health interact from early childhood into midadolescence, a period that encompasses different developmental periods and transitions.

Estimates of co-occurrence showed that comorbidity was more likely as children aged. These findings have bearing on the issue of multimorbidity across the physical-mental health axis, and the implications of comorbidity in childhood and its age at onset for later health, social, and economic consequences are currently poorly understood. However, the increasing prevalence of these chronic health outcomes in childhood in recent generations may increase the likelihood of their co-occurrence at earlier ages,^{9,11} and future research would benefit from understanding the later health implications of comorbidity in these children.

Temporal analysis of rates of change demonstrated no cross-domain associations between rates of change in one developmental period with rate of change in the subsequent developmental period across these outcomes. Cross-lag analysis indicated emergence of a BMI-to-internalizing pathway in

Table 2. Intercept and Slope Estimates From the Piecewise Latent Growth Model and Their Pairwise Correlations^a

Variable	Mean (SE)	Correlations					
		1	2	3	4	5	6
BMI							
1. Intercept	16.62 (0.72)						
2. Slope for ages 3-7 y	-0.01 (0.22)	0.59 ^b					
3. Slope for ages 7-14 y	0.71 (0.21)	0.29 ^b	0.73 ^b				
Internalizing symptoms							
4. Intercept	0.77 (0.62)	-0.09 ^b	0.00	0.01			
5. Slope for ages 3-7 y	0.02 (0.21)	0.06 ^b	0.01	0.02 ^b	0.15 ^b		
6. Slope for ages 7-14 y	0.03 (0.15)	-0.01	0.13 ^b	0.23 ^b	-0.37 ^b	-0.03 ^b	

Abbreviation: BMI, body mass index.

^a Model fit for piecewise growth model: root mean square error of approximation, 0.04; comparative fit index, 0.94; Tucker-Lewis index, 0.90; and standardized root mean square residual, 0.06.^b $P < .05$.Table 3. Predicting Development in BMI and Internalizing Symptoms^a

Variable	Standardized Coefficient (SE)	
	Baseline Model	Model With Socioeconomic Controls
Predicting BMI Development		
Predicting BMI slope ages 3-7 y		
Intercept BMI	0.26 (.05) ^b	0.26 (0.05) ^b
Intercept internalizing symptoms	-0.02 (.02)	-0.03 (0.02)
Predicting BMI slope ages 7-14 y		
Intercept BMI	0.06 (0.04)	0.06 (0.04)
Intercept internalizing symptoms	0.01 (0.02)	-0.01 (0.02)
BMI slope ages 3-7 y	0.49 (0.06) ^b	0.48 (0.06) ^b
Internalizing symptoms slope ages 3-7 y	-0.01 (0.02)	-0.01 (0.02)
Predicting Internalizing Symptom Development		
Predicting internalizing symptoms slope at ages 3-7 y		
Intercept internalizing symptoms	-0.18 (0.05) ^b	-0.19 (0.05) ^b
Intercept BMI	0.02 (0.02)	0.01 (0.02)
Predicting internalizing symptoms slope ages 7-14 y		
Intercept internalizing symptoms	-0.27 (0.03) ^b	-0.27 (0.03) ^b
Intercept BMI	-0.02 (0.03)	-0.02 (0.03)
Internalizing symptoms slope ages 3-7 y	-0.25 (0.05) ^b	-0.26 (0.04) ^b
BMI slope ages 3-7 y	0.05 (0.03) ^b	0.05 (0.03)

Abbreviation: BMI, body mass index.

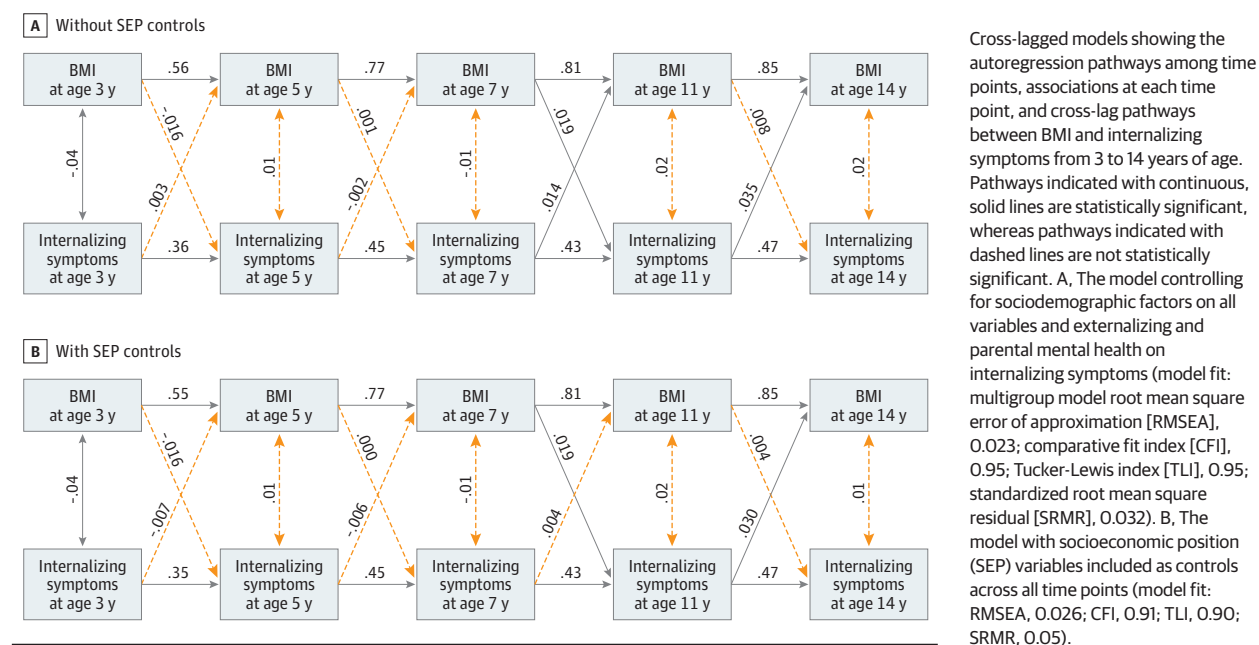
^a Baseline model and model with socioeconomic controls. Model fit for baseline model: root mean square error of approximation, 0.03; comparative fit index, 0.93; Tucker-Lewis index, 0.93; and standardized root mean square residual, 0.04. Model fit for socioeconomic position-adjusted model: root mean square error of approximation, 0.03, comparative fit index, 0.90, Tucker-Lewis index, 0.91, and standardized root mean square residual, 0.06.^b $P < .05$.

middle childhood, a similar finding from previous studies in childhood.^{20,21} However, in early adolescence, we observed an internalizing symptoms-to-BMI pathway, suggesting that the salience of these bidirectional pathways might be developmentally varied. Our study does not provide information on the mechanisms in the bidirectional associations between in-

ternalizing symptoms and BMI, but varied explanations have been hypothesized.³⁸ Children with higher BMI may experience weight-related discrimination, which over time leads to increased depressive symptoms, as has been found in adults.^{39,40} Previous research indicates that prejudice toward children with obesity is prevalent in later childhood but less so among 5-year-old children.⁴¹ Awareness and internalization of societal attitudes about weight also increase during childhood.⁴² Body dissatisfaction and negative body image begin to predict depressive symptoms around the transition to adolescence.⁴³ Dietary restraint begins to emerge in later childhood,⁴⁴ and futile attempts to control one's weight while in an obesogenic environment might result in feelings of failure, guilt, impaired self-esteem, and lack of perceived control,⁴⁵ leading to depressive symptoms. Taken together, this could explain our finding that the association between BMI and internalizing symptoms did not emerge until midchildhood. Potential explanations for the internalizing symptoms to BMI longitudinal pathway include emotional eating of high-calorie "comfort foods" and stress-induced alterations to metabolic signals that control energy balance.^{38,46} These mechanisms are also potential mediators of the observed socioeconomic differentials in both health outcomes. These mechanisms and their possible socioeconomic differential effects should be examined by future studies in longitudinal pediatric populations. Recent research in adults has used genetic instruments related to BMI to demonstrate that there is a potential association between higher BMI and the likelihood of depressive symptoms⁴⁷; such approaches applied to developmental samples and examining both directional pathways may help us understand how these health outcomes are associated during the life course.

The lack of sex differences in the association between BMI and internalizing symptoms diverges from adult studies^{15,18,19} in which sex differences were delineated. This finding replicates the lack of sex differences found in a previous study²¹ that investigated temporal precedence in childhood. Our findings suggest that at least until midadolescence, there are no sex differences in how BMI and internalizing symptoms are developmentally associated. In combination with the adult literature, these findings highlight the possibility that sex dif-

Figure 2. Temporal Precedence in Body Mass Index (BMI) and Internalizing Symptom Associations



ferences in how these outcomes are associated might emerge in later adolescence. Investigating when and why these differences occur might help us understand sex-specific risk factors, if any, in their longitudinal association.

Our study was the first, to our knowledge, to investigate whether the observed associations between BMI and internalizing symptoms are merely a function of the known socioeconomic disparities in the experience and development of both weight gain and internalizing symptoms in childhood or whether they have unique temporal associations over time. We found that the few cross-domain predictors of longitudinal BMI and internalizing symptom development as measured by latent slopes are attenuated by the inclusion of socioeconomic factors, implicating a shared origin in terms of socioeconomic risk. In the cross-lagged model adjusted for SEP, 2 pathways remained: BMI at 7 years of age still predicted internalizing symptoms at 11 years of age, which, in turn, predicted BMI at 14 years of age. Understanding of the mechanisms through which socioeconomic disadvantage is associated with risk for both obesity and poor mental health and their associations during development might help in designing prevention and health promotion interventions that are sensitive to socioeconomic disadvantage. The shared socioeconomic risk in the development of these health outcomes might be explained by many factors. For instance, socioeconomically deprived areas tend to have poorer access to healthy food and green spaces, which may be a contributing factor to social disparities in both health outcomes, compounding effects of family-level socioeconomic disadvantage.^{48,49}

In light of our findings of an increasing association between BMI and internalizing symptoms during development and shared socioeconomic factors, efforts to reduce risk factors associated with socioeconomic disadvantage and the obesogenic or psychopathogenic environments for chil-

dren might be positively associated with both outcomes. For example, a systematic review⁵⁰ in adults reported evidence of the efficacy of “green interventions,” whereby exposure to the natural environment improved mood, increased physical activity, and reduced cardiovascular disease-related mortality. There has been little consideration of the efficacy of such approaches in children beyond effects on physical activity.⁵¹

Limitations

This analysis uses a large, nationally representative sample with objective anthropometric measures at all study waves. As with all longitudinal cohorts, there was attrition in the study waves; however, we accounted for attrition using a well-established method, and any remaining bias in the findings is likely to be an underreporting in the size of the associations. Because of the size and population-based nature of the study, clinical interviews are not used to assess internalizing symptoms, instead relying on parent reports. It is possible that the biases associated with parental report masked the stronger associations between these factors because parent- and self-report of symptoms were weakly associated in this cohort⁵²; however, comparisons of rates of comorbidity at 14 years of age identified a similar proportion of those with high rates of symptoms having comorbid obesity, albeit higher overall prevalence rates based on self-reported symptoms.

Conclusions

Using longitudinal data from early childhood to midadolescence, we found that a codevelopmental and bidirectional association between BMI and internalizing symptoms emerged in midchildhood, with little evidence of an asso-

ciation between the occurrence and development in these domains earlier in childhood. Although sex differences existed in prevalence, there were no observable sex differences in how BMI and internalizing symptoms codeveloped and interacted through these years. Socioeconomic factors attenuated some of the observed associations between

these variables, stressing the role socioeconomic disadvantage plays in the development and association between these 2 key childhood health outcomes. Our findings highlight the importance of early interventions that target both weight and mental health in childhood to minimize negative outcomes later in adulthood.

ARTICLE INFORMATION

Accepted for Publication: January 2, 2019.

Published Online: March 20, 2019.

doi:10.1001/jamapsychiatry.2019.0169

Author Contributions: Dr Patalay had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

Concept and design: Both authors.

Acquisition, analysis, or interpretation of data: Both authors.

Drafting of the manuscript: Both authors.

Critical revision of the manuscript for important intellectual content: Both authors.

Statistical analysis: Patalay.

Conflict of Interest Disclosures: Dr Hardman reported receiving grants from the American Beverage Association and personal fees from International Sweeteners Association outside the submitted work. No other disclosures were reported.

Additional Contributions: The Economic and Social Research Council and a consortium of UK government departments provided funding for the Millennium Cohort Study (MCS) through the Centre for Longitudinal Studies (CLS) at the UCL Institute of Education, London, United Kingdom. A large number of stakeholders from academic, policymaker, and funder communities and colleagues at CLS were involved in data collection and management. We are grateful for the cooperation of the MCS families who voluntarily participated in the study.

REFERENCES

- Whiteford HA, Degenhardt L, Rehm J, et al. Global burden of disease attributable to mental and substance use disorders: findings from the Global Burden of Disease Study 2010. *Lancet*. 2013;382(9904):1575-1586. doi:10.1016/S0140-6736(13)61611-6
- Afshin A, Forouzanfar MH, Reitsma MB, et al; GBD 2015 Obesity Collaborators. Health effects of overweight and obesity in 195 countries over 25 years. *N Engl J Med*. 2017;377(1):13-27. doi:10.1056/NEJMoa1614362
- Singh AS, Mulder C, Twisk JWR, van Mechelen W, Chinapaw MJM. Tracking of childhood overweight into adulthood: a systematic review of the literature. *Obes Rev*. 2008;9(5):474-488. doi:10.1111/j.1467-789X.2008.00475.x
- Kessler RC, Berglund P, Demler O, Jin R, Merikangas KR, Walters EE. Lifetime prevalence and age-of-onset distributions of DSM-IV disorders in the National Comorbidity Survey Replication. *Arch Gen Psychiatry*. 2005;62(6):593-602. doi:10.1001/archpsyc.62.6.593
- Kessler RC, Foster CL, Saunders WB, Stang PE. Social consequences of psychiatric disorders. I: educational attainment. *Am J Psychiatry*. 1995;152(7):1026-1032. doi:10.1176/ajp.152.7.1026
- Goodman A, Joyce R, Smith JP. The long shadow cast by childhood physical and mental problems on adult life. *Proc Natl Acad Sci U S A*. 2011;108(15):6032-6037. doi:10.1073/pnas.1016970108
- Sahoo K, Sahoo B, Choudhury AK, Sofi NY, Kumar R, Bhadoria AS. Childhood obesity: causes and consequences. *J Family Med Prim Care*. 2015;4(2):187-192. doi:10.4103/2249-4863.154628
- Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. *Pediatrics*. 1998;101(3, pt 2):518-525.
- Johnson W, Li L, Kuh D, Hardy R. How has the age-related process of overweight or obesity development changed over time? co-ordinated analyses of individual participant data from five United Kingdom birth cohorts. *PLoS Med*. 2015;12(5):e1001828. doi:10.1371/journal.pmed.1001828
- Fink E, Patalay P, Sharpe H, Holley S, Deighton J, Wolpert M. Mental health difficulties in early adolescence: a comparison of two cross-sectional studies in England from 2009 to 2014. *J Adolesc Health*. 2015;56(5):502-507. doi:10.1016/j.jadohealth.2015.01.023
- Patalay P, Gage S. Trends in millennial adolescent mental health and health related behaviours over ten years: a population cohort comparison study Preprint. Published online September 5, 2018. bioRxiv. doi:10.1101/407585
- Swinburn BA, Sacks G, Hall KD, et al. The global obesity pandemic: shaped by global drivers and local environments. *Lancet*. 2011;378(9793):804-814. doi:10.1016/S0140-6736(11)60813-1
- US Department of Health. *No Health Without Mental Health: A Cross-government Mental Health Outcomes Strategy for People of All Ages*. London, England: Crown; 2011.
- Sutaria S, Devakumar D, Yasuda SS, Das S, Saxena S. Is obesity associated with depression in children? systematic review and meta-analysis. *Arch Dis Child*. 2019;104(1):64-74.
- Luppino FS, de Wit LM, Bouvy PF, et al. Overweight, obesity, and depression: a systematic review and meta-analysis of longitudinal studies. *Arch Gen Psychiatry*. 2010;67(3):220-229. doi:10.1001/archgenpsychiatry.2010.2
- de Wit L, Luppino F, van Straten A, Penninx B, Zitman F, Cuijpers P. Depression and obesity: a meta-analysis of community-based studies. *Psychiatry Res*. 2010;178(2):230-235. doi:10.1016/j.psychres.2009.04.015
- Kelly Y, Patalay P, Montgomery S, Sacker A. BMI development and early adolescent psychosocial well-being: UK Millennium Cohort Study. *Pediatrics*. 2016;138(6):e20160967. doi:10.1542/peds.2016-0967
- Konttinen H, Kiviruusu O, Huurre T, Haukka A, Aro H, Marttunen M. Longitudinal associations between depressive symptoms and body mass index in a 20-year follow-up. *Int J Obes (Lond)*. 2014;38(5):668-674. doi:10.1038/ijo.2013.151
- Forman-Hoffman VL, Yankey JW, Hillis SL, Wallace RB, Wolinsky FD. Weight and depressive symptoms in older adults: direction of influence? *J Gerontol B Psychol Sci Soc Sci*. 2007;62(1):S43-S51. doi:10.1093/geronb/62.1.S43
- Jansen PW, Mensah FK, Clifford SA, Tiemeier H, Nicholson JM, Wake M. Development of mental health problems and overweight between ages 4 and 11 years: a population-based longitudinal study of Australian children. *Acad Pediatr*. 2013;13(2):159-167. doi:10.1016/j.acap.2012.12.001
- Bradley RH, Houts R, Nader PR, O'Brien M, Belsky J, Crosnoe R. The relationship between body mass index and behavior in children. *J Pediatr*. 2008;153(5):629-634. doi:10.1016/j.jpeds.2008.05.026
- Wardle J, Brodersen NH, Cole TJ, Jarvis MJ, Boniface DR. Development of adiposity in adolescence: five year longitudinal study of an ethnically and socioeconomically diverse sample of young people in Britain. *BMJ*. 2006;332(7550):1130-1135. doi:10.1136/bmj.38807.594792.AE
- Sacker A, Wiggins RD. Age-period-cohort effects on inequalities in psychological distress, 1981-2000. *Psychol Med*. 2002;32(6):977-990. doi:10.1017/S0033291702006013
- Patalay P, Moulton V, Goodman A, Ploubidis GB. Cross-domain symptom development typologies and their antecedents: results from the UK Millennium Cohort Study. *J Am Acad Child Adolesc Psychiatry*. 2017;56(9):765-776.e2. doi:10.1016/j.jaac.2017.06.009
- Kuh D, Ben-Shlomo Y, Lynch J, Hallqvist J, Power C. Life course epidemiology. *J Epidemiol Community Health*. 2003;57(10):778-783. doi:10.1136/jech.57.10.778
- Connelly R, Platt L. Cohort profile: UK Millennium Cohort study (MCS). *Int J Epidemiol*. 2014;43(6):1719-1725. doi:10.1093/ije/dyu001
- Millennium Cohort Team. *Millennium Cohort Study: A Guide to the Datasets*. 8th ed. London, UK: Centre for Longitudinal Studies; 2014.
- Ketende S. *Millennium Cohort Study: Technical Report on Response*. London, UK: Centre for Longitudinal Studies; 2010.
- Cole TJ, Lobstein T. Extended international (IOTF) body mass index cut-offs for thinness, overweight and obesity. *Pediatr Obes*. 2012;7(4):284-294. doi:10.1111/j.2047-6310.2012.00064.x
- Goodman R. The Strengths and Difficulties Questionnaire: a research note. *J Child Psychol Psychiatry*. 1997;38(5):581-586. doi:10.1111/j.1469-7610.1997.tb01545.x
- Angold A, Costello EJ, Messer SC, Pickles A, Winder F, Silver D. The development of a short questionnaire for use in epidemiological studies of depression in children and adolescents. *Int J Methods Psychiatr Res*. 1995;5:237-249.

32. McIntosh S, Steedman H. *Qualifications in the United Kingdom 1985-1999*. London, UK: London School of Economics Research; 1999.
33. Office of National Statistics. SOC2010 volume 3: the National Statistics Socio-economic classification (NS-SEC rebased on SOC2010). <https://www.ons.gov.uk/methodology/classificationsandstandards/standardoccupationalclassification/soc/soc2010/soc2010volume3thenationalstatisticssocioeconomicclassificationnssecrebasedonsoc2010>. Accessed March 13, 2019.
34. Muthén LK, Muthén BO. *Mplus User's Guide*. 7th ed. Los Angeles, CA: Muthén & Muthén; 2012.
35. Patalay P, Fonagy P, Deighton J, Belsky J, Vostanis P, Wolpert M. A general psychopathology factor in early adolescence. *Br J Psychiatry*. 2015; 207(1):15-22. doi:10.1192/bjp.bp.114.149591
36. Ringoot AP, Tiemeier H, Jaddoe VW, et al. Parental depression and child well-being: young children's self-reports helped addressing biases in parent reports. *J Clin Epidemiol*. 2015;68(8):928-938. doi:10.1016/j.jclinepi.2015.03.009
37. Kessler RC, Barker PR, Colpe LJ, et al. Screening for serious mental illness in the general population. *Arch Gen Psychiatry*. 2003;60(2):184-189. doi:10.1001/archpsyc.60.2.184
38. Stunkard AJ, Faith MS, Allison KC. Depression and obesity. *Biol Psychiatry*. 2003;54(3):330-337. doi:10.1016/S0006-3223(03)00608-5
39. Robinson E, Sutin A, Daly M. Perceived weight discrimination mediates the prospective relation between obesity and depressive symptoms in U.S. and U.K. adults. *Health Psychol*. 2017;36(2):112-121. doi:10.1037/hea0000426
40. Jackson SE, Beeken RJ, Wardle J. Obesity, perceived weight discrimination, and psychological well-being in older adults in England. *Obesity (Silver Spring)*. 2015;23(5):1105-1111. doi:10.1002/oby.21052
41. Charsley JS, Collins SC, Hill AJ. The bigger picture: young children's perception of fatness in the context of other physical differences. *Pediatr Obes*. 2018;13(9):558-566. doi:10.1111/ijpo.12280
42. Puhl RM, Latner JD. Stigma, obesity, and the health of the nation's children. *Psychol Bull*. 2007; 133(4):557-580. doi:10.1037/0033-2909.133.4.557
43. Patalay P, Sharpe H, Wolpert M. Internalising symptoms and body dissatisfaction: untangling temporal precedence using cross-lagged models in two cohorts. *J Child Psychol Psychiatry*. 2015;56(11): 1223-1230. doi:10.1111/jcpp.12415
44. Hill AJ, Oliver S, Rogers PJ. Eating in the adult world: the rise of dieting in childhood and adolescence. *Br J Clin Psychol*. 1992;31(Pt 1):95-105. doi:10.1111/j.2044-8260.1992.tb00973.x
45. Polivy J, Herman CP. If at first you don't succeed: false hopes of self-change. *Am Psychol*. 2002;57(9):677-689. doi:10.1037/0003-066X.57.9.677
46. Hemmingsson E. A new model of the role of psychological and emotional distress in promoting obesity: conceptual review with implications for treatment and prevention. *Obes Rev*. 2014;15(9): 769-779. doi:10.1111/obr.12197
47. Tyrrell J, Mulugeta A, Wood AR, et al. Using genetics to understand the causal influence of higher BMI on depression. [published online November 13, 2018]. *Int J Epidemiol*. 2018. doi:10.1093/ije/dyy223
48. Beaulac J, Kristjansson E, Cummins S. A systematic review of food deserts, 1966-2007. *Prev Chronic Dis*. 2009;6(3):A105.
49. Macdonald L, Olsen JR, Shortt NK, Ellaway A. Do 'environmental bads' such as alcohol, fast food, tobacco, and gambling outlets cluster and co-locate in more deprived areas in Glasgow City, Scotland? *Health Place*. 2018;51:224-231. doi:10.1016/j.healthplace.2018.04.008
50. van den Bosch M, Ode Sang Å. Urban natural environments as nature-based solutions for improved public health: a systematic review of reviews. *Environ Res*. 2017;158:373-384. doi:10.1016/j.envres.2017.05.040
51. McGrath LJ, Hopkins WG, Hinckson EA. Associations of objectively measured built-environment attributes with youth moderate-vigorous physical activity: a systematic review and meta-analysis. *Sports Med*. 2015;45(6): 841-865. doi:10.1007/s40279-015-0301-3
52. Patalay P, Fitzsimons E. Development and predictors of mental ill-health and wellbeing from childhood to adolescence. *Soc Psychiatry Psychiatr Epidemiol*. 2018;53(12):1311-1323. doi:10.1007/s00127-018-1604-0