Journal of Clinical & Translational Endocrinology 2 (2015) 150–156

Contents lists available at ScienceDirect

Journal of Clinical & Translational Endocrinology

journal homepage: www.elsevier.com/locate/jcte

The development of youth-onset severe obesity in urban US girls

Kathleen M. McTigue [a,b,*], Stephanie D. Stepp [c,d], Charity G. Moore [a,e], Elan D. Cohen [a], Alison E. Hipwell [c,d], Rolf Loeber [c,d], Lewis H. Kuller [b]

[a] Department of Medicine, University of Pittsburgh, 230 McKee Place, Suite 600, Pittsburgh, PA 15213, USA
[b] Department of Epidemiology, University of Pittsburgh, 230 McKee Place, Suite 600, Pittsburgh, PA 15213, USA
[c] Department of Psychiatry, University of Pittsburgh, 230 McKee Place, Suite 600, Pittsburgh, PA 15213, USA
[d] Department of Psychology, University of Pittsburgh, 230 McKee Place, Suite 600, Pittsburgh, PA 15213, USA
[e] Department of Biostatistics, University of Pittsburgh, 230 McKee Place, Suite 600, Pittsburgh, PA 15213, USA

ARTICLE INFO

Article history:
Received 6 November 2014
Received in revised form 11 April 2015
Accepted 22 April 2015

Keywords
Obesity
Adolescent
Urban health
Epidemiology
Body weight
Longitudinal studies

ABSTRACT

Objective: To understand the incidence and persistence of severe obesity (≥1.2 × 95th BMI percentile-for-age) in girls across the transition to adolescence, and map developmental trajectories of adolescent severe obesity in a high-risk sample.
Methods: We examined ten years of prospectively collected data from a population sample of urban girls (n = 2226; 53% African American, aged 7–10 in 2003–2004). We determined severe obesity prevalence and incidence by age. Logistic regression evaluated for secular trend in the association between age and severe obesity prevalence. Unconditional latent growth curve models (LGCMs) compared BMI development through the adolescence transition between girls with severe obesity versus healthy BMI.
Results: Severe obesity prevalence was 8.3% at age 7–10 and 10.1% at age 16–19 (white: 5.9%; African American: 13.2%; $p < 0.001$). Age-specific prevalence increased more rapidly among the latest-born, versus earliest-born, girls ($p = 0.034$). Incidence was 1.3% to 2.4% annually. When we compared 12–15 year-old girls with severe obesity versus healthy BMI, average body weight was already distinct 5 years earlier (16.5 kg versus 25.7 kg; $p < 0.001$) and the BMI difference between groups increased annually. LGCMs between ages 7–10 and 11–14 indicated an increase of 3.32 kg/m^2 in the healthy-BMI group and 8.50 kg/m^2 in the severe obesity group, a 2.6-fold difference.
Conclusions: Youth-onset severe obesity warrants particular concern in urban girls due to high prevalence and an increasing secular prevalence trend. Late childhood and early adolescence may represent a key developmental window for prevention and treatment, but is too late to prevent youth-onset severe obesity entirely.

Introduction

Between 1976–1980 and 1999–2004, the prevalence of severe obesity in US youth aged 2–19 years more than tripled [1,2]. Over the same time frame, severe obesity prevalence increased disproportionately among the poor [1,2]. One recent analysis shows that among major racial/ethnic groups, non-Hispanic black girls, aged 12–19 years, have the highest prevalence (12.6%; SE 1.0) [2]. Prevalence estimates for non-Hispanic white girls and Hispanic girls of the same age were 4.8% (SE 0.8) and 6.3% (SE 0.8), respectively [2]. Since poor and minority youth are not only at particular risk for severe obesity, but are also likely to lack resources for treatment, an understanding of the natural history of severe obesity in that group is critical as a first step towards prevention strategies.

As youth-onset severe obesity prevalence has risen, its definition has been in flux. In 2007, an expert committee defined severe childhood obesity as a body mass index (BMI) ≥ 99th percentile-for-age, calculated by extrapolation from CDC growth charts [3]. However, since then, an alternative approach of defining severe obesity (≥120% of the 95th percentile-for-age) has been widely adopted [2,4–7]. It provides a better fit to empirical 99th BMI percentile values than do 99th percentile values extrapolated from the CDC growth chart lambda-mu-sigma parameters. In addition, the definition based on 120% of the 95th percentile-for age avoids the concern that CDC growth-chart values above the 97th

Abbreviations: BMI, body mass index; CFI, comparative fit index; CI, confidence Interval; CVRF, cardiovascular risk factors; EM, expectation maximization algorithm; LGCMs, unconditional latent growth curve models; PGS, Pittsburgh Girls Study; RMSEA, root mean-square error of approximation; SD, standard deviation; TLI, Tucker–Lewis index.

* Corresponding author. Tel.: +1 412 692 2940; fax: +1 412 692 4838.
E-mail address: kmm34@pitt.edu (K.M. McTigue).

http://dx.doi.org/10.1016/j.jcte.2015.04.001

percentile are beyond the range of the data from which growth chart parameters were calculated [8].

National data from 1999 to 2004 show that among youth aged 5–17 years with BMI ≥99th percentile-for-age, 59% exhibited at least 2 cardiovascular risk factors (CVRF) and 11% at least 4 CVRF. For those with BMI in the general obesity range (≥95th percentile-for-age), the corresponding estimates were substantially lower at 39% and 5% [9]. Furthermore, over 30% of US youth with BMI ≥99th percentile-for-age have findings consistent with the metabolic syndrome, versus only 13–17% of youth with a BMI-for-age in the 95–97th percentile ($p \leq 0.04$) [1]. They also show significantly higher systolic and diastolic blood pressure, insulin, and liver function tests, along with lower HDL cholesterol [1]. Youth-onset severe obesity (BMI≥1.2 × 95th percentile-for-age), has likewise been cross-sectionally associated with gallstone disease [6], fatigue and diminished mobility [7]. Furthermore, youth-onset severe obesity may be unlikely to reverse; in one analysis of severely obese children, 100% of individuals with adult follow-up data were found to be obese and 65% to have extreme obesity (BMI ≥ 40 kg/m^2) in adulthood [9]. In another sample, 70.5% of severely obese adolescents remained severely obese in adulthood [10]. This persistence of adolescent severe obesity into adulthood heightens concerns for long-term health implications because adult severe obesity is linked with numerous adverse outcomes, including mortality, coronary heart disease, hypertension, diabetes and poor physical health-related quality of life [11–13].

Common weight management practices for overweight and obesity are less effective for severe obesity, suggesting that prevention is essential [14]. Yet longitudinal data on how severe obesity develops – essential for informing preventive interventions – are lacking. Furthermore, published studies have aggregated data across a wide range of ages, so the timing of youth-onset severe obesity is poorly understood beyond its incidence prior to adulthood. To better understand the development and health implications of youth-onset severe obesity, we examined a population-based sample of urban US girls. We (a) estimated prevalence and incidence rates of severe obesity and determined the persistence of severe obesity in late childhood and early adolescence in a population of particular importance for cardiovascular prevention, due to high lifetime risk for severe obesity and weight-related health complications; (b) examined for a secular trend in youth-onset severe obesity, as an indicator of how rapidly the development of extreme body weight in youth has shifted; (c) compared rates of increase in weight development across early adolescence from ages 7 to 10 among girls who were severely obese, versus those with BMI in the healthy range, at 12–15 years of age; and (d) investigated the role of poverty in accelerated weight development in girls.

Methods

Sample description

The Pittsburgh Girls Study (PGS; n = 2451) comprises urban girls aged 5–8 upon initial assessment (2000–2001) and their primary caretakers [15]. They have been followed annually since enrollment. Low income urban neighborhoods in Pittsburgh were oversampled; neighborhoods in which at least 25% of families were living at or below poverty level were fully enumerated and approximately 50% of households in other neighborhoods in Pittsburgh were randomly selected for enumeration. Overall, 83.7% of girls listed by the US Census were identified. Analyses presented here use 10 annual data waves collected from wave 3 (ages 7–10; the first year with weight measurement) to wave 12 (ages 16–19). We excluded 225 PGS participants: 114 did not complete the PGS wave 3 survey and an additional 111 did not provide wave 3 weight data. Of our sample (n = 2226), on average, 92.6% (range: 88.0% to 97.3%) completed a PGS interview and 86.5% (range: 75.8% to 94.4%) provided data for BMI calculations annually in waves 4–12.

Data collection

Separate in-home interviews for girls and caretakers were conducted annually by trained interviewers. Interviewers also measured girls' height and weight. Families were compensated for their participation. Study procedures were approved by the University of Pittsburgh Institutional Review Board.

Measures

Caregivers' report of several demographic variables were assessed in wave 3: girl's age and race, family poverty (i.e., receipt of public assistance), whether the girl lived in a single-parent household, and caregiver education. Girls' BMI was calculated from annually measured height and weight. Weight data were excluded for any data wave in which a girl was pregnant. National reference data were used to calculate BMI-for-age percentiles and then determine age-and sex-specific weight status: underweight (<5th percentile); healthy weight (5th percentile to <85th percentile); overweight (85th to <95th percentile); obese (≥95th percentile) [16]. We used the ≥120% of the 95th percentile-for-age definition for severe obesity in these analyses, due to measurement advantages described above, widespread adoption [2,4–7], and clinical relevance [17]. Girls who were obese, but not severely obese, were categorized as mild/moderately obese.

Analytic plan

We examined wave 3 (baseline; age 7–10) descriptive statistics for the full sample and for those girls with wave 8 (age 12–15) BMI in the healthy or severely obese ranges (the LGCM subgroup). To capitalize on the richness of the repeated measures data, we used several approaches to examine the development of severe obesity in this cohort. Prevalence and incidence of severe obesity in each of ten annual sample waves (waves 3–12) were calculated for the sample. To examine for a secular trend in weight development, we examined mean BMI and severe obesity prevalence for girls in the oldest and youngest birth cohorts. For this analysis, we fit a quadratic regression to the mean BMI by age with a cohort interaction using random effects modeling, and a logistic regression to severe obesity with a linear trend and a cohort interaction. To examine the persistence of severe obesity in childhood, we identified the girls who were severely obese in wave 3 (age 7–10), and (a) estimated the prevalence of severe obesity in this group annually through wave 12 (age 16–19) then (b) examined the distribution of body weight categories in this group in wave 12.

We examined weight development specifically across the transition to adolescence, comparing girls in wave 8 (12–15 years) who had (a) severe obesity or (b) healthy BMI. We compared demographic variables between the two groups at ages 7–10 and 12–15. We used logistic regression to assess for a cross-sectional relationship between family poverty and severe obesity, modeling the outcome of severe obesity at wave 8 (versus healthy BMI) as a function of race and family poverty in wave 3. Unconditional latent growth curve models (LGCMs) characterized the developmental trajectories of girls' BMI separately for the two weight-defined groups across waves 3–7. These models were estimated using a robust maximum likelihood estimator in Mplus 5.2 [18]. Missing data on dependent variables were handled with the expectation maximization (EM) algorithm. Model fit was evaluated using the χ^2 goodness of fit test, comparative fit index (CFI), Tucker–Lewis index (TLI), and root-mean-square error of approximation (RMSEA). For CFI and TLI, we