

THE FALSE PREMISES OF THE OBESITY CRUSADE

Introduction. The crusade on overweight and obesity now expanding worldwide is poised to change drastically those traditional ways of life that in most developed countries – countries that nevertheless have witnessed a near doubling of life expectancy in little more than a century, parallel to a steady increase in average body weight. Common sense would expect that epidemiologic studies of obesity and overweight are based not on nebulous guesses but on actual measurements with sufficient and testable precision, on measurements that have measured what is said to have been measured and not something else, and on conclusions strong enough to make a difference and repeatedly consistent. In reality, these requirements are never met in epidemiologic studies of overweight and obesity.

The majority of those studies do not actually measure overweight or obesity but rely on what people report offhand about their own height and weight during telephone interviews or by responding to written questions. Even when height and weight may be measured, any excess of body mass over an arbitrary threshold of normalcy is assumed to be fat when in reality it could be muscle, water, or heavy bones. If an overweight person dies of lung cancer it is not possible to say that overweight was the cause of death without asking if the person was a smoker, exposed to asbestos or radiation, or to scores of other possible hazards for lung cancer. Still, no study has adequately inquired about the so many possible causes of death and disease to which study participants could have experienced. Ethnic, behavioral, occupational, environmental, physical fitness, and many more differences could easily bias the results and were never adequately addressed by epidemiologic studies. Thus, as could be expected, studies often come to divergent conclusions.

Is it truly an epidemic? In reality, reports indicate that while a large majority of people may have gained very few pounds – an apparently healthy pounds - over the last decades, it is only a small fraction of obesity-prone subjects who have registered excessive weight increases. The same conclusions hold for children. This means that by far the majority of people have been impervious to obesity and self-regulate their weight remarkably well. In fact, more recent information indicates that the trend of small weight gain for the majority of people may be coming to an end, reflecting observations that the average caloric intake may have been stationary or decreasing.

The claim of an obesity epidemic rests on statistical equivocations, and most of the problem lays in how overweight and obesity are measured and analyzed. The common method is a combination of weight and height measures called the body mass index (BMI), for which an arbitrary normalcy standard of BMI 25.0 has been set. Most studies assess average BMI values for segments of the BMI range of values, so that the average for a group of people in the segment of BMI 25.0 to 30.0 will be skewed upward by the presence of truly obese subjects at the high end, even though people at the low end of the segment might be quite normal. Clearly, what needs to

be done is to analyze segments of the BMI range that are homogeneous for the characteristics that count, namely mortality or disease rates. However, to achieve a realistic segmentation of the BMI range it will be necessary first to define normal BMI ranges for different groups of subjects, depending on age, sex, ethnicity, prevalent diseases, and many other characteristics: a daunting task whose incompleteness casts heavy uncertainties on claims about overweight, obesity, and their possible consequences.

Even when normalcy ranges were to be known, the task of counseling dangerously thin or obese individuals would still be daunting, after considering the problematic record of bariatric medicine and surgery, and the pitiable histories of self-correcting attempts by obese and overweight people. The literature is replete with reports of the dangers of losing weight even under supervised regimens.

What is an intelligent person to conclude? A clear impression is left that epidemiology is truly powerless in this case, given the barrage of heavyweight endorsements from all sort of professed authorities. For overweight and obesity the risk differentials over the middle range of BMI values are usually less than 1 and could be easily trumped by questionable measurements and many uncontrolled biases and confounders. It is only for very thin and definitely obese people that higher risks are recorded and raise legitimate concern.

Lest these remarks – and other to follow - be misconstrued, the analysis in the following pages should bring confirmation to anyone willing to look at the evidence with an open mind and eyes. It will not be an analysis in the technical sense – which would be useless for the purpose at hand – but a highlight of the uncertainties and inconsistencies that affect the studies. Fortunately, the construct of epidemiology is not rocket science and is fully accessible to any person of average education, most epidemiologists included.

The flawed evidence is not scientifically justified. The current interest on body weight is specifically directed at body fat. The visible clue of overweight and obesity is the extent of swelling of a given body's configurations, but it is important to note that such swelling is not always or exclusively attributable to an accumulation of fat, but is also the result of unrelated effects of muscle and organ swelling, and of the conditions of hydration – or water-logging – of both muscle and other organs. So it is, for instance, that body builders may have little fat in their oversized bodies, while other individuals may be swollen by excessive water that accumulates on account of disease or excessive water intake. With this in mind, it is apparent that a direct and precise measurement of the amount of body fat in a given living subject is beyond the pale, unless at autopsy.

The method of choice for clinical and epidemiologic applications has been the Quetelet Index or Body Mass Index (BMI), defined as the measure of the weight of an individual in kilograms divided by the square of the height in meters (kg/m^2). As such, BMI is a representation of body swelling, but it is clearly inadequate as a measure of body fat because it cannot account for the composition of a body's swelling. BMI also does not account for body frame, for instance the differences

between men and generally shorter women, nor is it a good index for children, being strictly age and body frame dependent. ¹ BMI also does not account whether body fat might be well distributed in the body or located at the waist, the latter condition being more linked to health risks. ²

Still, because of its easy applicability in epidemiologic surveys, the BMI has been and still is universally used in defining underweight, overweight, and obesity, despite its manifest shortcomings and ambiguities. **It follows that epidemiologic studies based on BMI are assuredly wrong in assuming that recorded BMI values are factual representations of body fat, and therefore in claiming that BMI values are solely and proportionally linked to food intake excesses and to dietary composition.** In effect, and counter to all evidence, we are asked to accept BMI assessments at face value in the numerous studies that have attempted to link BMI with mortality and diseases, and to abide by the official public health linkage of BMI with eating disorders and their fanciful and still vague remedies.

Health, diseases, and the BMI classifications. Detailed surveys show that the vast majority of people have gained small increments of weight over the last decades, whereas relatively few heavy obese are the ones showing the excessive gains.³ Official public health pronouncements speak of four segments within the range of BMI values in open populations, namely low weight, normal weight, overweight, and obese individuals. The official BMI threshold of normal weight has been scaled down in the last decade from 29 to 25, below which the health risks associated with being thin are said to progressively increase, and above which the health risks associated first with overweight up to a BMI of 30, and then with obesity upward of BMI 30 are said to progressively increase.

As a first consideration, it should be clear that declaring a specific cut-off point as the threshold for normal weight is wholly arbitrary, given the uncertain meaning of BMI in relation to obesity, and the manifest variability of individuals in relation to normalcy. Furthermore, the definition of wide segment of the BMI range that inclusive of normal weight, overweight, and obese people is also arbitrary because it ignores the natural overlapping among those segments. Thus defining as overweight those with BMI 25 to 30 and obese those from 30 and up, shortchanges the people at the high end of the segments and overloads those at the beginning.

Most illustrative of the vast uncertainties and of the arbitrariness of BMI

¹ Siervogel RM, Roche AF, Guo S, Mukherje ED, Chumlea WC. Patterns of change in weight/stature² from 2 to 18 years: findings from long-term serial data for children in the Fels Longitudinal Growth Study. *Int J Obes.* 1991;15:479-485.

² Berg C, Rosengren A, Aires N, Lappas G, Toren K, Thelle D, et al. Trends in overweight and obesity from 1985 to 2002 in Goteborg, West Sweden. *Int J Obes Relat Metab Disord* 2005;29(8):916-24.

³ [Freedman DS](#), [Khan LK](#), [Serdula MK](#), [Galuska DA](#), [Dietz WH](#). Trends and correlates of class 3 obesity in the United States from 1990 through 2000. [JAMA](#). 2002 Oct 9;288(14):1758-61.

classifications is a study that made USA and international news in March 2004.⁴ The report comes from the Centers for Disease Control and Prevention (CDC), an agency of the USA Department of Health and Human Services. For an added measure of impact the report includes the director of the CDC agency among its authors, and was published by the Journal of the American Medical Association (JAMA), a most prestigious medical and public health weekly. Largely based on four consecutive National Health and Nutrition Examination Surveys (NHANES) by the same CDC, the report asserts that overweight and obesity in the USA cause upward of 414,000 annual deaths, based on a selective review of past epidemiologic studies. Predictably, this CDC report made for an extensive and alarming media coverage, even though it exceeds by 134,000 annual deaths the assessment of a previous report, also published in JAMA in 1999. (Allison et al., 1999).

Astonishingly, that report was contradicted a few months later by a second report also from the CDC agency, and also published in JAMA.⁵ This second official study from the same government agency (do they talk to each other?) severely criticized the previous one because it *“used adjusted relative risks in an attributable fraction formula appropriate only for unadjusted relative risks, and thus only partially adjusted for confounding factors, did not account for variations by age in the relation of body weight to mortality, and did not include measures of uncertainty in the form of SEs or confidence intervals (Cis). [It] used data from a variety of studies to estimate relative risk, but the studies had some limitations. Four of the 6 included only older data (2 studies ended follow-up in the 1970s and 2 in the 1980s), 3 had only self-reported weight and height, and 1 study included only women. Only 1 data set, the National Health and Nutrition Examination Survey (NHANES) was nationally representative.”*

Using what was billed as a more credible approach, the study concluded that in the USA, relative to a normal weight category of BMI between 18.5 and 25, obesity over 30 BMI was associated with an excess of 111,909 deaths, while underweight was associated with 33,746 excess deaths. Surprisingly, it also found that for the category of BMI between 25 and 30 **overweight reduced mortality by 86,094 units annually**. Actually, Table 2 in the report lists data for nonsmokers where mortality trends in relation to BMI should be better and unaffected by cigarette use. For those the apparent mortality protection of body fat likely extends into the beginning of the 30-35 BMI category, which means that the estimated lives saved by body fat could be considerably more numerous than reported for the 25-30 BMI category alone.

⁴ [Mokdad AH, Marks JS, Stroup DE, Gerberding JL](#). Actual causes of death in the United States, 2000. [JAMA](#). 2004 Mar 10;291(10):1238-45

⁵ [Flegal KM, Graubard BI, Williamson DE, Gail MH](#). Excess deaths associated with underweight, overweight, and obesity. [JAMA](#). 2005 Apr 20;293(15):1861-7.

Table 2. Relative Risks by Age Group and BMI Level From the Combined NHANES I, II, and III Data Set

BMI Level	Relative Risk (95% Confidence Interval) by Age Category		
	25-59 y	60-69 y	≥70 y
	Overall		
<18.5	1.38 (0.82-2.32)	2.30 (1.70-3.13)	1.69 (1.38-2.07)
18.5 to <25	1.00	1.00	1.00
25 to <30	0.83 (0.65-1.06)	0.95 (0.80-1.13)	0.91 (0.83-1.01)
30 to <35	1.20 (0.84-1.72)	1.13 (0.89-1.42)	1.03 (0.91-1.17)
≥35	1.83 (1.27-2.62)	1.63 (1.16-2.30)	1.17 (0.94-1.47)
	Never-Smokers Only		
<18.5	1.25 (0.29-5.49)	2.97 (1.17-7.54)	1.50 (1.11-2.02)
18.5 to <25	1.00	1.00	1.00
25 to <30	0.66 (0.38-1.16)	0.81 (0.56-1.16)	0.90 (0.79-1.04)
30 to <35	0.77 (0.46-1.28)	1.21 (0.83-1.77)	1.13 (0.96-1.31)
≥35	1.25 (0.76-2.06)	2.30 (1.47-3.59)	1.12 (0.87-1.45)

Abbreviations: BMI, body mass index (measured as weight in kilograms divided by the square of height in meters); NHANES, National Health and Nutrition Examination Survey.

Table 3. Prevalence of BMI Levels in 1999-2002 (From NHANES 1999-2002). by Age Group

(From 5)

Notice in any event how the mortality risks reported are extremely weak and mostly without statistical significance. Data are also much weaker for nonsmokers than for smokers and nonsmokers together, which raises the question of how much weaker the data might be if the innumerable causes known to impinge on mortality had been accounted for, just as the study did for smoking alone. Could the study seriously mean that, smoking aside, the overall mortality observed was due exclusively to thinness, overweight, obesity, and nothing else? Yet, this is the ground on which aggressive public health policies are made and rapacious legal actions are made to advance.

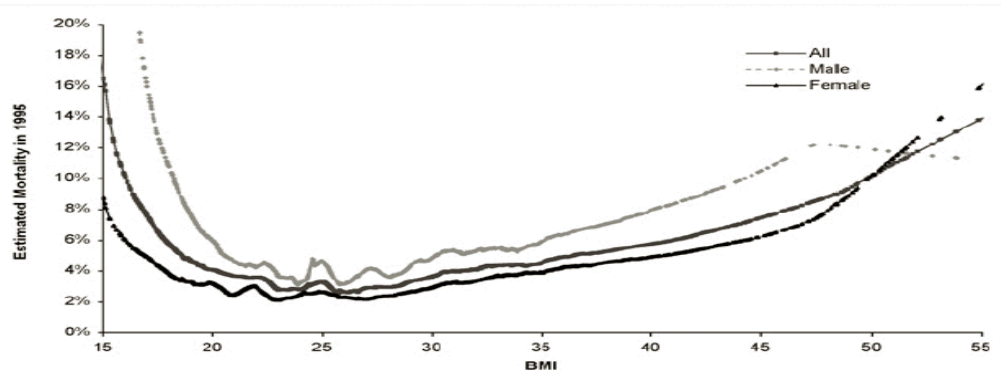
A 1999 epidemiologic study dealing with the different subjects of the large CPS-II study of the American Cancer Society essentially agrees with this second CDC report.⁶ Needless to say, all reports rely on the precarious meaning of the BMI, and none of the studies could account for the effects of changing lifestyles and medical improvements and other causes of mortality during the periods of observation. Indeed a commentary published in the same JAMA issue of the second CDC report expressed shock, noting that “[t]he magnitude of the differences cries out for an explanation of the reasons behind these differences. Some might wonder: If well-intentioned efforts to calculate this number can result in such widely varied estimates, is it worth trying to do at all?”⁷

A 2005 study of subjects from the National Health Interview Survey – presumably representative of the US population – concluded that “[n]ormal weight

⁶ Calle EE, Thun MJ, Petrelli JM, Rodriguez C, Heath CW Jr. Body-mass index and mortality in a prospective cohort of U.S. adults. *N Engl J Med.* 1999 Oct 7;341(15):1097-105.

⁷ Mark DM. Deaths Attributable to Obesity JAMA. 2005;293:1918-1919.

individuals of both genders did not appear to be relatively more long-lived than mildly obese individuals (BMIs of 30-35) whereas overweight people (BMIs of 25-30) appeared healthiest of all.”⁸ Gronniger, 2006). Virtually identical conclusions were reported in an earlier analyses of NHANES data. (Durazo-Arvizu et al., 1998; Troiano et al., 1996). In fact, the precariousness of the claimed association of excess weight and mortality is further illustrated by other recent and large studies here reported.



Note. Data were derived from the National Health Interview Survey 1987 Cancer Control Supplement File, 1989 Diabetes Supplement File, and 1995 Multiple Cause of Death File.²³⁻²⁶ Plots represent predictions for an average individual at a given BMI according to semiparametric regression results (bandwidth = 0.15). All covariates other than BMI were treated parametrically. Analyses were limited to respondents aged between 18 and 64 years with available mortality, BMI, and smoking information. All mortality estimates were adjust, via stratification, for race, age, limited smoking history, education level, income level, and marital status.

FIGURE 3—Semiparametric mortality estimates for 1995, by gender and body mass index (BMI), derived from self-reported height and weight: US adults aged 18 to 64 years in 1987 or 1989.

(From 8)

Also by CDC investigators, an earlier meta-analysis summary of US survey data concluded that the mortality risk increased below BMI 23 and above BMI 28 for men, but found no association of BMI and mortality for women, even if nonsmokers.⁹ A study of US 115,000 US nurses found that among nonsmokers the group below BMI 19.0 had the lowest mortality, while mortality increased progressively with BMI, up to a relative risk of 2.2 for BMI in excess of 32.0.¹⁰ By contrast, a large Canadian study in women found an increased risk of mortality for subjects of BMI below 18.5, and a progressive increase of the risk for BMI values above 22.0, up to a relative risk of 1.4 for subjects with BMI 35.0 and over.¹¹ A study of over 19,000 alumni of

⁸ [Gronniger JT](#). A semiparametric analysis of the relationship of body mass index to mortality. [Am J Public Health](#). 2006 Jan; 96(1):173-8. Epub 2005 Aug 30.

⁹ [Troiano RP](#), [Frongillo EA Jr](#), [Sobal J](#), [Levitsky DA](#). The relationship between body weight and mortality: a quantitative analysis of combined information from existing studies. [Int J Obes Relat Metab Disord](#). 1996 Jan;20(1):63-75

¹⁰ Manson JA, Willett WC, Stampfer MJ, Colditz GA, Hunter DJ, Hankinson SE, Hennekens CH, Speizer FE. Body Weight and Mortality among Women. [New England Journal of Medicine](#) Volume 1995; 333:677-685.

¹¹ [Jain MG](#), [Miller AB](#), [Rohan TE](#), [Rehm JT](#), [Bondy SJ](#), [Ashley MJ](#), [Cohen JE](#), [Ferrence RG](#). Body mass index and mortality in women: follow-up of the Canadian National Breast Screening Study cohort. [Int J Obes \(Lond\)](#). 2005 Jul;29(7):792-7.

Harvard University found no increased mortality risk for lean men, but an increase of the risk in subjects above BMI 22.5.¹²

A Polish study of 5,200 men and 5,600 women controlled for age, place of residence, blood cholesterol, hypertension and smoking over a mean period of 10 years. For men at BMI below 20.0 the risk of death was 2.2 compared to the low risk group of BMI 20.0 – 32.0, while the risk was 1.4 for the group at BMI 32.0 – 35.0, and 1.7 for BMI over 35.0. Women, by contrast, had an increased risk of mortality of 1.66 below BMI 20.0, but no increase of risk for higher BMI values.¹³ Similarly, a study of 7,900 European men found that the relative risk of mortality of people with BMI below 18.5 was 2.1, compared to the group of BMI 25.0 –30.0, while noting a relative risk of 1.8 for people with BMI over 30.0.¹⁴ A study of 16,000 Japanese men and women concluded that “[t]aking only never-smokers, the highest risk for all-cause mortality was observed in the lowest BMI category for men and women”.¹⁵

In a study of 2,100 Finnish men and women, “BMI did not prove to be an independent factor for mortality”, but physical fitness did. The study controlled for age, marital and employment status, and smoking and alcohol consumption.¹⁶ Somewhat different relationships were reported for a group of 5,500 men and 5,800 women from Japan. For subjects of BMI below 18.5 the study report an increased mortality risk of 2.66 for men and 3.14 for women. For the highest group over BMI 28.0, only women showed an increased risk of 3.25.¹⁷

An Italian study of 32,700 men and 30,300 women controlled for age, smoking and systolic blood pressure but failed to demonstrate in all cases a relation of BMI and mortality, except for some differences between young (higher) and mature women (lower). The study concluded that “uncommon high values of BMI carrying the

¹² [Lee IM](#), [Manson JE](#), [Hennekens CH](#), [Paffenbarger RS Jr](#). Body weight and mortality. A 27-year follow-up of middle-aged men. [JAMA](#). 1993 Dec 15;270(23):2823-8.

¹³ [Pajak A](#), [Topor-Madry R](#), [Waskiewicz A](#), [Sygnowska E](#). Body mass index and risk of death in middle-aged men and women in Poland. Results of POL-MONICA cohort study. [Kardiol Pol](#). 2005 Feb;62(2):95-105; discussion 106-7

¹⁴ [Visscher TL](#), [Seidell JC](#), [Menotti A](#), [Blackburn H](#), [Nissinen A](#), [Feskens EJ](#), [Kromhout D](#). Underweight and overweight in relation to mortality among men aged 40-59 and 50-69 years: the Seven Countries Study. [Am J Epidemiol](#). 2000 Apr 1;151(7):660-6.

¹⁵ [Miyazaki M](#), [Babazono A](#), [Ishii T](#), [Sugie T](#), [Momose Y](#), [Iwahashi M](#), [Une H](#). Effects of low body mass index and smoking on all-cause mortality among middle-aged and elderly Japanese. [J Epidemiol](#). 2002 Jan;12(1):40-4.

¹⁶ [Haapanen-Niemi N](#), [Miilunpalo S](#), [Pasanen M](#), [Vuori I](#), [Oja P](#), [Malmberg J](#). Body mass index, physical inactivity and low level of physical fitness as determinants of all-cause and cardiovascular disease mortality--16 y follow-up of middle-aged and elderly men and women. [Int J Obes Relat Metab Disord](#). 2000 Nov;24(11):1465-74.

¹⁷ [Hayashi R](#), [Iwasaki M](#), [Otani T](#), [Wang N](#), [Miyazaki H](#), [Yoshiaki S](#), [Aoki S](#), [Koyama H](#), [Suzuki S](#). Body mass index and mortality in a middle-aged Japanese cohort. [Epidemiol](#). 2005 May;15(3):70-7.

minimum risk of death seems to be in contrast with weight guidelines".¹⁸ A study of Aboriginal Australians found that *"BMI and mortality are inversely associated in Aboriginal adults in a remote community. Individuals with relatively higher BMI have a lower risk of death"*.¹⁹

High BMI values seem less of concern for elderly subjects. A most recent study found that nursing home residents with BMI over 28.0 had a lower relative risk of mortality (RR=0.89) compared to controls, and concluded *"suggesting that a higher BMI may be protective among long-stay residents"*, in line with the findings of an earlier study by the authors.²⁰ Also, a study of 6,400 elderly Norwegian men and women found that *"BMI was not associated with all-cause mortality"* – mortality being highly correlated with hypertension.²¹ Similarly, a study of 3,700 elderly Japanese-Americans from Honolulu, Hawaii, found *"a consistent inverse association"* of BMI and mortality risk, namely that increasing BMI values related to decreasing mortality risk.²²

What could be the meaning of epidemiologic studies of BMI and mortality? The uncertainties and ambiguities should not surprise, in the context of population surveys incapable to control beyond an obvious handful of what must be numerous competing hazards that impinge on mortality.²³

In light of previous considerations on the limitations of epidemiology, it is also necessary to recall that many primary surveys have not actually measured height, weight, and other characteristics of the individual approached. Rather they have collected such information from the subjects themselves or next of kin, who were responding to a written or telephone questionnaire, or to a verbal interview. Such data do not qualify as measures, cannot lead to calculating justifiable error rates, and therefore do not warrant to be called scientific, let alone objective.

Indeed, the apparent association is questioned by several studies showing that

¹⁸ [Seccareccia F](#), [Lanti M](#), [Menotti A](#), [Scanga M](#). Role of body mass index in the prediction of all cause mortality in over 62,000 men and women. The Italian RIFLE Pooling Project. Risk Factor and Life Expectancy. J Epidemiol Community Health. 1998 Jan;52(1):20-6.

¹⁹ [Wang Z](#), [Hoy WE](#). Body mass index and mortality in aboriginal Australians in the Northern Territory. Aust N Z J Public Health. 2002 Aug;26(4):305-10.

²⁰ [Grabowski DC](#), [Campbell CM](#), [Ellis JE](#). Obesity and mortality in elderly nursing home residents. J Gerontol A Biol Sci Med Sci. 2005 Sep;60(9):1184-9.

[Grabowski DC](#), [Ellis JE](#). High body mass index does not predict mortality in older people: analysis of the Longitudinal Study of Aging. J Am Geriatr Soc. 2001 Jul;49(7):968-79.

²¹ [Ellekjaer H](#), [Holmen J](#), [Vatten L](#). Blood pressure, smoking and body mass in relation to mortality from stroke and coronary heart disease in the elderly. A 10-year follow-up in Norway.

²² [Kalmijn S](#), [Curb JD](#), [Rodriguez BL](#), [Yano K](#), [Abbott RD](#). The association of body weight and anthropometry with mortality in elderly men: the Honolulu Heart Program. Int J Obes Relat Metab Disord. 1999 Apr;23(4):395-402.

²³ [Flegal KM](#), [Graubard BI](#), [Williamson DF](#). Methods of calculating deaths attributable to obesity. [Am J Epidemiol](#). 2004 Aug 15;160(4):331-8.

mortality may exacerbate after deliberate attempt to reduce BMI by dietary restrictions, and consistent studies indicate that mortality is definitely enhanced following involuntary weight loss and weight cycling, especially in the elderly.²⁴ In fact, there are reports that even being morbidly obese confers survival advantages in

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- ²⁴ [Eilat-Adar S, Goldbourt U, Resnick HE, Howard BV](#). Intentional weight loss, blood lipids and coronary morbidity and mortality. [Curr Opin Lipidol](#). 2005 Feb;16(1):5-9.
- [Droyvold WB, Lund Nilssen TI, Lydersen S, Midthjell K, Nilsson PM, Nilsson JA, Holmen J; the Nord-Trondelag Health Study](#). Weight change and mortality: the Nord-Trondelag Health Study. [J Intern Med](#). 2005 Apr;257(4):338-45.
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- [Diaz VA, Mainous AG 3rd, Everett CJ](#). The association between weight fluctuation and mortality: results from a population-based cohort study. [J Community Health](#). 2005 Jun;30(3):153-65.
- [Sorensen TI](#). Weight loss causes increased mortality: pros. [Obes Rev](#). 2003 Feb;4(1):3-7.
- [Nilsson PM, Nilsson JA, Hedblad B, Berglund G, Lindgarde F](#). The enigma of increased non-cancer mortality after weight loss in healthy men who are overweight or obese. [J Intern Med](#). 2002 Jul;252(1):70-8.
- [Wedick NM, Barrett-Connor E, Knoke JD, Wingard DL](#). The relationship between weight loss and all-cause mortality in older men and women with and without diabetes mellitus: the Rancho Bernardo study. [J Am Geriatr Soc](#). 2002 Nov;50(11):1810-5.
- [Torgerson JS, Sjostrom L](#). The Swedish Obese Subjects (SOS) study--rationale and results. [Int J Obes Relat Metab Disord](#). 2001 May;25 Suppl 1:S2-4.
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- [Wallace JL, Schwartz RS, LaCroix AZ, Uhlmann RE, Pearlman RA](#). Involuntary weight loss in older outpatients: incidence and clinical significance. [J Am Geriatr Soc](#). 1995 Apr;43(4):329-37

certain disease situations.²⁵

According to the World Health Organization overweight and obesity are mostly associated to cardiovascular disease (CVD) and cancer risks, the risk of hypertension, and of non-insulin dependent diabetes mellitus (NID diabetes).²⁶(WHO 2006). Still, there are dozens of known cancer hazards, hundreds of hazards for cardiovascular diseases, and many more conditions besides body weight that influence hypertension and diabetes. None of the epidemiologic studies did weigh in with a comprehensive analysis of these hazards, meaning that their claims remain just as perplexing as in the case of the association of BMI and mortality.

BMI and cardiovascular diseases (CVD). Although not measuring CVD incidence itself, investigators from the US Centers for Disease Control and Prevention reported in 2005 about US trends in the prevalence of major CVD risk factors, using data from 5 nationally representative surveys conducted from 1960 to 2000. The authors of the report concluded that *"[e]xcept for diabetes, CVD risk factors declined considerably over the past 40 years in all BMI groups. Although obese persons still have higher risk factors than lean persons, the levels of these risk factors are much lower than in previous decades."* They further stated: *"[t]otal diabetes prevalence was stable within BMI groups over time, as non-significant 1-2 percentage-point increases occurred between 1976-1980 and 1999-2000."*²⁷ The study categorized BMI as less than 25, 25 to 30, and over 30: a segmentation that likely missed important and potentially revealing overlaps, as suggested by the mentioned Gronniger report.²⁸

According to the Gregg et al. study (²⁷), the prevalence of high cholesterol, high blood pressure, and cigarette smoking declined 21, 18, and 12 percentage points respectively since 1960-1962 for obese persons with BMI over 30. Those risks were lower for obese persons in 1999-2000 than for lean people in 1960-1962. At the same time the study reports that overall BMI values and the prevalence of overweight and obesity increased better than 10% from 1960-1962 to 1999-2000, noting also how several official reports had documented a much greater decline of CVD over the same

²⁵ [Kalantar-Zadeh K, Kopple JD, Kilpatrick RD, McAllister CJ, Shinaberger CS, Gjertson DW, Greenland S.](#) Association of morbid obesity and weight change over time with cardiovascular survival in hemodialysis population. [Am J Kidney Dis.](#) 2005 Sep;46(3):489-500.

[Chaturvedi N, Fuller JH.](#) Mortality risk by body weight and weight change in people with NIDDM. The WHO Multinational Study of Vascular Disease in Diabetes. [Diabetes Care.](#) 1995 Jun;18(6):766-74.

²⁶ WHO, World Health Organization. Obesity and overweight. 2006. <http://www.who.int/dietphysicalactivity/publications/facts/obesity/en/> (Accessed 1/15/06).

²⁷ Gregg EW, Cheng YJ, PhD; Cadwell BL, Imperatore G, PhD; Williams DE, Flegal KM, Venkat Narayan KM, Williamson DF. Secular Trends in Cardiovascular Disease Risk Factors According to Body Mass Index in US Adults. [JAMA.](#) 2005;293:1868-1874.

²⁸ [Gronniger JT.](#) A semiparametric analysis of the relationship of body mass index to mortality. [Am J Public Health.](#) 2006 Jan; 96(1):173-8. Epub 2005 Aug 30

period.²⁹ Mindful of the limitations of epidemiologic surveys that explore possible BMI roles, these contrasting trends strongly suggest that the claimed association of CVD and BMI is at least questionable, and may be a mirage caused by contingent hazards, only incidentally related to overweight and obesity, and therefore unrelated to diet. In fact, at least two studies indicate that CVD mortality in obese subjects that are physically fit is comparable to that of normal weight individuals: a finding that would seem to exonerate obesity itself as a cause of CVD mortality.³⁰

While there are earlier reports associating CVD and BMI, more recent and arguably better ones tend to indicate no association or even a protective effect of BMI and CVD. A US study of 15,000 patients suffering from coronary heart disease found that “[o]verweight and obese BMI classifications were associated with better intermediate-term survival after acute coronary syndromes than normal weight and very obese, but death of myocardial infarction were similar.”³¹

A large French study of 140,000 men and 104,000 women concluded that with regard to mortality “[i]n both genders, the association of overweight with diabetes alone or hypercholesterolemia alone did not increase the risk. Bu contrast, in the presence of hypertension, cardiovascular mortality dramatically increased in overweight subjects with hypercholesterolemia ... or diabetes...”, again indicating a major role of hazard factors other than high BMI.³²

A US study of 7,700 patients that had experience heart failure, but were in stable condition, found : “Crude all-cause mortality rates **decreased in a near linear fashion** [emphasis added] across successively higher BMI groups, from 45.0% in the underweight group to 28.4% in the obese group (P for trend <.001). After multivariable adjustment, overweight and obese patients were at lower risk for death (hazard ratio [HR], 0.88; 95% confidence interval [CI], 0.80-0.96, and HR, 0.81; 95% CI, 0.72-0.92, respectively), compared with patients at a healthy weight (referent). In contrast, underweight

²⁹ [Cooper R](#), [Cutler J](#), [Desvigne-Nickens P](#), [Fortmann SP](#), [Friedman L](#), [Havlik R](#), [Hogelin G](#), [Marler J](#), [McGovern P](#), [Morosco G](#), [Mosca L](#), [Pearson T](#), [Stamler J](#), [Stryer D](#), [Thom T](#). Trends and disparities in coronary heart disease, stroke, and other cardiovascular diseases in the United States: findings of the national conference on cardiovascular disease prevention. [Circulation](#). 2000 Dec 19;102(25):3137-47. [Rosamond WD](#), [Chambless LE](#), [Folsom AR](#), [Cooper LS](#), [Conwill DE](#), [Clegg L](#), [Wang CH](#), [Heiss G](#). Trends in the incidence of myocardial infarction and in mortality due to coronary heart disease, 1987 to 1994. [N Engl J Med](#). 1998 Sep 24;339(13):861-7.

³⁰ [Farrell SW](#), [Braun L](#), [Barlow CE](#), [Cheng YI](#), [Blair SN](#). The relation of body mass index, cardiorespiratory fitness, and all-cause mortality in women. [Obes Res](#). 2002 Jun;10(6):417-23. [Lee CD](#), [Blair SN](#), [Jackson AS](#). Cardiorespiratory fitness, body composition, and all-cause and cardiovascular disease mortality in men. [Am J Clin Nutr](#). 1999 Mar;69(3):373-80.

³¹ [Eisenstein EL](#), [McGuire DK](#), [Bhappkar MV](#), [Kristinsson A](#), [Hochman JS](#), [Kong DE](#), [Califf RM](#), [Van de Werf F](#), [Yancy WS Jr](#), [Newby LK](#). Elevated body mass index and intermediate-term clinical outcomes after acute coronary syndromes. [Am J Med](#). 2005 Sep;118(9):981-90.

³² [Thomas E](#), [Bean K](#), [Pannier B](#), [Oppert JM](#), [Guize L](#), [Benetos A](#). Cardiovascular mortality in overweight subjects: the key role of associated risk factors. [Hypertension](#). 2005 Oct;46(4):654-9. Epub 2005 Sep 12

patients with stable HF were at increased risk for death (HR 1.21; 95% CI, 0.95-1.53).” The study concluded: “In a cohort of outpatients with established HF, higher BMIs were associated with lower mortality risks; overweight and obese patients had lower risk of death compared with those at a healthy weight. Understanding the mechanisms and impact of the “obesity paradox” in patients with HF is necessary before recommendations are made concerning weight and weight control in this population.”³³ This study was corroborated by independent and similar evidence.³⁴

A Swedish study of elderly subjects found that high BMI values were a hazard for stroke in men but not in women.³⁵ A meta-analysis study of 13 international surveys of elderly subjects concluded that “[m]ost studies showed a negative or no association between BMI and all-cause mortality.”³⁶ Also, a study of over 900 patients with coronary disease found that the electrocardiograms “of overweight and obese coronary patients showed no significant differences when compared with electrocardiograms of normal-weight patients.”³⁷

The massive MONICA study conducted by the World Health Organization in 38 different communities in the world found **no association** in women between BMI and coronary heart disease – the major subset of CVD – and a **declining** risk for men as BMI increases.³⁸ A study of 5000 male physicians concluded that the “findings indicate that elevated BMI may not be strongly associated with total cardiovascular mortality among men with previously manifested coronary disease”. In fact none of the weak associations was statistically significant in this study.³⁹ A study of 2,600 subjects in a

³³Curtis JP, Selter JG, Wang Y, Saif S, Rathore SS, Jovin IS, Jadbabaie F, Kosiborod M, Portnay EL, Sokol SI, Bader F, Krumholz HM. The Obesity Paradox: Body Mass Index and Outcomes in Patients With Heart Failure. *Arch Intern Med.* 2005;165:55-61.

³⁴ [Tanko LB](#), [Christiansen C](#). Can the obesity paradox be explained by the protective effects of peripheral adiposity? *Arch Intern Med.* 2005 Aug 8-22;165(15):1796-7.
[Kalantar-Zadeh K](#), [Kopple JD](#), [Kilpatrick RD](#), [McAllister CJ](#), [Shinaberger CS](#), [Gjertson DW](#), [Greenland S](#). Association of morbid obesity and weight change over time with cardiovascular survival in hemodialysis population. *Am J Kidney Dis.* 2005 Sep;46(3):489-500.

³⁵ [Dey DK](#), [Rothenberg E](#), [Sundh V](#), [Bosaeus I](#), [Steen B](#). Waist circumference, body mass index, and risk for stroke in older people: a 15 year longitudinal population study of 70- year-olds. *J Am Geriatr Soc.* 2002 Sep;50(9):1510-8.

³⁶ [Heiat A](#), [Vaccarino V](#), [Krumholz HM](#). An evidence-based assessment of federal guidelines for overweight and obesity as they apply to elderly persons. *Arch Intern Med.* 2001 May 14;161(9):1194-203.

³⁷ Nomura A, Zareba W, Moss AJ. Obesity does not influence electrocardiographic parameters in coronary patients. *Am J Cardiol.* 2000 Jan 1;85(1):106-8, A9.

³⁸ [Kuulasmaa K](#), [Tunstall-Pedoe H](#), [Dobson A](#), [Fortmann S](#), [Sans S](#), [Tolonen H](#), [Evans A](#), [Ferrario M](#), [Tuomilehto J](#). Estimation of contribution of changes in classic risk factors to trends in coronary-event rates across the WHO MONICA Project populations. *Lancet.* 2000 Feb 26;355(9205):675-87.

³⁹ [Widlansky ME](#), [Sesso HD](#), [Rexrode KM](#), [Manson JE](#), [Gaziano JM](#). Body mass index and total and cardiovascular mortality in men with a history of cardiovascular disease. *Arch Intern Med.* 2004 Nov

US Health Maintenance Organization who had experienced myocardial infarction, during a follow up of 3.4 years found that persons in the highest quartile of BMI values had the highest rate of re-infarction but the lowest rate of mortality.⁴⁰ A similar study of 2000 Welshmen who had suffered myocardial infarction found the highest mortality rate in the leaner group (BMI 15-24) and a lower mortality risk for all subjects with higher BMI values.⁴¹

A study of 6,100 obese German subjects concluded that "...morbid obesity (BMI of $> \text{or} = 40$) was a strong predictor of premature death due to CVD. Excess mortality risks associated with gross obesity (BMI from 32 to $<40 \text{ kg/m}^2$) were considerably lower than hitherto assumed; moderate degrees of obesity (BMI from 25 to $<32 \text{ kg/m}^2$) were not significantly associated with excess mortality."⁴²(Bender et al., 1998). Such results are remarkably in line with the already cited studies of Flegal and Gronniger.⁴³

Thus, and while some earlier and arguably less experienced studies may have hinted at a correlation of BMI and CVD, more recent and likely better run studies tend to play down the correlation, and many actually suggest that obesity may exert a protective role in CVD, especially in the elderly. Obese people who are physically fit may not experience an increased risk of CVD. Thus it is likely that independent CVD hazards, such as hypertension, hypercholesterolemia, and cigarette smoking, and others, were more prevalent in the obese in the past decades, at which time they caused diseases and mortality that erroneously become imputed to overweight and obesity. Indeed, the more recent studies find a marked decrease of those hazards previously accompanying high BMI, a substantial reduction in CVD mortality, and also a noticeably increased BMI average – a convergence of circumstances that tends to exonerate whatever BMI per se may represent of overweight and obesity as a significant hazard for CVD, and with possible exceptions for unfit obese subjects.

BMI and cancer. In a most recent example, a Swedish study of 25,000 women and 33,000 men found no association of BMI and overall cancer in men, but an

22;164(21):2326-32.

⁴⁰ [Kaplan RC](#), [Heckbert SR](#), [Furberg CD](#), [Psaty BM](#). Predictors of subsequent coronary events, stroke, and death among survivors of first hospitalized myocardial infarction. [J Clin Epidemiol](#). 2002 Jul;55(7):654-64.

⁴¹ [Ness AR](#), [Gunnell D](#), [Hughes J](#), [Elwood PC](#), [Davey Smith G](#), [Burr ML](#). Height, body mass index, and survival in men with coronary disease: follow up of the diet and reinfarction trial (DART). [J Epidemiol Community Health](#). 2002 Mar;56(3):218-9.

⁴² [Bender R](#), [Trautner C](#), [Spraul M](#), [Berger M](#). Assessment of excess mortality in obesity. [Am J Epidemiol](#). 1998 Jan 1;147(1):42-8

⁴³ [Flegal KM](#), [Graubard BI](#), [Williamson DF](#), [Gail MH](#). Excess deaths associated with underweight, overweight, and obesity. [JAMA](#). 2005 Apr 20;293(15):1861-7. [Gronniger JT](#). A semiparametric analysis of the relationship of body mass index to mortality. [Am J Public Health](#). 2006 Jan; 96(1):173-8. Epub 2005 Aug 30.

increase of cancer in women with BMI above 27.1 - much too broad a segment of the BMI range, and one that leaves open the all important question of whether the risk progresses linearly from BMI 27.1 on, or at what point over BMI 27.1 the risk may begin to increase. ⁴⁴

A worldwide WHO study representing 7 World Bank regions found an increased incidence of cancers of the colon, uterus, and post-menopausal breast cancer in relation to body mass scores in excess of BMI 21, also an excessively broad category leaving no credible or useful guidance as to the specific shape of the relationship and possible public health action. ⁴⁵ A study of 18,000 middle aged London men followed for an average of 35 years found an increase in the incidence of cancers of the rectum, bladder, colon, liver and lymphoma for obese and overweight people with BMI over 25 - results that are clearly not interpretable in relation to a sensible guidance for possible weight control. ⁴⁶

By contrast, a large Japanese study of 42,000 men and 46,000 women with a 10 year follow up found a U shaped association between BMI and cancer incidence in men, with the lowest risk for the BMI interval 23 – 24.9. The risk was most significant for underweight males with BMI 14.0 – 18.9. Remarkably, the study found no association of BMI and cancer risk in women. ⁴⁷

With a median follow up of 14 years, investigators at the US Centers for Disease Control and Prevention studied breast cancer mortality trends in a group of nearly 4,000 women who had been diagnosed with breast cancer. The study found a slightly increased risk of mortality in women with BMI above 22.9, but concluded that “[f]urther study is needed to determine how these findings might affect recommendations to reduce breast cancer mortality.” A most appropriate conclusion given the

⁴⁴ [Lukanova A](#), [Bjor O](#), [Kaaks R](#), [Lenner P](#), [Lindahl B](#), [Hallmans G](#), [Stattin P](#). Body mass index and cancer: Results from the Northern Sweden Health and Disease Cohort. [Int J Cancer](#). 2006 Jan 15;118(2):458-66.

⁴⁵ [Danaei G](#), [Vander Hoorn S](#), [Lopez AD](#), [Murray CJ](#), [Ezzati M](#); [Comparative Risk Assessment collaborating group \(Cancers\)](#). Causes of cancer in the world: comparative risk assessment of nine behavioural and environmental risk factors. [Lancet](#). 2005 Nov 19;366(9499):1784-93.

⁴⁶ [Batty GD](#), [Shiple M](#), [Jarrett J](#), [Breeze E](#), [Marmot M](#), [Davey Smith G](#). Obesity and overweight in relation to disease-specific mortality in men with and without existing coronary heart disease in London: The original Whitehall study. [Heart](#). 2005 Nov 3; [Epub ahead of print] [Heart](#) 2006;000:1-8. doi: 10.1136/hrt.2005.072637.

[Batty GD](#), [Shiple MJ](#), [Jarrett RJ](#), [Breeze E](#), [Marmot MG](#), [Smith GD](#). Obesity and overweight in relation to organ-specific cancer mortality in London (UK): findings from the original Whitehall study. [Int J Obes \(Lond\)](#). 2005 Oct;29(10):1267-74.

⁴⁷ [Inoue M](#), [Sobue T](#), [Tsugane S](#); [JPHC Study Group](#). Impact of body mass index on the risk of total cancer incidence and mortality among middle-aged Japanese: data from a large-scale population-based cohort study--the JPHC study. [Cancer Causes Control](#). 2004 Sep;15(7):671-80.

unjustifiable range of the considered segment of BMI values.⁴⁸ A study of women with breast cancer in Hong Kong found that "...present BMI and BMI 5 years before diagnosis were poorly associated with breast cancer risk among pre- and post-menopausal women."⁴⁹ A French study followed nearly 70,000 women 45 to 70 years old. It found a nonsignificant reduction of breast cancer with higher BMI values among pre-menopausal women, and an equally non-significant increase of breast cancer for increasing BMI values in post-menopausal women.⁵⁰

A study by the American Cancer Society followed 900,000 men and women adults who were free of cancer when first interviewed in 1982. The study found that in relation to normal weight persons, there was a 52% increased cancer incidence for men with BMI over 40 in 1982, and a 62% increased incidence for corresponding women. Specific cancers were stomach and prostate for men and breast, uterus, cervix, and ovary for women. The study found no increased cancer incidence for underweight persons. Being a study of mortality and not of cancer incidence, the reported risks could be inflated by other independent mortality risks, including obesity - which prompted the author to warn that "[i]t is also likely that the stronger associations seen in our study reflect a greater effect of body mass index on mortality than on incidence of cancer at some sites."⁵¹ Indeed, a multi-country study by the International Agency for Research on Cancer, a unit of the World Health Organization, found that "increasing body weight may confer a protection against ovarian cancer."⁵²

A study followed 8,000 Japanese men for 15 years and found an increased risk of colon cancer in relation to increasing BMI for subjects who were 55 or older at the initial examination. The study found that "[n]o other cancer had a significant positive association with either BMI at the time of examination or with weight gain since age 25."⁵³

Thus the picture of a possible association of BMI and cancer is anything but

⁴⁸ [Whiteman MK](#), [Hillis SD](#), [Curtis KM](#), [McDonald JA](#), [Wingo PA](#), [Marchbanks PA](#). Body mass and mortality after breast cancer diagnosis. [Cancer Epidemiol Biomarkers Prev](#). 2005 Aug;14(8):2009-14.

⁴⁹ [Chow LW](#), [Lui KL](#), [Chan JC](#), [Chan TC](#), [Ho PK](#), [Lee WY](#), [Leung LH](#), [Sy WM](#), [Yeung CC](#), [Yung AK](#). Association between body mass index and risk of formation of breast cancer in Chinese women. [Asian J Surg](#). 2005 Jul;28(3):179-84.

⁵⁰ [Tehard B](#), [Clavel-Chapelon F](#). Several anthropometric measurements and breast cancer risk: results of the E3N cohort study. [Int J Obes \(Lond\)](#). 2006 Jan;30(1):156-163.

⁵¹ [Calle EE](#), [Rodriguez C](#), [Walker-Thurmond K](#), [Thun MJ](#). Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. [N Engl J Med](#). 2003 Apr 24;348(17):1625-38.

⁵² [Lukanova A](#), [Toniolo P](#), [Lundin E](#), [Micheli A](#), [Akhmedkhanov A](#), [Muti P](#), [Zeleniuch-Jacquotte A](#), [Biessy C](#), [Lenner P](#), [Krogh V](#), [Berrino F](#), [Hallmans G](#), [Riboli E](#), [Kaaks R](#). Body mass index in relation to ovarian cancer: a multi-centre nested case-control study. [Int J Cancer](#). 2002 Jun 1;99(4):603-8.

⁵³ [Nomura A](#), [Heilbrun LK](#), [Stemmermann GN](#). Body mass index as a predictor of cancer in men. [J Natl Cancer Inst](#). 1985 Feb;74(2):319-23.

clear and consistent. In the available epidemiologic studies, the pervasive methodology of dividing the BMI range in segments that are too broad is bound to distort the interpretation of a possible association. Given the observation that baseline mortality rates are essentially stationary in the BMI range 18-35⁽⁵⁴⁾, it would be possible to think that an eventual association of body mass and cancer might show heightened risk at BMI levels below and above that baseline range. Such an assumption may not hold if there could be a tradeoff between – for instance – a reduce mortality for CVD and an increased one for cancer, but all presently available information is unable to shed light on that issue. In any event, and with a possible exception for the morbidly obese, the epidemiologic record is ambiguous about a possible association of BMI and cancer.

Children and body weight. For adults, there has been a continuing trend of body weight increases during many decades of the last century, parallel to a steady increase in life expectancy.⁵⁵ Such increases in body mass have occurred over the entire spectrum of BMI values in adult populations. Conversely, the weight gain for children has been mostly confined to the obese segments of children populations, while children of normal weight have not experienced significant gains in body mass.⁵⁶

Still, despite the reported BMI increases, most but not all US reports show that the amount of calories ingested by children and adolescents has declined during the time in which average body mass increased. Reports from other countries tend to show that energy intakes of children remained rather unchanged as body mass

⁵⁴ [Gronniger JT](#). A semiparametric analysis of the relationship of body mass index to mortality. [Am J Public Health](#). 2006 Jan; 96(1):173-8. Epub 2005 Aug 30.

[Durazo-Arvizu RA](#), [McGee DL](#), [Cooper RS](#), [Liao Y](#), [Luke A](#). Mortality and optimal body mass index in a sample of the US population. [Am J Epidemiol](#). 1998 Apr. 15;147(8):739-49.

[Troiano RP](#), [Frongillo EA Jr](#), [Sobal J](#), [Levitsky DA](#). The relationship between body weight and mortality: a quantitative analysis of combined information from existing studies. [Int J Obes Relat Metab Disord](#). 1996 Jan;20(1):63-75.

⁵⁵ Costa DL, Steckel RH. Long-term trends in health, welfare, and economic growth in the United States. In: Steckel RH, Floud R, editors. Health and welfare during industrialization. Chicago: University of Chicago Press; 1997.

⁵⁶ [Kautiainen S](#), [Koivusilta L](#), [Lintonen T](#), [Virtanen SM](#), [Rimpela A](#). Use of information and communication technology and prevalence of overweight and obesity among adolescents. [Int J Obes \(Lond\)](#). 2005 Aug;29(8):925-33.

Kalies H, Lenz J, von Kries R. Prevalence of overweight and obesity and trends in body mass index in German pre-school children, 1982-1997. [Int J Obes Relat Metab Disord](#) 2002;26(9):1211-7.

Flegal ICM, Troiano RP. Changes in the distribution of body mass index of adults and children in fee US population. [Int J Obes Relat Metab Disord](#) 2000;24(7):807-18.

Romon M, Duhamel A, Collinet N, Weill J. Influence of social class on time trends in BMI distribution in 5-Year-old French children fem 1989 to 1999. [Int J Obes Relat Metab Disord](#) 2005;29(1):54-9.

Troiano RP, Flegal KM. Overweight children and adolescents: Description, epidemiology, and demographics. [Pediatrics](#) 1998; 101(3):497-504.

increased.⁵⁷

Here it is necessary to again to warn that dietary surveys are among the weakest of epidemiologic exercises, dependent as they are on vague individual recalls of dietary intakes in previous months and years. As a result, dietary intakes are not measured but simply guessed, and no statistical deftness could remedy the intrinsic uncertainty. This said, studies leave the general impression that dietary calorie intakes of children may not have changed much over the last 4 decades in many countries, and might have decreased in the USA.

Official crusades and the media have tended to focus concern on foods that are simply caloric: soda drinks, dietary fats, dairy products, regular meals versus frequent nibbling, snacks and vending machines. A US study of beverage consumption from school vending machines found “*no impact on BMI by removing [regular carbonated soft drinks] consumption in schools*”.⁵⁸ A US study followed for a year nearly 11,000 children 2-3 years old who were consuming sweet drinks. For children of normal weight at start, the consumption of sweet drinks made no significant difference on weight gain, but for children who were classified as

⁵⁷ [Jungjohann SM](#), [Luhmann PM](#), [Bender R](#), [Blettner M](#), [Neuhauser-Berthold M](#). Eight-year trends in food, energy and macronutrient intake in a sample of elderly German subjects. [Br J Nutr](#). 2005 Mar;93(3):361-78.

[Kersting M](#), [Alexy U](#), [Kroke A](#), [Lentze MJ](#). Nutrition of children and adolescents. Results of the DONALD Study. [Bundesgesundheitsblatt Gesundheitsforschung Gesundheitsschutz](#). 2004 Mar;47(3):213-8.

[Cavadini C](#), [Siega-Riz AM](#), [Popkin BM](#). US adolescent food intake trends from 1965 to 1996. [Arch Dis Child](#). 2000 Jul;83(1):18-24.

[Nielsen SJ](#), [Siega-Riz AM](#), [Popkin BM](#). Trends in energy intake in U.S. between 1977 and 1996: similar shifts seen across age groups. [Obes Res](#). 2002 May;10(5):370-8.

[Rolland-Cachera MF](#), [Bellisle F](#), [Deheeger M](#). Nutritional status and food intake in adolescents living in Western Europe. [Eur J Clin Nutr](#). 2000 Mar;54 Suppl 1:S41-6.

Rolland-Cachera MF, Bellisle F. Nutrition. In: Child and Adolescence Obesity: Causes and Consequences, Prevention and Management. Burniat w, Cole T, Lissau I, Poskitt E, Editors. Cambridge University Press, Cambridge, 2002.

[Troiano RP](#), [Briefel RR](#), [Carroll MD](#), [Bialostosky K](#). Energy and fat intakes of children and adolescents in the united states: data from the national health and nutrition examination surveys. [Am J Clin Nutr](#). 2000 Nov;72(5 Suppl):1343S-1353S.

[Heini AF](#), [Weinsier RL](#). Divergent trends in obesity and fat intake patterns: the American paradox. [Am J Med](#). 1997 Mar;102(3):259-64.

FASEB. Federation of American Societies for Experimental Biology, Life Sciences Research Office. Prepared for Interagency Board for Nutrition Monitoring and Related Research. Third Report on Nutrition Monitoring in the United States. Vol 2. Washington, DC: US Government Printing Office; 1995.

[Kennedy E](#), [Goldberg J](#). What are American children eating? Implications for public policy. [Nutr Rev](#). 1995 May;53(5):111-26.

[Adamson A](#), [Rugg-Gunn A](#), [Butler T](#), [Appleton D](#), [Hackett A](#). Nutritional intake, height and weight of 11-12-year-old Northumbrian children in 1990 compared with information obtained in 1980. [Br J Nutr](#). 1992 Nov;68(3):543-63.

⁵⁸ [Forshee RA](#), [Storey ML](#), [Ginevan ME](#). A risk analysis model of the relationship between beverage consumption from school vending machines and risk of adolescent overweight. [Risk Anal](#). 2005 Oct;25(5):1121-35.

overweight at start, the consumption of 2 to 3 drinks a day increased significantly the risk of overweight. The results suggest the role of susceptibility factors independent from the use of sweet drinks.⁵⁹

A longitudinal 1981-1999 Norwegian study followed subjects from age 15 to 33 in relation to their consumption of carbonated soft drinks, and found "...no differences in body mass index, overweight or obesity in 1999 between long term high and low consumers."⁶⁰ A counterintuitive Nebraska study of 164 children followed for 2 years and found that more diet soda was consumed by subject overweight at the start and those who gained weight after 2 years. The study left unclear how diet soda without caloric content could account for an increased BMI score.⁶¹

A Tennessee study concluded that fruit juice intake in children 2 to 5 years old "was not associated with either stature or overweight."⁶² The Bogalusa Heart Study followed 1,500 children from 1973 to 1994 in relation to sweetened beverage consumption, and concluded that "there was no linear relationship between sweetened-beverage consumption and BMI and total energy intake."⁶³

A US Harvard study followed for two years over 10,000 boys and girls who were 9-14 years old in 1996, in relation to consumption of sugar-added beverages and weight gain. No significant differences were observed after adjustments for total energy intakes.⁶⁴ A North Dakota study of 1,300 low-income preschool children aged 2 to 5 years found that weight change was not related to intakes of fruit juices, milk, and sugared soda beverages. The same study found no significant associations of BMI an energy-adjusted dietary fat or fiber.⁶⁵

In a German study of 4,300 children 5 to 6 year old, "[a] protective effect of increased daily meal frequency on obesity in children was observed and appeared independent of other risk factors for childhood obesity." even though it also noted "a higher energy intake in nibblers compared to gorgers." The authors attribute the difference to a possible

⁵⁹ [Welsh JA](#), [Cogswell ME](#), [Rogers S](#), [Rockett H](#), [Mei Z](#), [Grummer-Strawn LM](#). Overweight among low-income preschool children associated with the consumption of sweet drinks: Missouri, 1999-2002. [Pediatrics](#). 2005 Feb;115(2):e223-9.

⁶⁰ [Kvaavik E](#), [Andersen LF](#), [Klepp KI](#). The stability of soft drinks intake from adolescence to adult age and the association between long-term consumption of soft drinks and lifestyle factors and body weight. [Public Health Nutr](#). 2005 Apr;8(2):149-57.

⁶¹ [Blum JW](#), [Jacobsen DJ](#), [Donnelly JE](#). Beverage consumption patterns in elementary school aged children across a two-year period. [J Am Coll Nutr](#). 2005 Apr;24(2):93-8.

⁶² [Skinner JD](#), [Carruth BR](#). A longitudinal study of children's juice intake and growth: the juice controversy revisited. [J Am Diet Assoc](#). 2001 Apr;101(4):432-7.

⁶³ [Rajeshwari R](#), [Yang SJ](#), [Nicklas TA](#), [Berenson GS](#). Secular trends in children's sweetened-beverage consumption (1973 to 1994): the Bogalusa Heart Study. [J Am Diet Assoc](#). 2005 Feb;105(2):208-14.

⁶⁴ [Berkey CS](#), [Rockett HR](#), [Field AE](#), [Gillman MW](#), [Colditz GA](#). Sugar-added beverages and adolescent weight change. [Obes Res](#). 2004 May;12(5):778-88.

⁶⁵ [Newby PK](#), [Peterson KE](#), [Berkey CS](#), [Leppert J](#), [Willett WC](#), [Colditz GA](#). Beverage consumption is not associated with changes in weight and body mass index among low-income preschool children in North Dakota. [J Am Diet Assoc](#). 2004 Jul;104(7):1086-94.

increased mobilization of insulin.⁶⁶ At the same time, a US study arrived to different conclusions and recommended limiting food intake to “no more than six times per day”.⁶⁷

A study at the Children’s Hospital in Boston, Massachusetts, asked why some adolescents who frequently eat fast food do not become overweight. In the study, all subjects “over-consumed fast food regardless of body weight, although the phenomenon was more pronounced in overweight participants.” The implication is that characteristics of susceptibility are likely to play a role in overweight beyond diet itself.⁶⁸ A Harvard study of some 13,000 adolescents found that children who drank the most milk gained more weight, but that dairy fat was not responsible for weight increases.⁶⁹ By contrast, a contemporary Italian study found “a significant inverse association between frequency of milk consumption and body mass in children.”⁷⁰

Again in Boston, a study followed for 3 years some 14,000 children 9 to 14 year old at start, based on self-reported questionnaire data. The study found virtually no differences in relation to consumption of fried food intake away from home (FFA). Subjects who consumed 1 or no FFA per week had an average BMI of 19.1, for a consumption of 1-3 time a week the average BMI was 19.2, and for a consumption of 4 to 7 FFA per week the average BMI was 19.3.⁷¹ In a different study, the same authors followed the same cohort of children in relation to frequency of having dinner with the family and BMI. The study found that the frequency of eating a family dinner was slightly (RR=0.85) inversely associated with overweight prevalence at the start of the study, but that such an association was no longer holding at the end of a 3 year observation period.⁷²

A study of 137,000 children 10 to 16 year old from 34 mostly European countries concluded that overweight “was not associated with the intake of fruits, vegetables, and soft drinks or time spent on the computer.”⁷³ Another study followed for

⁶⁶ [Toschke AM](#), [Kuchenhoff H](#), [Koletzko B](#), [von Kries R](#). Meal frequency and childhood obesity. [Obes Res](#). 2005 Nov;13(11):1932-8.

⁶⁷ [Thompson OM](#), [Ballew C](#), [Resnicow K](#), [Gillespie C](#), [Must A](#), [Bandini LG](#), [Cyr H](#), [Dietz WH](#). Dietary pattern as a predictor of change in BMI z-score among girls. [Int J Obes \(Lond\)](#). 2006 Jan;30(1):176-82.

⁶⁸ [Ebbeling CB](#), [Sinclair KB](#), [Pereira MA](#), [Garcia-Lago E](#), [Feldman HA](#), [Ludwig DS](#). Compensation for energy intake from fast food among overweight and lean adolescents. [JAMA](#). 2004 Jun 16;291(23):2828-33.

⁶⁹ [Berkey CS](#), [Rockett HR](#), [Willett WC](#), [Colditz GA](#). Milk, dairy fat, dietary calcium, and weight gain: a longitudinal study of adolescents. [Arch Pediatr Adolesc Med](#). 2005 Jun;159(6):543-50.

⁷⁰ [Barba G](#), [Troiano E](#), [Russo P](#), [Siani A](#). Total fat distribution and blood pressure according to eating frequency in children living in southern Italy: the ARCA project. [Int J Obes](#) 2006;30:1166-1169.

⁷¹ [Taveras EM](#), [Berkey CS](#), [Rifas-Shiman SL](#), [Ludwig DS](#), [Rockett HR](#), [Field AE](#), [Colditz GA](#), [Gillman MW](#). Association of consumption of fried food away from home with body mass index and diet quality in older children and adolescents. [Pediatrics](#). 2005a Oct;116(4):e518-24.

⁷² [Taveras EM](#), [Rifas-Shiman SL](#), [Berkey CS](#), [Rockett HR](#), [Field AE](#), [Frazier AL](#), [Colditz GA](#), [Gillman MW](#). Family dinner and adolescent overweight. [Obes Res](#). 2005b May;13(5):900-6.

⁷³ [Janssen I](#), [Katzmarzyk PT](#), [Boyce WF](#), [Vereecken C](#), [Mulvihill C](#), [Roberts C](#), [Currie C](#), [Pickett W](#); [Health Behaviour in School-Aged Children Obesity Working Group](#). Comparison of overweight and

10 years 196 non-obese girls 8 to 12 years old at the start, in relation to BMI and the intake of energy-dense snacks (EDS), such as baked goods, ice cream, chips, sugar-sweetened soda, and candy. At entry, the girls consumed an average 2.3 +/- 1.7 servings of EDS per day, amounting to 15.7 +/- 8.1% of total energy intake. After 10 years observation there was no relationship of BMI-body fat and consumption of EDS foods. The study also found a significant association of EDS intake and time spent on television viewing: a finding that seems to exonerate television viewing as a cause of BMI gain, given that the study found no association of EDS consumption and BMI. ⁷⁴

Another large US study followed for 3 years almost 15,000 children and adolescents of both sexes who were 9-14 years old at the start, in relation to BMI and consumption of fruit and vegetables. As for other studies, this study relied on questionnaire data self-reported by the children themselves. After controlling for developmental variables, no correlation was found between intake of fruit and vegetables and BMI scores. The study concluded that *"the recommendation for consumption of fruit and vegetables may be well founded, but should not be based on a beneficial effect on weight regulation."* ⁷⁵

Under the aegis of the European Commission, a British study in young adults found that different individuals display different susceptibility to obesity depending on several characteristics, among which: weak satiety response to fatty meals, preference for high-fat over low energy food, an hedonic attraction to palatable foods and eating, low basal metabolic rate, a binge preference, different profiles of allelic variations in a variable number of genes, differences in hormonal responses to food and to fat accumulation, and more. The consequences of susceptibility traits would be enhanced in a permissive environment, but subjects who were not susceptible would maintain normal weight even in a permissive environment. ⁷⁶ Although the study examined young adults, there is no reason to doubt that susceptibility characteristics would also be common in children.

In fact, a study of 3,000 children followed from 1 to 8 years of age suggested further conditions of susceptibility in children, and concluded that *"prenatal characteristics, particularly race, ethnicity, maternal smoking during pregnancy, and maternal pregnancy obesity, exert influence on the child's weight states through an early*

obesity prevalence in school-aged youth from 34 countries and their relationships with physical activity and dietary patterns. [Obes Rev.](#) 2005 May;6(2):123-32.

⁷⁴ [Phillips SM](#), [Bandini LG](#), [Naumova EN](#), [Cyr H](#), [Colclough S](#), [Dietz WH](#), [Must A](#). Energy-dense snack food intake in adolescence: longitudinal relationship to weight and fatness. [Obes Res.](#) 2004 Mar;12(3):461-72.

⁷⁵ [Field AE](#), [Gillman MW](#), [Rosner B](#), [Rockett HR](#), [Colditz GA](#). Association between fruit and vegetable intake and change in body mass index among a large sample of children and adolescents in the United States. [Int J Obes Relat Metab Disord.](#) 2003 Jul;27(7):821-6.

⁷⁶ [Blundell JE](#), [Stubbs RJ](#), [Golding C](#), [Croden F](#), [Alam R](#), [Whybrow S](#), [Le Noury J](#), [Lawton CL](#). Resistance and susceptibility to weight gain: individual variability in response to a high-fat diet. [Physiol Behav.](#) 2005 Dec 15;86(5):614-22.

tendency toward overweight, which then is perpetuated as the child ages.”⁷⁷ A second study on the same cohort of 3,000 children followed between the ages of 4 and 24 months, reached the somewhat divergent conclusion that infants eating small portions eat more frequently and vice versa. The study warned about “the potential adverse effects of coercive feeding behaviors...on children’s innate ability to regulate energy intake. This includes not only admonitions to “clean your plate”, but overrestrictions of intake that may be motivated by concerns that children are overeating.”⁷⁸

The studies just reviewed leave a rather confusing impression about the possible role of excess food intake and the observed increase in BMI scores. They seem to hint to underlying conditions of susceptibility to overweight, especially in permissible environments – conditions that are possibly linked to genetic, environmental and behavioral characteristics of individual children. Such conclusion also would be compatible with the observation that BMI enhancements have been mostly apparent in children who were already decidedly overweight or obese, but not in children who were in the normal BMI range.⁷⁹

If in fact BMI scores have increased while caloric intakes remained flat or actually may have decreased, the reasonable conclusions would be: first that most children have an innate capacity to self-regulate food intake despite the easy and abundant variety at hand, and second that the observed BMI enhancements may be more specifically related to a decline in physical activity and energy expenditures.

Most but not all studies find a correlation of overweight and obesity with low physical activity, and time spent on television and computer. A 2004 meta-analysis of prior studies – for whatever a meta-analysis is worth – found that “a statistically significant relationship exists between TV viewing and body fatness among children and youth although it is likely to be too small to be of substantial clinical relevance.” The study concluded that “Relationships between sedentary behavior and health are unlikely to be explained using single markers of inactivity, such as TV viewing or video/computer game use.”⁸⁰

More recently, a British study found that youth physical activity can both decrease and increase BMI at different developmental stages, but offers little guidance about possible causes and remedies.⁸¹ Other studies found a modest reduction of BMI values associated with poorly characterized levels of self-reported

⁷⁷ [Salsberry PJ](#), [Reagan PB](#). Dynamics of early childhood overweight. [Pediatrics](#). 2005 Dec;116(6):1329-38.

⁷⁸ [Fox MK](#), [Devaney B](#), [Reidy K](#), [Razafindrakoto C](#), [Ziegler P](#). Relationship between Portion Size and Energy Intake among Infants and Toddlers: Evidence of Self-Regulation. [J Am Diet Assoc](#). 2006 Jan;106(1S):77-83.

⁷⁹ Troiano RP, Flegal KM. Overweight children and adolescents: Description, epidemiology, and demographics. [Pediatrics](#) 1998; 101(3):497-504.

⁸⁰ [Marshall SJ](#), [Biddle SJ](#), [Gorely T](#), [Cameron N](#), [Murdey I](#). Relationships between media use, body fatness and physical activity in children and youth: a meta-analysis. [Int J Obes Relat Metab Disord](#). 2004 Oct;28(10):1238-46.

⁸¹ [Parsons TJ](#), [Manor O](#), [Power C](#). Physical activity and change in body mass index from adolescence to mid-adulthood in the 1958 British cohort. [Int J Epidemiol](#). 2005 Dec 22; doi:10.1093/ije/dyi291

increases in physical activity.⁸²

A group of over 8,000 Japanese children were followed from age 3 to age 6 in regard to BMI and body build and lifestyle data, as obtained by questionnaires filled by parents and caretakers. "Significant factors associated with overweight children were diet (eating rice, green tea, eggs, meat, but less breads and juice), rapid eating, short sleep duration, early bedtime, long periods of television viewing, avoidance of physical activity, and frequent bowel movement."⁸³ A Swiss study found that in "this sample of children living in Switzerland, the use of electronic games was significantly associated with obesity, independently of confounding factors. The association of obesity with television use and lack of physical activity confirms results from other populations and points to potential strategies for obesity prevention."⁸⁴

By contrast, a US study found that "In a national sample of preschool children, mothers' perception of neighborhood safety was related to their children's TV viewing time but not to their outdoor play time or risk for obesity."⁸⁵ A British cohort was followed up at 5, 10, and 30 years of age since 1970, and concluded that "Weekend TV viewing in early childhood continues to influence BMI in adulthood. Interventions to influence obesity by reducing sedentary behaviors must begin in early childhood."⁸⁶ A national representative sample of 6,500 Finnish children 14, 16, and 18 year old found that overweight was associated with television and computer use (RR=2.0 for over 4 hours daily), but not with the playing of videogames.⁸⁷

A study of UK children found that watching television for more than 8 hours per week at age 3 was one of 8 factors associated with the risk of overweight/obesity

⁸² [Kimm SY](#), [Glynn NW](#), [Obarzanek E](#), [Kriska AM](#), [Daniels SR](#), [Barton BA](#), [Liu K](#). Relation between the changes in physical activity and body-mass index during adolescence: a multicentre longitudinal study. [Lancet](#). 2005 Jul 23-29;366(9482):301-7.

[Berkey CS](#), [Rockett HR](#), [Gillman MW](#), [Colditz GA](#). One-year changes in activity and in inactivity among 10- to 15-year-old boys and girls: relationship to change in body mass index. [Pediatrics](#). 2003 Apr;111(4 Pt 1):836-43.

[Berkey CS](#), [Rockett HR](#), [Field AE](#), [Gillman MW](#), [Frazier AL](#), [Camargo CA Jr](#), [Colditz GA](#). Activity, dietary intake, and weight changes in a longitudinal study of preadolescent and adolescent boys and girls. [Pediatrics](#). 2000a Apr;105(4):E56.

⁸³ [Sugimori H](#), [Yoshida K](#), [Izuno T](#), [Miyakawa M](#), [Suka M](#), [Sekine M](#), [Yamagami T](#), [Kagamimori S](#). Analysis of factors that influence body mass index from ages 3 to 6 years: A study based on the Toyama cohort study. [Pediatr Int](#). 2004 Jun;46(3):302-10.

⁸⁴ [Stettler N](#), [Signer TM](#), [Suter PM](#). Electronic games and environmental factors associated with childhood obesity in Switzerland. [Obes Res](#). 2004 Jun;12(6):896-903.

⁸⁵ [Burdette HL](#), [Whitaker RC](#). A national study of neighborhood safety, outdoor play, television viewing, and obesity in preschool children. [Pediatrics](#). 2005 Sep;116(3):657-62.

⁸⁶ [Viner RM](#), [Cole TJ](#). Television viewing in early childhood predicts adult body mass index. [J Pediatr](#). 2005 Oct;147(4):429-35.

⁸⁷ [Kautiainen S](#), [Rimpela A](#), [Vikat A](#), [Virtanen SM](#). Secular trends in overweight and obesity among Finnish adolescents in 1977-1999. [Int J Obes Relat Metab Disord](#) 2002;26(4):544-52.

at age 7 (RR=1.55).⁸⁸ A US study of 3 to 7 year old children found that “Physical activity and TV viewing were the only significant predictors (other than baseline BMI) of BMI among a tri-ethnic cohort of 3-4-y-old children followed for 3 y, with both physical activity (negatively associated) and TV viewing (positively associated) becoming stronger predictors as the children aged. It appears that 6 or 7 y is a critical age when TV viewing and physical activity may affect BMI. Therefore, focusing on reducing time spent watching television and increasing time spent in physical activity may be successful means of preventing obesity among this age group.”⁸⁹

A study of 137,000 youth aged 10 to 16 years from 34 prevalently European countries - based on self-reported data from questionnaires – found that overweight was associated with television viewing, but not with time spent on a computer.⁹⁰ A group of over 1,000 New Zealand children was followed from birth and every 2 years from age 3 to age 15. The study concluded that “Time spent watching television is a significant predictor of BMI and overweight in childhood. Although the effect size appears small, it is larger than the effect sizes commonly reported for nutritional intake and physical activity. Television viewing should be regarded as an important contributing factor to childhood obesity.”⁹¹

In all, it make sense that reduced physical activity would contribute to reduce energy expenditure and thus to an accumulation of body mass, even though available studies are incapable of producing useful quantitative estimates of the relationship. Direct measures of physical activity by accelerometry techniques might offer better quantitative insights (⁹²), but their meaning would relate to immediate local conditions, and would still be unable to provide crucial longitudinal evidence over periods of time.

Other studies also highlight the likely role of psychosocial, socioeconomic, and environmental factors that are independent of dietary intake, and likely to interfere significantly with an understanding of the relationship of physical activity, overweight and obesity.⁹³ Such interferences are most likely to be material, given

⁸⁸ [Reilly JJ, Armstrong J, Dorosty AR, Emmett PM, Ness A, Rogers I, Steer C, Sherriff A; Avon Longitudinal Study of Parents and Children Study Team](#). Early life risk factors for obesity in childhood: cohort study. [BMJ](#). 2005 Jun 11;330(7504):1357.

⁸⁹ [Jago R, Baranowski T, Baranowski JC, Thompson D, Greaves KA](#). BMI from 3-6 y of age is predicted by TV viewing and physical activity, not diet. [Int J Obes \(Lond\)](#). 2005 Jun;29(6):557-64.

⁹⁰ [Janssen I, Katzmarzyk PT, Boyce WE, Vereecken C, Mulvihill C, Roberts C, Currie C, Pickett W; Health Behaviour in School-Aged Children Obesity Working Group](#). Comparison of overweight and obesity prevalence in school-aged youth from 34 countries and their relationships with physical activity and dietary patterns. [Obes Rev](#). 2005 May;6(2):123-32.

⁹¹ [Hancox RJ, Poulton R](#). Watching television is associated with childhood obesity: but is it clinically important? [Int J Obes \(Lond\)](#). 2006 Jan;30(1):171-5.

⁹² [Page A, Cooper AR, Stamatakis E, Foster LJ, Crowne EC, Sabin M, Shield JP](#). Physical activity patterns in nonobese and obese children assessed using minute-by-minute accelerometry. [Int J Obes \(Lond\)](#). 2005 Sep;29(9):1070-6.

⁹³ [Kelly LA, Reilly JJ, Fisher A, Montgomery C, Williamson A, McColl JH, Paton JY, Grant S](#). Effect of socioeconomic status on objectively measured physical activity. [Arch Dis Child](#). 2006 Jan;91(1):35-8.

that physical activity, psychosocial, socioeconomic, and environmental determinants seem all to have comparable effects of similar small force on BMI scores. Could food and excess body weight be the product of self-therapy attempts in the face of complex psychosocial stressors? ⁹⁴

Generally overlooked has been the likely contribution to population weight gain of the progressive cigarette smoking cessation during the last two decades. Smokers are notoriously lighter than non smokers, and a recent review of the literature concluded that smokers weigh from 1.18 to 9.79 kilograms less than nonsmokers. ⁹⁵ Such differentials amount to average deficits considerably in excess of population weight gains registered over the last two decades. In addition, there is much evidence that smoking cessation can result in weight gains averaging to several kilograms. ⁹⁶ The evidence suggests that smoking cessation likely provided a significant contribution to the population weight gain of the last two decades, both in adults and adolescents. Of note, the weight gain attributable to smoking cessation could not be unhealthy, for it lifts former smokers to the weight of presumably healthier nonsmokers.

In any event, and with regard to adolescents and children, it appears that the spike in overweight has been mostly confined to children who were consistently

[Norman GJ](#), [Schmid BA](#), [Sallis JF](#), [Calfas KJ](#), [Patrick K](#). Psychosocial and environmental correlates of adolescent sedentary behaviors. [Pediatrics](#). 2005 Oct;116(4):908-16.

[Salmon J](#), [Timperio A](#), [Telford A](#), [Carver A](#), [Crawford D](#). Association of family environment with children's television viewing and with low level of physical activity. [Obes Res](#). 2005 Nov;13(11):1939-51.

[Manios Y](#), [Panagiotakos DB](#), [Pitsavos C](#), [Polychronopoulos E](#), [Stefanadis C](#). Implication of socio-economic status on the prevalence of overweight and obesity in Greek adults: the ATTICA study. [Health Policy](#). 2005 Oct;74(2):224-32.

[Oliver LN](#), [Hayes MV](#). Neighbourhood socio-economic status and the prevalence of overweight Canadian children and youth. [Can J Public Health](#). 2005 Nov-Dec;96(6):415-20.

[Swallen KC](#), [Reither EN](#), [Haas SA](#), [Meier AM](#). Overweight, obesity, and health-related quality of life among adolescents: the National Longitudinal Study of Adolescent Health. [Pediatrics](#). 2005 Feb;115(2):340-7.

⁹⁴ [Flodmark CE](#). The happy obese child. [Int J Obes \(Lond\)](#). 2005 Sep;29 Suppl 2:S31-3.

[Wardle J](#), [Cooke L](#). The impact of obesity on psychological well-being. [Best Pract Res Clin Endocrinol Metab](#). 2005 Sep;19(3):421-40.

⁹⁵ Klesges RC, Meyers AW, Klesges LM, LaVasque ME. Smoking, body weight, and their effects on smoking behavior. A comprehensive review of the literature. *Psychological Bulletin* 1998; 106(2):204-230.

⁹⁶ [Filozof C](#), [Fernandez Pinilla MC](#), [Fernandez-Cruz A](#). Smoking cessation and weight gain. [Obes Rev](#). 2004 May;5(2):95-103.

[Chou SY](#), [Grossman M](#), [Saffer H](#). An economic analysis of adult obesity: results from the Behavioral Risk Factor Surveillance System. [J Health Econ](#). 2004 May;23(3):565-87.

[Froom P](#), [Melamed S](#), [Benbassat J](#). Smoking cessation and weight gain. [J Fam Pract](#). 1998 Jun;46(6):460-4.

[Klesges RC](#), [Winders SE](#), [Meyers AW](#), [Eck LH](#), [Ward KD](#), [Hultquist CM](#), [Ray JW](#), [Shadish WR](#). How much weight gain occurs following smoking cessation? A comparison of weight gain using both continuous and point prevalence abstinence. [J Consult Clin Psychol](#). 1997 Apr;65(2):286-91.

overweight over time, and was not apparent in those in the normal weight range.⁹⁷ The observation again suggests the likely influence of susceptibility traits of genetic, physiologic, or pathologic origin, which are apt to conspire with dietary habits in causing a fraction of children to become overweight. The potential of such a constellation of susceptibilities is reinforced by the observation that children with excessive BMI are prone to be overweight when becoming adults.⁹⁸

Thus, the picture emerging from the available studies on children and BMI is anything but clear. The comparatively smaller fraction of heavy and susceptible children seem to have become heavier within the span of some two decades, but normal weight children may have remained normal, at least in the United States. The observation seems congruous with the observation that overall caloric intake was likely constant or slightly reduced during the period. Remedies for the children at risk are not clear. The feared roles of dietary components such as sodas, fast food items, milk, fats, high calorie snacks, and others, were not confirmed in several studies, with a possible exception for the heavier children. At the same time, the presence of conditions of susceptibility independent of diet – such as physical activity, psychosocial and other stressors - has been confirmed.

The basis for a rational and decisive course of action is not immediately apparent, and a congeries of uncertain signals demands caution. Conceivably, remedial action should focus on the heavier children, for whom a reduction of caloric intake would seem the easy answer. Still, the long clinical and anecdotal experience of voluntary weight loss is less than reassuring and is fraught with alarming safety issues, recommending extreme caution and personalized attention, rather than sweeping generic guidelines. It is to be hoped that new safe interventions will emerge from genetic studies, and from the understanding of the intricate physiologic and psychological conditions that influence appetite, energy utilization and waste, and storage and release of energy as body fat.

⁹⁷ Troiano RP, Flegal KM. Overweight children and adolescents: Description, epidemiology, and demographics. *Pediatrics* 1998; 101(3):497-504.

⁹⁸ [Sandhu J](#), [Ben-Shlomo Y](#), [Cole TJ](#), [Holly J](#), [Davey Smith G](#). The impact of childhood body mass index on timing of puberty, adult stature and obesity: a follow-up study based on adolescent anthropometry recorded at Christ's Hospital (1936-1964). *Int J Obes (Lond)*. 2006 Jan;30(1):14-22.

[Field AE](#), [Cook NR](#), [Gillman MW](#). Weight status in childhood as a predictor of becoming overweight or hypertensive in early adulthood. *Obes Res*. 2005 Jan;13(1):163-9.

[Freedman DS](#), [Khan LK](#), [Serdula MK](#), [Dietz WH](#), [Srinivasan SR](#), [Berenson GS](#). The relation of childhood BMI to adult adiposity: the Bogalusa Heart Study. *Pediatrics*. 2005 Jan;115(1):22-7.

[Guo SS](#), [Chumlea WC](#). Tracking of body mass index in children in relation to overweight in adulthood. *Am J Clin Nutr*. 1999 Jul;70(1 Part 2):145S-148S.

[Valdez R](#), [Greenlund KJ](#), [Wattigney WA](#), [Bao W](#), [Berenson GS](#). Use of weight-for-height indices in children to predict adult overweight: the Bogalusa Heart Study. *Int J Obes Relat Metab Disord*. 1996 Aug;20(8):715-21.

[Serdula MK](#), [Ivery D](#), [Coates RJ](#), [Freedman DS](#), [Williamson DF](#), [Byers T](#). Do obese children become obese adults? A review of the literature. *Prev Med*. 1993 Mar;22(2):167-77.

A summation. A crusade on overweight and obesity that focuses primarily on dietary changes and restrictions seems counterfactual against an historical trend that in little more than a century has seen unprecedented advances in food quality, safety, and availability, parallel to a near doubling of life expectancy and fast declining mortality rates.

Tampering with current dietary habits would be foolhardy unless based on more than secure scientific footing, and the contrasting studies just reviewed prove that such footing is not presently at hand. Given the many complexities, at the present rate of discovery it will take a long time before it is possible to say safely and with objective confidence what the long term consequences of dietary and body weight changes and restrictions might be. Clearly, therefore, generic dietary and body weight guidelines are dangerously arrogant, and based on the foolish assumption that populations are made of uniform individuals.

Available hints suggest that there is a range of normal body mass rather than a point threshold, and in fact they suggest different normal ranges for different ages, sexes, ethnicities, and other markers including different conditions of lifestyle and stress. Such normalcy ranges will be moving targets, for they are bound to change as culture, technology, and affluence will change. Advice about dietary and nutritional requirements would not be of much use unless it can be specifically tailored to each individual. The current inability to do so is a principal reasons why bariatric medicine and surgery have mixed record in reducing and controlling the weight of obese people.

Children undoubtedly would be the best subject of study because of the relative plasticity of their conditions and development. Such plasticity, however, remains individual and is determined by genetic traits and environmental and cultural pressures, which in turn are apt to determine what would be desirable body mass and diet. Today no one, in good faith and with testable data in hand, could assert to know how to best answer what the preferred body mass and dietary goals of a child ought to be. Currently used standard growth charts remain simplistic approximations based on outdated records of development that will have to be revisited in view of the historically sudden and profound changes in behavior, stress, and affluence that children populations have experiences in developed and developing countries. Those changes are here to stay and to cause even more changes, simply because they portend what is perceived as an inevitable and attractive new way of life.

In this context it is fair to ask whether much success can be hoped from hopeful exhortations to greater physical activity. Much could be gained if in fact such activity could meaningfully increase, but much may militate against that goal. The information technology, innumerable common chore robots, energy increasingly less expensive, amusements and work patterns that mimic and surrogate physical activity, food at hand for the asking... But also an ease of life that makes less evident the need of learning and work, relaxed and casual learning methods, increasing prospects of entering make-pretend professions, less emphasis on focused personal goals, transient and superficial social connections, broken families... All this and

more is linked to health and disease and to ideal body mass and diet, although our knowledge of these complexities can again be compared to the blind men of Hindustan touching an elephant, and groping to figure out what it might be. Presently, and even if sweeping government impositions were conceivable in free societies, no one could say to possess objective knowledge that would justify changing those diets and lifestyles that have allowed billions of people to reach levels of health and longevity unprecedented in human history. It is conceivable that changes in diet, weight, and physical activity might be desirable, but testable knowledge to do so is not available. Such undisputable ignorance brands as reckless any crusade by self-serving authorities professing too.