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Phenotyping children and adolescents with obesity using behavioral, psychological, and familial data

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Abstract

Objective: This prospective cohort study aimed to empirically derive phenotypes of children and adolescents with overweight and obesity.

Methods: Latent class analyses (LCA) using Mplus were carried out in the Growing Up Today Study. Information on participants' weight status, disordered eating behaviors, body image and weight concerns, depressive symptoms, and pubertal timing as well as and maternal weight status were included in the LCA, which were stratified by sex. Mixed effects regression was used to examine associations of the obesity phenotypes with adult weight gain, between 20 and 35 years, independent of weight at beginning of follow up and duration of follow up.

Results: Among the girls, four obesity phenotypes were identified: 1) early puberty, 2) mothers with obesity, 3) high weight concerns, and 4) mixed. Only three phenotypes were identified among the boys: 1) high weight concerns, 2) mothers with obesity and 3) mixed. Participants who had overweight or obesity in childhood or adolescence gained more weight in young adulthood than their leaner peers, but the patterns of weight gain in young adulthood varied by phenotype of obesity in childhood and adolescence.

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Conclusion: Our results support examining risk factors for and treatment outcomes by obesity phenotypes.

Introduction

Approximately 35% of youth in the US have overweight or obesity,[1,2] and are at risk for developing numerous chronic diseases.[3–5] Although numerous clinical trials and observational studies have identified a range of genes and behavioral and environmental factors predictive of weight change and obesity,[6–8] relatively few obesity prevention interventions have had a significant impact on body mass index (BMI) or weight status. One possible reason is that most studies have classified weight status based only on BMI. This approach assumes that all individuals with high weight are similar, which seems unlikely. Moreover, utilizing one homogenous category (obesity) effectively averages associations across different potential phenotypes of obesity, attenuating the impact of interventions that could be highly effective for one group, but not others. The inconsistent results from treatment efforts across studies may be partially due to differences in distribution of obesity phenotypes across studies.[9]

There is a vital need to move beyond trying to find the optimal prevention strategy for all youth or treatment for all children and adolescents with overweight/obesity because it is highly unlikely one approach will work equally well for everyone.[10] Several small studies have found success using a targeted approach where obesity prevention and treatment strategies have been tailored to subgroups of individuals with distinct psycho-behavioral or genetic characteristics.[11–13] For example, in a trial comparing interpersonal therapy vs. health education for gain weight prevention among high risk adolescent girls, those with high anxiety had greater declines in BMI z-scores at 36 months in the interpersonal psychotherapy group vs. the health education arm of the trial.[13] In addition, Huang et al. also found that a high protein diet was most beneficial to individuals with the *FTO* rs9939609 A allele,[11] thus suggesting that diet recommendation should consider genotype. However, to develop targeted prevention and treatment strategies that could be widely used, we must first identify obesity phenotypes based on combinations of psychological, behavioral, and familial characteristics.

Previous obesity classifications have focused on severity[14] or primarily on the presence or absence of metabolic risk factors. Additionally, they have mostly taken a “variable-centered” approach in which factors associated with overweight and obesity are studied in isolation.[15] Person-centered approaches, such as latent class analysis (LCA), in which participants are grouped into mutually exclusive classes based on their observed response patterns,[16] have been used to identify multiple phenotypes of obesity characterized by appetitive behaviors, eating behaviors, and/or physical activity.[17,18] No large studies, however, have amalgamated those behaviors with psychological factors, physiologic factors, and family history of obesity into a person-centered classification of obesity. Furthermore, additional research is needed on the differential association between obesity phenotypes and risk of future weight gain and health consequences.

The goal of this project was to determine if there were distinct, clinically meaningful obesity phenotypes based on familial characteristics, expressed behaviors, cognitions, and other established risk factors and correlates (e.g., physical activity, dietary behaviors). We subsequently aimed to assess prospective association between the identified phenotypes and weight gain in adulthood.

Methods

Sample

Data were from the Growing Up Today Study (GUTS), an ongoing prospective cohort study in the United States (US), which was established in 1996, when participants were aged 9–14 years. All participants are children of women in the Nurses' Health Study II (NHS II).[19] A total of *9,033* girls and *7,843* boys whose mothers consented agreed to participate. Our sample for the phenotyping analyses was restricted to individuals who were classified as having overweight (n=*4,885*) or obesity (n=*2,509*), based on their self-reported weight and height, in one or more years between ages 9 and 19 years.

Measures

Obesity Phenotype Indicators:

Weight Status: BMI (kg/m^2) was calculated using self-reported weight (lbs) and height (inches), which was converted to kg and meters, respectively. Self-reported weight and height are valid in adolescent and young adult populations, despite some underestimation.[20,21] Participants were classified as having overweight or obesity based on the International Obesity Task Force age and sex-specific BMI cutoffs.[22]

Maternal Weight Status: Adult weight status was assessed from self-reported weight and height measures in NHS II. Those with a BMI ≥ 25 kg/m^2 were categorized as having overweight or obesity. Maternal weight status in childhood was measured by recalled body size at age 10y using a series of 9 silhouettes.[23] Women who recalled a body size larger than figure 3 were classified as having childhood overweight or obesity.

Pubertal Timing: We used age at menarche (girls) and Tanner stage of pubic hair development [24,25] (boys and girls) as indicators of pubertal timing. Female participants were classified as having early pubarche if stage was ≥ 3 at ages 9, 10, or 11. Age at menarche was also categorized into early (1 SD below the sample mean) vs normative or late (within 1 SD from the mean or 1 SD above the mean). Male participants were classified as having early pubarche if stage was ≥ 3 at ages 9, 10, or 11 or ≥ 4 at age 12.

Concerns with weight and shape and social influence on eating: The McKnight Risk Factor Survey (MRFS)[26] assessed attitudes and influences on concerns with weight and shape. Response options for the questions ranged from 1 ("never" or "not at all") to 5 ("always" or "totally"). Participants with a mean of ≥ 4 on the subscales were categorized as having high weight and shape concerns or highly influenced by peers, respectively.

Disordered eating behaviors: The questions on binge eating, self-induced vomiting, and use of laxatives to control weight, assessed frequency of engaging in the behaviors during the past year. The response options for binge eating included “a couple of times”, “less than once a month”, “1–3 times a month”, “once a week”, and “more than once a week”. Whereas the response options for use of self-induced vomiting or laxatives use to control weight ranged from “never” to “every day”. The questions have been validated in the GUTS sample and found to have high sensitivity and predictive value.[27]

Frequency of overeating episodes was assessed using the question *“During the past year, how often have you eaten so much food in a short period of time that you would be embarrassed if others saw you?”.* Those who reported engaging in the behavior were asked if they felt out of control during the episodes. If they reported a loss of control and at least monthly episodes, they were classified as binge eating. To assess frequency of purging, participants were asked how often they made themselves throw up or took laxatives to keep from gaining weight, in the past year. We defined purging as using vomiting or laxatives to keep from gaining weight at least monthly in the past year.

Depressive symptoms: Depressive symptoms were assessed by a five item scale on the MRFS in 1999, 2001, and 2003.[26] In 2007, 2010, 2013, and 2014 the Center for Epidemiologic Studies Depression scale (CESD-10) was used to assess depressive symptoms. Participants in the top 10th percentile of depressive symptoms were considered to have high depressive symptoms, whereas those in the lower 90th percent were classified as having lower depressive symptoms (coded as 0=low depressive symptoms and 1= high depressive symptoms).

Correlates of obesity phenotypes: Physical activity was measured with the Youth Adolescent Activity Questionnaire (YAAQ) in 1996, 1997, 1998, 1999, and 2001 questionnaires.[28] Hours per week of vigorous activity were computed as the sum of average hours per week engaged in the following sports: basketball, dance/aerobics, hockey/lacrosse, running, swimming, skating, soccer, tennis, football (boys only), cheerleading/gymnastics, volleyball, and martial arts. Participants in the top quartile of vigorous activity were classified as highly active (coded as 1=highly active and 0= not highly active).

Dietary intake was assessed with the Youth/Adolescent Questionnaire[29] in 1996–1998. Participants who reported consuming > 1 sugar sweetened beverages (SSB)/week were classified as regular SSB consumers. Fried food away from home (FFA) intake, a proxy measure for fast food, was measured in 1996–1998. Participants who reported consuming FFA > 1 time/week were classified as regular FFA consumers. Frequency of breakfast consumption was assessed in 1996–1998. Participants who reported eating breakfast < 5 days/week were classified as breakfast skippers.

In 1996 and 1997 participants were asked about their frequency of consuming prepared foods. Participants who reported consuming prepared foods > 1 time/week were classified as high consumers.

Outcome: Weight gain in adulthood was modeled as the difference between first and last follow up weight where both were obtained between age 20 and 35 years. Among girls, weights within one year of reporting a pregnancy were set to missing. Adolescents and young adults are consistent in underreporting their weight, thus weight change based on self-report is highly valid.[30]

Statistical Analysis

All analyses were stratified by sex assigned at birth and age groups (children: 9–13 years old and adolescents: 14–19 years old). Analyses examining the associations between the obesity phenotypes and weight change included GUTS participants with and without overweight or obesity (N=16,875).

Latent class analysis (LCA) was used to identify phenotypes of obesity. Through an iterative process, we fit two through six-class models in each age group and biological sex combination. Starting with a 2-class solution, LCA models with increasing numbers of classes were fit. To determine relative model fit and appropriate number of classifications, we used use a series of fit indices: (a) the lowest Bayesian Information Criterion (BIC),[31] (b) the lowest sample-size adjusted BIC (aBIC),[32] (c) a significant Bootstrapped Likelihood Ratio Test (BLRT) [33], and (d) an entropy value indicating good class separation (closer to 1). We decided a priori that each class would need to contain at least 3% of the sample for interpretability and stability.

Following the identification of obesity phenotypes, we assessed associations with a range of behavioral risk and protective factors across phenotypes using the Bolck, Croon, and Hagenaar's (BCH) approach (for continuous variables) [34] and DCAT approach (for categorical variables) [35] within Mplus.

Using the "savedata" option in Mplus, we were able to create a new dataset with membership into the obesity phenotypes as a variable. This new dataset was then merged with the original GUTS dataset in SAS. We then used mixed effects regression models (SAS version 9, proc mixed), to examine prospective associations between the obesity phenotypes with weight change in adulthood among GUTS participants with or without overweight or obesity. We specified a compound symmetry covariance structure to account for non-independence due to clustering within mothers with multiple children participating in GUTS. These models controlled for weight and age of participants at the beginning of follow up and duration of follow up.

Results

Descriptive Characteristics

The final sample included *1,766* girls and *1,867* boys in the 9–13 age group. In the 14–19 age group, the sample consisted of *2,038* girls and *2,177* boys (Table 1).

Obesity Phenotypes

Among girls, we examined 2- to 6-class models. Among girls 9–13 years, although the 5- and 6-class models had higher entropy values (Table 2), a lower BIC, ssBIC, significant

BLRTs, and class sizes (> 3% of the sample) indicated that the 4-class model was the best fitting solution. Compared to the 2- and 3-class model, the 4-class model also provided a better fit based on lower AIC and ssBIC values as well as significant BLRTs. The “early puberty” phenotype (7%) was predominantly defined by high probabilities of early puberty. The “mothers with obesity” phenotype (16%) was characterized by high probabilities of mothers having overweight at age 10 and obesity in adulthood. Participants in the “high weight concern” phenotype (17%) were characterized by high probabilities of concerns with weight and shape and social influence on eating and had higher probabilities of depression than the other phenotypes. Those participants also had the highest probabilities of binge eating and purging. The “mixed” phenotype included the most participants (60%) and had low probabilities of all obesity-related indicators (see supplementary file for probability plots).

Among girls in the 14–19 age group, lower BIC and ssBIC and a significant BLRT provided support for the 4-class model. The four phenotypes identified in this age group aligned with those identified in the younger age group: 1) “early puberty” (11%), 2) “mothers with obesity” (25%), 3) “high weight concerns” (22%), and 4) “mixed” (42%).

Among boys, LCA models with up to 6 classes were run. In the 9–13 age group, although the four-class model had slightly higher entropy, the model with three classes was chosen due to lower BIC, ssBIC, a significant BLRT and more interpretability of the classes (Table 2). Three of the previously identified phenotypes emerged among the boys: 1) “high weight concerns” (9%), 2) “mothers with obesity” (12%), and 3) mixed (79%). In the 14–19 age group, BIC and ssBIC values, along with significant BLRTs indicated that the 3-class model had the best fit to the data. Boys in this age group were similarly distributed across the same three phenotypes identified in the younger age group: 1) “high weight concern” (10%), 2) “mothers with obesity” (12%), and 3) “mixed” (78%).

Correlates of obesity phenotypes

Among both girls and boys with obesity, those in “high weight concerns” phenotype were the most likely to report skipping breakfast (girls: 64% and boys: 55%, $p<0.001$) (data not shown). In both age groups, girls in the “mothers with obesity” phenotype had lower levels of vigorous activity (9–13 years: 5.0 ± 0.2 and 14–19 years: 2.8 ± 0.1, $p<0.001$).

Associations between obesity phenotypes and weight change

Among 9-13-year-old girls, those who were members of the obesity phenotypes all gained more weight than their leaner peers. Independent of age and weight at the beginning of follow-up and duration of follow-up, those in the “mothers with obesity” phenotype had the greatest weight gain in adulthood (β= 6.3 kgs, 95% Confidence Interval (CI) 4.5 – 8.1) followed by the “early puberty” phenotype (β= 3.9 kgs, 95% CI 1.4 – 6.5), the “high weight concerns” (β = 2.5 kgs, 95% CI 0.8 – 4.3), and the “mixed” phenotype (β = 1.4 kgs, 95% CI 0.4 – 2.4), respectively (Table 3). A similar pattern was observed among the 14-19-year-old girls. Compared to their peers without obesity, the “mothers with obesity” phenotype was predictive of the greatest weight gain in adulthood (β = 6.2 kgs, 95% CI 4.2 – 8.2), followed by the early puberty obesity phenotype (β = 5.1 kgs, 95% CI 3.8 – 6.5), the

mixed phenotype (β = 3.0 kgs, 95% CI 1.9 – 4.0), and the high weight concerns phenotype (β= 2.3 kgs, 95% CI 1.1 – 3.6).

Among boys the “mothers with obesity” phenotype was predictive of similar weight gain in adulthood (9–13 years: β= 6.5 kgs, 95% CI 4.1 – 8.9; 14–19 years: β= 4.5 kgs, 95% CI 2.3 – 6.7) as the high weight concerns phenotype (9–13 years: β = 5.2 kgs, 95% CI 2.6 – 7.8; 14–19 years: β = 4.6 kgs, 95% CI 2.4 – 6.8). (Table 3). The “mixed” phenotype was predictive of weight gain but only in the 14–19 age group (β =1.1 kgs, 95% CI 0.1 – 2.0).

Discussion

Although previous studies have used person-centered approaches, including LCA, to derive phenotypes of obesity,[36,37] our study included indicators of familial characteristics, expressed behaviors, cognitions, and family history of obesity in a comprehensive classification of pediatric and adolescent obesity. Moreover, we examine how obesity phenotypes relate to future weight gain. We identified four phenotypes among the girls: 1) *early puberty*, 2) *mothers with obesity*, 3) *high weight concerns*, and 4) *mixed*. Only three of those phenotypes emerged among the boys: 1) *high weight concerns*, 2) *mothers with obesity*, and 3) *mixed,* which may reflect gender differences in body fat changes during puberty. Participants who had overweight or obesity in childhood or adolescence gained more weight in young adulthood than their leaner peers, but the patterns of weight gain in young adulthood varied by phenotypes of obesity identified in the present study.

Previous studies that have investigated obesity phenotypes in youth have not examined whether the associations with health outcomes vary by obesity phenotypes.[38,39] The cross-sectional studies have primarily focused on measures of dietary intake, smoking, alcohol consumption, screen time, and physical activity for identifying obesity phenotypes. [38,40] These studies have found that phenotypes of youth characterized by unhealthy eating behaviors, low activity levels, high screen time, and substance use have a higher prevalence of youth with overweight or obesity.[39,41] Additional studies have included indicators of appetitive traits, such as food and satiety responsiveness,[42,43] loss of control eating, [44,45] and psychological functioning (e.g., depression, anxiety), [46,47] to characterize phenotypes of children and adolescents with higher prevalence of overweight and obesity.

An advantage of our study is that it included a wider range of indicators than previous studies and examined the associations between the identified phenotypes and subsequent weight gain; however, the phenotyping was restricted to youth with overweight or obesity. Therefore, we cannot directly compare our results to previous studies phenotyping youth, which were cross-sectional, did not restrict the phenotyping to youth with overweight or obesity, and included a more limited range of indicators. However, we can place our results in the context of what is known about obesity risk factors and consequences. Our finding that the largest weight gains were among youth with obesity in the “mother with obesity” phenotype supports the previous literature on the intergenerational transmission of obesity.[48,49] The increased weight gain in this phenotype could be due to genetic predispositions, epigenetic factors, or learned behaviors. Some of the associations between the “mother with obesity” phenotype and greater weight gain in young adulthood may be

due to the role of family environments. [48,50] For example, the lower level of physical activity in this subgroup could be due to learned behaviors or patterns in the family.

Puberty is characterized by marked changes in body composition, with considerable sex differences in adiposity and fat free mass.[51] During puberty, girls experience significantly greater increases in fat mass thanboys.[52] Conversely, while gain in fat-free mass begins to stabilize around age 12 in boys begin to gain fat-free mass at an increased rate during this time.[52,53] Children with obesity experience accelerated growth[54] and particularly in girls, earlier puberty. [55,56] A meta-analysis and systematic review showed that girls with overweight and obesity are at increased risk of early puberty (4.67-fold and 2.22-fold, respectively), compared to those with normal weight.[57] This relationship was not statistically significant among boys. This may, in part, explain why the early puberty phenotype only emerged among the girls. Additionally, congruent with our findings, it has previously been shown that both childhood obesity and early puberty are independently predictive of obesity in adulthood,[58,59] again with stronger associations in girls than boys.[60,61] Previous studies have observed that eating behavior phenotypes characterized by disordered eating (e.g., binge eating, emotional eating) and dysregulated appetitive traits (e.g., food responsiveness, enjoyment of food) are associated with higher weight status in youth.[36,43] Among children and adolescents, disordered eating behaviors are associated with high levels of body dissatisfaction [62] and depressive symptoms.[63,64] Our results indicate that there is a phenotype of children and adolescents with obesity that is characterized by the co-occurrence of disordered eating (i.e., binge eating and purging), concerns with body weight and shape, and depression and that this group experiences greater weight gain in adulthood compared to their leaner peers and some other types of obesity. In line with restraint theory,[65] association between this phenotype and future weight gain could be, in part, due to dysregulated appetite and subsequent dietary compensation and overeating, which could be at least partially driven by genetics.

There are some limitations to this study. First, a considerable proportion of the participants were classified as having the mixed obesity phenotype, which could indicate that there are other phenotypes of obesity that we were unable to identify because defining variables were not measured in GUTS (e.g., anxiety, drive for thinness). Nevertheless, our results suggest that, compared with those without overweight or obesity, the “mixed” phenotype is predictive of greater weight gain in adulthood, highlighting its clinical relevance. Second, BMI and weight change were calculated using self-reported height and weight. BMI based on self-reported measures does result in some underestimation, [20,21] but people are consistent in their underreporting of weight. Therefore, weight change based on self-reports has little underestimation (Field et al. 2007). Third, it is likely that there are other factors related to obesity that were not measured in GUTS and thus not included in the analysis. Lastly, more than 95% of participants were white and all participants being children of nurses. Thus, our results may not be generalizable to racially/ethnically or socio-economically diverse populations. Our large, community-based sample, however, represents individuals across all regions in the US.

Conclusion

In summary, our study highlights the utility of person-centered approaches to classify obesity in children and adolescents. Using a wide range of psychological, behavioral, familial, and physiological factors, we identified distinct obesity phenotypes that were differentially associated with weight gain in adulthood. Thus, our results suggest that these phenotypes were clinically meaningful and may confer disparate levels of risk. Future studies should replicate these results in more diverse samples and assess whether the inclusion of additional risk factors for obesity as indicators will lead to the emergence of additional clinically meaningful phenotypes. Moreover, these data provide a foundation to further explore whether treatment tailored to obesity phenotypes results in greater weight loss and weight loss maintenance.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Information regarding the Growing Up Today Study and questionnaires can be found at https://gutsweb.org/.

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Study Importance

What is already known about this subject?

- Relatively few obesity prevention interventions or treatment trials have had a large impact on body mass index (BMI) or weight change, which partially could be due to using only BMI-defined cut-points to define weight status and therefore assuming youth with obesity are a homogenous group.
- Previous studies that have investigated obesity phenotypes in youth have not examined whether health outcomes vary by obesity phenotypes.

What are the new findings in your manuscript?

- In both girls and boys, we identified multiple phenotypes of obesity. Among the girls we found four phenotypes - 1) *early puberty*, 2) *mothers with obesity*, 3) *high weight concerns*, and 4) *mixed*. Among the boys we found three phenotypes: 1) *high weight concerns*, 2) *mothers with obesity*, and 3) *mixed.*
- Weight gain in young adulthood varied by phenotype of obesity, even after controlling for weight at the beginning of follow-up.

How might your results change the direction of research or the focus of clinical practice?

- Our results highlight that the field should move away from examining predictors and consequences of obesity to looking at associations with phenotypes of obesity.
- Clinicians should consider whether treatment tailored to obesity phenotypes might result in greater weight loss and weight loss maintenance.

Table 1.

Obesity indicators, and risk and protective factors among girls and boys by age group

	Girls		Boys	
	Age 9–13y (n= 1766) n (%) or mean (sd)	**Age 14–19y (n=2038) n (%) or mean (sd)**	**Age 9–13y (n= 1867) n (%) or mean (sd)**	**Age 14–19y (n=2177) n (%) or mean (sd)**
Weight status (0 vs 1)				
Overweight	1,360 (77.0%)	1,435 (70.4%)	1403 (75.1%)	1500 (68.9%)
Obesity	406 (23.0%)	603 (29.6%)	464 (24.9%)	677 (31.1%)
Maternal weight status				
Overweight at 10	308 (17.4%)	343 (16.8%)	318 (17.0%)	350 (16.1%)
Obesity in adulthood	538 (30.5%)	664 (32.6%)	528 (28.3%)	571 (26.2%)
Binge eating (0 vs 1)	104 (5.9%)	260 (12.8%)	51 (2.7%)	47 (2.2%)
High social eating influence (0 vs 1)	272 (15.4%)	247 (12.1%)	161 (8.6%)	69 (3.2%)
High concerns with weight and shape (0 vs 1)	581 (32.9%)	986 (48.4%)	349 (18.7%)	262 (12.2%)
High depression (0 vs 1)	45 (2.6%)	341 (16.7%)	49 (2.6%)	265 (12.2%)
Pubertal Development (0 vs 1)				
Early	634 (35.9%)	523 (25.7%)	640 (34.3%)	575 (26.4%)
Age at menarche	11.8 (1.1)	11.9 (1.1)	--	--
Vigorous activity (hrs/wk)	6.2 (4.3)	5.4 (4.7)	8.2 (5.0)	7.7 (5.2)
Breakfast skipping (5–7/days/wk vs < 5 days/wk)	759 (43.0%)	496 (24.3%)	645 (34.6)	369 (17.0)
Sugar-sweetened beverages	1.0 (0.9)	1.0 (1.1)	1.2 (1.1)	1.3 (1.2)

Table 2.

Fit indices for latent class solutions among girls and boys in both age group

Girls										
	9–13y age group (n= 1,766)					**14–19y age group (n=2,038)**				
	2 class	3 class	4 class *	5 class	6 class	2 class	3 class	4 class *	5 class	6 class
AIC	13166.1	13071.2	13017.4	13011.1	13015.3	19041.6	18915.1	18837. 6	18818.3	18804.8
BIC	13281.1	13246.4	13252.9	13306.8	13371.2	19159.6	19094.9	19079.2	19121.8	19170.1
ssBIC	13214.4	13144.8	13116.3	13135.2	13164.8	19092.9	18993.3	18942.6	18950.2	18963.6
Entropy	0.67	0.69	0.63	0.70	0.70	0.59	0.66	0.63	0.59	0.63
VLMR-LRT	p<0.001	p <0.01	p<0.01	p=0.18	p=0.13	p<0.001	p<0.001	p=0.25	p=0.07	p=0.08
BLRT	p<0.001	p<0.001	p<0.001	p< 0.05	p=1.00	p<0.001	p<0.001	p<0.001	p<0.001	P< 0.001
Class Size										
1	360 (20%)	1269 (72%)	122 (7%)	11 (0.6%)	11 (0.6%)	585 (29%)	591(29%)	442 (11%)	129 (6%)	107 (5%)
2	1406 (80%)	138 (8%)	290 (16%)	121 (7%)	18 (1%)	1453 (71%)	974 (48%)	513 (25%)	287 (14%)	224 (11%)
3		359 (20%)	303 (17%)	244 (14%)	330 (19%)		473 (23%)	442 (22%)	464 (23%)	398 (20%)
4			1051 (60%)	346 (20%)	561 (32%)			858 (42%)	405 (20%)	264 (13%)
5				1044 (59%)	713 (40%)				753 (37%)	819 (40%)
6					133 (8%)					226 (11%)
Boys										
	9–13y age group (n= 1,867)					**14–19y age group (n=2,177)**				
	2 class	3 class *	4 class	5 class	6 class	2 class	3 class *	4 class	5 class	6 class
AIC	11859.4	11788.3	11781.3	11786.3	11795.7	13098.7	13016.0	13007.1	13005.1	13009.4
BIC	11964.5	11948.8	11997.0	12057.4	12122.1	13206.7	13180.9	13228.8	13283.7	13344.9
ssBIC	11904.2	11856.6	11873.1	11901.7	11934.6	13146.4	13088.8	13104.9	13128.0	13157.4
Entropy	0.83	0.70	0.75	0.55	0.59	0.79	0.65	0.54	0.60	0.63
VLMR-LRT	p<0.001	p =0.001	P=0.47	p=0.49	p=0.24	p<0.001	p =0.01	P=0.19	p=0.27	p=0.57
BLRT	p<0.001	p<0.001	P=0.07	p= 1.0	p=1.0	p<0.001	p<0.001	P<0.000	p= 0.67	p=0.33
Class Size										

1	182 (10%)	1471 (79%)	113 (6%)	94 (5%)	10 (0.5%)	217 (10%)	1707 (78%)	143 (7%)	35 (1.6%)	81 (4%)
2	1685 (90%)	221 (12%)	207 (11%)	63 (3%)	72 (4%)	1960 (90%)	270 (12%)	983 (45%)	110 (5%)	304 (14%)
3		175 (9%)	1483 (79%)	1281 (69%)	199 (11%)		200 (10%)	261 (12%)	1346 (62%)	1394 (64%)
4			64 (3%)	238 (13%)	226 (12%)			790 (36%)	266 (12%)	35 (2%)
5				191 (10%)	86 (5%)				420 (19%)	287 (13%)
6					1274 (68%)					76 (3%)

*: denotes the chosen LCA solution

Table 3.

Prospective association between obesity phenotypes and weight change in adulthood (20–35 years)*

	Girls		Boys	
Age group	**9–13y**	**14–19y**	**9–13y**	**14–19y**
	Weight change	**Weight change**	**Weight change**	**Weight change**
	Beta Est. (95% CI)	**Beta Est. (95% CI)**	**Beta Est. (95% CI)**	**Beta Est. (95% CI)**
Early puberty	3.9 (1.4 – 6.5)	5.1 (3.8 – 6.5)	---	---
Mothers with obesity	6.3 (4.5 – 8.1)	6.2 (4.2 – 8.2)	6.5 (4.1 – 8.9)	4.5 (2.3 – 6.7)
High Weight Concerns	2.5 (0.8 – 4.3)	2.3 (1.1 – 3.6)	5.2 (2.6 – 7.8)	4.6 (2.4 – 6.8)
Mixed	1.4 (0.4 – 2.4)	3.0 (1.9 – 4.0)	0.9 (−0.2 – 1.9)	1.1 (0.1 – 2.0)

*From mixed effects models accounting for repeated measures and clustering within families and controlling for age and weight at the beginning of follow-up and duration of follow-up