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Title

Longitudinal modelling of Body Mass Index from birth to 14 years.

Running Title

Longitudinal modelling of BMI: birth to 14 years.

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Keywords: adiposity rebound, adolescent, Body Mass Index, children, Linear Mixed Models, longitudinal survey, obesity, Raine Study.

SUMMARY

Objective: To examine the tracking of Body Mass Index (BMI), from birth to age 14 years.

Method: Linear mixed model analysis was used to model the trajectories of BMI in a sample of 1403. Adiposity rebound was investigated for a subset of 173 individuals.

Results: Adolescents who are overweight or obese at 14 years followed a different BMI trajectory from birth compared to those of normal weight. There was a difference between weight status groups for the timing of adiposity rebound ($p<.001$) and BMI at nadir ($p<.001$). The linear mixed model depicted a significant difference in rate of change of BMI over time for males and females ($p<0.001$) with female BMI increasing at a faster rate than males, and for weight status groups ($p<.005$), with the obese cohort having the faster increase in BMI over time. BMI at birth was significantly lower for the normal weight cohort compared to the overweight ($p=.029$), and obese ($p=.019$) cohorts.

Conclusion: The longitudinal modelling of BMI show that weight status at 14 years is the result of a distinct path in earlier years. Compared to their normal weight peers, overweight and obese adolescents experience an earlier adiposity rebound, with a higher BMI at rebound.

Introduction

In Australia, unhealthy weight status has overtaken smoking as the major cause of preventable disease [1], supporting a similar trend in the United States [2]. Childhood obesity is associated with an increase in adverse health consequences in adulthood, with typically 'adult' health concerns now being diagnosed more often in children [3].

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Infant birth weight is seen as a marker of intra-uterine health and well-being with influence on the child's developmental outcome. Controversy remains as to the role of infant birth weight on obesity when older. Some studies have reported a relationship between high birth weight and later obesity [4-6], whereas low birth weight and rapid weight gain in infancy and childhood have also been shown [7, 8], while others state that regardless of birth weight, rapid weight gain in the first six months is associated with obesity [9, 10].

Adiposity Rebound

Adiposity rebound is thought to be an important marker for identifying the development of later obesity [11-14]. Adiposity rebound refers to the second rise in BMI curve that usually occurs between the ages of 5-7 years, or more specifically the upward trend in BMI after its nadir [12-17]. This adiposity rebound has been argued to reflect upward BMI centile crossing, which at any age can predict later obesity [18]. Infancy sees the greatest height and weight growth rate, reducing to a relatively constant growth rate during early to middle childhood [10, 19]. Some argue that the timing of adiposity rebound in early childhood can accurately predict up to 30% of later obesity [12-15, 20]. Rolland-Cachera and colleagues [12, 15] found the occurrence of adiposity rebound at 3 years corresponded to obese individuals, while adiposity rebound at 6 years corresponded to normal weight individuals.

In the last decade, biological and environmental factors that influence the timing of adiposity rebound have been investigated [13, 17]. While parental obesity was strongly associated with earlier adiposity rebound [13, 17, 20], dietary variables such as high protein intake were not [13]. It may be that early adiposity rebound is the result of some factors yet to be identified [16, 21, 22], which may program later weight status [16, 23, 24].

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The purpose of this study was to model BMI from birth, using a linear mixed model, for children using the IOTF cut-off categories of normal weight, overweight, or obese at 14 years, with particular interest in the adiposity rebound for each weight status group, and gender differences. Multilevel models are becoming popular in health and social sciences for the analysis of longitudinal data and provide an opportunity to investigate within-person and between-person change over time. The multilevel model for change is used to separate differences between individuals at baseline and changes over time within individuals. The model is a mathematical representation of population behaviour [25, 26], which enables correct modelling of correlated errors for repeated, continuous and correlated observations [25].

Methods

Sample

The Western Australian Pregnancy Cohort (Raine) Study enrolled mothers of 2,979 children *in utero* from antenatal clinics at King Edward Memorial Hospital for Women (KEMH), Perth's primary specialist obstetric health care facility, and has followed 2,868 live birth children. Women were enrolled into the project over 30 months from May 1989 to November 1991. Enrolment criteria included gestational age of 16-20 weeks, basic proficiency in English for informed consent, expectation of delivery at KEMH, and intention to remain in Western Australia. All mothers gave written informed consent and the study was approved by the institutional ethics committees. The protocol for the original study has been previously reported describing the antenatal [27] and postnatal periods [28, 29].

The study has followed 2868 live born children who have been assessed at survey wave's birth, 1, 2, 3, 6, 8, 10 and 14 years, with consent at each follow-up. The Raine Study families are broadly representative of the WA population: 10.7% of parents being never married (vs. 9.8%),

7.5% children were born <37 weeks (vs. 6.9%), with a slight overrepresentation children born <2500g, 8.6 (vs. 6.5%). The Raine cohort is well established and there is frequent contact between enrolled families and study organisers [30]. There has been attrition over time in sample sizes from each survey wave and among variables. Overall retention rates are high for each survey wave (92% at 1-year, 74% at 2-years, 85% at 6-years, 82% at 8- and 10-years, and 79% at 14-years), with enthusiasm amongst participants to provide high quality information. In survey wave two funding limitations restricted the number of individuals assessed across physical measures, which impacted upon the sample size for BMI.

In this study, multiple birth, congenital abnormality and preterm birth (gestational age <37 weeks) cases were excluded from the sample. Only participants with a BMI at the survey year 14 were included, resulting in a total of 1403 participants being available for analysis from birth to 14 years, 674 (48%) females and 729 (52%) males. ANOVA tests were used to compare the weight status of individuals in years 2, 3, 6, 8 and 10 selected for this study, to those excluded, with no statistical difference at each survey year in proportions of normal, overweight and obese. In survey year two funding limitations restricted the collection of physical measures with only 400 individuals with valid BMI data. There was a slight selection bias in this sub-sample, tending to have a higher proportion of professional fathers, high income families and older mothers, but similar to the retention trends seen across the survey waves in the Raine Study. There were no statistical differences in gestational age, gender or family structure. At 14 years, 73% (1031) of adolescents were classified normal weight (501 female, 530 male), 19% (263) overweight (127 female, 136 male), and 8% (109) obese (46 female, 63 male). BMI cut-off points of 25 and 30, age and gender adjusted for children, were used to classify children as normal weight, overweight or obese as defined by the IOTF criteria [31] at survey year 14. Gender separated mean age, height, weight, BMI and sample size at each survey wave is detailed in Table 1.

Anthropometric Measures

Anthropometric measures were taken by a small group of extensively trained staff of the Telethon Institute of Child Health Research using strict protocols, with intra and inter-rater reliability established at the beginning of each follow-up. The neonatal examination was conducted between 24 and 72 hours following birth. Length was measured by two people using the Harpenden Neonatometer to the nearest 0.1cm. Newborns and infants (survey wave year one) were laid in supine position, with their head held by one person against a curved head plate in mid-line. The other person stretched the legs straight, knees held together, ankle flexed at rights angles to the lower leg, moving the mobile plate to rest against the baby's feet. Weight was measured to the nearest 100g, using calibrated hospital scales at birth and a Wedderburn digital chair scales in survey wave year one.

In survey waves year two and on, where appropriate, children were standing in the anatomical position, palms facing forward. Each area was measured at least twice in sequence with measures within one centimeter. Unless stated, measures were taken at expiration. Height was measured using a Holtain stadiometer with shoes off, and heels, bottom and head against the board. The chin was positioned to straighten the neck and the measure taken with a breath intake. Weight was measured with children wearing light clothing (running shorts and singlet top), to the nearest 100g, using a Wedderburn digital chair scales.

In each survey wave year BMI was calculated from measured height and weight using the formula weight (kg)/height $(m)^2$, as defined by the IOTF criteria [31] at survey year 14. Adiposity rebound in this study was determined as the last minimum (nadir) BMI before the continuous increase in BMI over time [11], in a subset of individuals with BMI at every survey wave (n=173).

Statistical Processes

The statistical software SPSS version 15 and 17 (SPSS Inc., Chicago, IL) was used for all statistical processes. Gender difference for mean height, weight and BMI across survey waves was analyzed using independent t-tests. Mean height, weight, BMI, adiposity rebound nadir for normal, overweight and obese weight status were compared across survey waves using ANOVA and post hoc tests. These statistically processes used the raw BMI data.

A linear mixed model was performed to model the trajectory of BMI over time from birth to 14 years. Fixed and random effects, interactions and covariance structure were all investigated in the determination of the final model, with model diagnostics performed. Time was used as a repeated measure. A non-linear transformation was applied to 'age' to determine the best model fit (based on Akaike's Information Criterion (AIC)). Polynomial fits were investigated to the fifth order, but the quadratic (age squared), compared to higher form transformations, provided the best model fit (AIC). The linear term in the model did not account for variation in the early years, nor describe the functional form, particularly the initial peak around age one year, or the nadir. Consequently a natural log function of age was added into the model. This final model included age, age squared and the natural log of age and provided the best model fit (AIC) and model diagnostic results. Effects of age, gender, gestational age and weight status (determined at age 14) were investigated. BMI was treated as the dependent variable; with gender, age, gestational age and weight status treated as factors (fixed effects).

Results

There were no gender differences in age at assessment, with survey year eight having the largest age range (Table 1). There were significant gender differences in height and weight for every survey wave ($p<0.05$), with males taller and heavier than females, except for weight in

year 8, and height and weight in year 10, where males and females were similar. Gender differences for BMI were significant for years 1, 2, 3, and 14 only ($p<0.05$), with males higher in BMI for years 1, 2, and 3, while females higher than males at year 14. The BMI model (Figure 1) depicts this cross over point at around 8 years of age.

BMI data were grouped according to gender and IOTF BMI weight status groups at year 14. Using the weight status groupings at year 14, BMI group means were calculated at each survey wave (birth, 1,2,3,6,8,10, and 14) for males and females, with results presented in Table 2. The mean BMI trajectories from birth to 14 years, for each weight category are plotted in Figure 2. Evaluation of the height and weight data according to weight status and gender found that these differences were mostly attributable to changes in weight rather than height over time (Figure 3). This was confirmed by the ANOVA tests. Post hoc results were the same for BMI and weight, but not for height.

Adiposity rebound

BMI at nadir and age at nadir were calculated for a subset of individuals with actual BMI at every survey wave (n=173) and is detailed in Table 3. There were significant weight status group differences for BMI at Nadir and Age at Nadir ($p<0.001$), but no significant gender differences. In the sub-sample, adiposity rebound occurred for the normal weight group at 5.3 years, for the overweight group at 3.8 years, and for the obese group at 2.6 years. Post hoc analysis of weight status groups found a significant difference for females between BMI and age at Nadir of the normal weight compared to overweight group, and normal weight compared to obese groups. For males, there was only a significant difference in BMI at nadir between the normal weight and overweight and obese groups. There were no statistically significant differences between the overweight and obese groups. Adiposity rebound results were similar for the subsample (n=173) and full sample (n=1403) and is shown pictorially for

the full sample in Figure 2. The BMI trajectory paths for each weight status are distinct in their different profiles, particularly in the timing of the peaks and troughs (Figure 2) and is based on the mean BMI for each weight status group, and mean age at each survey wave.

Apart from birth year, there were significant between group differences in BMI means for males and females in all survey waves ($p<0.001$) . Post hoc analysis found a significant difference for males and females between the normal weight and overweight group, between the normal weight and obese groups from survey year 1 onwards, and between overweight and obese groups for males from survey year 3 onwards, and for females from survey year 6 onwards.

BMI Longitudinal Modelling

The final linear mixed model (unstructured covariance) found that there were no significant gender-weight category; age/age^2/Logage-gestational age; gender-gestational age; or weight category-gestational age interactions, and these were removed from the final model.

The final model can be described by the following equation:

*BMI (predicted) = intercept + weight status + gender + age(actual age) + age(actual age)^2 + logAge (actual age +1) + gestational age (actual gestational age) + [age(actual age)*gender] + [age(actual age)^2 x gender] + [logAge (actual age +1) x gender] +[age(actual age) x weight status] + [age(actual age)^2 x weight status] + [logAge (actual age +1) x weight status].*

The model estimates and statistic results are depicted in Table 4. By interchanging estimates into the equation, predicted BMI can be determined for any individual dependent upon their gender, weight status, age and gestational age.

Even with preterm children (<37 weeks) removed from the sample, there was a significant gestational age effect ($p<0.001$), with every additional week in gestation resulting in an increase in BMI at birth. At birth (model intercept) there was a significant difference between BMI of normal weight children compared to overweight ($p=0.029$) and obese ($p=0.019$), while the difference between overweight and obese was not significant. For example, for a female child born at 40 weeks gestation, their BMI at birth would be, if normal weight at age 14 14.2 kg/m^2 compared to 14.4 kg/m^2 if overweight at age 14, or 14.5 kg/m^2 if obese at age 14. There was no significant gender effect between male and female BMI at birth. However, females increased their BMI at a faster rate than males ($p<0.001$), which accounts for the overlap seen in the trajectory at about eight years. The increase in BMI over time was statistically different for each weight status group, with the obese cohort having the largest rate of increase ($p<0.001$) as shown in Figure 1. The modelled BMI trajectories, with an overlay of mean age-adjusted BMI at each survey wave is depicted in Figure 1 indicating a relative good fit, supported by the residual diagnostics which followed an approximate normal distribution, although there was a slight deviation at the positive tail.

Discussion

The BMI trajectories followed a distinct pathway from birth to 14 years for individuals within different weight categories. As suggested by others [12, 16], the timing of the adiposity rebound can be seen as a marker for later obesity. Our data shows more clearly than Rolland-Cachera's figures [11, 12], the distinct and significantly different pathways followed by the three weight status groups for both the raw means and modeled data. There are statistically different pathways, in particular between the normal weight group compared to the overweight and obese groups. A major limitation is the absence of data at years four and five, the smaller sample at year two, and this was reflected in the lack of statistical difference

between the overweight and obese weight status groups only, although pictorially a difference is shown. We also found a statistical difference in BMI at Nadir, with the normal weight group having a lower BMI at rebound compared to the overweight and obese groups.

The limitation in the number of data points up to age six and in particular between age three and six years may play a significant part in the adiposity rebound age and BMI nadirs reported. Although the ages for the obese and normal weight groups appear clear, those for the overweight group are not. The STRIP study reported by Lagstrom and colleagues (2008), collected annual measurements until age 13, and reported adiposity rebound at 4.3years for overweight boys and 3.8 years for overweight girls. In reviewing our BMI trajectory plots (Figure 2), it is likely that there may have been a lower BMI point somewhere between assessments at three and six years of age. Irrespective, it is clear that the overweight adiposity rebound occurs at some point at, or after, three years and before six years in this cohort. This is well before that of the normal group, and those originally reported by Rolland-Cachera and colleagues [11], yet similar to more recent studies [12, 32].

Our finding on adiposity rebound supports others who found that the earlier the occurrence of the adiposity rebound the higher the BMI with age [12, 15, 32]. However, unlike Rolland-Cachera and colleagues [15] we have pre-classified our children into weight status at age 14 and then calculated their average adiposity rebound in the earlier years. This difference in methodology may account for our finding that the higher the BMI at year one, the earlier the age of rebound. Cole [18] argues that the timing of adiposity rebound is reflective of children with high centile BMI, or upward centile crossing. Like others, we believe the occurrence of the earlier adiposity rebound is a marker for early determinants or mechanisms of obesity [16, 18, 21, 22]. These include environmental, behavioural and individual factors (such as breastfeeding, diet, physical activity and genetics), that may program later weight status [16,

23, 24]. To date however the value of identifying the timing of adiposity rebound for use in a clinical setting is yet to be confirmed [14].

Although our findings on adiposity rebound are similar to others [12, 32], the meaning of the distinct peaks and troughs in BMI in relation to child behaviour is as yet unclear. Analysis of the height and weight data found that the changes observed in BMI were principally related to changes in weight and not height (Figure 3). This can be accounted for by height and weight growth rates that occur during childhood [10]. While height trajectories were very similar for all three weight categories, the overweight and obese groups were gaining weight at a faster rate than their normal weight counterparts. This was most pronounced by three years of age, although statistically significant from age one. Adiposity rebound for the obese group occurred at around two years, the same as the beginning of the second critical period for adiposity proposed by Botton and colleagues [10].

The linear mixed model analysis found that the trajectories of BMI were different between males and females, and different for each weight category (Figure 1). This difference was in both the rate of change over time (acceleration) and their BMI at birth. The interaction effects showed that within the weight categories the pathways for males and females were significantly different, with females slightly leaner than males when young, with a cross over in later childhood / early adolescence. The obese group increased their BMI over time at a faster rate, and this difference began from birth. Our results include more frequent collection points and have a larger sample, and support results reported by Blair and colleagues [4] for their New Zealand cohort. Like them, we believe the preschool years are the critical time period for the development of obesogenic behaviours.

The overlay of mean age-adjusted BMI at each survey wave on the modeled BMI trajectories provided an overview of goodness of fit of our proposed model (Figure 1), with residual

diagnostics supporting this. The tight clustering of mean data points in the first few years again highlights the need for greater in-depth analysis of this important period with more frequent data collection points, perhaps at three-monthly intervals.

It is therefore apparent that in this cohort, the increased rate in weight gain accounts for later adiposity, however the underlying causes of the accelerated weight gain are unclear. An examination of growth patterns may assist in the understanding of the development of obesity [33]. Parents often observe children undergoing increased hunger and appetite, weight gain, followed by decreased appetite and height growth, or episodes of spurt and lag [34]. One explanation may be that established food behaviours do not follow the pendulum return of decreased appetite during growth for the overweight and obese groups. Physical and social aspects of the home environment may also influence food choices based on types of food available, parent food behaviours and feeding practices [35]. There may also be a developmental switch triggered by the environment, that is present or absent in the overweight and obese [4, 36]. In addition, the level of physical activity and sedentary past times could be important. Dubois and Girard [21] concluded that influences of behavioural and social factors were critical to the possible onset of obesity in the early pre-school years. Regardless of cause, rapid weight gain after age one is associated with adverse health effects [36], and this critical period of development requires intensive investigation in regards to obesity.

Strengths and limitations

The sample described was not drawn randomly, but enrolled *in utero* from the major Women's Hospital in Perth, Western Australia, and therefore the findings may not be truly representative, with a slight selection bias in this study's sample. There was an expected

attrition rate, with variation across survey waves and among measured variables, although overall participant numbers remained high.

Specific to this study, data collection at survey waves is in years, with the time point an average age at that assessment. In some survey years there was a large range of ages for collection of data. This is particularly significant in respect to the mean BMI trajectories presented, and the calculation of adiposity rebound, although the clear distinction in time points still provide a valid assessment of the difference between the weight status categories. The Raine Study did not collect data in years four and five and these are important years for adiposity rebound, although this seemed relevant to the overweight and obese group distinctions only. Notably, we believe our data underestimate the occurrence of adiposity rebound because of this limitation. All other results presented have been calculated using actual age of the child. Lastly, accurate puberty data for this study was not available across gender; therefore no control for maturity was able to be made in the later survey waves.

The strength of this study is the unique mixed modelling used which accounts for correlated errors normally associated with repeated, continuous and correlated observations. The mixed model permits the evaluation of age as a covariate, rather than predetermined averaged time points (survey waves), increasing the validity of the model. The large sample, even with attrition, provides for more accurate distinctions to be made among gender and weight categories. The longitudinal nature of the cohort, from birth to year 14, provides an opportunity to examine early pathways of weight status, in particular a re-examination of the adiposity rebound. This modeling approach provides an opportunity to test mechanisms that might drive accelerated and early increases in BMI.

Conclusion

Birth to six years is a key developmental period for adiposity. For children who are obese at 14 years the critical period was earlier, between birth and 2 years, than normal and overweight children. The adiposity rebound occurred earlier among the obese cohort. These findings indicate that obesity determinants may be individual, behavioural or environmental, but most likely multi-factorial, probably gender specific, with each factor contributing its own level of risk to the individual. This modeling approach provides a mechanism to further investigate the contribution of possible determinants to adiposity during this period.

To conclude, this study supports the importance of the early years (birth to age six) in the development of adolescent obesity. Further research may provide a clearer insight into the interplay of individual, behavioural and environmental factors which lead to a healthy or unhealthy weight in later life.

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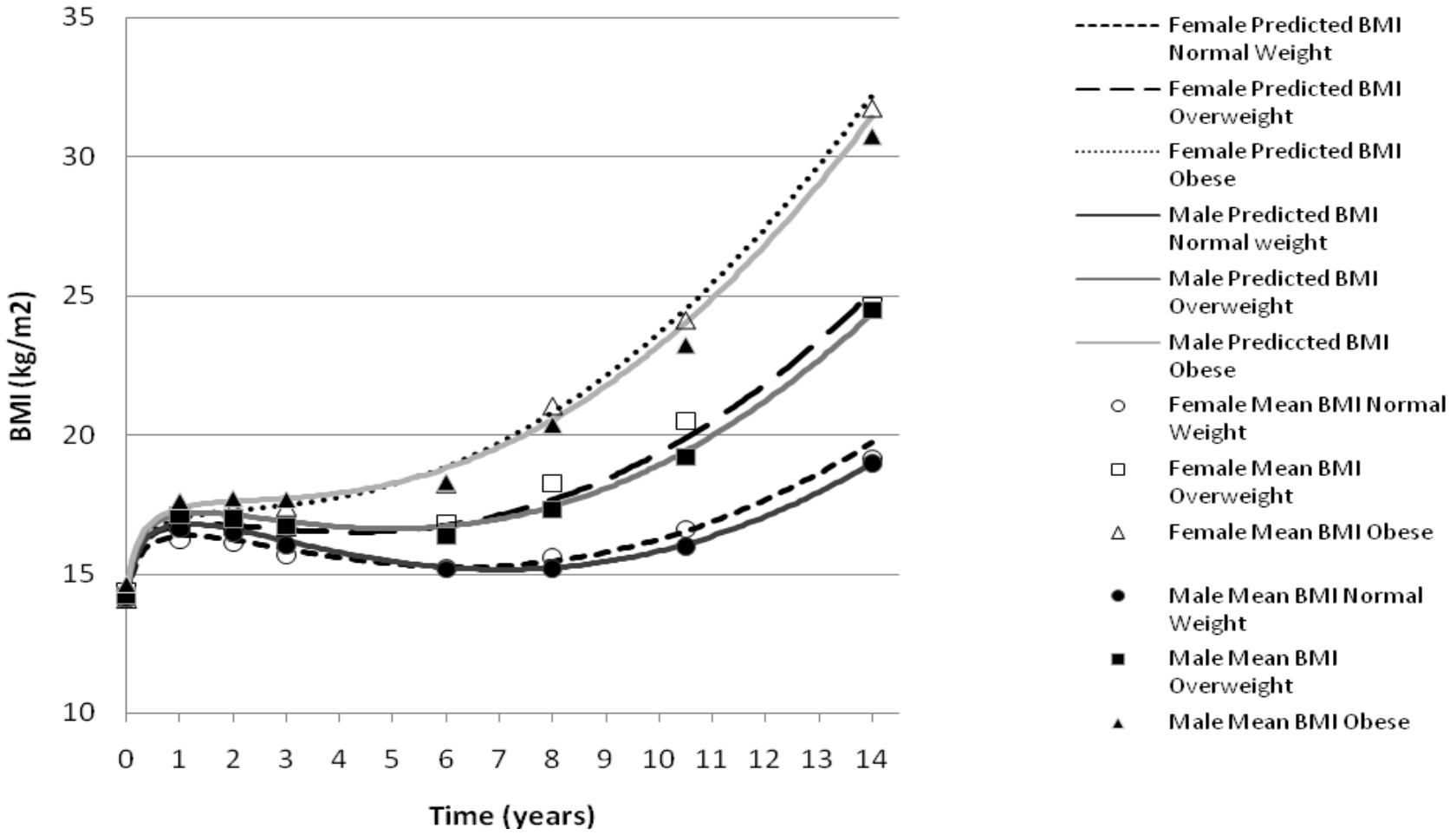

Figure 1. Predicted BMI trajectories from birth to 14 years, separated by weight categories normal weight, overweight and obese (determined at 14 years using IOTF cut-offs) and gender. Mean age-adjusted BMI calculated for each survey wave is overlayed for each weight category and gender to demonstrate goodness of fit to the predicted BMI trajectory model.

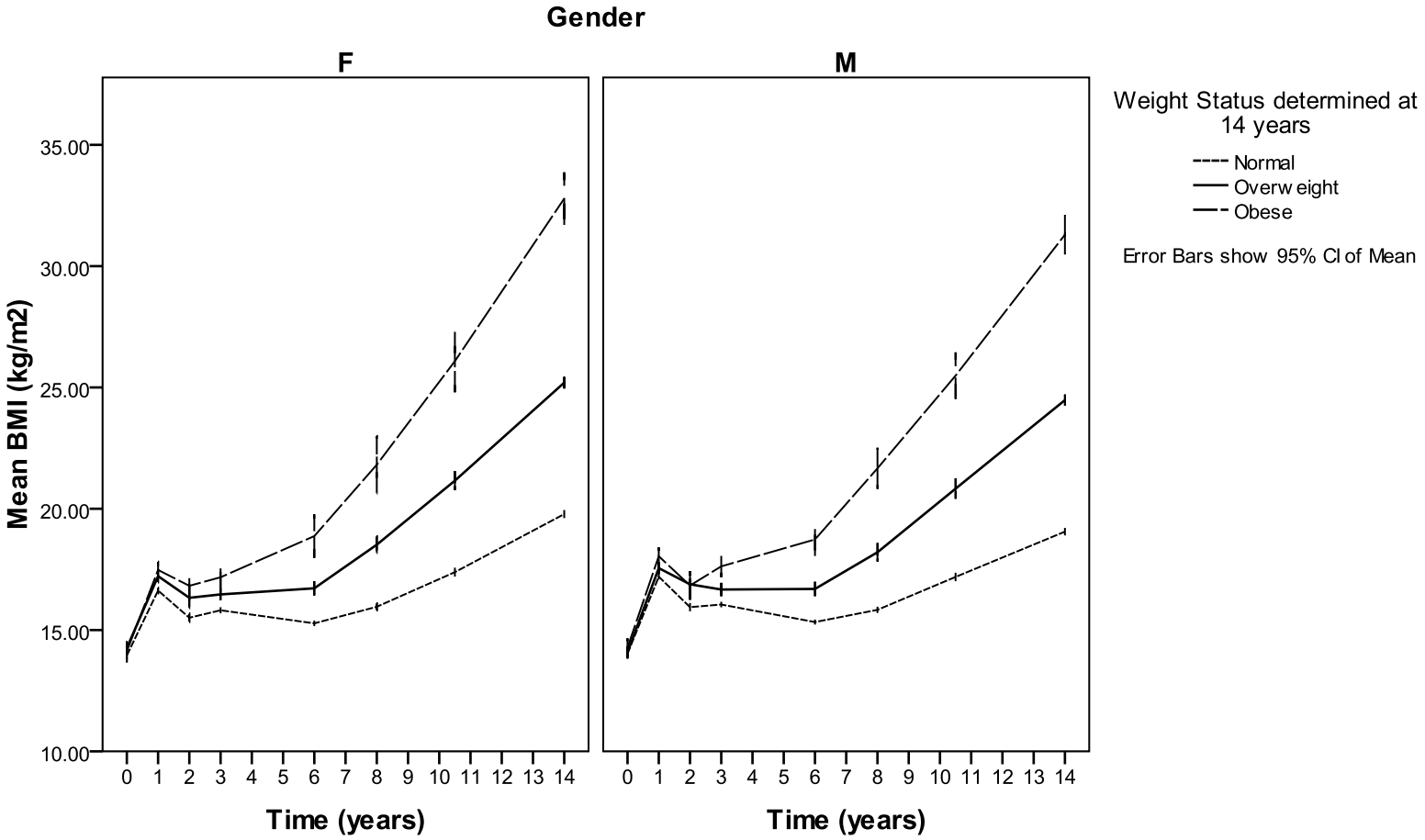

Figure 2. Mean BMI over time based on BMI IOTF weight category as determined at survey year 14 (n=1403). Adiposity rebound in this study was determined as the minimum BMI value.

Table 1. Cohort characteristics of the whole sample, males and female at each survey wave (birth, 1,2,3,6,8,10 and 14) for age, height, weight, BMI.

Variable	Total		Male		Female		Gender Difference	
	N	Mean(SD)	N	Mean(SD)	N	Mean(SD)	t	P
BIRTH								
Gestational age (weeks)	1403	39.31(1.30)	729	39.34(1.31)	674	39.28(1.28)	0.80	0.421
Weight (kg)	1402	3.44(0.47)	728	3.50(0.47)	674	3.37(0.46)	5.26	<0.001
Height (cm)	1388	49.4(2.1)	722	49.8(2.1)	666	49.0(2.0)	7.64	<0.001
BMI (kg/m^2)	1388	14.0(1.4)	722	14.1(1.3)	666	14.0(1.4)	0.48	0.631
SURVEY YEAR 1								
Age (mths)	1317	13.3(1.2)	679	13.3(1.2)	638	13.3(1.2)	-0.82	0.412
Weight (kg)	1312	10.3(1.2)	677	10.7(1.2)	635	10.0(1.1)	10.7	<0.001
Height (cm)	1305	77.6(3.1)	673	78.3(3.1)	632	76.9(2.9)	8.28	<0.001
BMI (kg/m^2)	1305	17.1(1.4)	673	17.3(1.4)	632	16.8(1.4)	7.23	<0.001
SURVEY YEAR 2								
Age (mths)	449	25.6(1.7)	238	25.7(1.6)	211	25.5(1.8)	1.14	0.255
Weight (kg)	438	13.0(1.5)	234	13.3(1.5)	204	12.6(1.4)	4.97	<0.001

Height (cm)	401	90.1(3.7)	208	90.6(3.6)	193	89.5(3.7)	2.97	0.003
BMI (kg/m^2)	400	16.0(1.3)	207	16.2(1.3)	193	15.7(1.21)	3.82	<0.001
SURVEY YEAR 3								
Age (mths)	981	36.9(1.5)	505	36.9(1.6)	476	36.8(1.5)	0.73	0.467
Weight (kg)	969	15.1(1.8)	498	15.4(1.8)	471	14.7(1.7)	6.27	<0.001
Height (cm)	964	96.4(3.8)	494	97.0(3.9)	470	95.6(3.5)	5.91	<0.001
BMI (kg/m^2)	956	16.2(1.3)	488	16.3(1.3)	468	16.0(1.3)	3.39	0.001
SURVEY YEAR 6								
Age (mths)	1327	70.5(2.3)	690	70.6(2.4)	637	70.5(2.2)	0.33	0.741
Weight (kg)	1261	21.4(3.3)	656	21.7(3.4)	605	21.1(3.2)	3.24	0.001
Height (cm)	1260	116.0(4.8)	657	116.6(5.1)	603	115.3(4.5)	4.66	<0.001
BMI (kg/m^2)	1259	15.8(1.8)	656	15.9(1.8)	603	15.8(1.8)	1.02	0.308
SURVEY YEAR 8								
Age (mths)	1272	96.7(4.1)	651	96.8(4.1)	621	96.6(4.1)	0.98	0.327
Weight (kg)	1271	28.2(5.6)	652	28.4(5.7)	619	27.9(5.5)	1.67	0.095
Height (cm)	1272	129.1(6.0)	652	129.8(6.1)	620	128.4(5.7)	4.10	<0.001
BMI (kg/m^2)	1271	16.8(2.5)	652	16.8(2.5)	619	16.8(2.5)	-0.34	0.738
SURVEY YEAR 10								

Age (mths)	1247	126.6(2.2)	651	126.6(2.3)	596	126.5(2.0)	1.10	0.270
Weight (kg)	1246	38.7(8.8)	650	38.6(9.0)	596	38.8(8.6)	-0.48	0.633
Height (cm)	1247	143.7(6.6)	651	143.7(6.7)	596	143.7(6.4)	-0.12	0.904
BMI (kg/m^2)	1329	18.1(3.8)	692	18.0(3.9)	637	18.1(3.8)	-0.37	0.708
SURVEY YEAR 14								
Age (mths)	1403	168.3(2.3)	729	168.2(2.4)	674	168.3(2.3)	-0.54	0.592
Weight (kg)	1403	58.0(13.3)	729	58.8(14.3)	674	57.1(12.1)	2.40	0.017
Height (cm)	1403	164.3(8.1)	729	166.3(8.9)	674	162.1(6.3)	10.23	<0.001
BMI (kg/m^2)	1403	21.4(4.2)	729	21.1(4.2)	674	21.7(4.2)	-2.51	0.012

Table 3. BMI and Age at Nadir for a subset of individuals (n=173) with BMI scores at every survey wave, years birth to age 14 years.

		Male		Female		Total	
	BMI Weight Status determined at year 14	**N**	**Mean (SD)**	**N**	**Mean (SD)**	**N**	**Mean (SD)**
BMI Nadir*	Normal Weight	71	15.1(0.9) §	61	14.9(1.0)§	132	15.0(1.0)§
(kg/m2)	Overweight	19	16.7(1.3)	13	16.1(1.2)†	32	16.4(1.3)†
	Obese	5	16.3(1.2)	4	16.6(0.3)	9	16.4(0.9)
Age Nadir*	Normal Weight	71	5.4(2.0)	61	5.1(2.3)§	132	5.3(2.2)§
(years)	Overweight	19	4.2(2.4)	13	3.2(1.9)†	32	3.8(2.2)†
	Obese	5	3.1(1.7)	4	2.0(0.7)	9	2.6(1.4)

* Significant difference in Anova between groups test $p<0.005$.

§ Significant difference between normal weight and overweight groups $p<0.05$.

† Significant difference between normal weight and obese weight groups $p<0.005$.

Table 2. BMI summary statistics for each survey year based on BMI IOTF weight categories determined for each individual in follow-up year 14, separated for males and females.

		Male		Female	
Survey Wave	**BMI Weight Status determined at year 14**	**N**	**Mean (SD)**	**N**	**Mean (SD)**
Birth	Normal Weight	526	14.0(1.3)	494	14.0(1.4)
	Overweight	133	14.1(1.5)	127	14.3(1.3)
	Obese	63	14.3(1.5)	45	14.1(1.6)
Year 1	Normal Weight	490	17.2(1.3)	472	16.6(1.3)
	Overweight	124	17.6(1.4)	117	17.2(1.4)
	Obese	59	18.0(1.4)	43	17.5(1.1)
Year 2	Normal Weight	153	15.9(1.1)	151	15.5(1.1)
	Overweight	40	16.9(1.8)	29	16.3(1.2)
	Obese	14	16.8(1.1)	13	16.8(1.0)
Year 3	Normal Weight	357	16.1(1.1)	347	15.8(1.2)
	Overweight	88	16.7(1.3)	93	16.5(1.2)
	Obese	43	17.6(1.5)	28	17.2(1.8)
Year 6	Normal Weight	476	15.3(1.2)	449	15.3(1.2)
	Overweight	122	16.7(1.7)	114	16.7(1.6)
	Obese	58	18.7(2.6)	40	18.9(2.9)
Year 8	Normal Weight	475	15.8(1.4)	459	15.9(1.6)

	Overweight	119	18.2(2.1)	119	18.5(1.9)
	Obese	58	21.7(3.3)	41	21.8(3.9)
Year 10	Normal Weight	506	16.8(2.5)	473	16.9(2.6)
	Overweight	127	19.9(3.8)	121	20.3(3.6)
	Obese	59	24.7(5.0)	43	25.0(5.8)
Year 14	Normal Weight	530	19.1(1.8)	501	19.8(1.9)
	Overweight	136	24.5(1.4)	127	25.2(1.4)
	Obese	63	31.3(3.3)	46	32.8(3.7)

Table 4. Final BMI Linear Mixed Model: Estimates of Fixed Effects for parameters.

Parameter	BMI Estimate	Standard Error	Significance
Intercept	8.334684	.762569	<.001
Weight status - Obese group	.314924	.134407	.019
Weight status - Overweight group	.201214	.092172	.029
Weight status - Normal weight group	0	0	.
Gender - female	-.038381	.071336	.591
Gender - male	0	0	.
Age	-.121952	.002623	<.001
Age^2	.000614	.000011	<.001
LogAge	1.546032	.033056	<.001
Gestational Age	.146582	.019332	<.001
Age*gender - female interaction	.015887	.003451	<.001
Age*gender - male interaction	0	0	.
Age^2*gender - female interaction	-.000027	.000015	.076
Age^2*gender - male interaction	0	0	.
LogAge*gender - female interaction	-.217211	.043482	<.001
LogAge*gender - male interaction	0	0	.
Age*Weight status - Obese interaction	.028256	.006479	<.001
Age*Weight status - Overweight interaction	.005560	.004472	.214
Age*Weight status – Normal weight interaction	0	0	.
Age^2*Weight status - Obese interaction	.000269	.000028	<.001

Age^2*Weight status - Overweight interaction	.000144	.000019	<.001
Age^2*Weight status – Normal weight interaction	0	0	.
LogAge*Weight status - Obese interaction	-.036766	.081960	.654
LogAge*Weight status - Overweight interaction	.030509	.056258	.588
LogAge*Weight status – Normal weight interaction	0	0	.