doi:10.1111/ijpo.12457

Remission of obesity among a nationally representative sample of US children

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Received 15 March 2018; revised 25 May 2018; accepted 15 June 2018

Summary

Background: Little is known about the incidence and natural history of obesity remission among children outside of weight loss programmes.

Objectives: The objectives are to characterize and identify socio-demographic and early life predictors of obesity remission between kindergarten and eighth grade among a nationally representative sample of US children.

Methods: The sample included children with obesity [age-specific and gender-specific body mass index percentile (BMI) ≥95] at the spring kindergarten assessment of the Early Childhood Longitudinal Study, Kindergarten Class of 1998–99. Weight categories across 8 years of follow-up were used to identify three transition patterns: persistent obesity remission, non-persistent obesity remission and non-remission. Weight, height and BMI changes between remission categories were examined and predictors of persistent remission were identified.

Results: One-third of children with obesity in kindergarten experienced remission during follow-up and 21.6% of children experienced persistent remission through eighth grade. Female gender and high socio-economic status predicted persistent remission; these associations were attenuated after accounting for baseline BMI. Children experiencing persistent remission gained less weight across waves than those experiencing non-remission.

Conclusions: A meaningful proportion of young children with obesity experience remission by eighth grade. Further study is needed to identify factors that support obesity remission among children outside of treatment contexts.

Keywords: Children, obesity, population-based, remission.

Introduction

Nearly all recent observational studies of growth among US children have focused on quantifying children's gains in body mass index (BMI) or risk of obesity over time (1–4). These efforts have identified that early, rapid growth among children is a risk factor for obesity (5); by kindergarten, a large proportion of young children have obesity, with substantial sociodemographic disparities in obesity prevalence (6–8); and once established, obesity is persistent, with the majority of young children with obesity remaining with obesity into adulthood (1,9–11).

However, several studies have also found that a portion of young children with obesity experience remission from obesity as they age (12–15). For example, in a longitudinal study of early adolescent females with obesity from the south-western USA, 1-year obesity remission rates fluctuated between 8% and 24% over 6 years of follow-up (12). In another study of low-income, minority children from Philadelphia between the ages of 3 and 18, 29% of children with obesity experienced remission after 2 years (13). Young children ages 3 to 5 had the highest rates of remission (46%), followed by adolescents ages 13 to 17 (28%). Lastly, in a nationally representative sample of US children enroled in kindergarten in 1998 and 1999, only 47% of children with a BMI percentile of 95 in kindergarten still had obesity by eighth grade (1). Together, these studies demonstrate that early childhood obesity, while persistent, can also remit over time.

While obesity remission among children has been identified among diverse study populations, our understanding of obesity remission stems primarily from paediatric obesity treatment trials (16). However,

examining predictors of obesity remission among children in treatment does not provide insight into the natural history of paediatric obesity remission in the general population, and therefore does not provide insight into factors that may support remission independent of treatment. For example, it is unclear whether exposures that contribute to obesity remission among children are merely the inverse of exposures that promote incident obesity. Only two studies have examined predictors of remission of overweight and/or obesity among population-based paediatric cohorts. One study found that children who ate breakfast at home more frequently were more likely to experience remission from overweight between the fifth and eighth grades (17). The second study found that children who ate school-provided lunches less frequently and who were viewed as possessing better interpersonal skills by their teachers were more likely to experience remission from obesity between the fifth and eighth grades (18). To our knowledge, no observational studies have examined predictors of remission earlier than the fifth grade.

Important insights can be gained from an in-depth examination of obesity remission among population-based paediatric cohorts. Identifying periods during childhood when obesity remission is most likely can shed light on opportunities during which it may be easier to help children normalize weight gain. Conversely, identifying periods during which remission is uncommon can help pinpoint times when developmentally specific interventions may be necessary. Given the limited reach and uptake of paediatric obesity treatment programmes (19), identifying factors that help children with obesity achieve remission outside of treatment can provide insight into community, family and individual-level exposures that could influence childhood obesity. The objectives of the current study are to examine patterns of obesity remission between kindergarten and eighth grade among a nationally representative cohort of children and identify sociodemographic and early-life predictors of persistent obesity remission.

Methods

Data come from the Early Childhood Longitudinal Study, Kindergarten Class of 1998–99 (ECLS-K) study, which followed children prospectively from kindergarten through eighth grade. ECLS-K was developed by the US Department of Education, National Center for Education Statistics to study children's early school experiences. National Center for Education Statistics used a multi-stage probability sampling strategy, using counties as primary-stage units, schools within primary sampling units as second-stage units, and students within schools as third-stage units, in order to select a nationally representative cohort of US kindergarten students (20). In total, 21 260 kindergarten students were recruited to participate in the study. Seven waves of data collection were performed during fall kindergarten in 1998 (Wave 1), spring kindergarten in 1999 (Wave 2), fall first grade in 1999 (Wave 3), spring first grade in 2000 (Wave 4), spring third grade in 2002 (Wave 5), spring fifth grade in 2004 (Wave 6) and spring eighth grade in 2007 (Wave 7). The study sample was limited to children with obesity, as defined by an age and gender-specific BMI percentile greater than or equal to 95, at Wave 2 (n = 2273). Children with missing height and weight information at Waves 4 through 7 (n = 1434) and biologically implausible values (n = 27) were further excluded, resulting in a final analytic sample of 812 children. Data from the first and third study waves were omitted to maintain intervals of at least 1 year between adjacent waves. For the present study, the baseline wave refers to Wave 2. Children with complete data were more likely to be of higher SES than children with missing data but were otherwise similar across baseline sociodemographic characteristics and BMI percentile.

Measures

BMI, weight status and obesity remission: Height and weight were collected via in-person examinations during each data collection wave. Trained research staff measured height using a Shorr board and weight using a digital scale. Age- and gender-specific BMI percentiles were calculated using the Centers for Disease Control and Prevention 2000 growth charts (21). Exact ages for Waves 5, 6 and 7 were not provided in the public-access data set to protect respondent confidentiality (20). In Wave 5, exact ages were recoded into categories spanning 3 months, while in Waves 6 and 7, exact ages were recoded into categories spanning 6 months. To calculate BMI percentiles in Waves 5, 6 and 7, ages in months were estimated using the midpoint of the categories. In sensitivity analyses, BMI percentiles in Waves 5, 6 and 7 were recalculated using estimated ages at the boundaries of the categories. Biologically implausible values were defined as (1) mean BMI changes of plus or minus three standard deviations; (2) height decrements of greater than one inch; and (3) mean height increases above three standard deviations between waves (22). In total, 27 children with biologically

implausible values were omitted, leaving a final sample of 812 children.

Obesity at each wave was defined as an age-specific and gender-specific BMI percentile ≥95. Children were divided into three remission categories based on changes in weight status categories between study waves. *Persistent obesity remission* was defined as transitioning to a BMI percentile <95 at any point after Wave 2 (kindergarten) and remaining without obesity at all waves thereafter. *Non-persistent obesity remission* was defined as transitioning to a BMI percentile <95 at any wave after Wave 2, but returning to obesity at a subsequent wave. Finally, *Non-remission* was defined as having a BMI percentile ≥95 at every study wave. In sensitivity analyses, *Persistent obesity remission* was redefined to exclude children who first experienced remission at Wave 7 as without future waves, it is unknown whether these children continued to remain without obesity. In separate sensitivity analyses, varying BMI percentile cut-offs were used to define obesity (94 and 96).

Sociodemographic characteristics

Children's gender and race/ethnicity were reported by parents/guardians at Wave 1. Household socioeconomic status (SES) was an ECLS-K-constructed variable that includes information on father/male guardian's education and occupation, mother/female guardian's education and occupation, and household income.

Early childhood characteristics

Potential predictors of persistent obesity remission were selected based on existing literature (23–25). Children's birthweight, maternal age and education at child birth, number of siblings at child birth and family structure at child birth were obtained from parents at Wave 1. Children's birthweight was categorized into quartiles. Family structure at child birth was categorized as single-parent vs. dual-parent families. Number of siblings at child birth was dichotomized as no siblings vs. one or more siblings.

Statistical analysis

Descriptive statistics were used to compare sociodemographic characteristics across remission categories. Two-sample *t*-tests were used to examine differences in changes in BMI percentile, height and weight since the previous wave between children experiencing persistent remission and those experiencing non-remission. Predictors of persistent remission were identified using logistic regression models, both in unadjusted models and in models adjusting for baseline BMI percentile. In all analyses, appropriate cross-sectional or longitudinal survey weights and survey procedures were used to account for the complex study design in order to obtain nationally representative results (20). All statistical analyses were performed with SAS 9.4 (SAS Institute Inc, Cary, NC) using survey procedures. An alpha of 0.05 was used in significance testing, unless otherwise specified.

Results

Table 1 contains the baseline characteristics of the sample by remission status. Among the 812 children who had obesity in kindergarten, 35.6% ($N = 294$) experienced obesity remission (persistent or non-persistent) by eighth grade, with 21.6% ($N = 181$) experiencing persistent remission. The largest proportion of children with persistent remission first experienced remission in eighth grade (11.6% of all children with obesity in kindergarten), followed by 5.1% in first grade, 3.3% in fifth grade and 2.3% in third grade (Fig. S1). Overall, 19.1% of boys and 24.5% of girls experienced persistent remission, while 32.7% of those in the highest SES category and 16.7% in the lowest experienced persistent remission. Among non-Hispanic whites, 27.0% experienced persistent remission compared to 13.4% of non-Hispanic Black and 14.6% of Hispanic children. Mean baseline BMI percentiles were similar across remission categories; by Wave 7, children who experienced persistent remission and those who experienced non-persistent remission had mean BMI percentiles of 83.3 and 94.6, respectively.

Across all study waves, children who experienced persistent remission weighed, on average, significantly less at the wave prior to the wave of remission than children who experienced non-remission. These children also gained less weight between the previous wave and the wave of remission compared to those who experienced non-remission (Table 2). For example, children who first experienced persistent remission at Wave 4 gained, on average, 1.43 kg since the previous wave, compared to 7.00 kg among those who experienced non-remission. In contrast, changes in height between children who experienced persistent remission and those who experienced non-remission were inconsistent. Children who first experienced persistent remission at Wave 4 gained greater height since the previous wave compared to those who experienced non-remission (8.21 cm vs. 7.15 cm, $P < 0.01$). However, in Waves 6 and 7, there were no differences in height changes since the

Table 1 Sociodemographic characteristics of study population by remission category[1]

	N	Persistent remissers	Non-persistent remissers	Non-remissers
Total (*n*, %)	812	181 (21.6)	113 (14.0)	518 (64.4)
Gender (%)				
Male	431	19.1	13.3	67.6
Female	381	24.5	14.9	60.6
Race/Ethnicity (%)				
Non-Hispanic White	431	27.0	12.9	60.1
Non-Hispanic Black	97	13.4	18.0	68.7
Hispanic	185	15.6	15.2	69.2
Other	99	18.5	11.4	70.1
SES (%)				
1	156	16.7	15.1	68.2
2	173	18.1	9.9	72.1
3	166	21.7	15.6	62.7
4	165	23.5	16.6	59.9
5	122	32.7	15.4	52.0
Baseline BMI percentile (mean, SE)	812	97.3 (0.11)	96.8 (0.17)	98.4 (0.10)
Wave 7 BMI percentile (mean, SE)	812	83.3 (2.15)	94.6 (0.77)	98.6 (0.06)

[1]All percentages are weighted.

previous wave ($P > 0.01$). Lastly, children who first experienced persistent remission at younger ages tended to have a lower BMI percentile after remission than children who first experienced remission at older ages. For example, persistent remission at Wave 4 was associated with a BMI percentile of 86.0 while persistent remission at Wave 6 was associated with a BMI percentile of 92.9. Among those who first experienced persistent remission at Wave 4, 9.8% remitted to normal weight compared to 0.11% of children who first experienced persistent remission at Wave 6.

Examining potential predictors of persistent remission, girls were more likely than boys to experience persistent remission in unadjusted models (Table 3). Additionally, children of Hispanic and other race/ethnicity were less likely to experience persistent remission compared to non-Hispanic white children. Older maternal age at birth was associated with higher odds of persistent remission (OR = 1.08, 95% CI: 1.01, 1.15). Finally, children in the second quartile of birthweight, with an average birthweight of 7.40 pounds, were more likely to experience persistent remission than children in the first quartile (OR = 2.71, 95% CI: 1.32, 5.58). However, after adjusting for baseline BMI, gender (OR = 1.66, 95% CI: 0.86, 3.20) and maternal age (OR = 1.06, 95% CI: 0.99, 1.12) were no longer associated with persistent remission. Similarly, after adjusting for baseline BMI, there were no differences by race/ethnicity. However, children with a birthweight in the second quartile remained more likely to experience persistent remission than children in the first quartile (OR = 2.41, 95% CI: 1.02, 5.71). In sensitivity analyses evaluating different BMI percentile cut-offs for obesity, patterns of association did not differ for cut-offs of 94 or 96 vs. 95 (data not shown). Additionally, in sensitivity analyses evaluating different ages for calculating BMI percentiles in Waves 5, 6 and 7, patterns of association did not differ when using the lower or upper bounds compared to the midpoint (data not shown). Finally, in sensitivity analyses excluding children who first experienced obesity remission at Wave 7 from the definition of persistent remission, baseline sociodemographic characteristics were consistent with findings from when these children were included in the definition (Table S1). Using this alternative definition, 10.4% of children experienced persistent remission. In analyses adjusted for baseline BMI, the second quartile of birthweight remained predictive of persistent remission. Additionally, children in some higher SES categories (Quintiles 2 and 4) were more likely to experience persistent remission than children in the lowest SES category (Table S2).

Discussion

In a nationally representative sample of kindergarten children followed through eighth grade, 21.6% of those with obesity in kindergarten became and remained without obesity through eighth grade.

Table 2 BMI percentile, height and weight changes across study waves between children experiencing persistent remission and those experiencing non-remission[b,2]

	Wave 4 (Spring 1st)		Wave 5 (Spring 3rd)		Wave 6 (Spring 5th)		Wave 7 (Spring 8th)	
	Initial remission at Wave 4	Non-remission	Initial remission at Wave 5	Non-remission	Initial remission at Wave 6	Non-remission	Initial remission at Wave 7	Non-remission
N	41	518	19	518	27	518	94	518
Normal weight *N* (%)	9 (9.8)	--	0 (0)	--	1 (0.11)	--	14 (4.8)	--
Mean (SE)								
Weight (kg) at prior wave	**26.6 (0.7)**	**32.0 (0.4)**	**32.5 (0.3)**	**38.9 (0.6)**	**42.8 (0.5)**	**53.9 (0.9)**	**62.6 (1.0)**	**70.8 (1.0)**
Weight (kg) at wave	**28.1 (0.5)**	**38.9 (0.6)**	**39.5 (0.4)**	**53.9 (0.9)**	**50.6 (0.6)**	**70.8 (1.0)**	**69.9 (0.8)**	**97.0 (1.2)**
Average change	**1.43 (0.3)**	**7.00 (0.2)**	**6.99 (0.5)**	**14.9 (0.4)**	**7.76 (0.7)**	**16.9 (0.3)**	**7.31 (0.9)**	**26.3 (0.7)**
Height (cm) at prior wave	**116 (1.8)**	**120 (0.3)**	126 (0.5)	128 (0.4)	**137 (0.9)**	**140 (0.5)**	152 (0.7)	152 (0.6)
Height (cm) at wave	**124 (0.6)**	**128 (0.4)**	**137 (0.6)**	**140 (0.5)**	**148 (0.6)**	**152 (0.6)**	167 (0.8)	167 (0.5)
Average change	**8.21 (1.6)**	**7.15 (0.1)**	**11.4 (0.5)**	**12.7 (0.2)**	11.9 (1.0)	11.6 (0.3)	15.4 (0.6)	15.5 (0.4)
BMI percentile at wave	**86.0 (2.4)**	**98.6 (0.1)**	**92.2 (0.3)**	**98.6 (0.1)**	**92.9 (0.1)**	**98.7 (0.1)**	**89.4 (0.9)**	**98.6 (0.1)**

[b]Initial remission is the wave in which children who experienced persistent remission first remised
[2]Bold type refers to a *p*-value of <0.01 by two-sample *t*-tests comparison between means within wave.

Table 3 Predictors of persistent remission in unadjusted and adjusted models comparing children who experienced persistent remission (n = 181) to those who experienced non-remission (n = 518)[1]

	Unadjusted	Adjusted for baseline BMI
	OR (95% confidence interval)	
Gender		
Male	Ref	Ref
Female	2.01 (1.09, 3.71)	1.66 (0.86, 3.20)
Race/ethnicity		
Non-Hispanic White	Ref	Ref
Non-Hispanic Black	0.54 (0.17, 1.73)	0.73 (0.24, 2.19)
Hispanic	0.50 (0.24, 1.05)	0.59 (0.26, 1.34)
Other	0.47 (0.24, 0.92)	0.46 (0.19, 1.11)
Socio-economic status		
1	Ref	Ref
2	1.56 (0.63, 3.82)	1.63 (0.56, 4.72)
3	0.93 (0.44, 1.98)	0.76 (0.29, 2.01)
4	2.40 (1.02, 5.65)	2.28 (0.87, 6.01)
5	1.85 (0.81, 4.22)	1.32 (0.46, 3.76)
Birthweight		
Quartile 1 (2.13–<6.38 lbs)	Ref	Ref
Quartile 2 (6.38–<7.44 lbs)	2.71 (1.32, 5.58)	2.41 (1.02, 5.71)
Quartile 3 (7.44–<8.50 lbs)	1.26 (0.65, 2.42)	1.01 (0.41, 2.46)
Quartile 4 (8.50–13.8 lbs)	1.65 (0.64, 4.26)	1.85 (0.73, 4.66)
Mother age at birth	1.08 (1.01, 1.15)	1.06 (0.99, 1.12)
Mother education at birth		
1 (Less than high school)	Ref	Ref
2 (High school diploma)	1.90 (0.81, 4.43)	2.08 (0.66, 6.52)
3 (College degree)	1.73 (0.74, 4.04)	1.66 (0.57, 4.84)
4 (Professional degree)	1.97 (0.65, 6.00)	1.78 (0.50, 6.31)
Number of siblings at birth		
1 (0 Siblings)	Ref	Ref
2 (1 or More siblings)	0.94 (0.40, 2.20)	0.75 (0.34, 1.64)
Family structure		
1 (Two parents)	Ref	Ref
2 (One parent)	0.75 (0.35, 1.60)	0.78 (0.31, 1.94)

Consistently as they aged, children who experienced persistent remission gained less weight prior to experiencing remission than those who experienced non-remission. These differences were often substantial with children who did not experience remission gaining two to three times as much weight as children who experienced persistent remission. Persistent remission was not consistently related to height gains, suggesting that children do not 'grow out' of obesity merely by gaining height. Together these findings suggest that weight gain must be limited to promote obesity remission. These data are in contrast with findings from clinical interventions that indicate that weight loss is necessary for children with obesity to achieve non-obesity (26). Additionally, findings suggest that children who experience persistent remission at earlier ages may be more likely to achieve a BMI percentile in the normal weight range compared to children who experience persistent remission later in childhood. These findings extend our understanding of early childhood obesity patterns by suggesting that limiting weight gain early in development may be predictive of healthier long-term weight trajectories for children with early childhood obesity (4).

Not all children are equally likely to achieve persistent remission. Male gender and lower socioeconomic status, which are known predictors of obesity, were differentially associated with remission (27). These differences, however, were explained by girls,

and children of higher SES having lower BMI percentiles at baseline compared to their counterparts. However, even after accounting for BMI percentile at baseline, moderate birthweight was associated with a higher odds of experiencing persistent remission compared to low birthweight. Given that prior studies have found that low birthweight is a risk factor for incident obesity (23,28), these findings suggest that some factors that promote remission among children (e.g. moderate birthweight) may be the inverse of factors that promote incident obesity. Longitudinal data sets with larger sample sizes and more extensive characterization of the childhood environment are necessary to further evaluate this question.

This study has several strengths. This longitudinal, nationally representative cohort included frequent, objective anthropometric measurements over an important developmental period. While our findings highlight the substantial proportion of children with obesity who experienced persistent remission, many children experienced frequent movement in and out of obesity. These findings emphasize the substantial heterogeneity in children's growth and challenge the notion that childhood obesity is intractable once developed. While many children with obesity do consistently experience obesity into adulthood, our findings suggest that there are windows of opportunity when many children's BMI falls into the overweight or normal weight categories. Capitalizing on these periods and supporting continued weight maintenance may help more children experience persistent remission. Finally, in the ECLS-K cohort, obesity remission likely occurred outside of clinical treatment, providing insight into the natural history of early childhood obesity in the USA. Given the low obesity diagnosis rates in ambulatory and well-child visits – 0.78% and 0.93%, respectively (29) – and the small percentage of hospitals in the USA that offer comprehensive weight management services (30), it is unlikely that many children in this cohort were engaged in clinical treatment for obesity.

Findings should be interpreted in light of study limitations. The analytic sample was limited to children with complete data across all study waves. However, the similarity between children with and without complete data, along with the use of appropriate survey weights, provides confidence in the results from our complete-case analytic approach. Still, this restriction limited our sample size, and thus our statistical power. Second, because this data set was designed for education research, we were limited in the potential predictors available to examine. Finally, given the truncated follow-up period, the prevalence of persistent remission may be overestimated since we assumed that children who first experienced remission at Wave 7 remained without obesity in the future. However, few differences were noted in analyses that excluded children who first experienced obesity remission at Wave 7 from our persistent remission definition. Namely, only 10.4% of children in the cohort experienced persistent remission if children who first experienced remission at Wave 7 are not considered to have experienced persistent remission. Longer follow-up studies are needed to determine whether remission up to early adolescence is maintained long-term.

Understanding factors that contribute to children with obesity experiencing obesity remission outside of clinical intervention is an important line of research as currently, paediatric obesity treatment programmes have limited capacity, reach and efficacy (19). In particular, further research is needed to understand if the predictors of obesity remission among children are the inverse of those that contribute to incident obesity; if the predictors are distinct, different interventions may be needed to treat vs. prevent obesity. Additional cohorts with sufficient numbers of children who have experienced obesity remission and extensive characterization of children's social, behavioural, familial and prenatal environments can advance this area of study and provide insight into novel population-based interventions for the treatment of paediatric obesity.

Conflict of interest statement

No conflict of interest was declared.

Acknowledgements

DL conducted analyses and wrote the manuscript. BM provided statistical guidance and edited the manuscript. KWB designed the research project, supervised data analyses and data interpretation and proofread and edited the manuscript. All authors had final approval of the submitted and published versions. None of the authors reported a conflict of interest.

References

1. Cunningham SA, Kramer MR, Narayan KM. Incidence of childhood obesity in the United States. *N Engl J Med* 2014; 370: 403–411.
2. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of obesity and trends in body mass index among US children and adolescents, 1999-2010. *JAMA* 2012; 307: 483–490.
3. Gordon-Larsen P, Adair LS, Nelson MC, Popkin BM. Five-year obesity incidence in the transition period between

adolescence and adulthood: the National Longitudinal Study of Adolescent Health. *Am J Clin Nutr* 2004; 80: 569–575.
4. Ward ZJ, Long MW, Resch SC, Giles CM, Cradock AL, Gortmaker SL. Simulation of growth trajectories of childhood obesity into adulthood. *N Engl J Med* 2017; 377: 2145–2153.
5. Nader PR, O'Brien M, Houts R, *et al*. Identifying risk for obesity in early childhood. *Pediatrics* 2006; 118: e594–e601.
6. Singh GK, Siahpush M, Kogan MD. Rising social inequalities in US childhood obesity, 2003-2007. *Ann Epidemiol* 2010; 20: 40–52.
7. Stamatakis E, Wardle J, Cole TJ. Childhood obesity and overweight prevalence trends in England: evidence for growing socioeconomic disparities. *Int J Obes (Lond)* 2010; 34: 41–47.
8. Taveras EM, Gillman MW, Kleinman KP, Rich-Edwards JW, Rifas-Shiman SL. Reducing racial/ethnic disparities in childhood obesity: the role of early life risk factors. *JAMA Pediatr* 2013; 167: 731–738.
9. Singh AS, Mulder C, Twisk JW, van Mechelen W, Chinapaw MJ. Tracking of childhood overweight into adulthood: a systematic review of the literature. *Obes Rev* 2008; 9: 474–488.
10. Whitaker RC, Wright JA, Pepe MS, Seidel KD, Dietz WH. Predicting obesity in young adulthood from childhood and parental obesity. *N Engl J Med* 1997; 337: 869–873.
11. Guo SS, Wu W, Chumlea WC, Roche AF. Predicting overweight and obesity in adulthood from body mass index values in childhood and adolescence. *Am J Clin Nutr* 2002; 76: 653–658.
12. Huh D, Stice E, Shaw H, Boutelle K. Female overweight and obesity in adolescence: developmental trends and ethnic differences in prevalence, incidence, and remission. *J Youth Adolesc* 2012; 41: 76–85.
13. Robbins JM, Benson BJ, Esangbedo IC, Ward RL, Haden RN. Childhood obesity in an inner-city primary care population: a longitudinal study. *J Natl Med Assoc* 2016; 108: 158–163.
14. Pan L, May AL, Wethington H, Dalenius K, Grummer-Strawn LM. Incidence of obesity among young U.S. children living in low-income families, 2008-2011. *Pediatrics* 2013; 132: 1006–1013.
15. Johannsson E, Arngrimsson SA, Thorsdottir I, Sveinsson T. Tracking of overweight from early childhood to adolescence in cohorts born 1988 and 1994: overweight in a high birth weight population. *Int J Obes (Lond)* 2006; 30: 1265–1271.
16. O'Connor EA, Evans CV, Burda BU, Walsh ES, Eder M, Lozano P. Screening for obesity and intervention for weight management in children and adolescents: evidence report and systematic review for the US Preventive Services Task Force. *JAMA* 2017; 317: 2427–2444.
17. Chang Y, Gable S. Predicting weight status stability and change from fifth grade to eighth grade: the significant role of adolescents' social-emotional well-being. *J Adolesc Health* 2013; 52: 448–455.
18. Chang Y, Halgunseth LC. Early adolescents' psychosocial adjustment and weight status change: the moderating roles of gender, ethnicity, and acculturation. *J Youth Adolesc* 2015; 44: 870–886.
19. Kelleher E, Davoren MP, Harrington JM, Shiely F, Perry IJ, McHugh SM. Barriers and facilitators to initial and continued attendance at community-based lifestyle programmes among families of overweight and obese children: a systematic review. *Obes Rev* 2017; 18: 183–194.
20. Tourangeau K NC, Le T, Sorongon AG, Najarian M. Early Childhood Longitudinal Study, Kindergarten Class of 1998-99 (ECLS-K): combined user's manual for the ECLS-K eighth-grade and K-8 full sample data files and electronic codebooks (NCES 2009-004). National Center for Education Statistics, Institute of Education Sciences 2009.
21. Kuczmarski RJ, Ogden CL, Guo SS, *et al*. 2000 CDC growth charts for the United States: methods and development. *Vital Health Stat 11*; 2002: 1–190.
22. Kim J, Must A, Fitzmaurice GM, *et al*. Incidence and remission rates of overweight among children aged 5 to 13 years in a district-wide school surveillance system. *Am J Public Health* 2005; 95: 1588–1594.
23. Reilly JJ, Armstrong J, Dorosty AR, *et al*. Early life risk factors for obesity in childhood: cohort study. *BMJ* 2005; 330: 1357.
24. Ouyang F, Parker MG, Luo ZC, *et al*. Maternal BMI, gestational diabetes, and weight gain in relation to childhood obesity: The mediation effect of placental weight. *Obesity (Silver Spring)* 2016; 24: 938–946.
25. Li C, Goran MI, Kaur H, Nollen N, Ahluwalia JS. Developmental trajectories of overweight during childhood: role of early life factors. *Obesity (Silver Spring)* 2007; 15: 760–771.
26. Goldschmidt AB, Wilfley DE, Paluch RA, Roemmich JN, Epstein LH. Indicated prevention of adult obesity: how much weight change is necessary for normalization of weight status in children? *JAMA Pediatr* 2013; 167: 21–26.
27. Wang Y, Beydoun MA. The obesity epidemic in the United States—gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. *Epidemiol Rev* 2007; 29: 6–28.
28. Parsons TJ, Power C, Manor O. Fetal and early life growth and body mass index from birth to early adulthood in 1958 British cohort: longitudinal study. *BMJ* 2001; 323: 1331–1335.
29. Cook S, Weitzman M, Auinger P, Barlow SE. Screening and counseling associated with obesity diagnosis in a national survey of ambulatory pediatric visits. *Pediatrics* 2005; 116: 112–116.
30. Wilfley DE, Staiano AE, Altman M, *et al*. Improving access and systems of care for evidence-based childhood obesity treatment: conference key findings and next steps. *Obesity (Silver Spring)* 2017; 25: 16–29.

Supporting information

Additional supporting information may be found online in the Supporting Information section at the end of the article.

Figure S1. Patterns of remission across study waves for (a) persistent remission, (b) non-persistent remission, and (c) non-remission.[1]

Table S1. Characteristics of study population by remission category.[1]

Table S2. Predictors of persistent remission in unadjusted and adjusted models comparing children who experienced persistent remission including in Wave 7 (n = 181) and children who experienced persistent remission excluding in Wave 7 (n = 77) to those who experienced non-remission (n = 518).[1]

Data S1 Supporting information