Published by Oxford University Press on behalf of the International Epidemiological Association
International Journal of Epidemiology 2009;**38**:499–509

doi:10.1093/ije/dyn214

SOCIAL EPIDEMIOLOGY

Social disparities in BMI trajectories across adulthood by gender, race/ethnicity and lifetime socio-economic position: 1986–2004

Philippa Clarke,* Patrick M O'Malley, Lloyd D Johnston and John E Schulenberg

Accepted 2 September 2008

Background The prevalence of obesity and overweight is rapidly increasing in industrialized countries, with long-term health and social consequences. There is also a strong social patterning of obesity and overweight, with a higher prevalence among women, racial/ethnic minorities and those from a lower socio-economic position (SEP). Most of the existing work in this area, however, is based on cross-sectional data or single cohort studies. No national studies to date have examined how social disparities in obesity and overweight differ by age and historical period using longitudinal data with repeated measures.

Methods We used panel data from the nationally representative Monitoring the Future Study (1986–2004) to examine social disparities in trajectories of body mass index (BMI) over adulthood (age 18–45). Self-reported height and weight were collected in this annual US survey of high-school seniors, followed biennially since 1976. Using growth curve models, we analysed BMI trajectories over adulthood by gender, race/ethnicity and lifetime SEP (measured by parents' education and respondent's education).

Results BMI trajectories exhibit a curvilinear rate of change from age 18 to 45, but there was a strong period effect, such that weight gain was more rapid for more recent cohorts. As a result, successive cohorts become overweight (BMI > 25) at increasingly earlier points in the life course. BMI scores were also consistently higher for women, racial/ethnic minority groups and those from a lower SEP. However, BMI scores for socially advantaged groups in recent cohorts were actually higher than those for their socially disadvantaged counterparts who were born 10 years earlier.

Conclusions Results highlight the importance of social status and socio-economic resources for maintaining optimal weight. Yet, even those in advantaged social positions have experienced an increase in BMI in recent years.

Keywords Obesity, growth curve models, age, cohort, socio-economic status, gender, race/ethnicity

Institute for Social Research, University of Michigan, Ann Arbor, MI, USA.

* Corresponding author. Institute for Social Research, University of Michigan, 426 Thompson Street, Ann Arbor, MI 48106-1248, USA. E-mail: pjclarke@umich.edu

Introduction

The prevalence of overweight and obesity is rapidly increasing in industrialized countries,[1–8] with extensive health, economic and social implications.[9–17] In the United States, currently two-thirds of adults are overweight or obese.[1,2,18,19] Increases in prevalence have taken place primarily since 1980 and have occurred in all age groups simultaneously,[1,3,6,18,20] prompting the use of the term 'epidemic' to describe these trends.

There is also repeated evidence of social disparities in the prevalence of obesity and overweight. Data from national surveys paint a consistent picture where women, individuals of lower socio-economic position (SEP) and minority racial/ethnic groups have the highest rates of obesity and overweight.[3,6,18,21–23] This is consistent with the argument that social disadvantage is a 'fundamental cause'[24,25] of obesity and overweight. People with more knowledge, money, power, prestige and beneficial social connections are better able to control weight gain, either through the ability to make healthy food choices[26,27] (by having greater awareness of, access to, and resources to purchase healthy foods), or through greater opportunities for exercise, and safe play.[28,29]

However, much of the literature on social disparities in obesity is based on data from repeated cross-sectional surveys[1,4,6] or from single cohort studies.[3,30,31] As a result, it is difficult to determine whether patterns of weight gain over the life course represent true age effects or simply age differences in the characteristics of cross-sectional samples over time. Given the rapid secular changes that have been occurring in obesity prevalence, social disparities in trajectories of weight gain are likely to vary across historical time. The timing of birth exposes each generation or cohort to a particular window in historical time with its own unique set of social and environmental constraints and opportunities.[32–34] Social patterns of weight gain are therefore inextricably linked to the social and cultural conditions of the larger society in which they are embedded across time. Longitudinal data with repeated measures are needed to identify how social disparities in obesity and overweight differ by age and historical period.

We use nationally representative panel data from the Monitoring the Future Study (MTF) in the United States to examine social disparities in adult trajectories of body mass index (BMI) from 1986 to 2004. The MTF study is ideally suited to examine the recent upsurge in obesity and overweight among Americans since the early 1980s. With 19 years of data on annual cohorts followed prospectively over adulthood (age 18–45), it is possible to simultaneously study individual weight change as well as period shifts in BMI over time. We hypothesize that BMI scores are positively associated with both age and historical time, such that BMI trajectories will become increasingly steep in more-recent time periods. We also hypothesize that BMI is inversely related to social and economic advantage, such that, within each cohort, adult BMI trajectories will be steeper for socially disadvantaged subgroups (i.e. women, racial/ethnic minorities and individuals of lower SEP).

Methods

Data

Data are drawn from the MTF project, a nation-wide school-based survey conducted annually in the United States since 1976.[35] The study design (described in more detail elsewhere)[36] involves a nationally representative sample of high-school seniors (age 18) surveyed in the spring of each year (~15 000 per year). For the panel component, a 16% representative subsample (~2400 students per year) was randomly selected from each cohort for six biennial follow-ups (to the age of 30), and then at 5-year intervals to the age of 45, using self-completed mailed questionnaires. All procedures are reviewed and approved on an annual basis by the University of Michigan's Institutional Review Board (IRB) for compliance with federal guidelines for the treatment of human subjects.

To ease respondent burden in the MTF panel study, multiple forms are used, with some questions appearing on only some of the forms. Since 1986 a random one-sixth of respondents received forms asking about height and weight. Thus, the final sample available for this analysis consists of 10 956 individuals who were age 18–45 between 1986 and 2004. (Sample sizes for each high-school cohort average 345 persons with up to nine observations per person.)

Since subjects are selected in their senior year, the survey does not include high-school dropouts.[35] Drug users were oversampled for participation in the panel survey by a ratio of 3:1 (and then re-weighted for analyses to reflect population estimates, as discussed below). Student response rates in their senior year average 82.7%. Retention rates in the panel respondents are highest in the first follow-up after high school (averaging 70% of the original cohort), and fall to an average of 64% in the biennial follow-ups through to age 32. Longer-term retention rates average 50% at age 35, 57% at age 40 and 60% at age 45.[35] Using logistic regression analysis (with backward elimination) we modelled the probability of participation at each wave according to a broad array of baseline socio-demographic characteristics that could potentially influence study retention. Five variables consistently predicted participation at each wave: female, White, coming from a two-parent family, enrolment in an academic high school and not living in the southern region of the US at baseline. By deriving the predicted probability of participation from these five variables, each observation at each wave of data collection was weighted to account for attrition (losses to follow-up as well as survey non-response) based on the inverse probability of participation. Using this weight in our models resulted in only trivial differences in the coefficients in our results, ruling out the possibility that losses due to attrition substantially bias our results.

Measures

We capture weight gain over adulthood with the BMI ($BMI = kg/m^2$). Height was self-reported in inches (converted to metres for these analyses), and weight in pounds (converted to kilograms). Item non-response for height and weight was small in this panel survey, ranging from <1% (0.7%) to 2.3% over the follow-up period. A BMI of 25 or higher is used to define 'overweight'.[37] BMI scores were excluded for pregnant women.

We focus on three key social indicators: (i) gender; (ii) race/ethnicity; and (iii) lifetime SEP. Female is a dummy variable coded 1 for females and 0 for males. Race/ethnicity is represented by four dummy variables: White, Black, Hispanic and other racial/ethnic group (includes Asians and Native Americans). Consistent with a life-course perspective we include SEP both in childhood and in adulthood. Childhood SEP is the maximum of the respondent's parents' education (less than high school, high-school diploma, college degree or higher). Adult SEP is a time-varying measure of completed education at each follow-up (high-school diploma, college degree or higher). [More sophisticated coding (differentiating associate's degree, bachelor's degree or graduate degree) did not yield differences in intercepts or slopes in the growth model]. In order to capture increases in socio-economic resources afforded by marriage, we take the maximum of the respondent's and spouse's education for those who were married at each follow-up. Although SEP can also be measured by income and occupation, social disparities in obesity have typically been observed by educational levels and less consistently by income.[4] As a result, we focus on educational attainment over the life course as our measure of SEP.

Statistical analyses

Growth curve models[38] were used to examine trajectories of BMI over adulthood. Growth curve models belong to a general class of mixed models that take into consideration the clustering of observations within persons and also have the capacity to handle unbalanced designs (inconsistent number of observations per person).[39] A two-level linear model was analysed, with multiple observations nested within persons over time (adulthood). Age was used as the indicator of time. In order to facilitate parameter interpretation, we centred age at the initial point of data collection (age 18).

The structure of this model can be expressed by equations at two levels. At level 1 (within-person model) BMI scores at time t are nested within individuals (i):

$$\mathrm{BMI}_{ti} = \pi_{0i} + \pi_{1i}(\mathrm{age} - 18)_{ti} + r_{ti} \quad (1)$$

where π_{0i} is the expected BMI score for person i at age 18 (since age is centred), π_{1i} captures the rate of change in BMI over the life course, and r_{ti} is the within-person residual (that part of an individual's BMI at time t not predicted by age).

To incorporate period effects we also include the year of data collection as a time-varying variable, again centred on the initial year that BMI data were collected (1986). This allows us to examine period shifts in BMI across all ages. (Cohort effects integrate age and period effects by capturing variations in BMI across groups of individuals in the same high-school year as they age. We do not focus on cohort effects in this paper. Rather, we examine period differences in age patterns of BMI over adulthood.) We examine how adult trajectory slopes vary across historical time by modelling interactions between the age and year variables:

$$\mathrm{BMI}_{ti} = \pi_{0i} + \pi_{1i}(\mathrm{age} - 18)_{ti} + \pi_{2i}(\mathrm{year} - 1986)_{ti} + \pi_{3i}(\mathrm{age} - 18)_{ti}(\mathrm{year} - 1986)_{ti} + r_{ti} \quad (2)$$

The level-1 parameters are then modelled as a function of individual characteristics (at level two). The level two (between person) submodel assumes that BMI trajectories vary across individuals, and we explicitly model these differences as follows:

$$\pi_{0i} = \beta_{00} + \beta_{01}(\mathrm{female})_i + e_{0i} \quad (2.1)$$

$$\pi_{1i} = \beta_{10} + \beta_{11}(\mathrm{female})_i + e_{1i} \quad (2.2)$$

$$\pi_{2i} = \beta_{20} + e_{2i} \quad (2.3)$$

$$\pi_{3i} = \beta_{30} \quad (2.4)$$

Here, for example, the intercept and age slope from equation (1) are modelled as a function of gender, where β_{01} represents the difference in the initial BMI score for females compared with males, and β_{11} captures the difference in the rate of change in BMI over adulthood for women compared with men. (We also modelled person-level differences in the slope for year, but found no effects.) The residual errors (e_{0i}, e_{1i}, e_{2i}) capture random variance in the intercept and slope values across persons, which we attempt to capture through the incorporation of additional person-level variables. (Due to model limitations on the number of estimated variance components, we set the random variance for the age by year term to zero.) Substituting equations (2.1) through (2.4) into equation (1) gives us the full composite model:

$$\mathrm{BMI}_{ti} = \beta_{00} + \beta_{01}(\mathrm{female})_i + \beta_{10}(\mathrm{age} - 18)_{ti} + \beta_{11}(\mathrm{female})_i(\mathrm{age} - 18)_{ti} + \beta_{20}(\mathrm{year} - 1986)_{ti} + \beta_{30}(\mathrm{age} - 18)_{ti}(\mathrm{year} - 1986)_{ti} + e_{1i}(\mathrm{age} - 18)_{ti} + e_{2i}(\mathrm{year} - 1986)_{ti} + e_{0i} + r_{ti}$$

We used the MIXED procedure in SAS to estimate all models using full maximum likelihood, assuming normally distributed residuals. (The distribution of the residuals showed a good approximation to normality, with little deviation from the diagonal in the normal probability plots.) All analyses were weighted to correct for the oversampling of drug users in the panel data. Nested models were compared according to three goodness-of-fit indices: (i) change in the $-2\log$ likelihood (or deviance statistic), which follows a χ^2 distribution with degrees of freedom equal to the difference in the number of parameters tested between models; (ii) change in the Bayesian Information Criterion (BIC), which makes an adjustment for model parsimony;[40] and (iii) proportion of variance in BMI that is explained by a model (pseudo R^2), calculated by squaring the correlation between the observed and predicted BMI values.

Results

Descriptive statistics

The individuals in this study were born between 1958 and 1985, with the majority (53%) born before 1971. Socio-demographic characteristics for the study sample at baseline (age 18) are presented in Table 1. Approximately one-quarter (26.2%) went on to receive a college degree over the follow-up period.

Table 1 Descriptive statistics for adults in study sample at baseline[a] MTF 1986–2004

	N	Weighted percent
Female	5587	52.04
Race/Ethnicity		
White	8256	75.17
Non-Hispanic black	1077	11.00
Hispanic	728	7.02
Other race/ethnicity	719	6.81
Childhood SEP[b]		
Parents <High school education	966	9.28
Parents with high school education	4911	46.50
Parents with college degree or higher	4626	44.22
Region of residence		
South	3551	33.34
Northeast	2398	21.23
North Central	3034	27.78
West	1971	17.66

[a]Baseline refers to senior year of high school.
[b]SEP, socioeconomic position (assessed by parents' education).

Table 2 presents the weighted mean BMI scores for the study sample over adulthood. Three specific high-school cohorts are illustrated. Mean BMI scores increase over adulthood, but at any given age BMI scores are higher in more recent cohorts.

Unconditional growth model

The first column in Table 3 (Model A) presents the results for the unconditional growth model without any person-level covariates. The value for the intercept (22.4) represents the average BMI score at age 18. The coefficients for age express the average rate of change in BMI over adulthood. A second-order polynomial indicates that BMI scores increase steadily over young adulthood and then begin to flatten out towards midlife, as illustrated in Figure 1. After incorporating the effects of age, variation in BMI remains both within persons and between persons (variance components), and additional person-level variables may explain this variation. Note that ageing itself explains about 6.5% of the total variance in BMI scores.

Incorporating period effects

The second column of Table 3 (Model B) incorporates the period effect. The positive coefficient for year indicates that BMI scores have increased linearly since 1986, at an average rate of 2.3 BMI units every decade since 1986. BMI trajectories were then allowed to vary by historical time by including interactions between age and the year of data collection (Model C in Table 3). Compared with the model including only age, the inclusion of secular time results in an improvement in model fit and accounts for about 34% of the residual variation between individual trajectories [(0.165–0.109)/0.165]. Together, ageing and historical time explain about 8% of the total variation in BMI scores.

A visual depiction of this age and period effect is presented in Figure 2. For simplicity, only three specific high-school cohorts are illustrated. Members of the earliest cohort (born in 1958) were high-school seniors in 1976. Although height and weight data are not available for this cohort until the age of 28 (in 1986), the BMI trajectory for this group exhibits a

Table 2 Mean BMI scores (± SD) for high school cohorts over adulthood (Age 18–45) MTF 1986–2004

	Age 18	Age 25	Age 35	Age 45
Class of 1976	n/a	n/a	25.38 (3.81)	26.63 (3.46)
			N = 249	N = 215
Class of 1986	21.97 (2.92)	24.41 (3.52)	26.62 (4.25)	n/a
	N = 490	N = 274	N = 185	
Class of 1996	23.26 (3.72)	25.39 (4.61)	n/a	n/a
	N = 384	N = 199		

n/a = Not available.

gradual increase over adulthood. However, for each successive cohort, BMI scores are higher and the early adult BMI trajectory becomes increasingly steep. As a result, successive cohorts become ‘overweight’ (BMI > 25) at increasingly earlier points in the life course.

Examining social disparities

The models in Table 4 examine social disparities in adult BMI trajectories over time. As a reference point, the first column in Table 4 replicates the coefficients from Model C in Table 3.

Disparities by gender and race/ethnicity

The second and third columns of Table 4 add the effects of gender and race/ethnicity to the age and period effect model. Females and racial/ethnic minority groups are at greater risk of weight gain over the adult life course than white males (Model C). (There were no statistical interactions between any of the socio-demographic characteristics and the period effect). However, since 1986 there has been a convergence in BMI scores by racial/ethnic groups across cohorts. Figure 3 illustrates racial differences in BMI trajectories across the same three high-school cohorts depicted in Figure 2, but for simplicity we plot trajectories for women only since there are no differences in the BMI trajectories between Whites and other race/ethnic groups and between Blacks and Hispanics (Table 4, Model C), predicted BMI trajectories are only plotted for Whites and Blacks for illustrative purposes.

As illustrated in Figure 3, BMI scores are consistently higher across adulthood for racial/ethnic minority groups than for whites. However, over time the BMI trajectories for whites tend to track those for Blacks from earlier cohorts. For example, BMI scores for white women in the class of 1986 tend to approach those of black women in the 1976 cohort over the equivalent age range (age 28–36, Figure 3). Similarly, early adult BMI scores for white women in the 1996 cohort track closely those for Black women who graduated from high school 10 years earlier.

Disparities by lifetime SEP

The fourth column of Table 4 (Model D) adds childhood SEP to the model. Consistent with a life-course perspective on health disparities, higher SEP in childhood is protective of weight gain both at the age of 18 and over adulthood. Individuals who have a background of lower SEP in childhood (parents with less than college degree) have a higher BMI at age 18 (initial status) and also have

Table 3 Linear growth model coefficients for BMI MTF: 1986–2004

	Age effect Model A	Period effect Model B	Age + period effect Model C
Fixed effects			
Initial Status			
Intercept	22.418*** (0.039)	21.931*** (0.043)	21.991*** (0.055)
Rate of change			
Age	0.332*** (0.007)		0.288*** (0.012)
Age (quadratic)	−0.005*** (0.0003)		−0.011*** (0.001)
Year		0.231*** (0.003)	0.091*** (0.006)
Age by year			0.002 (0.001)
Age (quadratic) by year			0.0002*** (0.0001)
Variance components			
Level 1 within person	1.727*** (0.016)	1.883*** (0.016)	1.714*** (0.016)
Level 2 intercept	11.661*** (0.206)	12.936*** (0.252)	10.187*** (0.285)
Level 2 age	0.165*** (0.002)		0.109*** (0.008)
Level 2 age (quadratic)	0.0002*** (0.00001)		0.0002*** (0.00001)
Level 2 Year		0.048*** (0.001)	0.024*** (0.004)
Goodness-of-fit statistics			
Pseudo R^2	0.0653	0.0311	0.0801
ΔDeviance (df)	–	–	733.1 (4 df)
ΔBIC	–	–	667.3

Standard errors are in parentheses under the parameter estimates.
***$P < 0.001$ (two-tailed tests).

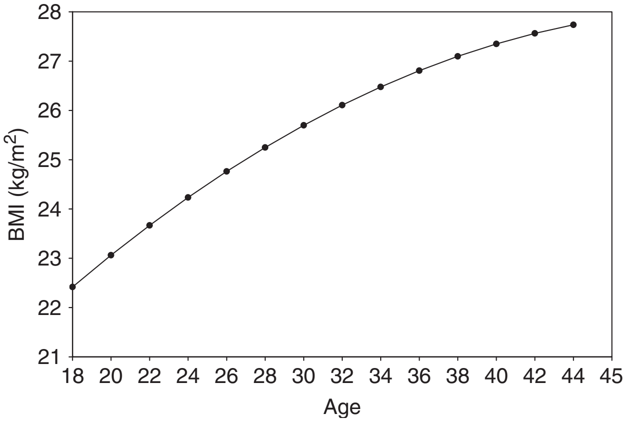

Figure 1 Predicted trajectories of BMI over adulthood: MTF (1986–2004)

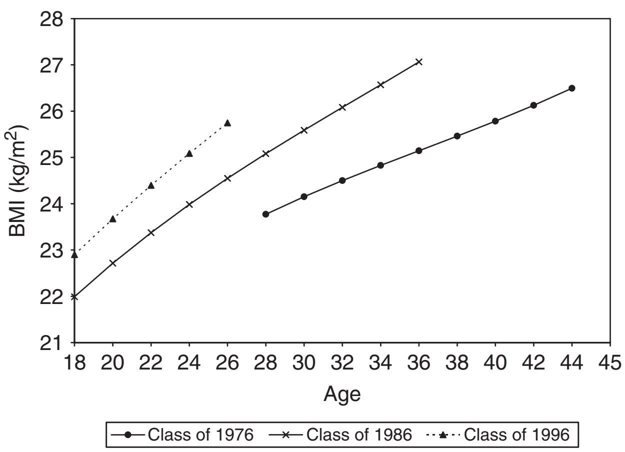

Figure 2 Predicted trajectories of BMI by high-school cohort: MTF (1986–2004)

higher rates of annual growth in BMI over adulthood than those whose parents had a college degree. Childhood SEP accounts for only a small part of the effect of racial/ethnic disadvantage [largely for Hispanics, see Table 4 (Model D)], indicating that there are other factors associated with racial/ethnic position that carry an increased risk for higher BMI that are not simply a function of SEP at birth.

However, because of the period effect, even white males from a high SEP have been gaining weight more rapidly over adulthood in more-recent cohorts. Figure 4 illustrates BMI trajectories for white males in the three separate high-school cohorts according to childhood SEP. While BMI scores are consistently higher in each cohort amongst those with lower childhood SEP (parents with less than a high-school education), at each age BMI scores for socially advantaged groups in later cohorts are actually higher than those for their socially disadvantaged counterparts who were born 10 years earlier.

The protective effects of adult SEP over the life course are weaker than for childhood SEP (Table 4, Model E). High-school seniors who go on to receive a college degree have somewhat flatter BMI trajectories over adulthood than their classmates who did not pursue further education. However, achieved SEP in adulthood does not fully mediate the lingering effects of childhood SEP. Model E simultaneously adjusts for adult SEP and examines the residual effect of childhood SEP on BMI after accounting for the mediating effects of adult SEP. Thus, social disadvantage in childhood carries a persisting risk for increased BMI throughout the life course. (There were no statistical interaction effects between gender and adult SEP, race and adult SEP or between childhood SEP and adult SEP.) Together, these socio-demographic characteristics, along with ageing and historical time, explain over 12% of the total variation in BMI scores over adulthood (final model Table 4).

Discussion

We use 19 years of nationally representative panel data for repeated cohorts of Americans to describe adult trajectories of BMI since 1986. We found that, not only do BMI scores follow a curvilinear course over adulthood, but there has also been a linear increase in adult BMI trajectories over historical time. Thus, historical conditions have differentially affected adult trajectories of weight gain since the mid-1980s. As a result, one's BMI later in adulthood may have less to do with one's age than with historical changes in social, environmental and cultural conditions that significantly influence caloric balance.[41–44]

We also found evidence of a 'social patterning'[45] in adult trajectories of BMI. Weight gain over adulthood is more rapid for disadvantaged social groups, including women, racial/ethnic minorities and those of lower SEP. As a result, social disparities in BMI become increasingly more pronounced over the adult life course, which has ominous implications for future health disparities among these cohorts as they age. Targeted interventions towards these groups,[46–48] particularly early in the life course,[49,50] remain an important and worthwhile strategy.

However, BMI scores have also been rising steadily in socially advantaged groups since 1986. Even white males in a high SEP are at an increased risk for weight gain at any given age over time. This has implications for preventive strategies. If the rise in obesity, as a public health concern, is approached from a 'fundamental cause' perspective,[24] weight gain is assumed to be the result of restricted access to health-enhancing knowledge and resources among disadvantaged social groups. But our results indicate that another strategy may need to be considered for the prevention of obesity in the more advantaged subgroups of society. These people presumably have access to resources but are failing to use them

Table 4 Growth models for BMI: fixed effect coefficients by gender, race/ethnicity and lifetime socioeconomic position MTF: 1986–2004

	Growth model + period effect Model A	+ Gender Model B	+ Race/ Ethnicity Model C	+ Childhood SEP Model D	+ Adult SEP Model E
Fixed effects					
Initial status					
Intercept (BMI at age 18 in 1986)	21.991***	22.635***	22.567***	22.398***	22.474***
	(0.055)	(0.068)	(0.071)	(0.083)	(0.135)
Female [a]		−1.248***	−1.272***	−1.284***	−1.289***
		(0.076)	(0.076)	(0.077)	(0.077)
Black[b]			0.890***	0.829***	0.835***
			(0.131)	(0.135)	(0.135)
Hispanic[b]			0.539***	0.418**	0.419**
			(0.152)	(0.162)	(0.612)
Other race[b]			−0.460**	−0.440**	−0.442**
			(0.150)	(0.154)	(0.154)
Childhood SEP[c]				0.617***	0.632***
<High school				(0.150)	(0.150)
High school[c]				0.262***	0.270***
				(0.081)	(0.081)
Adult SEP					−0.074
High School[d]					(0.101)
Rate of change					
Age	0.288***	0.299***	0.291***	0.249***	0.212***
	(0.012)	(0.014)	(0.014)	(0.016)	(0.027)
Age^2	−0.011***	−0.012***	−0.012***	−0.011***	−0.010***
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Year	0.091***	0.092***	0.090***	0.094***	0.093***
	(0.006)	(0.006)	(0.006)	(0.007)	(0.007)
Age by year	0.002	0.002	0.001	0.002	0.003*
	(0.001)	(0.001)	(0.001)	(0.001)	(0.001)
Age^2 by year	0.0002***	0.0002***	0.0002***	0.0002***	0.0001**
	(0.0001)	(0.0001)	(0.0001)	(0.0001)	(0.0001)
Age by female[a]		−0.026*	−0.030*	−0.027*	−0.025
		(0.014)	(0.014)	(0.014)	(0.014)
Age^2 by female[a]		0.003***	0.003***	0.003***	0.003***
		(0.001)	(0.001)	(0.001)	(0.001)
Age by black[b]			0.094***	0.107***	0.103***
			(0.026)	(0.027)	(0.027)
Age^2 by black[b]			−0.002	−0.002	−0.002
			(0.001)	(0.001)	(0.001)
Age by hispanic[b]			0.132***	0.117***	0.112***
			(0.032)	(0.034)	(0.034)
Age^2 by hispanic[b]			−0.004*	−0.003	−0.003
			(0.002)	(0.002)	(0.002)

(continued)

Table 4 Continued

	Growth model + period effect Model A	+ Gender Model B	+ Race/ Ethnicity Model C	+ Childhood SEP Model D	+ Adult SEP Model E
Age by other race[b]			−0.014	−0.016	−0.018
			(0.029)	(0.029)	(0.029)
Age^2 by other race[b]			−0.001	−0.001	−0.001
			(0.001)	(0.001)	(0.001)
Age by child SEP<HS[c]				0.064*	0.051
				(0.027)	(0.027)
Age^2 by child SEP<HS[c]				−0.001	−0.001
				(0.001)	(0.001)
Age by child SEP = HS[c]				0.067***	0.059***
				(0.014)	(0.015)
Age^2 by child SEP = HS[c]				−0.002**	−0.002**
				(0.001)	(0.001)
Age by adult SEP = HS[d]					0.044*
					(0.019)
Age^2 by adult SEP = HS[d]					−0.001
					(0.001)
Goodness-of-fit statistics					
Pseudo R^2	0.0801	0.0987	0.1114	0.1195	0.1223

Note: Standard errors are in parentheses under the parameter estimates.
[a]Reference group is male.
[b]Reference group is white.
[c]Reference group is parents with college degree.
[d]Reference group is college degree.
*$P < 0.05$.
**$P < 0.01$.
***$P < 0.001$ (two-tailed tests).
SEP, socioeconomic position; HS, high school.

effectively. Similar findings are beginning to appear in the literature, where social advantage does not confer the expected protective effects against weight gain in American adults.[51–53] However, if these advantaged social groups begin adopting better nutrition and exercise behaviours afforded to them by their flexible social resources,[25] we may observe a decline in their BMI trajectory over time, and this will only serve to magnify the socio-economic gradient in obesity and overweight in future years.

Limitations

Due to the underestimation of weight in self-reported data,[54] BMI scores in this sample may be higher if weight were measured directly. However, because there is no evidence of differential underreporting of weight in ethnic or racial minority groups, or by gender or SEP, the social patterns in BMI are not likely to differ markedly from those reported here. Because study subjects were selected in their senior year of high school, high-school dropouts are not represented. Although panel attrition in the MTF data is nontrivial, statistically adjusting for attrition yielded no differences in the results. Because follow-up is ongoing in this study, relatively fewer data points are available for estimation at the tail end of the growth curves (age 35–45) in these analyses.

Our purpose in this paper was to describe social disparities in BMI over adulthood as they change over historical time. The results highlight the importance of social status and socio-economic resources for maintaining optimal weight. Yet, even those in advantaged social positions, presumably with access to health-enhancing resources, have experienced an increase in BMI in more recent cohorts. Thus, promoting access to resources and opportunities for healthy eating and exercise may not be sufficient for overturning the obesity epidemic.

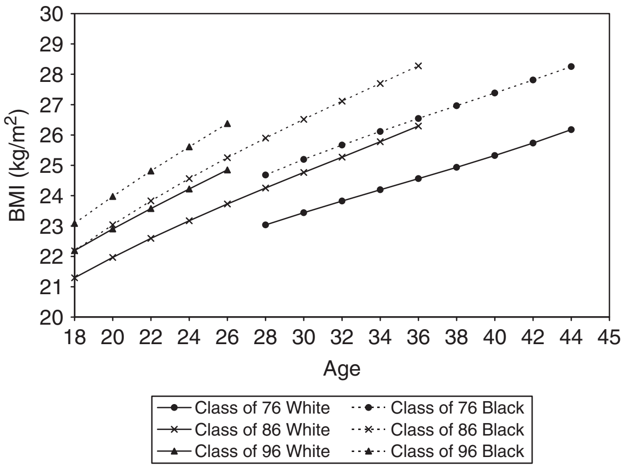

Figure 3 Predicted trajectories of BMI by high-school cohort and race/ethnicity for women: MTF (1986–2004)

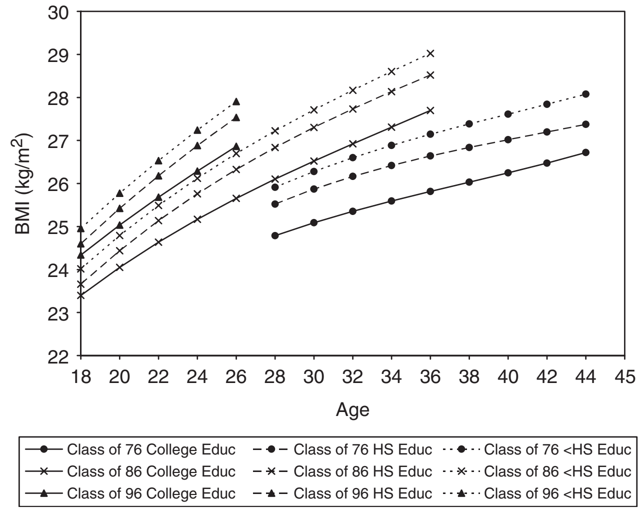

Figure 4 Predicted trajectories of BMI by high-school cohort and childhood SEP (parents' education) for White males: MTF (1986–2004)

Funding

Robert Wood Johnson Foundation, as part of the Youth, Education, and Society Project; National Institute on Drug Abuse, a part of the National Institutes of Health; University of Michigan Center for Social Inequality Mind and Body Life Course core [National Institutes of Health/National Institutes of Child Health and Human Development Grant (R24HD04786)].

Acknowledgements

An earlier version of this paper was presented at the Second North American Congress of Epidemiology in Seattle, WA, June 21–24, 2006. We thank Jersey Liang and Jim House for helpful comments on an earlier draft.

Conflict of interest: None declared.

KEY MESSAGES

- No national studies to date have examined how social disparities in obesity and overweight differ by age and historical period using longitudinal data with repeated measures.
- Using panel data from the nationally representative MTF Study (1986–2004) in the United States to examine social disparities in trajectories of BMI over adulthood (age 18–45), we found a strong period effect, such that weight gain was more rapid for more recent cohorts; as a result, successive cohorts become overweight (BMI $>$ 25) at increasingly earlier points in the life course.
- BMI scores were consistently higher for women, racial/ethnic minority groups and those from a lower SEP.
- However, BMI scores for socially advantaged groups in recent cohorts were actually higher than those for their socially disadvantaged counterparts who were born 10 years earlier.

References

1 Flegal KM, Carroll MD, Kuczmarski RJ, Johson CL. Overweight and obesity in the United States: prevalence and trends, 1960–1994. *Int J Obes Relat Metab Disord* 1998;**22:**39–47.

2 Hedley AA, Ogden CL, Johnson CL, Carroll MD, Curtin LER, Flegal KM. Prevalence of overweight and obesity among US children, adolescents, and adults, 1999–2002. *JAMA* 2004;**291:**2847–50.

3 Lewis CE, Jacobs DR, McCreath H *et al*. Weight gain continues in the 1990s: 10 year trends in weight and overweight from the CARDIA study. *Am J Epidemiol* 2000;**151:**1172–81.

4 Mokdad AH, Bowman BA, Ford ES, Vinicor F, Marks JS, Koplan JP. The continuing epidemics of obesity and diabetes in the United States. *JAMA* 2001;**286:**1195–200.

5 Maillard G, Charles MA, Thibult N *et al*. Trends in the prevalence of obesity in the French adult population

between 1980 and 1991. *Int J Obes Relat Metab Disord* 1999;**23:**389–94.

6 Kuczmarski RJ, Flegal KM, Campbell SM, Johnson CL. Increasing prevalence of overweight among US adults: the National Health and Nutrition Examination Surveys, 1960–1991. *JAMA* 1994;**272:**205–11.

7 WHO. *Obesity: Preventing and Managing the Global Epidemic*. Report of a WHO consultation on obesity. Geneva, Switzerland: World Health Organization, 1997.

8 WHO. *Life in the 21st Century – A Vision for All*. The World Health Report. Geneva, Switzerland: World Health Organization, 1998.

9 US Department of Health and Human Services. *The Surgeon General's Call to Action to Prevent and Decrease Overweight and Obesity*. Rockville, MD: US Department of Health and Human Services, Public Health Service, Office of the Surgeon General, 2001.

10 Thorpe KE, Florence CS, Howard DH, Joski P. The impact of obesity on rising medical spending. *Health Aff* 2004; **4:**480–86.

11 Olshansky SJ, Passaro DJ, Hershow RC *et al*. A potential decline in life expectancy in the United States in the 21st century. *New Engl J Med* 2005;**352:**1138–45.

12 Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. *New Engl J Med* 1993;**329:**1008–12.

13 Sturm R. The effects of obesity, smoking, and drinking on medical problems and costs. Obesity outranks both smoking and drinking in its deleterious effects on health and health costs. *Health Aff* 2002;**21:**245–253.

14 Field AE, Coakley EH, Must A *et al*. Impact of overweight on the risk of developing common chronic diseases during a ten year period. *Arch Intern Med* 2001;**161:**1581–86.

15 Must A, Spadano J, Coakley EH *et al*. The disease burden associated with overweight and obesity. *JAMA* 1999;**282:**1523–29.

16 Manson JE, Stampfer MJ, Hennekens CH, Willett WC. Body weight and longevity. A reassessment. *JAMA* 1987;**257:**353–58.

17 Troiano RP, Flegal KM. Overweight children and adolescents: description, epidemiology, and demographics. *Pediatrics* 1998;**101:**497–504.

18 National Center for Health Statistics (NCHS). *Health, United States 2005 with Chartbook on Trends in the Health of Americans*. Hyattsville, MD, 2005.

19 Ogden CL, Carroll MD, Curtin LR, McDowell MA, Tabak CJ, Flegal KM. Prevalence of overweight and obesity in the United States, 1999–2004. *JAMA* 2006;**295:**1549–55.

20 Flegal KM, Carroll MD, Ogden CL, Johnson CL. Prevalence and trends in obesity among US adults, 1999–2000. *JAMA* 2002;**288:**1723–27.

21 Sobal J, Stunkard AJ. Socioeconomic status and obesity: a review of the literature. *Psychol Bull* 1989;**105:**260–75.

22 Freedman DS, Khan LK, Serdula MK, Ogden CL, Dietz, WH. Racial and ethnic differences in secular trends for childhood BMI, weight, and height. *Obesity* 2006; **14:**301–8.

23 Paeratakul S, Lovejoy JC, Ryan DH, Bray GA. The relation of gender, race, and socioeconomic status to obesity and obesity comorbidities in a national sample of US adults. *Intern J Obes* 2002;**26:**1205–10.

24 Link BG, Phelan JC. Social conditions as fundamental causes of disease. *J Health Soc Behav* 1995;**35:80–94.**

25 Phelan JC, Link BG. Controlling disease and creating disparities: a fundamental cause perspective. *J Gerontol: Series B* 2005;**60B (Special Issue II):**27–33.

26 Morland K, Wing S, Diez Roux A. The contextual effect of the local food environment on residents' diets. *A J Public Health* 2002;**82:**1761–67.

27 Powell LM, Auld MC, Chaloupka FJ, O'Malley PM, Johnston LD. Associations between access to food stores and adolescent body mass index. *Am J Prev Med* 2007;**33:**S301–7.

28 Powell LM, Slater S, Chaloupka FJ. The relationship between physical activity settings and race, ethnicity, and socioeconomic status. *EvidBased Prev Med* 2004;**1:**135–44.

29 Johnston LD, Delva J, O'Malley PM. Sports participation and physical education in American secondary schools: current levels and racial/ethnic and socioeconomic Disparities. *Am J Prev Med* 2007;**33:**S195–208.

30 James SA, Fowlser-Brown A, Ragthunathan TE, Van Hoewyk J. Life-course socioeconomic position and obesity in African American Women: The Pitt County Study. *Am J Public Health* 2006;**96:**554–60.

31 Baltrus PT, Lynch JW, Everson-Rose S, Raghunathan TE, Kaplan GA. Race/ethnicity, life-course socioeconomic position, and body weight trajectories over 34 years: The Alameda County Study. *Am J Public Health* 2005; **95:**1595–601.

32 Riley MW. In: Riley MW (ed.). *On the significance of age in sociology*. Beverly Hills, CA: Sage, 1988, pp. 24–45.

33 Ryder NR. The cohort as a concept in the study of social change. *Am Sociol Rev* 1965;**30:**843–61.

34 Elder GH, Jr. *Children of the Great Depression: Social Change in Life Experience*. Chicago: University of Chicago Press, 1974.

35 Johnston LD, O'Malley PM, Bachman JG, Schulenberg JE. *Monitoring the Future National Survey results on Drug Use, 1975-2006:* Volume 1, Secondary school students (NIH Publication No. 07-6205). Bethesda, MD: National Institute on Drug Abuse, 2007.

36 Bachman JG, Johnston LD, O'Malley PM. *The Monitoring the Future Project after Twenty-Seven Years: Design and Procedures*. (Monitoring the Future Occasional Paper No. 54.) Ann Arbor, MI: Institute for Social Research, 2001.

37 National Institutes of Health (NIH). Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults: the evidence report [published correction appears in Obesity Research 1998;**6**:464]. *Obes Res* 1998;**6 (Suppl 2):**51S–209S.

38 Singer JD, Willet JB. *Applied Longitudinal Data Analysis*. New York: Oxford University Press, 2003.

39 Raudenbush SW, Wing-Shing C. Application of a hierarchical linear model to the study of adolescent deviance in an overlapping cohort design. *J Consult Clin Psychol* 1993;**61:**941–51.

40 Raftery AE. Bayesian model selection in social research. *Sociol Method* 1995;**25:**111–63.

41 Brown PJ. Culture and the evolution of obesity. *Hum Nat* 1991;**2:**31–57.

42 Cawley J. Markets and childhood obesity policy. *Future Child* 2006;**16:**69–88.

[43] Anderson PM, Butcher KF. Childhood obesity: trends and potential causes. *Future Child* 2006;**16:**19–45.

[44] Glass TA, McAtee MJ. Behavioral science at the crossroads in public health: Extending horizons, envisioning the future. *Soc Sci Med* 2006;**62:**1650–71.

[45] McIntyre S. The pattering of health by social position in contemporary Britain: directions for sociological research. *Soc Sci Med* 1986;**23:**393–415.

[46] Kumanyika S, Grier S. Targeting interventions for ethnic minority and low-income populations. *Future Child* 2006;**16:**187–207.

[47] Dallow CB, Anderson J. Using self-efficacy and a transtheoretical model to develop a physical activity intervention for obese women. *Am J Health Promot* 2003;**17:**373–81.

[48] Baker EA, Kelly C, Barnidge E *et al*. The Garden of Eden: Acknowledging the impact of race and class in efforts to decrease obesity rates. *Am J Public Health* 2006; **96:**1170–74.

[49] Fitzgibbon ML, Stolley MR, Schiffer L, Van Horn L, Kauferchristoffel K, Dyer AR. Two-year follow-up results for hip-hop to health jr.: a randomized controlled trial for overweight prevention in preschool minority children. *J Pediatr* 2005;**146:**618–25.

[50] Caballero B, Clay T, Davis SM *et al*. Pathways: a school-based, randomized controlled trial for the prevention of obesity in American Indian schoolchildren. *Am J Clin Nutr* 2003;**78:**1030–38.

[51] Chang VW, Lauderdale DS. Income disparities in body mass index and obesity in the United States, 1971–2002. *Arch Intern Med* 2005;**165:**2122–28.

[52] Zhang Q, Wang Y. Socioeconomic inequality of obesity in the United States: do gender, age, and ethnicity matter? *Soc Sci Med* 2004;**58:**1171–80.

[53] Mujahid MS, Diez Roux AV, Borrell LN, Nieto FJ. Cross-sectional and longitudinal associations of BMI with socioeconomic characteristics. *Obes Res* 2005;**13:**1312–21.

[54] Stewart AW, Jackson RT, Ford MA, Beaglehole R. Underestimation of relative weight by use of self-reported height and weight. *Am J Epidemiol* 1987;**125:**122–26.

Published by Oxford University Press on behalf of the International Epidemiological Association

International Journal of Epidemiology 2009;**38**:509–511
doi:10.1093/ije/dyn352

Commentary: Closing the disparity gaps in obesity

Boyd A Swinburn

Accepted 4 December 2008

The two big challenges that obesity presents us with are how to reduce its overall burden and how to reduce its associated disparities—especially the disparities by socio-economic position (SEP) and ethnicity. The paper by Clarke *et al*.[1] in this issue elegantly highlights the epidemiology of the obesity epidemic in relation to the changing patterns in the USA across gender, ethnicity, SEP, age and cohort. As shown in this and other US studies in adults and children,[2–4] the inter-relationships between these factors are complex and changing over time. Sophisticated studies, such as this analysis of the Monitoring the Future Study (MTS), are needed to help untangle the various contributions of these determinants to the prevalence and, more importantly, the trends in obesity. For example, the findings that the ethnic and SEP disparities in body mass index (BMI) seem to increase over the life course of the population (longitudinal analyses) but decrease over time (serial cross-sectional analyses) is subtle but important—the gaps between Whites and Blacks or between advantaged and disadvantaged are greater for older age groups but are overall less now than they used to be.

Longitudinal studies consistently show that people with lower SEP gain more weight than those with higher SEP,[5] but these findings need to be combined with monitoring of population trends to determine whether the SEP gap is narrowing or widening. The MTS data to 2004 seem to suggest that the SEP gap is narrowing over time. This is somewhat contrary to the expectation that any flattening of the obesity trajectory will occur earlier in the more advantaged sections of the community, thus widening the SEP gap. This will be a very important gap to keep a close eye on as the obesity epidemic unfolds.

Despite the complex, inter-related nature of the determinants of obesity, there are a few clear messages coming through from the research findings.

WHO Collaborating Centre for Obesity Prevention, Deakin University, 221 Burwood Highway, Melbourne 3125, Australia. E-mail: boyd.swinburn@deakin.edu.au