11-5-1992

Long Term Mobidity and Mortality of Adolescent Obesity: A Follow-up of Third Harvard Growth Study Participants of 1922 to 1935

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LONG-TERM MORBIDITY AND MORTALITY OF OVERWEIGHT ADOLESCENTS

A Follow-up of the Harvard Growth Study of 1922 to 1935

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Abstract *Background.* Overweight in adults is associated with increased morbidity and mortality. In contrast, the long-term effect of overweight in adolescence on morbidity and mortality is not known.

Methods. We studied the relation between overweight and morbidity and mortality in 508 lean or overweight adolescents 13 to 18 years old who participated in the Harvard Growth Study of 1922 to 1935. Overweight adolescents were defined as those with a body-mass index that on two occasions was greater than the 75th percentile in subjects of the same age and sex in a large national survey. Lean adolescents were defined as those with a body-mass index between the 25th and 50th percentiles. Subjects who were still alive were interviewed in 1988 to obtain information about their medical history, weight, functional capacity, and other risk factors. For those who had died, information on the cause of death was obtained from death certificates.

Results. Overweight in adolescent subjects was associated with an increased risk of mortality from all causes and disease-specific mortality among men, but not among women. The relative risks among men were 1.8 (95 percent confidence interval, 1.2 to 2.7; P = 0.004) for mortality from all causes and 2.3 (95 percent confidence interval, 1.4 to 4.1; P = 0.002) for mortality from coronary heart disease. The risk of morbidity from coronary heart disease and atherosclerosis was increased among men and women who had been overweight in adolescence. The risk of colorectal cancer and gout was increased among men and the risk of arthritis was increased among women who had been overweight in adolescence. Overweight in adolescence was a more powerful predictor of these risks than overweight in adulthood.

Conclusions. Overweight in adolescence predicted a broad range of adverse health effects that were independent of adult weight after 55 years of follow-up. (N Engl J Med 1992;327:1350-5.)

OVERWEIGHT in adults is associated with cardiovascular disease, hypertension, gallbladder disease, diabetes mellitus, atherosclerosis, gout, arthritis, and certain cancers.[1,2] In adolescents, overweight is less closely related to the later appearance of many of the same conditions. Some cardiovascular risk factors, however, may become established in childhood,[3] among them serum total cholesterol and lipoprotein subfraction concentrations and blood pressure.[4,5]

The long-term risks that arise from overweight in adolescents have been assessed mostly in studies of special populations or with short follow-up periods.[6-10] Although the studies generally suggested a long-term effect of overweight in adolescence, the age at which overweight confers excess risk and the magnitude of its lifelong effects are not known. Access to early results from the Harvard Growth Study of 1922 to 1935 provided a unique opportunity to study an elderly population for which detailed data on growth were available. From the growth records, we identified subjects who had been consistently lean or overweight in adolescence and then assessed their lifetime morbidity and mortality. We present here the results of a 55-year follow-up of the participants in the growth study.

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Supported in part by a contract (53-3K06-5-10) with the Agricultural Research Service of the U.S. Department of Agriculture. This article does not necessarily reflect the views or policies of the U.S. Department of Agriculture.

METHODS

Study Subjects and Design

The Third Harvard Growth Study, conducted from 1922 to 1935 by the Harvard School of Education, included measurements of height and weight in more than 3000 schoolchildren. The study subjects were first- and second-grade public-school children from three middle-class cities north of Boston who were enrolled in 1922 and 1923. The subjects were measured annually until they graduated from or left high school. Of these, 1857 schoolchildren were studied annually for a minimum of eight years. The studies included triplicate annual measurements of height and weight at the same time each year and detailed anthropometric and psychometric measurements, as previously described.[11]

In 1988 we contacted equal numbers of subjects whom we had designated as overweight or lean during adolescence. The overweight group consisted of all subjects with a body-mass index (defined as the weight in kilograms divided by the square of the height in meters) for any two years between 13 and 18 years of age that was greater than the 75th percentile in subjects of the same age and sex from the First National Health and Nutrition Examination Survey of 1971 to 1974.[12] The lean group was a random sample of subjects whose body-mass index remained between the 25th and 50th percentiles when they were between 13 and 18 years old. We did not include subjects with a body-mass index below the 25th percentile because they might have been ill. The 19 nonwhite subjects were excluded because they represented less than 1 percent of the growth-study group. We used the 75th rather than a higher percentile to define overweight in order to include sufficient subjects to provide adequate power. This percentile corresponds to the upper bound of suggested weights for young adults.[13] The age-specific values for the 75th percentile of the body-mass index from 13 to 18 years of age were 22 to 25 for male subjects and 22 to 24 for female subjects. Eighty-four percent of the overweight group had a body-mass index that was above the 85th percentile at least once during high school; 26 percent exceeded the 95th percentile at least once during the same period.

In 1968, 45 percent of the original growth-study cohort (mean age, 53 years) was contacted by mail for a mid-life follow-up study.[14] A total of 309 subjects who responded to the mailed questionnaire in 1968 were included in our follow-up study. We used their re-

sponses to questions about smoking and adult weight and height to estimate the effect of these factors on the relation between overweight in adolescence and subsequent mortality.

In 1988 we attempted to locate the subjects selected for this study according to the criteria for body-mass index described above through the use of the 1968 follow-up information, high-school–reunion committees, polling books, postal services, state offices of vital statistics, and credit bureaus. For subjects deemed lost to follow-up, these resources also helped to establish the last date on which the subject was known to be alive.

Subjects who resided in Massachusetts or southern New Hampshire (66 percent) were interviewed in their homes by one of the authors; telephone interviews were conducted with the other subjects. The structured interview included medical, reproductive, smoking, weight, and exercise histories. Functional capacity was assessed with a questionnaire on activities of daily living.[15,16] The subjects were asked whether they had ever been told by a doctor that they had coronary heart disease, angina, diabetes mellitus, atherosclerosis (site unspecified), stroke, cancer, hip fracture, arthritis, or any other condition; their age at diagnosis; and their current use of medications. Height and weight were measured at all the face-to-face interviews, and blood pressure was measured twice with a digital blood-pressure meter (Tycos Instruments, Arden, N.C.) and averaged. Information on height, weight, and blood pressure was reported by the subjects in the case of telephone interviews.

Death certificates for deceased subjects were obtained from the National Death Index,[17] state offices of vital statistics, and military records. Causes of death were coded in accordance with the *International Classification of Diseases, 9th Revision, Clinical Modification* (ICD-9-CM)[18] and reviewed by a physician. The study protocol was approved by the Human Investigations Review Committee of the New England Medical Center.

Statistical Analysis

The relative risks of mortality and morbidity according to weight in adolescence (overweight relative to lean), unadjusted and adjusted for potential confounders, were calculated by proportional-hazards analysis (with the BMDP 2L software program[19]). For the subgroup of subjects interviewed in 1968, we adjusted for body-mass index and smoking status at the age of 53 years to assess the effect of adult body-mass index and smoking on the association between mortality and weight in adolescence. For subjects alive at the 1988 follow-up, the additional covariates of past exercise levels and the presence of arthritis were included. We evaluated the effect of weight in adolescence on current functional capacity by logistic-regression analysis. These and all other analyses were performed with SAS version 6.06.[20] All t-tests were two-tailed.

Results

The frequency of follow-up did not differ significantly according to weight in adolescence (Table 1). Thirty-six percent of the subjects were interviewed, 32 percent were dead, and 16 percent declined or were unable to respond. Vital status could thus be determined for 84 percent of the cohort (425 subjects). The mean (± SD) age of the surviving subjects was 73±1 years. Fifty-two percent of the surviving subjects who had been overweight in adolescence were still overweight in 1988.

Mortality

The crude relative risks of mortality from all causes and cause-specific mortality according to weight in adolescence are shown in Table 2. For men, the relative risks of death from all causes and death from coronary heart disease were approximately two times higher among those who had been overweight in adolescence than among those in the lean group. The results were similar when a more restrictive definition of mortality from coronary heart disease that included only myocardial infarction, angina pectoris, and coronary artery disease (ICD-9-CM codes 410 to 414) was used. The relative risks of mortality from atherosclerotic cerebrovascular disease and colon cancer were also higher (13.2 and 9.1, respectively), but the number of cases was small. For men, the survival curves for mortality from all causes and from coronary heart disease (Fig. 1) reveal poorer survival in the overweight group starting at about 45 years of age. We found no increase in the relative risk of death from all causes or cause-specific death among women according to weight category in adolescence.

For the subgroup of 309 subjects for whom mid-life follow-up data were available, we adjusted for the influence of adult body-mass index on the relative risks associated with weight in adolescence by multi-

Table 1. Characteristics of the Subjects in the Harvard Growth Study of 1922 to 1935 Who Were Studied in 1988.

Variable	Lean as Adolescents	Overweight as Adolescents	Total
	*number (percent)**		
Interviewed	96 (36)	85 (36)	181 (36)
Men	49	32	81
Women	47	53	100
Deceased	81 (30)	80 (34)	161 (32)
Men	48	45	93
Women	33	35	68
Declined or unable to respond	50 (19)	33 (14)	83 (16)
Lost to follow-up	43 (16)	40 (17)	83 (16)
Total	270	238	508
Men	156	100	256
Women	114	138	252

*Percentages do not all sum to 100 because of rounding.

Table 2. Relative Risk of Mortality Associated with Overweight in Adolescence.*

Cause of Death	Men (N = 256)		Women (N = 252)	
	No. of Deaths	Relative Risk (95% CI)	No. of Deaths	Relative Risk (95% CI)
All causes	93	1.8 (1.2–2.7)†	68	1.0 (0.6–1.6)
Coronary heart disease‡	51	2.3 (1.4–4.1)§	19	0.8 (0.3–2.1)
Atherosclerotic cerebrovascular disease¶	8	13.2 (1.6–108.0)§	7	0.4 (0.1–1.8)
Colorectal cancer‖	6	9.1 (1.1–77.5)**	4	1.0 (0.1–7.0)
Breast cancer ††	0	—	8	0.9 (0.2–3.8)

*The relative risks are for the overweight group as compared with the lean group. CI denotes confidence interval.

†P = 0.004 for the comparison between the overweight and lean groups.

‡ICD-9-CM codes 402, 410 through 414, 428, 429, and 440.

§P = 0.002 for the comparison between the overweight and lean groups.

¶ICD-9-CM codes 431 through 437.

‖ICD-9-CM codes 153 and 154.

**P = 0.01 for the comparison between the overweight and lean groups.

††ICD-9-CM code 174.

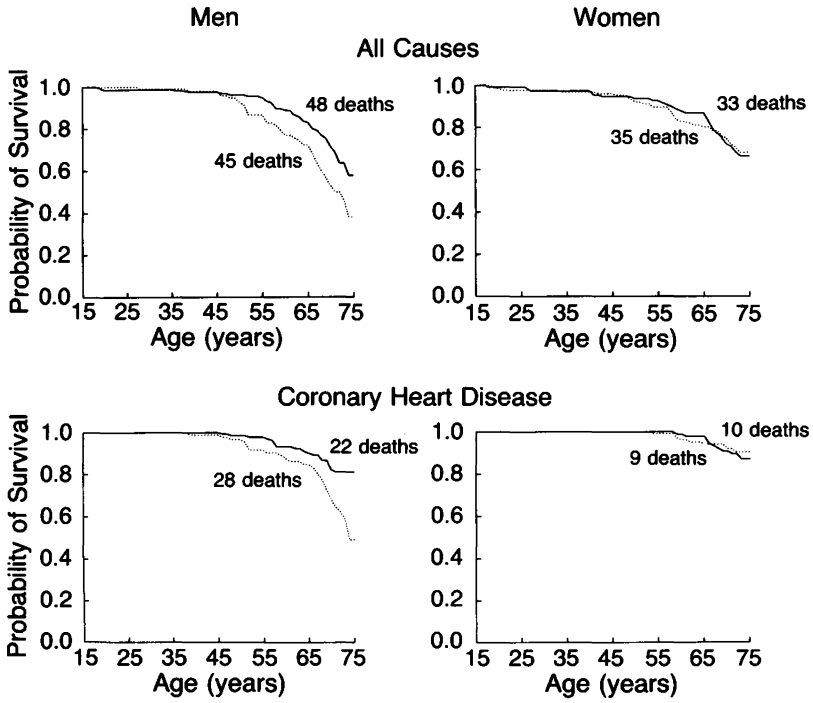

Figure 1. Mortality from All Causes and Mortality from Coronary Heart Disease According to Weight in Adolescence.
The solid line represents the lean group, and the broken line the overweight group.

variate regression analysis. Among the women, neither the unadjusted nor the adjusted relative risks were significantly different from 1. Among the men, the addition of adult body-mass index as a variable slightly decreased the relative risk of death from all causes from 2.9 (95 percent confidence interval, 1.5 to 5.8) to 2.4 (95 percent confidence interval, 1.1 to 5.0) and the relative risk of death from coronary heart disease from 2.6 (95 percent confidence interval, 1.1 to 6.6) to 1.9 (95 percent confidence interval, 0.7 to 5.2). Because there were no deaths in some subgroups, we could not make a comparable analysis of the effect of adult body-mass index on the risk of death from atherosclerotic cerebrovascular disease or colorectal cancer. The addition of smoking status to the regression model did not significantly change the estimated relative risks.

Morbidity

The crude and adjusted relative risks associated with weight in adolescence, as estimated by proportional-hazards analysis for selected diseases, are shown in Table 3. For coronary heart disease, atherosclerosis, colorectal cancer (in men), gout (in men), and arthritis (in women), the addition of adult body-mass index as a variable only slightly attenuated the risk associated with overweight in adolescence. In contrast, for non-insulin-dependent diabetes mellitus, all of the effect of weight in adolescence was accounted for by overweight in adulthood. The inclusion of smoking status and exercise history did not significantly change the relative risks. In addition, all six hip fractures reported by the subjects interviewed at the 1988 follow-up occurred in the group that was overweight in adolescence ($P = 0.01$ by Fisher's exact test).

After exclusion of the subjects who were taking antihypertensive medications (35 percent), systolic and diastolic blood pressure was normal in both weight groups and not significantly different between them. The prevalence of the use of antihypertensive medications was similar in the two groups (32 percent in the lean group and 39 percent in the overweight group). Stratification according to measured or reported blood pressure did not significantly alter the results.

Functional Capacity

Women who were overweight in adolescence were eight times more likely to report difficulty with personal care and routine needs in the activities of daily living than women who were lean in adolescence (Table 4). These risks decreased slightly after adjustment for arthritis. The specific tasks for which an elevated risk of difficulty was reported among women who were overweight in adolescence were walking ¼ mile (400 m), climbing stairs, and lifting. No increase in risk was found among the men who were overweight in adolescence as compared with the men who were lean.

Discussion

After 55 years of follow-up, mortality from all causes and from coronary heart disease, stroke, and colorectal cancer was greater among men who were overweight in adolescence than among those who were lean. Although this study involved only 508 persons, the lengthy follow-up period amassed 24,913 person-years of experience. Overweight in adolescence increased the risk of morbidity for several conditions in men, women, or both, and it compromised functional capacity in women. The increased risk was independent of adult body-mass index for all morbidity and mortality outcomes except morbidity from diabetes.

Bias due to loss to follow-up or misclassification of overweight or outcome could have distorted our results. Although vital status was determined for 84 percent of the cohort and although the rates of refusal to participate were comparable in the two weight groups, the possibility of bias due to loss to follow-up cannot

be entirely excluded. The close correlation between body-mass index and body fat as determined by densitometry[21] suggests that body-mass index represents a reasonable measure of fatness in adults. Use of the National Death Index for matching is highly sensitive and reliable.[22] We assessed mortality from all causes as well as cause-specific mortality because of potential misclassification in coding death certificates.[23]

Mortality from coronary heart disease accounted for most of the excess risk of death associated with overweight. The adverse effect of adult overweight on mortality from all causes and mortality from coronary heart disease and cerebrovascular disease has been previously demonstrated.[2,24,25] The few studies that have linked overweight in adolescence or young adulthood with adult health outcomes were based on shorter follow-up periods and were largely limited to young men 18 years old or older. Nonetheless, the results of those studies were similar to ours. Mortality from all causes and from coronary heart disease was increased among Dutch men with a body-mass index of more than 25,[7] Danish men with a body-mass index of more than 31,[10] college alumni with increased weight for height,[26] and hospitalized overweight children.[6] In a 10-year follow-up of 1.7 million Norwegians,[27] mortality among 15-to-19-year-old women did not vary with base-line body-mass index, but it was increased among 20-to-24-year-old women with a body-mass index of more than 23 and among 15-to-19-year-old men and 20-to-24-year-old men with a body-mass index of more than 27 (Waaler HT: personal communication).

The effect of overweight in adolescence on adult morbidity and mortality may reflect the central deposition of fat that occurs in adolescence.[28,29] Increased central-body fat affects a variety of cardiovascular risk factors, including blood pressure and lipoprotein profiles,[30,31] that cluster with overweight.[3] These risk factors are related to precursor atherosclerotic plaques in the arteries of children,[32-34] track from adolescence into adulthood,[4] and improve with weight reduction.[35]

The rate of death from colorectal cancer was increased among men who were overweight in adolescence, as was the risk of morbidity from colorectal cancer. A relative risk of 1.5 was found in the American Cancer Society's prospective study among men (but not women) who weighed more than 130 percent of ideal weight.[2] Earlier studies have identified increased height, body-mass index, adult weight gain, energy intake, and intake of animal fat as predictors of colorectal cancer.[36]

The incidence of heart disease, atherosclerosis, gout, colorectal cancer, hip fracture, and arthritis was elevated among the subjects in

Table 3. Crude and Adjusted Relative Risks of Morbidity at the Age of 73 in 181 Subjects Interviewed in 1988, According to Weight in Adolescence.*

Condition	No. of Cases	Crude Relative Risk (95% CI)	Adjusted Relative Risk† (95% CI)
Coronary heart disease			
Both sexes	38	2.1 (1.1–4.0)‡	1.8 (0.9–3.9)§
Men	19	2.8 (1.1–7.2)¶	2.5 (0.9–7.1)§
Women	19	1.6 (0.6–4.1)	1.4 (0.5–4.0)
Angina			
Both sexes	22	1.7 (0.7–4.0)	1.5 (0.6–4.1)
Men	16	1.6 (0.6–4.2)	1.3 (0.4–3.9)
Women	6	5.1 (0.6–43.4)	3.7 (0.4–37.4)
Diabetes mellitus			
Both sexes	32	1.8 (0.9–3.7)§	1.0 (0.5–2.3)
Men	16	1.6 (0.6–4.2)	0.9 (0.3–2.6)
Women	16	2.2 (0.8–6.4)	1.2 (0.3–4.3)
Atherosclerosis			
Both sexes	7	7.7 (0.9–63.7)§	7.3 (0.8–68.3)§
Men	4	5.0 (0.5–48.4)	3.4 (0.3–39.2)
Women	3	—‖	—‖
Stroke			
Both sexes	11	0.7 (0.2–2.4)	1.1 (0.3–4.5)
Men	7	0.6 (0.1–3.3)	0.8 (0.1–5.3)
Women	4	1.0 (0.1–7.1)	2.0 (0.1–28.9)
Colorectal cancer			
Both sexes	5	—**	—**
Men	5	6.5 (0.7–57.9)§	5.6 (0.6–57.5)
Women	0	—**	—**
Hip fracture			
Both sexes	6	—‖††	—‖
Men	3	—§‖	—‖
Women	3	—‖	—‖
Arthritis			
Both sexes	68	1.6 (1.0–2.5)§	1.2 (0.7–2.0)
Men	24	0.9 (0.4–2.0)	0.7 (0.3–1.7)
Women	44	2.0 (1.1–3.7)‡‡	1.6 (0.8–3.2)
Gout			
Both sexes	16	2.7 (0.9–7.7)§	2.7 (0.9–8.4)§
Men	14	3.1 (1.1–9.3)§§	2.2 (0.7–6.9)
Women	2	—‖	—‖

*The relative risks are for the overweight group as compared with the lean group. CI denotes confidence interval.

†All models were adjusted for adult body-mass index; the "both sexes" models were also adjusted for sex.

‡P = 0.029 for the comparison between the overweight and lean groups.

§P<0.10 but >0.05 for the comparison between the overweight and lean groups.

¶P = 0.022 for the comparison between the overweight and lean groups.

‖The relative risk is infinite; statistical significance was assessed by Fisher's exact test.

**Men accounted for all cases.

††P = 0.01 for the comparison between the overweight and lean groups.

‡‡P = 0.025 for the comparison between the overweight and lean groups.

§§P = 0.031 for the comparison between the overweight and lean groups.

Table 4. Crude and Adjusted Relative Risks of Difficulty with Activities of Daily Living in 181 Subjects Interviewed in 1988, According to Weight in Adolescence.*

Variable	Men (N = 81)		Women (N = 100)	
	Personal Care	Routine Needs	Personal Care	Routine Needs
	relative risk (95% confidence interval)			
Crude risk	3.2 (0.3–36.8)	5.0 (0.5–50.0)	8.2 (1.0–68.1)†	8.1 (1.7–37.8)‡
Risk adjusted for adult body-mass index	1.3 (0.1–18.4)	4.5 (0.4–52.8)	—§	6.1 (1.2–31.9)¶
Risk adjusted for arthritis	3.6 (0.3–43.1)	5.3 (0.5–54.5)	6.6 (0.8–56.3)	6.5 (1.3–31.1)‖

*The relative risks of difficulty are for the overweight group as compared with the lean group.

†P = 0.05 for the comparison between the overweight and lean groups.

‡P = 0.008 for the comparison between the overweight and lean groups.

§The relative risk could not be estimated because the numbers of subjects were too small.

¶P = 0.032 for the comparison between the overweight and lean groups.

‖P = 0.020 for the comparison between the overweight and lean groups.

our study who were overweight in adolescence and who survived to their early 70s, a finding consistent with previous studies of adult overweight.[1,2,24] The increased relative risks were independent of adult body-mass index for heart disease and atherosclerosis, but not for diabetes. Adult overweight is a strong risk factor for non-insulin-dependent diabetes,[37] and diabetes improves with weight loss.[38] In a large, prospective study, adult overweight, but not overweight at the age of 18, predicted the incidence of diabetes in women.[39]

Among women, we found a 1.6-fold increase in arthritis associated with being overweight during adolescence that was independent of adult body-mass index. In cross-sectional studies, overweight was associated with arthritis at specific sites.[40,41] Our results confirm and extend the findings of the 35-year follow-up of the Framingham Heart Study, in which adjustment for serum cholesterol and uric acid levels, diabetes, blood pressure, and estrogen use did not diminish the association between overweight and the subsequent development of osteoarthritis of the knees.[42]

Women who were overweight in adolescence were at increased risk of reported difficulty in the activities of daily living. Difficulty with walking and stair climbing suggests that weight-bearing activity was compromised; only some of the difficulty appeared to be attributable to arthritis. Asymptomatic arthritis or differential reporting could have biased these results or accounted for the lack of findings in men. Elderly women are more likely than elderly men to report functional limitations and seek help.[43] More objective measures of functional capacity are needed to separate real disability from self-perceived limitations.

A number of adverse health effects that occur in adulthood are associated with being overweight during adolescence. Furthermore, except for diabetes, the risks appear to be independent of later overweight. On the basis of the criteria used to define overweight in this study, approximately 25 percent of U.S. adolescents are at risk for adult consequences of increased body-mass index. Because body-mass index appears to be programmed early in life, the prevention of overweight in childhood and adolescence may be the most effective means of decreasing the associated mortality and morbidity in adults.

We are indebted to Dr. Francis E. Johnston and Dr. Eugenie Scott for sharing the original Harvard Growth Study materials and 1968 follow-up data, to Dr. Richard Frankel for bringing the existence of these data to our attention, and to the subjects of the Harvard Growth Study, without whose generous cooperation the work could not have been done.

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