

The Obesity Drift:  
Generational Weight (BMI) Patterns and Health Effects

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**October, 2001**

Preliminary Draft

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We gratefully acknowledge Daniel Hill and David Weir for helpful comments on an earlier draft.

## Introduction

Concern over rising body weight in the United States, measured by Body Mass Index (BMI;  $\text{kg}(\text{weight})/\text{m}^2(\text{height})$ ), is confirmed by data from the Panel Study of Income Dynamics (PSID). A common translation of BMI into weight categories is as follows: Underweight,  $<18.5$ ; Normal,  $18.5-24.9$ ; Overweight (OW),  $25.0-29.9$ ; Obese I (OB-I),  $30-34.9$ ; Obese II (OB-II),  $35-39.9$ ; and Extreme Obesity (EOB);  $\geq 40$ . The PSID data series is sponsored by the National Science Foundation (NSF). Supplements in 1986 and 1999 sponsored by the National Institute on Aging (NIA) provide measures for adults in those years. In 1997 measures of the BMI of children age 0-12 were gathered in a supplement sponsored the National Institute for Child Health and Human Development (NICHD).

Recently, the data from these different years of the PSID data archive ([www.umich.edu/~psid/](http://www.umich.edu/~psid/)) were assembled to address two questions. First, is there a shift through time to higher weights of adults? Second, is there significant individual mobility across weight patterns over time? Third, are there factors operating across generations in the same family? Preliminary results show that the answer to all these questions is 'yes'. In addition, we show an association between obesity, other health behaviors, and life course mortality risk. The genealogical design of the study permits us to study the extent to which being overweight is related across three different generations in the same family.

Because of the only recent availability of these repeated measures on BMI in the PSID, our primary goal is to provide an overview of the evolution of BMI through time for the overall population, for specific subgroups, and to explore some of the basic dynamic patterns at a more descriptive level. For now we will not delve into the complex issues of what family and individual circumstances lead to wide cross-sectional differences in BMI and differing dynamic patterns for particular subgroups.

## **I. Background and Descriptive Statistics**

### **A. Adults**

There is epidemiological evidence showing increases in mortality of adults with BMI's above 25. This increase is modest up to a BMI level of 30. For those with BMI of 30 and above, mortality rates from all causes, especially from cardiovascular heart diseases (CHDs) are increased by 50-100% above persons with BMIs in 20-25 range. Overweight and obesity are the second leading cause of preventable death in US today (Allison et al., 1999; U.S. DHHS, June 1998). The health risks have been identified as hypertension, type 2 diabetes, stroke, gallbladder disease, osteoarthritis, sleep apnea, respiratory problems, endometrial, breast, prostate, colon cancers. These conditions are known to lead to increased medical care and disability costs (U.S. DHHS, June 1998). In 1995, the total cost attributed to overweight was estimated to be \$99.2 billion, with more than half of all direct medical costs associated with diseases attributable to obesity; indirect costs are loss of output from reduced labor market activity (U.S. DHHS, November 2000).

Based on an NIH report<sup>1</sup>, women with low education or income are more likely to be obese than those of higher SES; this association is less consistent in men. Below we show that even for men there is an educational factor. So the shift to higher BMI occurs despite the long-term shift toward higher levels of educational attainment.

"The number of overweight (OW) and obese (OB) men and women has risen between 1960 (National Health and Nutrition Survey - NHANES-II) and 1994 (OW increased from 30.5 to 32% and OB from 12.8 to 22.5%); in the last decade the percentage of people in these categories has increased to 54.9% of adults age 20 and older (NHANES III). OW and obesity are especially evident in some minority groups, as well as in those with lower incomes and less education... NHANES III

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<sup>1</sup> "CLINICAL GUIDELINES ON THE IDENTIFICATION, EVALUATION, AND TREATMENT OF OVERWEIGHT AND OBESITY IN ADULTS"

estimated that 13.7% of children and 11.5% of adolescents are overweight, while a number of smaller, ethnic-specific studies suggest that OW and OB may afflict up to 30 to 40% of children and youth from minority populations.....”.

**TABLE 1  
PERCENTAGE WHO ARE OVERWEIGHT OR OBESE, 1960-1994**

Gender, race/ethnicity, age 20 years and older, age adjusted:	NHES I 1960-62 (age 20-74)	NHANES I 1971-74 (age 20-74)	NHANES II 1976-80 (age 20-74)	NHANES 1982-84 (age 20-74)	NHANES III 1988-94 (age >=20)
Both Sexes	43.3	46.1	46.0		54.9
Men	48.2	52.9	51.4		59.4
Women	38.7	39.7	40.8		50.7
White men	48.8	53.7	52.3		61.0
White women	36.1	37.6	38.4		49.2
Black men	43.1	48.9	49.0		56.5
Black women	57.0	57.6	61.0		65.8
White, non-Hispanic men			52.0		60.6
White, non-Hispanic women			37.6		47.4
Black, non-Hispanic men			48.9		56.7
Black, non-Hispanic women			60.6		66.0
Mexican American men				59.7	63.9
Mexican American women				60.1	65.9
Age and gender-specific categories:					
Men					
20-29	39.9	38.6	37.0		43.1
30-39	49.6	58.1	52.6		58.1
40-49	53.6	63.6	60.3		65.5
50-59	54.1	58.4	60.8		73.0
60-69	52.9	55.6	57.4		70.3
70-79	36.0	52.7*	53.3*		63.1
80+	N/A**	N/A**	N/A**		50.6
Women					
20-29	17.0	23.2	25.0		33.1
30-39	32.8	35.0	36.8		47.0
40-49	42.3	44.6	44.4		52.7
50-59	55.0	52.2	52.8		64.4
60-69	63.1	56.2	56.5		64.0
70-79	57.4	55.9*	58.2*		57.9
80+	N/A**	N/A**	N/A**		50.1

\*Prevalence for age 70 to 74 years. \*\* Not available  
Overweight or obese = BMI≥25

Data from the NHANES cross-sections are presented in Table 1. These data document the upward temporal trend of obesity and its concentration in different demographic groups. Close to two-thirds of many subgroups are overweight or obese. The data also

suggest a life course pattern with weight rising to the seventies and then declining. This is, of course, oversimplified and inaccurate if high BMI does increase mortality risks, leaving more of the persistently thinner adults to survive to older ages.

We compared PSID data for selected years to the 1999 cross-section from the NHANES national sample. From both Tables 1 and 2, it is clear that weight is rising for both men and women over time. Data from PSID 1986 shows 10.5% as obese, a percent that more than doubled to 24.2% in PSID 1999. And in all BMI categories for 1999, PSID and NHANES show similar values. Preliminary work shows this is not just attributable to a changing age distribution. Why are body weights rising? Or are they really rising that much?

Table 3 presents cross-sectional median BMI tables by age group, 1986 and 1999, for the entire PSID sample disaggregated by key elements in the weight calculation and by groups defined by age, education, and race. Here, in contrast to the strong rise in the percentages of people crossing the 25 or 30 BMI thresholds in Tables 1 and 2, we see a far more gentle upward movement in the medians from 24.3 for the overall 1986 average to 25.9 by 1999. For the subgroups, much of the movement upward is on the same order of magnitude, but given the large share of individuals in the range just below 25.0 as of 1986 (i.e., the cutoff value for 'overweight'), the percent crossing the 25.0 threshold into 'overweight' by 1999 is quite dramatic. So the issue of the 'epidemic' seems in part a matter of how the data are presented.

BMI Categories		overall	men	women
overweight				
25=<BMI<30				
	PSID86	26.5	33.4	21.1
	PSID99	35.9	44.2	28.7
	NHANES99	34.0	NA	NA
obese				
BMI>=30				
	PSID86	10.5	10.2	10.5
	PSID99	24.2	25.1	23.4
	NHANES99	27.0	NA	NA
obese or overweight				
BMI>=25				
	PSID86	37.0	43.6	31.6
	PSID99	60.0	69.3	52.1
	NHANES99	61.0	NA	NA

		age 20-29	age 30-39	age 40-49	age 50-59	age 60-69	age 70 plus	All ages
	PSID YEAR							
All	86	22.95	23.82	24.50	25.18	25.20	24.72	24.31
	99	25.05	25.77	25.94	26.51	26.70	25.48	25.89
White men	86	24.30	25.11	25.89	25.94	25.90	24.50	25.18
	99	25.59	26.24	26.48	27.16	27.22	25.86	26.48
White women	86	21.53	21.89	22.89	23.86	24.70	24.71	22.89
	99	23.11	23.15	23.86	24.63	25.05	24.89	23.86
Black men	86	23.65	25.16	25.18	25.94	25.94	25.92	25.16
	99	25.86	27.30	27.56	26.68	28.41	25.20	27.14
Black women	86	23.52	24.72	27.42	27.56	28.29	24.53	25.48
	99	26.80	27.31	27.88	28.75	30.34	27.04	27.56

A number of possible factors come to mind as an explanation for this upward drift in BMI. One has been the argument that there has been a long term shift to a more sedentary life style which would possibly affect all ages. If so the upward shift should not be substantially reduced by the type of age disaggregation in Table 3, but the upward shift of the medians was pervasive. Here we take a closer look at the question of whether there is a basic upward shift in BMI versus a simple compositional change in which there are,

between 1986 and 1999, simply more adults in the ‘weight prone categories’ as predicted by a simple BMI model.

The BMI for each individual was regressed on  $X$ , a vector of explanatory variables. From estimated coefficients, a conditional mean of BMI at time  $t$  and  $s$  is can be written as  $E(BMI | X, t) = \hat{\beta}_t X$  and  $E(BMI | X, s) = \hat{\beta}_s X$ , respectively. A difference in the conditional mean of BMI through time is  $E(BMI | X, s) - E(BMI | X, t) = (\hat{\beta}_s - \hat{\beta}_t) X$ . In practice, for each gender/race group, we estimated a basic BMI equation for each 1986 and 1999 PSID samples. The set of  $X$  variables included age, age<sup>2</sup>, age<sup>3</sup>, dummies for smoking and regular exercise, and their interactions. Using the estimated coefficients,  $\hat{\beta}_{86}$  and  $\hat{\beta}_{99}$ , from each gender/race group sample, we calculated conditional mean of BMI, evaluated at midpoint of age ranges (age=25, 35, 45, 55, and 65), a non-smoker (smoke=0) and a non-regular exerciser (exercise=0).

Table 4 presents the estimated average BMI by gender/race. This more focused analysis supports the main implication of Table 3 in demonstrating that there is almost always an increase in average BMI between 1986 and 1999 across all gender/race groups, over all age groups.

Race/gender	PSID year	Age group				
		20-29	30-39	40-49	50-59	60-69
White men	86	25.49	26.45	26.98	27.07	26.69
	99	27.48	28.07	28.51	28.62	28.22
White women	86	23.30	24.06	24.95	25.69	26.05
	99	25.42	25.49	25.99	26.53	26.72
Black men	86	24.68	26.24	27.12	27.44	27.32
	99	26.92	28.32	28.96	28.91	28.25
Black women	86	24.75	26.81	28.40	29.20	28.87
	99	28.44	28.95	29.84	30.55	30.54

## B. Children Age 2-12

Why are body weights rising over such a wide range of the U.S. population? This is a question with only partial answers to date. If it is a shift to sedentary work, as opposed to a generally more sedentary life style, we might not expect to find this pattern affecting young children. Yet, as shown below, the prevalence of OW/OB is rising among American youth. According to a recent National Center of Health Statistics report (U.S. DHHS, March 2001), an estimated 13% of children ages 6-11 and 14% of adolescents ages 12-19 are overweight, an increase from 11% in the previous survey conducted between 1988 and 1994 (Table 5).

This conclusion is based on initial results from the most recent National Health and Nutrition Examination Survey (NHANES 1999), which used measured heights and weights and based its definition of overweight on the body mass index concept (weight/height<sup>2</sup>; kg/m<sup>2</sup>). To identify children who are overweight or at risk of overweight, NHANES used the Centers for Disease Control BMI-for-age-growth charts for the U.S. and a cutoff criterion of BMI values at or above the 95<sup>th</sup> percentile. This increase in the prevalence of overweight exists despite a national health objective for 2010 to reduce these rates in children and thus lower the risk of becoming overweight in adulthood.

Age (years)*	1963-65, 1966-70**	1971-74	1976-80	1988-94	1999
6-11	4	4	7	11	13
12-19	5	6	5	11	14

\*Source: Table 1 from <http://www.cdc.gov/nchs/products/pubs/pubd/hestats/overwght99.htm>

\*\*Excludes pregnant women starting with 1971-74. Pregnancy status not available for 1963-65 and 1966-70

\*\*\*Data for 1963-65 are for children 6-11 years of age; data for 1966-70 are for adolescents 12-17 years of age, not 12-19 years.

From the Child Development Supplement (CDS) to the PSID-1997 we have measures of BMI for children for 2-12 ages. These are derived using the same methodology as used in the NHANES, from in-home measures using a scale and a tape measure, with specific training of professional interviewers to obtain accurate measures. Table 6 presents the



weighted results using the 95<sup>th</sup> percentile as overweight and the 85<sup>th</sup> percentile as ‘risk for overweight.’ These data match quite well to the NHANES data. Overall, almost one-fifth of these young children can be considered overweight. More young boys are overweight or at risk of overweight (38.5 %) than are young girls (28.1 %).

BMI group	Boy	Girl	Overall
underweight	7.62	9.91	8.76
normal	53.87	62.02	57.93
risk of overweight	14.95	12.68	13.82
overweight	23.56	15.39	19.49
Total	100	100	100

Thus, our initial exploration indicates that the PSID measures of BMI match quite well to other national data. What is distinctive about the PSID is that it has a family and generational design. Starting from the original sample in 1968 the families are re-interviewed and as the children leave home to form their own households, they become new members of the active sample of adults. See “The Sample” in the Overview section in See <http://www.isr.umich.edu/src/psid>. These features, an intergenerational panel design with high response rates, create an opportunity to study BMI transitions across time for a given individual and across generations in the same family. To explore some of the nature of BMI levels over the life course and across generations we look more closely at the inter-temporal and intergenerational links of obesity in the PSID.

## **II. Intertemporal and Intergenerational Patterns of Overweight.**

### **A. Intertemporal Weight Patterns**

Are most overweight people persistently overweight? Everyone has heard of the crash diet in which weight may be gone temporarily but then reappear as the discipline or novelty wears off, and those with major illnesses may lose weight because of the illness itself or in response to medical advice to lose weight to control health risks. Here we provide an overview of the transitions across weight categories over a 13-year span.

The 1986 BMI categories are those previously used, but for 1999 we have supplemented the categories with two additional categories: non-response and deceased.

In addition, we have converted the initial 1986 BMI values into decile values and have created a new set of decile values for 1999. The need for new decile values arises because of the upward drift seen in the repeated cross sectional data and, for this panel sample, the fact that the individuals have aged approximately 13 years (depending on precise interview dates). This aging effect is somewhat complex to account for since, as we will demonstrate, there is a strong life cycle effect over the age range 30-40. Nonetheless, the basic transition table for adult men, age 20-60 as of 1986, gives a picture of how much overall ‘migration’ there is through time.

Table 7. BMI Transition Probabilities, Men's BMI Deciles between 1986 and 1999

1986 BMI group		1999 BMI group												% of retained sample		
decile	BMI range	BMI range	<23.04	To 23.84	To 25.09	To 25.86	To 26.64	To 27.60	To 28.80	To 30.52	To 32.67	>32.67	deceased	non-rep	Rising	Falling
1	<21.70		26.94	16.50	6.33	6.13	1.39	2.30	1.26	1.06	0.00	0.58	6.09	31.41	35.55	0.00
2	To 22.89		16.17	19.25	9.28	4.89	5.91	6.59	2.51	1.18	0.00	0.59	3.11	30.53	30.95	16.17
3	To 23.71		7.98	13.10	13.00	12.15	6.20	3.30	2.95	2.64	0.47	0.45	5.05	32.71	28.16	21.08
4	To 24.45		5.54	8.02	13.92	9.70	11.72	8.70	4.27	4.97	0.23	0.55	5.82	26.56	30.44	27.48
5	To 25.18		2.51	3.42	7.91	10.79	13.14	9.48	5.51	6.52	4.13	1.15	4.02	31.44	26.79	24.63
6	To 25.92		0.06	2.21	9.50	8.47	13.63	11.06	8.59	5.21	6.82	1.80	5.09	27.54	22.42	33.87
7	To 26.93		0.85	3.58	1.60	4.55	6.90	14.06	12.16	15.49	9.68	2.47	1.91	26.76	27.64	31.54
8	To 28.23		1.89	1.07	3.25	3.38	4.31	6.32	11.01	16.91	14.75	6.90	4.40	25.80	21.65	31.23
9	To 30.45		1.20	0.00	1.18	0.56	2.27	2.96	5.56	14.91	17.58	15.03	5.00	33.75	15.03	28.64
10	>30.45		0.01	1.08	0.57	0.56	2.76	2.73	2.18	6.38	10.91	37.44	7.92	27.45	0.00	27.18

The results indicate that there is substantial migration or transition as can be seen from the summary columns, “Percent changing BMI deciles 1986-1999”. The values for the decile ranges shifting upward from 1986-1999 are evident from the entries across the top descriptive row of the table. To illustrate, in this panel sample the 1986 median BMI is 25.18 (upper end of the 40-50 percentile range) and for 1999 this value is 26.64. The results from Table 7 show that there is quite a bit of mobility for weight across time. Some of this is no doubt measurement error, but there is enough transition upward and downward to suggest that people do not necessarily ‘lock in’ to a weight pattern as young adults. However, some of this change in relative position is certainly dependent on how many initial sample members are in the early 30’s range and then migrate through to their mid 40’s by 1999.

To begin, consider all of those in the lowest BMI decile (decile 1) as of 1986. By 1999 what is their BMI position? Some (26.94 percent) remained alive, were respondents in 1999, and remained in the lowest BMI decile (decile 1). This is our ‘no change’ group. For our purpose, another group is ‘barely moved’. In this group, an individual moved only to the second decile, a neighboring decile, in the relative distribution of 1999. Specifically, 16.50 percent did so. Because of possible measurement error and the close proximity to decile 1, we will call this the ‘barely moved group’. A temporary weight gain or misreporting could have caused their decile reclassification. Summing the entries in the 1986 BMI decile 1 row across 1999 decile values 3 through 10, we have 19.05 percent. These we refer to as substantial weight gain or ‘moved up’. Finally adding the 6.09 percent known deceased with 31.41 percent non-respondents<sup>2</sup> to the ‘no change’, ‘the barely moved’, and ‘moved up’, we account for all 100 percent of those in the first BMI decile of the 1986 sample.

In a parallel fashion we can examine those in the top (10<sup>th</sup>) 1986 decile. The ‘no change group’ accounts for 37.44 percent of them as of 1999. That is, in each year they were in the top decile. The ‘barely moved’ group represents 10.91 percent. An interesting result is that approximately 16 percent moved into the range of normal (18.5-24.9) or overweight (24.9-29.9). Or another way of saying it is that of those in the top 1986 decile and remaining in the sample in both years, somewhat over half (58 percent) remained in the top weight decile, 17 percent were ‘barely moved’ and 25 percent had a significant weight loss, moving into categories of ‘overweight’ and ‘normal’.

In the middle deciles, there is room to move in both upward and downward directions. At 5<sup>th</sup> decile only 13.14 percent remain in the same 1999 decile (for non-deceased who remain respondents). Note that the 5<sup>th</sup> decile (median) BMI value moves upward from 25.18 to 26.64. Two reasons for this are the overall cross-sectional drift noted above, the

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<sup>2</sup> The relationship between non-response and deceased becomes important when we examine below the relation between BMI and mortality, 1986-1999.

possible influence of life cycle weight gain, and possible sample selectivity from the categories of deceased and non-respondent.

Extending the discussion in parallel with that for the top and bottom deciles, there are 20.27 percent in the ‘barely moved’ group (10.79 percent in the 4<sup>th</sup> decile in 1999 plus 9.48 percent in decile 6<sup>th</sup> in 1999). Of those in the 5<sup>th</sup> decile in 1986 and remaining in the sample in both years, something around 20 percent (20.4) remained in the 5<sup>th</sup> weight decile, around a third (31.4) ‘barely moved’ (one decile up or down), around one quarter (26.8) gained and one fifth (21.4) lost.

The impression we have is that there is substantial BMI mobility over the life course. However, it is difficult to put into perspective without some comparison. A somewhat non-traditional comparison may be to the mobility of economic measures, such as income or wealth decile mobility. A common simple index of relative mobility is simply based on off-diagonal elements in a transition table from a balanced panel (excluding those who became non-response or deceased by 1999), known as the Shorrocks index<sup>3</sup>.

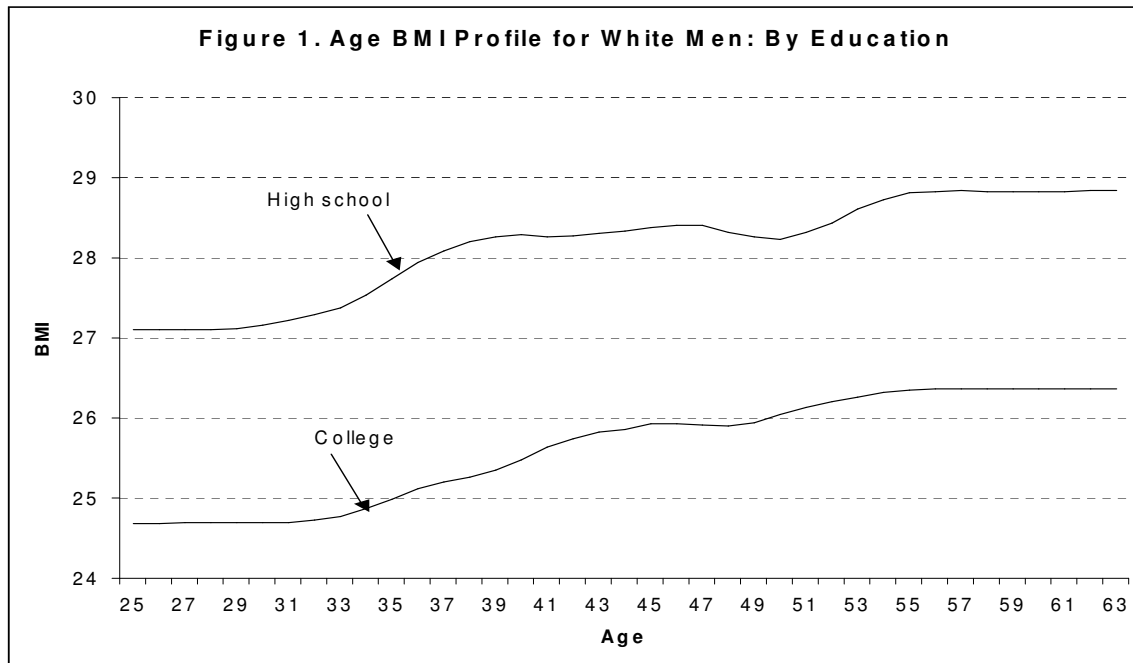
In studying ten-year decile mobility of household net worth, 1984-1994, it was estimated that the Shorrocks index had a value of .804 (U.S). For Sweden the wealth mobility over the nine-year period from 1983-84 to 1992-1993, the index has been estimated as .870 (Hurst, Louh, and Stafford, 1998). In Table 7 for BMI transition probabilities, the Shorrocks index has a value of .813. This suggests the intriguing result that relative BMI and household wealth mobility are of the same order to magnitude. Stylizing some, we might say that ‘losing weight’ is about as likely as ‘becoming rich’.

In studying the mobility of BMI or wealth one should reflect on the fact that a balanced panel will very likely include and aging or life course process. People often gain both weight and net worth as part of the life-cycle aging process.

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<sup>3</sup> The Shorrocks measure,  $S$ , is given by  $S = [N - tr(P)]/(N-1)$ , where  $N$  is the number of fractiles (ten for decile groups) and  $tr(P)$  is the trace of the  $N \times N$  transition matrix  $P$ ; For complete immobility,  $S=0$  and for perfect mobility,  $S=N/(N-1)$ .

This key aging role is highlighted by the age-BMI charts for white men. This is a more ambitious (non-parametric) analysis which shows, for high school or less and some college or more, the age path net of other factors, specifically two other important health behaviors -- smoking and getting regular exercise as a part of market work or from an exercise program. The specific question wording can be found in the [http://www.isr.umich.edu/src/psid/pdf\\_doc/psid86w19v1.pdf](http://www.isr.umich.edu/src/psid/pdf_doc/psid86w19v1.pdf) (question H23: “Do you get any regular exercise, such as doing hard physical work, or walking a mile without stopping, or playing an active sport?”) This clear acceleration of BMI in the age range of 30s to early 40s could create some upward drift in a simple repeated cross-section if the initial sample is populated by a large share in (or moving into) the BMI acceleration age range. We will return to this question of - in some sense – standardizing for age, in examining BMI and mortality (Section III).



### **B. Intergenerational Weight Pattern**

As discussed above, the scale for children’s BMI differs from that of adults and is age and gender specific. It is common to use an age and gender-specific percentile measure for obesity in children. A BMI value for children in the percentile of over 95 is defined as overweight and the range of 85-95 percent is defined as risk of overweight. Using these

measures for the PSID children, age 2-12 as of 1997, we created a matched sample of their parents and compared BMI values across the generations. The tabulation, Table 8, below shows the weight of parents (father) by BMI class of children for the full sample and for two subgroups, black and white and all other. The results show a generally positive association between the father's and child's weight. The relationship is more obvious for the full sample. For black and white separately, the relationship is not as clear, but this mostly reflects smaller sample size for these subgroups.

	Father's BMI	Children's BMI		
		normal	risk of overweight	oveweight
All family (N=1470)	normal	74.6	9.6	15.8
	overweight	66.1	14.8	19.2
	obese	52.7	17.7	29.6
Black family (N=408)	normal	61.9	8.5	29.7
	overweight	53.8	20.8	25.4
	obese	45.3	15.4	39.3
White family (N=1062)	normal	79.3	10.0	10.7
	overweight	70.1	12.8	17.1
	obese	56.9	19.0	24.2

A quite similar pattern holds for weight of the mother and the children (Table 9). The reasons for the association between the weight of parents and their children could be common diet and social environment or the link may be, in part, genetic. This is yet to be explored, but there is surely a generational link.

	Mother's BMI	Children's BMI		
		normal	risk of overweight	oveweight
All family (N=1908)	normal	70.9	11.8	17.3
	overweight	61.3	15.9	22.8
	obese	57.6	14.7	27.8
Black family N=798	normal	61.9	11.7	26.3
	overweight	60.7	14.6	24.7
	obese	54.3	14.5	31.3
White family N=1110	normal	74.1	11.8	14.1
	overweight	61.9	17.0	21.1
	obese	64.1	15.0	20.9

To extend the exploration of intergenerational relationships, the link between weight of the mothers and their own mothers was explored. This inquiry is possible because of the generational nature of the PSID study, a feature that supports a cross-sectional snapshot in which the older adults are the parents of the younger adults. Moreover, by linking the mother generationally forward to her own mother and then backward to her own young child, we are able to create a match between children age 2-12 and their grandmothers. An overview of these cross-generational patterns is presented in Table 10. An intriguing result is that there is not only a substantial relationship between the BMI of mothers and their children, but the relationship between the BMI of grandmothers and their grandchildren is about as strong.

	Grand Mother's BMI	Children's BMI		
		normal	risk of overweight	oveweight
All family (N=962)	normal	65.37	14.5	20.13
	overweight	58.69	16.6	24.71
	obese	55.19	14.11	30.71

### III. The Effects of BMI on Mortality

Here we examine BMI as of 1986 and mortality outcomes by 1999. Recent work (Hill, 2000) shows there is potentially great bias in mortality estimation unless non-response is considered. However, this may be less a problem in the PSID since mortality – even of non-respondents – is reported from relatives and from the 1994-1996 recontact initiative. What we have adopted is a bivariate probit model where the states are survival, death, and non-response<sup>4</sup>. The non-response from sample attrition from a separate selection equation which is to be estimated with the main mortality equation. As a practical matter the full model was not estimable, so various assumed values or value ranges in the attrition equation were specified as part of an estimation and sensitivity analysis. This exercise convinced us that the results on the predictors of mortality were not strongly sensitive to the problem of attrition from non-response relating to conditions – such as a serious health condition prior to mortality, which induces non-response before the death occurs.

The results from this analysis can be summarized as follows: The effects on mortality by 1999 from a select set of variables were estimated from categorical variables assessed in 1986. These were “whether smoked as of 1986”, “whether had vigorous physical exercise on a regular basis (from either work or recreational activities)”, a small number of BMI categories – For men: < 24.0, 24.0-26.9, 27.0-29.0, 30.0-32.9, and 33.0 and above; for women: <20.0, 20.0-22.9, 23.0-26.9, 27.0-30.9, and 31.0 and above – and a few demographic controls (male/female, white/black) used to define the same four samples as in Table 3. These results are presented in Tables 11 and 12. Model 1 estimates the effect on mortality using CDC-defined BMI categories. In this model, the reference category is ‘normal’ ( $20 \leq \text{BMI} \leq 24.9$ ). As expected, age and smoking are associated with increases in mortality, and exercise reduces mortality. Compared to the ‘normal’ category, there is a reduced probability of mortality among white men for all BMI ranges except ‘obese 1,’ (BMI between 30 and 34.9) although none of these coefficients is statistically significant. Among black men, compared to ‘normal,’ there is a lower probability of mortality at all categories of BMI, and particularly for the ‘very

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<sup>4</sup> Alternatively, competing hazard model



thin' category. The pattern of results is somewhat different for women. White women in the lowest BMI category have an elevated risk of mortality compared to white women in the normal category. None of the coefficients for black women are significant, presumably due to the small sample size in the gender disaggregated model. Yet, it is noteworthy that the direction of the coefficients in the model for black women is consistent with the results of black men – lower risk of death for all BMI categories, compared to 'normal.'

Our next step was to generate an estimate based on a quadratic model specification, Model 2 in Table 11 and 12. This model simply estimates the effect of 'BMI' and 'BMI-squared' on mortality (i.e., specifies a simple u-shape to the relation between mortality and BMI), along with the same covariates for smoking, age, exercise, and interaction terms as in Model 1. The result was that there was quite a higher minimum BMI mortality risk (BMRM) for black men and women (men=33.1, women=32.2) than for white men and women (men=28.3, women=27.2). Interestingly, these results for our nationally representative sample are similar to preliminary results derived using panel data (unadjusted for attrition) from the Health and Retirement Study (HRS) among much older individuals. These data showed a BMRM for blacks as 34 and for whites as 30 (Weir, unpublished note 2001). At least one other study has found that obesity may be protective compared with thinness or more normal weight for older community dwelling individuals (Grabowski & Ellis, 2001). As we will point out below, however, our small sample size precludes us from precisely pinpointing an 'ideal' BMI value; instead we attempt to specify a *range* of BMI values that are associated with lower mortality risk and to assess the extent to which mortality risk is elevated for BMI values outside this range.

To develop range values, we departed from the quadratic functional form and used gender-specific BMI definitions created from the results of the quadratic estimation reported above. The interval results in Model 3 show that for white males, being 'very thin' (BMI <24) is associated with higher rates of mortality, compared with a mid-level BMI (between 27 and 29.9). Rates of mortality also increase significantly at BMI levels over 33. For black men, being 'very thin' is also associated with higher rates of mortality, compared to that of mid-level BMI, a risk that is nearly double that of 'very

thin' white men. This risk continues to be significantly elevated among 'thin' (BMI between 24 and 26.9) black men, compared to those in the mid-level category. Thus, having a low BMI appears to be a more serious risk to mortality for black men than for white men.

Among women, the mortality risk of being 'very thin' compared to 'thin' (BMI between 20 and 22.9) appears particularly pronounced for *white* women, compared to black women. There is also an increase in mortality for white women in higher BMI categories, compared to the reference category of 'thin,' and especially among women in the highest obesity category (BMI  $\geq 31$ ). Among black women, higher levels of BMI are associated with *decreases* in mortality, but these results need to be replicated using larger sample sizes.

**Table 11. Effect of 1986 BMI on Probability of Mortality by 1999 Among Males, by Race**

Variables	White Males			Black Males		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
<b>Traditional Categories</b>	<u>b</u>	<u>b</u>	<u>b</u>	<u>b</u>	<u>b</u>	<u>b</u>
Underweight: (BMI < 20)	-.06			-.74*		
Normal: (20<BMI<24.9)	Reference			Reference:		
Overweight: (25<BMI<29.9)	-.10			-.38**		
Obese 1: (30<BMI<34.9)	.22			-.44*		
Obese 2: (BMI >=35)	-.30			-.07		
<b>Simple Categories</b>						
BMI		-.18			-.16	
BMI*BMI		.003			.002	
<b>Quadratic Defined Categories</b>						
Very Thin: (BMI<24)			.21*			.41**
Thin: (24<=BMI<=26.9)			.01			.29*
Middle: (27<=BMI<=29.9)			Reference			Reference
Overweight 1 (30<=BMI<=32.9)			.20			.02
Overweight 2 (BMI>=33)			.39*			.30
<b>Covariates</b>						
Smoke	.31**	.32**	.31**	.16	.13	.16
Exercise	-.19*	-.22*	-.18*	-.17	-.17	-.18
Smoke * Exercise	.05	.06	.05	.02	.04	.03
Exercise*Obese (BMI>=30)	.004	.20	.03	-.25	-.27	-.27
Age	.06***	.06***	.06***	.05***	.05***	.05***
Constant	-4.09	-1.86	-4.26	-3.08	-3.28	-3.45

**Table 12. Effect of 1986 BMI on Probability of Mortality by 1999 Among Females, by Race**

Variables	White Females			Black Females		
	Model 1	Model 2	Model 3	Model 1	Model 2	Model 3
<b>Traditional Categories</b>	<u>b</u>	<u>b</u>	<u>b</u>	<u>b</u>	<u>b</u>	<u>b</u>
Underweight: (BMI < 20)	.37***			-.74*		
Normal: (20<BMI<24.9)	Reference			Reference		
Overweight: (25<BMI<29.9)	.01			-.38**		
Obese 1: (30<BMI<34.9)	.02			-.44*		
Obese 2: (BMI >=35)	.32			-.07		
<b>Simple Categories</b>						
BMI		-.15**			-.16	
BMI*BMI		.003**			.002	
<b>Quadratic Defined Categories</b>						
Very Thin: (BMI<20)			.57***			.41**
Thin: (20<=BMI<=22.9)			Reference			.29*
Middle: (23<=BMI<=26.9)			.32**			Reference
Overweight 1 (27<=BMI<=30.9)			.21			.02
Overweight 2 (BMI>=31)			.48***			.30
<b>Covariates</b>						
Smoke	.44**	.42**	.44***	.14	.15	.13
Exercise	-.22*	-.17	-.24*	-.25*	-.22	-.26*
Smoke * Exercise	-.13	-.17	-.10	-.11	-.12	-.11
Exercise*Obese (BMI>=30)	-.35	-.37	-.47	-.12	-.19	-.11
Age	.06***	.05***	.06***	.04***	.04***	.04***
Constant	-3.93	-1.03	-4.24	-3.03	-1.96	-3.10

\*p&lt;.01.

Given our small sample sizes when we disaggregate by race and gender, we are unable to provide precise estimates of an ‘ideal’ BMI. The data suggest instead that there is a range of BMI values that are neither deleterious nor protective. This may be thought of as the ‘safe BMI’ range. The range values differ, especially between black and white sample members. Beyond this group-specific ‘safe BMI’ range, in both directions, risks of mortality increase. For a given ‘low’ BMI value, blacks have a risk of mortality that is substantially higher than for whites (i.e., being ‘underweight’ is a greater

risk factor for blacks). And whites seem to have mortality risks substantially higher than blacks at a common 'high' level of BMI.

Table 13 presents predicted mortality percentages based on the analyses presented in Tables 11 and 12. These results show a consistent race difference in the effect of BMI on mortality over time among males. For example, among white males aged 55, the lowest predicted mortality in 1999 is among those in 1986 BMI ranges between 24 and 30 and the highest mortality rates occur among the most obese. For black men, the reverse is true: the lowest predicted mortality in 1999 occurs among those in the 1986  $30 \leq \text{BMI} < 33$  category – currently defined as 'obese' by the CDC – while the highest mortality rates are among those who were the thinnest in 1986. This pattern is even more pronounced among black women in the most obese BMI categories in 1986 that have the lowest mortality rates by 1999. A similar pattern of high future mortality among the thinnest also holds for white women. These results are generally stable across age categories.

What is most intriguing and deserves more careful assessment connects to the pattern illustrated in our earlier Table 3. Over the period 1986-1999 a rather larger number of African Americans are now in the lower risk BMI category of 25-30, which should reduce their mortality hazards if our simple model has predictive power.

Table 13. Predicted Mortality (%) for Selected Age

AGE	MEAN				STD			
	25	35	45	55	25	35	45	55
<b>White men</b>								
BMI<24	0.5	2.2	7.6	19.8	0.2	0.6	1.8	4.1
24<=BMI<27	0.3	1.4	5.2	14.8	0.1	0.4	1.3	3.1
27<=BMI<30	0.3	1.3	5.0	14.5	0.1	0.4	1.3	3.1
30<=BMI<33	0.5	2.2	7.6	19.8	0.2	0.9	2.3	4.9
BMI>=33	0.8	3.5	10.9	25.8	0.4	1.5	3.9	7.5
<b>Black men</b>								
BMI<24	3.7	9.8	21.2	38.0	4.0	8.1	12.7	15.6
24<=BMI<27	2.9	8.0	18.0	33.8	3.3	7.0	11.6	15.0
27<=BMI<30	1.4	4.4	11.3	23.8	1.7	4.2	8.1	12.1
30<=BMI<33	1.1	3.7	9.8	21.3	1.5	3.8	7.6	12.0
BMI>=33	2.4	6.8	16.0	30.9	3.0	6.6	11.6	15.8
<b>White women</b>								
BMI<20	1.5	5.7	15.9	33.9	1.2	3.2	6.1	8.2
20<=BMI<23	0.3	1.6	5.8	16.2	0.3	1.1	2.9	5.3
23<=BMI<27	0.8	3.3	10.6	25.3	0.7	2.0	4.2	6.3
27<=BMI<31	0.5	2.4	8.3	21.2	0.5	1.5	3.5	5.8
BMI>=31	0.9	3.7	11.5	26.9	0.7	2.2	4.6	7.0
<b>Black women</b>								
BMI<20	2.7	6.9	14.9	27.4	2.8	5.4	8.6	11.3
20<=BMI<23	2.3	6.1	13.5	25.3	2.4	4.6	7.5	10.1
23<=BMI<27	2.8	7.2	15.3	28.0	2.6	5.0	7.7	9.8
27<=BMI<31	2.0	5.3	12.0	23.2	2.0	4.1	6.9	9.3
BMI>=31	1.6	4.4	10.3	20.4	1.6	3.4	5.9	8.3

predicted mortality is evaluated for a non-smoker with no exercise.

#### IV. Conclusion

Repeated measures of BMI are a recent addition to the Panel Study of Income Dynamics. In this paper we utilized data from the PSID Data Center (<http://stat0.isr.umich.edu/psid/data-center/data-center.html>) from 1986 and 1999 files to explore the main features of BMI in the U.S. adult population. Using samples of adults as of 1986 and 1999 we found the following:

1. There appears to be a BMI drift through time, not an epidemic.
2. This drift appears pervasive across all age and gender groups and applies to African-American as well as others.
3. This drift appears not the result of changes in the age distribution or levels of smoking and regular exercise.
4. Children are not exempt from this obesity drift.
5. There is substantial mobility across BMI deciles through time. It is on the order of mobility for economic variables such as household wealth.

6. Children's BMI and that of their parents are quite highly correlated. Further, the BMI of grandmother's and their grandchildren is also high.
7. BMI is a risk factor in mortality, but other measures indicating health behaviors smoking and regular exercise, play a role. Regular exercise appears to offset the disadvantage of being overweight, but the measure of the estimated effect is low. Of course, our results show that both being in 'normal' weight ranges in combination with exercise is the best policy.
8. The range of BMI values that have lower mortality risks for African-American men and women are quite wide. There seems to be more a 'safe BMI' range rather than an ideal as might be suggested by a single quadratic relationship. The range of values for white men and women appear also quite wide, but with lower values for the range endpoints. Being 'underweight' by the definitions developed for whites appears to be a far greater mortality risk for African-American men and women.

These are results from our research. Various colleagues have offered valuable comments. It is prudent to regard our findings as provisional until our work receives careful peer reviews and replication in other studies.

## REFERENCES

Calle, E., Thun, M., Petrelli, J., Rodriguez, C., and Heath, C., 1999, "Body-Mass Index and Mortality in a Prospective Cohort of U.S. Adults", The New England Journal of Medicine. Vol. 341, No. 15, pp. 1097-1105.

Child Development Supplement (home page): <http://www.isr.umich.edu/src/child-development/home>

Hill, Daniel

Hurst, E., Luoh, M., and Stafford, F., 1999, "The Wealth Dynamics of American Families, 1984-94", Brookings Papers on Economic Activity. Vol. 1, pp. 267-337.

Lantz, Paula, M., James S. House, James M. Lepkowski, David R. Williams, Richard P. Mero, J. Chen, 1998, "Socioeconomic Factors, Health Behaviors, and Mortality," Journal of the American Medical Association, Vol. 270, No. 21, pp. 1703-1708.

Panel Study of Income Dynamics (home page) <http://www.isr.umich.edu/src/psid>.

Shorrocks, A., 1978, "The Measurement of Mobility", Econometrica. Vol. 46, No.5, pp. 103-124.

U.S. Department of Health and Human Services, June 1998. "Clinical Guidelines on the Identification, Evaluation, and Treatment of Overweight and Obesity in Adults: The Evidence Report." Washington, D.C.: U.S. Government Printing Office.

U.S. Department of Health and Human Services, November, 2000. "Healthy People 2010: Understanding and Improving Health," 2<sup>nd</sup> ed., Washington, D.C.: U.S. Government Printing Office.