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The bigger the healthier: Are the limits of BMI risk changing over time?

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Abstract

The body mass index (BMI) is often used as a predictor of overweight and obesity. There is, however, an important debate among international specialists as to what the risk limits should be, and where the cut-off points should be located. In the United States, for instance, adults with a BMI between 25 and 30 are considered overweight, while adults with a BMI of 30 or higher are considered obese. Nevertheless some researchers, especially in developing countries, claim that the limits established for the US are too permissive, and that the threshold to define obese adults should be set lower for other nationalities and ethnicities.

This paper analyzes the mortality risks for different BMI levels of two populations of American adult men. The first population lived during the last quarter of the 19th century and the early 20th century. These men were drawn from a random sample of Union Army veterans who fought during the American Civil War (1861–1865). A contemporary sample of men was drawn from the first wave of the National Health and Nutrition Examination Survey (NHANES I) conducted between 1971 and 1975. The results indicate that the frontier of overweight and obesity are expanding over time, such that the potential risk is nowadays associated with higher levels of BMI. The finding may imply that differences in BMI cut-off points are not only cross ethnic, but also occur for similar ethnicities across time.

Keywords

BMI; Obesity; Mortality; Health; Union Army; NHANES I

1. Introduction

Over the course of the 20th century, obesity has grown dramatically in the United States. During the last decade of the 19th century, around 3.5% of white middle aged men were obese. A century later, more than 35% of the comparable population was considered obese (Helmchen and Henderson, 2004).¹ As Komlos and Baur (2004) point out, the American population went from being the tallest in the world during the 19th century to one of the heaviest at the end of the 20th century. Yet the obesity problem is not confined to the United States. International organizations such as the World Health Organization (WHO) and the International Obesity Task Force (IOTF) have recognized obesity as a worldwide epidemic (James et al., 2001). Even in developing countries obesity has become a public health problem, to the point that some households now share both undernutrition and obesity within its members (Popkin, 2002).²

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¹The prevalence of obesity is defined as a BMI of 30 kg/m² or higher. Most of the growth in obesity happened during the last quarter of the 20th century, and seems to keep growing into the beginning of the new millennium.

²The whole supplement of *Public Health Nutrition* 5(1A) 2002 presents the papers of the Bellagio meeting, where worldwide experts discussed the growth and prevalence of obesity in developing countries.

One of the problems that researchers face when analyzing obesity prevalence and trends, is the comparability of measurements across populations. In order to measure overweight and obesity, the body mass index (BMI) is widely utilized as a predictor of the healthy levels of weight for height among populations and individuals. The BMI or Quetelet index relates height to weight (weight (kg)/height (m²)) and is commonly used as an indirect measure of adiposity in the human body. Different levels of BMI are associated with morbidity and mortality risk levels, and the level of body fat or adipose tissue stored in the body. The evidence shows that across populations in time and space both very low and very high levels of BMI are unhealthy and increase the probability of morbidity and mortality (Troiano et al., 1996; Waaler, 1984; Costa, 1993; Costa and Steckel, 1997; Fogel, 1993; Fogel, 1994; Visscher et al., 2000).

For more than a decade, analysts in the nutrition and anthropometrics fields have been debating whether a uniform set of BMI ranges can be applied to all populations and subpopulations across time, space and groups. For instance, most evidence points to a different physiology among contemporary Caucasians and Asians. Associated risks tend to appear at lower BMI ranges for Asians than for Caucasians. But this has not always been the case. In the United States, men born during the mid-19th century had higher risks at lower BMIs than contemporary Americans.

This paper compares BMI risk across populations by contrasting two cohorts of American men that were born almost a century apart. A 19th century cohort is represented by a group of Union Army veterans, whereas the 20th century cohort was drawn from the first wave of the National Health and Nutrition Examination Survey (NHANES I). I argue that the problems for comparing cut-off points for overweight and obesity are found not only across different racial and ethnic groups, but also across comparable populations over time. Nineteenth century adult men had a higher mortality risk at lower BMI levels than the contemporary cohort.

2. Overweight adults

When humans ingest more energy than they spend, the excess energy is stored in the form of adipose tissue. Energy storage is fundamental for survival. Whenever food is scarce, the body uses the stored fat to provide additional energy. As more fat is stored, resulting from an energy surplus, the risks of morbidity and mortality increase. In other words, “obesity may be defined as the degree of fat storage associated with clearly elevated health risks.” (WHO, 1995). Therefore for health purposes obesity is not an aesthetic consideration, but rather a risk factor. It represents excessive body mass for stature, and more specifically an excessive body fat content. The effects of obesity are debilitating both socially and physically (Bray et al., 1998).

There are several methods to measure obesity or excess body fat. Among the alternatives, BMI is often used as an indicator of obesity. While there are other alternatives to BMI that are more accurate in terms of measuring areas of regional fat, BMI is a good and reliable predictor of total fat, and it is relatively inexpensive to measure.³ BMI measurements facilitate the comparison between past and present populations and across countries, especially given that other measurements are not readily available for poor countries. Even in richer countries, like the US, other obesity tests remain too expensive and prevent the use of large samples. In addition, the use of BMI as a proxy for overweight and obesity is a relatively easy method for policy recommendations and communicating targets to the population at large.

Given the considerations mentioned, BMI is widely accepted as a predictor of obesity. The consensus, however, breaks down among the international community of researchers when

³See Bray et al. (1998) for alternatives to measuring body fat.

establishing the boundaries that predict obesity and overweight. In the United States, for instance, adults with a BMI of 30 or more are considered to be obese (CDC, 2004), a limit that has been also established by the World Health Organization (WHO, 1998). Nevertheless, some researchers find a BMI of 30 too permissive for certain ethnic groups. In the year 2000, a panel of experts coordinated by the World Health Organization produced a report to assess the risks of different BMIs in Asian Populations (WHO, 2000). The report concluded that Asians in general had higher health risks than Caucasians for the same BMIs and therefore that the limits of overweight and obesity should be set lower for Asian adults. Popkin argues that there is limited but strong evidence that the biology among sub-populations is different in relation to the BMI risks, “[t]hat is, Asians, Africans, and Latin Americans are more likely than whites in the USA and Europe to have greater body fat and central fat for the same BMI and to have a higher likelihood of experiencing cardiovascular disease (CVD) outcomes of importance at lower BMI levels” (Popkin, 2002, p. 100).

Table 1 presents the recommendations of a number of selected international boards of experts. The table clearly shows that there are marked differences in the recommendations, not only internationally and among different study groups, but the same expert boards have established different limits at different times. In the United States, most boards agree on cut-off points of 25 and 30 kg/m² for overweight and obesity respectively. The 1989 report of the National Research Council (NRC), however, considered a BMI of 27 as the threshold for overweight. In Australia and Canada the recommendations are similar to those in the United States. By contrast, Asians are considered overweight at 23 kg/m² and obese at 25 kg/m². In Mexico, adults of short stature (women below 1.5 m and men below 1.6 m) share the same cut-off points as Asians. As in the Mexican case, some experts have differentiated obesity risk limits by gender showing the importance of sex specific cutoff points. For example, during the mid 1980s, the National Institutes of Health (1985) concluded that men with a BMI over 27.8 and women with a BMI over 27.3 were obese.

To a large degree the recommendations of some panels respond to pragmatic considerations and convenience. While overweight and obesity increase the risk of morbidity and mortality, the risk of particular diseases may increase at different BMI levels. In order to simplify the use of cutoff points, Garrow (1981) suggested adopting a single set of divisions using 5 BMI unit intervals (Bray et al., 1998). The WHO and most expert committees in the United States have followed Garrow’s approach. The National Institutes of Health recommend to the general public the following guidelines⁴ (Table 2).

Yet, not only overweight generates health problems, being underweight is also linked to poor health.⁵ In an influential 1984 study of a very large sample of Norwegians, Hans Waaler outlined the U-shaped relationship between BMI and morbidity and mortality, such that extreme BMIs are riskier than those at the center of the distribution. The graphical depiction of these relationships has been called “Waaler curves,” an analytical instrument that has served to indicate optimal anthropometrics from a health standpoint.⁶ Several follow-up studies have further supported Waaler’s findings and support the U-shape relationship between BMI and mortality for other populations in different regions and historical periods (Fogel, 1993; Kim, 1996; Allison and Faith, 1996; Allebeck and Bergh, 1992; Engeland et al., 2003; Song et al., 2003; Strandberg, 1997). The proposed weight-to-height minimum risks vary according to age, race, gender, disease history and behavioral factors such as smoking.

⁴NIH website revised on 18 May 2004 http://www.nhlbi.nih.gov/health/public/heart/obesity/lose_wt/risk.htm.

⁵Underweight increases the risk of death even when adjusting for presence of disease and smoking history (Cornoni-Huntley et al., 1991; Calle et al., 1999).

⁶Waaler also pointed out the inverse J-shape relationship between height and mortality.

3. Data: The Union Army and NHANES I

The present study compares two samples of American adult men. The first sample was drawn from a group of Union Army (UA) veterans who fought during the American Civil War (1861–1865). Over the past two decades, Robert Fogel and his associates at the Center for Population Economics have compiled the life histories of around 40,000 UA volunteer white soldiers who were randomly chosen at the National Archives (Fogel, 1999, 2000a). The American Civil War marked a whole generation and a large number of northern men served in the Union Army. The sample is therefore representative of Northern white men of military age in the early 1860s (Fogel, 2001). Veterans who survived the war were eligible for pensions, a program that became so large that by the early 20th century 90% of the survivors were on the pension rolls (Glasson, 1918).⁷ Pension applicants were examined by a board of surgeons who determined the degree of disability and the amount of money that should be awarded. During the medical examination, the height and weight of pension applicants were recorded. Hence, it is possible to calculate the BMI of the veterans at the time of the examination. For most recruits, the date of death was also recorded, so we can trace them from the time of the examination to their death, and some cases, the cause of death was documented.

The second sample of men was drawn from the first wave of National Health and Nutrition examination Surveys (NHANES I-NCHS, 1973) conducted by the National Center for Health Statistics from 1971 to 1975. NHANES I is a stratified multi-stage clustered probability survey of the non-institutionalized population of the United States.⁸ Trained personnel recorded among other variables the height and weight of examinees. A follow-up study (NCHS, 1992) traces the survivors of NHANES I into the early 1990s, and therefore we are able to determine who died within two decades of the examination. In addition, the cause of death for non-survivors was recorded.

The sub-sample of UA veterans included in the present study consists of 9509 war survivors that were examined after 1865 between the ages of 45 and 64 for whom there is information on their date of death.⁹ For comparative purposes, the NHANES I sub-sample was restricted to men in the same age range and includes 1912 examinees.¹⁰ Both sub-samples were further subdivided into two groups. At some stages of the analysis, the Union Army sample was restricted to veterans who were examined after 1890. In this year, the rules for entry into the pension system were liberalized through the Dependent Pension Act, and any veteran who had honorably served for ninety days and was disabled for manual labor was eligible for a pension (US Bureau of Pensions, 1925).¹¹ The number of pensioners soared after 1890. In order to keep genetic and ethnic characteristics constant, portions of the analysis restrict NHANES I to non-Hispanic white men.¹² Finally, the cause of death was recorded for sub-samples of the UA ($n = 6321$) and NHANES I ($n = 1837$) who died within 20 years after the medical examination.¹³

⁷The wives and children of deceased soldiers were also eligible for pensions.

⁸Given the sample design of NHANES I the appropriate weights were used for all calculations.

⁹The sub-sample of UA veterans may present a number of randomness problems due to the survival probabilities and the selection effect of undergoing a medical examination. Previous tests, however, show that those who survived and were examined had similar anthropometric characteristics to the rest of the Union Army cohort (Helmchen and Henderson, 2004).

¹⁰The age distribution of the UA sample is less uniformly distributed than the NHANES I (see Table A.1 in Appendix A). As a result, UA veterans were almost a year and a half younger at examination than NHANES I subjects.

¹¹Over time, as legislation liberalized old age became sufficient to claim disability.

¹²It is important to be cautious about the level of generalization that can be drawn from the UA sample and the level of comparability to NHANES I. In addition to the aforementioned selection problems in the UA sample, genetic and ethnic changes due to different migration patterns between the 19th and 20th century should be acknowledged, even when restricting the NHANES I sample to non-Hispanic white men. In addition, the UA was mainly a volunteer army and some of the volunteers were rejected because of health problems, whilst NHANES I is a sample drawn from the whole population. Note, however, that there is no alternative or better dataset to study BMI distributions and longevity in the 19th century US other than the UA.

4. BMI and mortality: the changing role

Over the past century, the risk of having a higher BMI has decreased for middle-aged American men. The comparison between UA veterans and NHANES I cohorts reveals that there has been an upward shift in healthy BMI. Nowadays individuals with higher BMIs are healthier than their counterparts a century earlier. Underweight and extremely obese contemporary men, however, are at a higher relative risk than their ancestors.

Fig. 1a–d show the incidence rate of death of UA veterans and NHANES I examinees censored 20 years after the medical examination. There are five possible BMI classes with 5 unit intervals—BMI < 20, $20 \leq \text{BMI} < 25$, $25 \leq \text{BMI} < 30$, $30 \leq \text{BMI} < 35$, and BMI ≥ 35 . The samples were further subdivided into 5-year age groups.

For UA veterans, a BMI between 20 and 25 was clearly the healthiest for all age groups. Fig. 1a presents all men who survived the war and that were examined between the ages of 45 and 64. Fig. 1b, on the other hand, restricts the sample to veterans that were examined after the general law of 1890. Both figures follow a similar pattern. The inclusion of all those examined before 1890 does not make a big difference in the general trend. Veterans with a BMI between 20 and 25 had the lowest incidence rate. Conversely, the most likely to die were those with a BMI of 35 and above (except for the 55–59 age group in Fig. 1a). Veterans with a BMI between 30 and 35 were the second group at highest risk of death. In general terms, it was healthier to have a BMI below 20 than a BMI of 30 or larger. Men with a BMI in the 25–30 range were at a higher risk than the 20–25 group, but were in general healthier than most other groups. Arguably, men with a BMI of 25 were at a higher risk of death than those under 25.

In NHANES I, a BMI between 20 and 25 was not consistently the healthiest for all age groups. In some cases, examinees with a BMI in the 25–30 group were at a similar or even lower risk of death. In Fig. 1c and d, we can observe the incidence rate of death for men in NHANES I. Fig. 1c corresponds to all men between the ages of 45 and 64 years, while Fig. 1d displays only non-Hispanic white men within the same age range. Both groups of men present similar trends. The healthiest men appear to be those with a BMI of 20–25 and those with a BMI of 25–30 at the time of the interview. Clearly, men with a BMI of 30–35 were unhealthier than examinees with a BMI between 20 and 30. The worst possible situation was to have a BMI below 20 or a BMI of 35 or larger. In other words, underweight and very obese people had significantly shorter lifespans.

Not surprisingly, men from NHANES I had higher survival rates than their counterpart a century earlier. For instance, 20 years after the first examination, 62% of the healthiest examinees in NHANES I—that is men with a BMI between 20 and 25 were alive. Comparatively, only 54% of the Union Army veterans with similar BMI characteristics had survived. This group ($20 \leq \text{BMI} < 25$) remained the healthiest for both time periods. Nevertheless, the importance of other BMI categories shifted through time as a determinant of longevity. First, the difference between those with a BMI between 25 and 30 and those with a BMI between 20 and 25 deserves notice in the UA sample. For the NHANES I cohort the difference practically vanished, such that it is no longer clear that one category is healthier than the other. Second, being underweight (BMI < 20) became riskier relative to the other categories over time. For the UA it was riskier to be extremely obese (BMI ≥ 35) than being underweight, whilst for men in NHANES I both situations were equally risky.

¹³For the UA both samples $n = 9509$ and $n = 6321$ are relatively similar in terms of age at measurement, BMI, and birthplace (see Tables A.2 and A.3 in Appendix A). Furthermore, for veterans who died within 20 years after the medical examination, those without information on the cause of death lived on average slightly longer (11.79 years) than those for whom we know the cause of death (11.05 years).

In Fig. 2 we can further observe the differences in survival rates by BMI class and the changes in risk through time. The figure presents the mean relative risk of death within 20 years following the first medical examination.¹⁴ Once again we can see that Union Army veterans with a BMI between 25 and 30 were at a higher risk than those in the normal range ($20 \leq \text{BMI} < 25$). The difference in risk between both BMI categories withered away for NHANES I examinees. Fig. 2 also shows a U-shaped relationship between risk and BMI – such that risk increases at low and high BMI levels – a pattern that has been found by previous research (Fogel, 1993,1994;Kim, 1996;Waalder, 1984). Note, however, that the relative risk by BMI class is steeper for NHANES I than for the UA. Underweight men in NHANES I were on average almost 50% more likely to die in relative terms than underweight UA veterans. Even in absolute terms, underweight men in NHANES I appeared to have a higher risk than the UA sample (see Table A.4).¹⁵ Extreme obesity was also riskier for NHANES I examinees, especially for non-Hispanic white men. This result may be questionable, given that only a very small number of examinees in both samples were extremely obese, and therefore the confidence intervals are very large and not statistically distinguishable.

A breakdown into two unit BMI intervals further shows the U-shaped relationship between BMI and risk of death. Fig. 3 displays the mean relative risk of death within 20 years of the first examination by two unit BMI groups. Once again the association between BMI and risk appears to be steeper for NHANES I than for the UA. In addition, for Union Army veterans a BMI between 22 and 24 had the lowest risk, closely followed by the 20–22 and 24–26 ranges. The minimum risk for the UA sample is situated at a plateau between 20 and 26. Conversely, the minimum risk for NHANES I appears in the 26–28 BMI interval, closely followed by the 22–24 and 24–26 intervals, therefore forming a plateau between 22 and 28 BMI points.¹⁶ In fact, fitting a cubic function shows that the minimum risk shifted by almost 3 BMI points between both time periods, from 23.13 for the UA to 25.99 for NHANES I. Nevertheless, the cubic fit may be too restrictive by forcing the data into a curvilinear function, and actually erases the risk plateaus for both time periods.¹⁷ Figs. 2 and 3 show that Waaler curves may be getting steeper over time, given that extreme values are becoming relatively riskier.

The upward shift in healthy BMI from the UA to NHANES can be further evidenced in Table 3, where a Cox proportional hazard model is presented. The regression compares the hazard ratios for different BMI classes (by 5 unit intervals) against a BMI range of 20–25 (the omitted category). For the cohort born during the first half of the 19th century, having a BMI between 20 and 25 was the healthiest situation. Although veterans with a BMI of 25–30 were the second healthiest group, they were clearly at a higher risk of death than those in the normal range. This group was closely followed in risk by those with a BMI lower than 20. The veterans with the highest death risk were those with a BMI of 30 or more, especially those extremely obese who had an average hazard ratio 66% higher than those with a BMI between 20 and 25.

Comparatively, the men in NHANES I with a BMI of 25–30 were as healthy as those with a BMI between 20 and 25.¹⁸ The relative risk of the other BMI classes in relation to those with a BMI between 20 and 30 is much higher for NHANES I examinees than for the UA, a finding that is consistent with the curves shown in Figs. 2 and 3 above. A further breakdown (see Table

¹⁴Relative risk in relation to the mean risk. Table A.1 in Appendix A shows the absolute risk of death within 20 years of the first examination by BMI class. The table includes the 95% confidence intervals.

¹⁵The absolute mean risk of death was higher for NHANES I than for the UA. Yet, there is no statistical difference between the 95% confidence intervals.

¹⁶Breaking down BMI categories into one-unit intervals provides a similar pattern, but far more erratic due to the low sample size within each interval.

¹⁷The regression used to fit a cubic function is not shown in the paper, but is available upon request. Other authors have found an even higher nadir for BMI risk using quadratic fits in NHANES I (Allison et al., 2002; Su, 2003) and for HRS (Henderson, 2001).

¹⁸Although the hazard ratio for men in NHANES I with a BMI of 25–30 was a little higher than those with a BMI of 20–25 the level of statistical significance is questionable. Non-Hispanic white men with a BMI between 25 and 30 have a higher hazard ratio than the all the men together, but once again the level of statistical significance is low.

A.5 in Appendix A) into two unit intervals shows that the lowest risk for the UA was in the 20–26 BMI range, while the lowest risk for NHANES appears for men with a BMI between 22 and 28 at the time of the interview.¹⁹ The regression includes a linear term for age to control for the effect of aging, as expected, each additional year increased the risk of death.²⁰

Given that age is such an important determinant of survivorship, the samples were broken down into 10-year age intervals. Tables 4a and 4b show the results for the proportional hazard models of these subdivisions. A BMI under 20 was riskier than the 20–25 BMI group for all age groups, but even riskier in the case of NHANES I. Once again, veterans with a BMI between 25 and 30 were at higher risk than the benchmark group ($20 \leq \text{BMI} < 25$), a result that does not hold for NHANES I. Finally, having a BMI of 30 or larger increased the risk of death for both the UA and NHANES I in comparison to those with a BMI between 20 and 25. As we can see breaking down the samples into age groups, still reveals the general pattern that the risk of having a BMI between 25 and 30 has become less risky over time.

We should take into consideration that the analysis was done looking at all-cause mortality and not the emergence of particular morbidities. The risk of specific diseases associated with overweight and obesity may be different than those related to death as the final outcome. Nevertheless, it is possible to further analyze the interaction between different BMI levels and the specific cause of death.²¹ Tables 5a and 5b show the results for the Cox proportional hazard competing risk models for the UA and NHANES I. The sample was restricted to men with a known cause of death, excluding those who died of external causes such as accidents. The causes of death were classified into four categories: infectious diseases, cancer, cardiovascular diseases (CVD) and other.²² The tables also present the results for all-cause mortality.

All-cause mortality remains relatively similar to the results presented in Table 4 when restricting the sample to examinees with a known cause of death. The basic conclusion that men in the UA with a BMI of 25–30 have a higher risk than those with a BMI in the 20–25 range still holds, and the difference between these two categories in NHANES I is still non-existent with respect to mortality. In addition, men in NHANES I with extreme BMIs (below 20 and above 35) were at a higher relative risk of death than their UA counterpart. Note, however, that UA examinees with a BMI below 20 are not at a higher risk than the 20–25 group. Although the hazard ratio is larger than 1, indicating a higher probability of death, the low statistical significance makes the result questionable.

Death from infectious diseases declined substantially since the Civil War. As the epidemiologic transition progressed, the relative number of deaths from infectious diseases declined giving way to a higher percent of deaths from chronic degenerative diseases.²³ In the case of UA veterans with a known cause of death, 23% died from infectious diseases within the first 20 years after the medical examination. Comparatively, only less than 3% of NHANES I examinees died from such cause. Being underweight increased the odds of dying from infectious diseases in the past. Underweight veterans ($\text{BMI} < 20$) were twice as likely to die from infectious diseases than those in the 20–25 BMI range.²⁴ Underweight NHANES I

¹⁹The actual low risk range for NHANES I may in the 20–22 BMI range. For all men the hazard ratio ($\text{HR} = 1.14$) is relatively larger for this group than for the 24–24 BMI range, but the statistical significance is not very robust ($P > |z| = 0.497$). For white men only, however, the hazard ratio of the 20–22 MBI group is even larger ($\text{HR} = 1.36$) with a higher level of statistical significance ($P > |z| = 0.174$).

²⁰The results are very similar if the “normal” category runs from a BMI of 18.5 to 25 instead of 20–25. The main difference is that underweight men are at a higher risk when the limit is set to a BMI of 18.5 instead of 20.

²¹The recorded cause of death may have some accuracy problems especially in the UA cohort when the methods of diagnosis were not as precise as in the late 20th century. In addition, only the first cause of death was used and in many cases people have multiple causes of death.

²²Alternative causes of death were treated as right censored in the competing risk model.

²³The term “epidemiologic transition” was first introduced by Abdel Omran (1971).

examinees, on the other hand, were as likely to die than their 20–25 BMI counterpart.²⁵ In addition, men in both samples with a BMI of 25 or more had the same risk of death from infectious diseases as the 20–25 BMI group.

In the recent years, the association between adiposity and a higher risk of cancer has been well documented (Calle et al., 2003). UA veterans show a clear relationship between higher BMI and death from cancer. Veterans with a BMI of 25 or higher had a higher probability of death from cancer than those with a lower BMI, and the risk keeps increasing as BMI raises, such that veterans with a BMI between 30 and 35 were almost three times as likely to die from cancer than those in the 20–25 BMI range. On the other hand, having a BMI under 20 had no effect on the odds of cancer in the UA. For NHANES I having a BMI between 25 and 30 did not increase the probability of dying from cancer in comparison to the 20–25 BMI group. In addition, there was a weak relationship between having a BMI over 30 and death from cancer. For all men, having a BMI over 30 increased the risk of cancer as the cause of death. For non-Hispanic whites, a BMI over 30 increased the odds of death from cancer, however, the statistical significance is not as satisfactory to solidly claim a higher risk.²⁶ Yet, a BMI of 35 or more increased over three times the risk of death from cancer for non-Hispanic whites. Note also that for all men in NHANES I, the risk of death from cancer increased with a BMI under 20, an effect that was not clear for non-Hispanic men. Underweight men may have been more prone to death from cancer because of the disease itself. Given that many cancer patients' suffer from cachexia and, therefore, loose weight and body mass.

Several studies demonstrate that a higher fat concentration increases the risk of cardiovascular diseases (CVD) and death from CVD (Gordon and Kannel, 1976; Hubert et al., 1983; Tuomilehto et al., 1987; Lee et al., 1993; Calle et al., 1999; Jonsson et al., 2002). The link between obesity and CVD has been one of the main concerns among epidemiologists and public health specialists to recommend healthier BMI levels across populations. Accordingly, in the UA and NHANES I samples, the risk of death from CVD increases with higher levels of BMI. The risk of CVD death in both samples augments with a BMI of 25 or more. Furthermore, the risk intensifies as we move from a BMI category to a higher one. The effect of being underweight is not clear when we look at both samples. While in the UA, having a BMI under 20 reduced the risk of death from CVD, for NHANES I examinees there seems to be no consequence.²⁷

Over two fifths of the deaths catalogued as “other” in the UA were from respiratory diseases and over a quarter died from genitourinary problems. Digestive disorders caused close to 17% of these deaths. Comparatively, almost half of the deaths labeled as “other” in NHANES I were from diseases of the respiratory system and around a fifth from diseases of the digestive system. The other two leading causes in order of importance were endocrine, nutritional and metabolic diseases, and immunity disorders and diseases of the nervous system and sense organs. In the UA, men with a BMI of 25–30 were at the lowest risk of death from respiratory disease, while being underweight had no impact. In NHANES I men with a BMI 25–30 were also at the lowest risk of death from respiratory diseases. Men with a BMI under 20 were six times as likely to die from respiratory disease than those in the 20–25 BMI group, to a large degree explaining

²⁴The actual causality between those who had a low BMI and died of infectious disease is hard to predict, especially for those who died rapidly after the examination. Some may have had a low BMI because they were infected while some others may have been more susceptible to infection because of their low weight.

²⁵Note that the actual hazard ratios are substantially larger than 1 for NHANES I examinees with a BMI below 20, especially for non-Hispanic white men. Yet, it is hard to draw any conclusions given the statistical significance of the estimates.

²⁶The hazard ratio for all men in NHANES I with a BMI of 30 or above was 1.716 ($P > |z| = 0.050$) in comparison to those in the 20–30 BMI group. The hazard ratio for non-Hispanic white men with a BMI of 30 or larger was 1.457 ($P > |z| = 0.179$). The regression is available upon request.

²⁷Note that being underweight for non-Hispanic white men in NHANES I may increase the risk of death from CVD since the hazard was 1.723 ($P > |z| = 0.181$).

the huge hazard ratio for underweight examinees who died from “other” causes (Table 5b). BMI differences had no consequence on the odds of death from digestive disorders in either the UA or NHANES I.²⁸ Death from endocrine, nutritional and metabolic diseases, and immunity disorders increased substantially for veterans with a BMI of 20 or higher, and the risk increased with higher BMI levels, such that men with a BMI of 35 or higher were eight times as likely to die from these diseases than those in the 20–25 BMI range. Although a very small number of examinees ($n = 13$) in NHANES I died from endocrine, nutritional and metabolic diseases, and immunity disorders, underweight (BMI < 20) men and men with a BMI over 30 were at a much higher risk than the 20–25 BMI group.²⁹ Yet, the 25–30 BMI group was not at a higher risk. Finally, men with a BMI 35 or higher in the UA had a higher risk of death from genitourinary diseases. Underweight veterans were at a lower risk of death from this cause, particularly in the case of those examined after 1890. Very few examinees died in NHANES I from genitourinary diseases to make a reliable comparison.³⁰

5. Discussion

American white men living during the last third of the 20th century had different BMI cut-off risk limits than their counterpart at the end of the 20th century. Over the course of the past decades, the BMI cut-off points that define overweight and obesity may have grown, which in turn may imply a long-term secular shift toward healthier bodies at higher BMI levels. A plausible explanation for the expansion in BMI risk limits is the amount of body fat stored for the same BMI level. As stated earlier, BMI is used as a predictor of body fat concentration: higher amounts of adipose tissue increase the risks of morbidity and mortality. The association between BMI and percent body fat has been well documented in a number of studies (Womersley and Durnin, 1977; Norgan and Ferro-Luzzi, 1985; Garrow and Webster, 1985; Deurenberg et al., 1991; Gallager et al., 1996). Yet, different BMI levels may predict different body fat by gender and age. A second important finding is that extreme BMI's may be riskier in contemporary populations than they were in the past. In other words, Waaler curves may have steep ended in the modern era.

The upward shift in BMI risk limits over time for American men may be the result of changes in the human frame. While there is no way to test differences in body complexion beyond BMI during the late 19th century with the present sample, according to Costa (2004), since the 19th century the body frame of American men has increased in terms of height and weight.³¹ Yet, the amount of abdominal fat has decreased. In the past, men had higher concentrations of body fat for the same BMI than their contemporaries. In the case of NHANES I the evidence suggests that the amount of fat mass in the body decreased monotonically the probability of survival for the same BMI level. Conversely, the amount of fat free mass (mainly muscle) increased the probability of survival (Allison et al., 2002). Additionally, other anthropometric changes may have occurred such as a change in the relative leg length to frame size.³² Thus, making contemporaries leaner for the same BMI.

²⁸The risk of death from digestive disorders for all veterans was the highest for those with a BMI between 25 and 30, an effect that did not apply to those examined after 1890.

²⁹The effect of a BMI over 35 is unclear probably due to the low proportion of deaths from endocrine, nutritional and metabolic diseases, and immunity disorders.

³⁰The regressions for causes of death included in “other” are available upon request.

³¹The description provided by the boards of surgeons that examined the Union Army veterans provides some evidence of the adipose accumulation in some of the men with a BMI under 30. In some cases, the board of physicians described some of the general characteristics of the examinee. Surgeons described among other factors the complexion of the veterans, and in some cases made allusion to their weight and fat. Physicians were not required to describe the weight complexion of their subject, and therefore the record was not made systematically. We can find, however, a small number of references to veterans being “fat”, “obese” or with “abundance of adipose tissue”. Veterans that were described as “obese” had a significantly shorter lifespan than the rest of the population. On average they lived over 5 years less after the examination. Although the sample was very small altogether, most veterans described as “obese” had a BMI above 30, a few (24%) with a BMI between 25 and 30, and even that group died earlier than the rest.

The change in survivorship for similar BMI levels through time may respond to physiological transformations that are not determined by differences in ethnicity or genetics. Fogel and Costa coined the term technophysio evolution to describe the “synergism between technological and physiological improvements [that] has produced a form of human evolution that is biological but not genetic, rapid, culturally transmitted, and not necessarily stable” (Fogel, 2000b: 74). Technophysio evolution, unlike genetic theory, only applies to the past three 100 years of human history, and in particular to the past century when the human body increased in size and has become more robust with a stronger vital organ system.

One of the trademarks of such transformation is the increase in adult stature. Populations throughout the world have grown taller over the past three centuries as a reflection of better nutrition and less disease. Differences in body build and height can be appreciated across time for similar ethnicities. The populations of many countries have grown taller and gained body mass.³³ Changes in heights have been observed for populations that have had little genetic and ethnic change, such as in Sweden (Sandberg and Steckel, 1997).

Taller people tend to live longer because their adult stature is a product of their nutritional and disease history during their developmental years.³⁴ In a study of Norwegian mortality, Kim (1996) found that for the same BMI levels shorter people lived less.³⁵ A similar result can be found for the UA and NHANES I as shown in Tables 6a and 6b. Both tables present the absolute and relative risk of death within a 20-year interval by height and BMI for each sample respectively. The sample sizes are very small and therefore some of the cells have few observations or none. Nevertheless, it is possible to appreciate the general trend: taller men had a higher probability of survival for the same BMI range. For example, in Table 6a we can see that for UA veterans who had a BMI between 20 and 25 their risk of death decreased progressively for higher statures. Men who were between 1.5 and 1.6 m had a relative risk of death of 1.191, while men between 1.9 and 2 m had a relative risk of death of 0.819. Correspondingly, Table 6b shows a similar pattern for men within different groups of BMI.

During the 19th century, the American population was probably the tallest in the world. As Komlos and Baur (2004) point out, Americans did not grow much during the 20th century, especially in comparison to some European populations. Men in NHANES I were on average over 1.5 cm taller than the UA veterans, an increase in stature that explains a small proportion of survivorship differential.³⁶ The increase in mean height between both populations is larger when we look only at men ages 45–49. In this case, UA veterans were close to 2.5 cm shorter than NHANES examinees. Restricting the sample to this age group may give us a more accurate

³²Differences in relative leg length and frame size are such that two people may share the same amount of body fat, but have different leg sizes. Hence, the person with the shorter legs will have a higher BMI. On the other hand, two people can have the same BMI but one has a big frame and therefore low body fat, while the other has a small frame and a high body fat content (Deurenberg et al., 2002).

³³Adult height reflects the cumulative history of nutrition during developing years, while BMI responds to the current level of nutrition. A huge body of literature has developed over the past three decades on the changes in heights (see for example, Fogel, 1993; Komlos, 1994; Steckel and Floud, 1997). Studies in long-term trends in BMI are more scattered because the data on weight is harder to find. There is ample evidence, however, of growing BMIs in the United States, especially during the 20th century (see for example Costa and Steckel, 1997; Helmchen and Henderson, 2004).

³⁴The association between adult stature and survival has been found for past and present populations (see Alter, 2003 for a review). Some researchers, however, argue that shorter people are not healthier (Seckler, 1982).

³⁵Analogous results have been found across ethnicities that may be explained due to differentials in stature. In a comparative study, Gurrici et al. (1998) reported that Indonesians had more body fat at a lower BMI than Dutch Caucasians, and recommended that the cut-off points for obesity in Indonesia should be 27 kg/m² instead of 30 kg/m². It is worth mentioning that Indonesians were substantially shorter on average than the Dutch, one of the tallest contemporary populations. For instance Indonesian men (1.64 m) were 16 cm shorter than the Dutch (1.8 m), while Indonesian women were 14 cm shorter (1.53 and 1.67 m, respectively). The study was made with relatively low samples, and as the authors acknowledge “[t]he two study groups are difficult to compare with relation to socio-economic class, because of the different states of development in both countries.” (Gurrici et al., 1998, p. 781).

³⁶Around 3.4% of the increase in the probability of survivorship within the first 20 years can be explained by the increase in 1.5 cm between the UA and NHANES. The overall probability of death within the first 20 years declined from 0.489 (UA) to 0.396 (NHANES) —a 19% decrease. Additionally for both samples, an extra centimeter reduced the probability of death by ~0.003. So a 1.5 cm increase in mean height would explain around 3.4% of the change in survival probability.

picture of the change in height. Humans get shorter as they age, mainly because the cartilage between joints withers away. People begin to decrease in height during their 50s, and it is difficult to assume that everyone decreases evenly. So by looking at those men examined before age 50 we may be looking at a more accurate description of the adult heights of both cohorts.

While European populations grew substantially taller over the same time period, a 2.5 cm increase is far from negligible. Not only were the UA veterans shorter than NHANES I examinees, but they also had a more spread height distribution. The coefficient of variation, for instance, passed from 0.415 to 0.395. Fig. 7a and b present the density functions for both cohorts. Fig. 4a displays all men ages 45–64, while 4b restricts the sample to ages 45–49. As we can see, the overall NHANES I distribution shifted outward in comparison to the UA, especially for men ages 40–49.³⁷ The shortest 5% of this age group grew by more than 3.6 cm, while the median increased by 2.4 cm. Furthermore, the tallest 95% of men in NHANES were close to 3.8 cm bigger than their UA counterpart.

The mean height of UA veterans decreased as BMI categories increased, while the stature of NHANES I examinees remained more stable across BMI groups. Such difference in mean height by BMI group explains to some degree why the Waaler curves are getting steep ended in modern times. Further research has developed Waaler curves into Waaler surfaces where the simultaneous effect of BMI, height and weight on mortality can be analyzed (Fogel, 1993; Kim, 1996). A key finding from the Waaler surfaces is that the ideal BMI varies with height, such that shorter people tend to be healthier at higher BMIs. As Fogel explains in the case of Norwegians “[a] BMI of 25 is the ideal for men in the neighborhood of 176 cm (69 inches), but for short men (under 168 cm or 66 inches) the ideal BMI is about 26” (Fogel, 2004: 27). Hence stunted individuals—those with a low height for age— need a higher BMI to equalize the risk of death in comparison to taller people. Table 7 presents the mean height and weight by BMI category. The tallest UA army veterans were those with a BMI under 20 and mean height diminished as we move toward higher BMIs, such that those with a BMI of 35 or larger were almost 10 cm shorter than those with a BMI under 20. Comparatively, the differences in mean height across BMI groups for NHANES I examinees are smaller and not monotonically decreasing. So while the mean height for NHANES I examinees remains relatively constant across BMI groups, the height and BMI of UA veterans is inversely related. As a result, underweight veterans were slightly taller than underweight NHANES I examinees and obese veterans were shorter than obese NHANES I examinees. Thus extreme BMIs in the modern cohort are relatively riskier.

Another factor that may explain relative riskier extremes in NHANES I is the wider dispersion of BMI in the modern cohort, where a higher percent of the examinees are in the tails of the distribution, especially at the upper tail. We can also see in Table 7 that underweight men in NHANES I are around 3 kg lighter than UA veterans, while overweight NHANES I examinees are around 15 kg heavier than UA veterans with a BMI of 35 or more.

In addition to the overall increase in height it is important to consider that throughout the 19th century adult statures oscillated in the United States. In the three decades that preceded the Civil War, heights declined and mortality increased, while at the same time income per capita income grew rapidly. Some authors have named this paradox of rising incomes and decreasing health the “Antebellum Puzzle” (Steckel, 1995; Komlos, 1996; Haines, 1998). Heights declined during this era because of changes in diet and the deterioration of the disease environment due to urbanization, transport improvements and increased geographic mobility. Nonetheless, during the 20th century adult statures did not oscillate significantly. In fact, there is an upward trend in height for men ages 25–75 in NHANES I by birth cohort, such that men born in the

³⁷The UA height distribution is multimodal, maybe due to heaping. For example, the first mode is 1.524, which is equivalent to 5 feet.

late 1920s are about 1.4 cm shorter on average than men born in the late 1940s. The fact that men who grew up during the 19th century had stature oscillations shows that they grew up under more precarious conditions than men who were born in the 20th century.

UA veterans grew up under unhealthier conditions. A situation that not only reflects in height, but that may also produce less robust vital organs. Further evidence on the robustness of the body can be seen in the age of onset of disease, which has expanded over the past century (Fogel and Canavese, 2004). In other words, people who grew up under healthier conditions may have stronger muscle tissue, heavier organs, and a thus bigger frame for the same stature. As discussed earlier, people with a bigger frame tend to have lower adiposity for the same BMI. Additionally, some of the overweight people in the past may have also suffered from undernutrition, as it is the case today in some developing countries where obesity and undernutrition are found in the same households (Popkin, 2002).

An important component of human development extends back to the womb. The situation under which fetuses grow remains an important determinant of health in infancy and late in life. Some chronic conditions during adulthood, such as cardiovascular disease and non-insulin dependent diabetes, are highly determined by insults in utero.³⁸ The birth weight of term babies has been used as a proxy to in utero conditions and the health status of newborns. Middle-aged people who were born underweight tend to have less glucose tolerance, high blood pressure, and higher rates of obesity (Susser and Stein, 1994).³⁹ In addition, underweight newborns may have more adiposity accumulation as adults (Yajnik, 2004).

There is no information on birth weights during the first half of the 19th century in the United States. Since the mid-19th century, scattered data show that mean birth weights have remained fairly constant (Goldin and Margo, 1989; Ward, 1993; Costa, 1998). The percent of underweight children, however, decreased almost by half between the third quarter of the 19th century and the first third of the 20th century. In their study of newborns in Philadelphia's Almshouse Hospital, Goldin and Margo (1989) found that 10% of the babies born between 1848 and 1873 were underweight. Additionally, there was a sharp decrease in birth weights for those born between 1866 and 1870. A sign that birth weights also oscillated during the 19th century. Dora Costa argues that 5.5% of the children born in New York's Lying-In between 1910 and 1931 had low weight.⁴⁰ While the mean birth weight did not increase considerably ever since, the variance has declined. Disparities in weight between newborns have always been small, but they were larger at the beginning of the 20th century (Costa, 1998).

Given the trends into the past, it is likely that children born during the first half of the 19th century had lower birth weights, had a higher prevalence of low birth weight and more dispersion in birth weight than those born a century later.⁴¹ UA army veterans, therefore, were born and grew up under less favorable conditions than NHANES I examinees. During the 1840s, a period when a large number of UA veterans were born, available proteins and calories per capita fell even though agricultural output was growing (Haines et al., 2000).⁴² In addition,

³⁸The "fetal origins" hypothesis, advanced by Barker (1993, 1997, 1998), suggests that conditions in utero (most likely nutritional) will affect the development of chronic conditions later in life. Barker's hypothesis is still controversial, for a review see Rasmussen (2001).

³⁹The World Health Organization considers babies born under 2.5 kg as underweight (WHO, 1995), but the measure may be different across time.

⁴⁰For children born in Boston Lying-In between 1884 and 1900, from 4.7 to 6.9 were underweight (Ward, 1993).

⁴¹Children born in the 19th century may have been born to shorter mothers than those born a century later. Babies born to shorter women tend to have lower birth weight than those born to taller women. Lagiou et al. (2003) argue that children born from Caucasian American women tend to weigh more than children born out of Chinese mothers. In both cases the height of the mother is positively related to the birth weight of babies, and American mothers are significantly taller than their Chinese counterpart. In addition, Chinese women who migrate to the United States tend to grow taller, heavier, and have higher weighting children.

⁴²Part of the growth in agricultural output went to non-food cash crops, such as cotton. Komlos (1987) argues that the relative price of food increased after 1940.

from 1830 to the Civil War the health environment deteriorated as heights and life expectancy declined (Steckel, 1992).

There are of course other factors beyond the developmental years that may explain the upward shift in BMI risk limits. The United States changed spectacularly during the past 200 years. UA veterans lived in a very different world than NHANES examinees did. Lifestyle, diet and the health environment were very different in both time periods. Occupations mechanized, hours of work throughout the life cycle declined, and people use their leisure time very differently.⁴³ As the United States got richer and technology advanced, the amount of food per capita increased and its flow became more constant; it got cheaper, cleaner and healthier. Technological changes such as refrigeration reduced the consumption of salted foods, an important source of hypertension in the past. Cleaning water supplies and installing sewage systems made people less vulnerable to infectious disease. Medical technology also transformed dramatically during the 20th century. Among other developments, NHANES I examinees had the availability of drugs and medical procedures to treat many conditions and reduce the adverse effect of higher BMIs.⁴⁴

6. Conclusion

The body mass index (BMI) is commonly used as a measure of current nutritional level in humans. Very high and very low BMIs increase the likelihood of morbidity and mortality. High BMIs are often used as predictors of overweight and obesity. Experts around the world have debated over the past years whether or not the same set of risk BMI cut-off points can be applied across populations and ethnicities. As the present paper shows, the cut-off points may be shifting across time. The range of healthiest BMI for American men born during the first half of the 19th century was around two BMI points below those born during the first half of the 20th century. The minimum risk for UA veterans was situated in the 20–26 BMI span, while the minimum risk for NHANES I examinees appeared in the 22–28 BMI range. The findings may imply that differences in BMI risk cut-off points are not only cross ethnic, but also occur across time for similar ethnicities. The dynamics over time in cut-off points suggests that experts should revise the risk limits on a periodic basis in order to transmit the best available information to policy makers and the population.

A plausible explanation for the upward shift in BMI risk cut-off points may be a difference in physiology between both cohorts. In comparison to the NHANES men, a higher proportion of UA veterans may have been born underweight, grew up in unhealthier environments and had less nutrients during their developmental years. As a result, UA veterans were shorter in stature and had less robust vital organs, which may have led to a smaller body frame. People with smaller body frames tend to accumulate more fat for the same BMI than people with larger body frames.

The results by no means imply that we should stop worrying about the obesity epidemic experienced in the United States and the rest world over the past decades, given that not only the mean and median BMIs have increased but also the proportion of people with extreme

⁴³As a proxy of adiposity, BMI may not reflect some of the changes in fat accumulation over time resulting from changes in the structure of labor and a transition toward less physically demanding jobs.

⁴⁴As a result of all these transformations, life expectancy for men aged 50 increased by more than 17% from 20.54 to 24.11 years from 1900 to 1975, respectively. (Bell et al., 1992; Human Mortality Database, 2005). The life expectancy of UA veterans may have been somewhat larger than the rest of the population. On average men aged 50 in 1900 lived 22.75 years more. In addition, due to many of these improvements the disease pattern and the causes of death have changed substantially. The transformation of the disease pattern makes the comparison across ethnicities complicated. As disease patterns change over time, we may only be able to infer that diseases vary across ethnicities at a particular point in time.

obesity has risen dramatically. In addition, obesity and extreme obesity have grown at alarming rates since NHANES I was conducted.

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Appendix A

See Tables A.1– A.5.

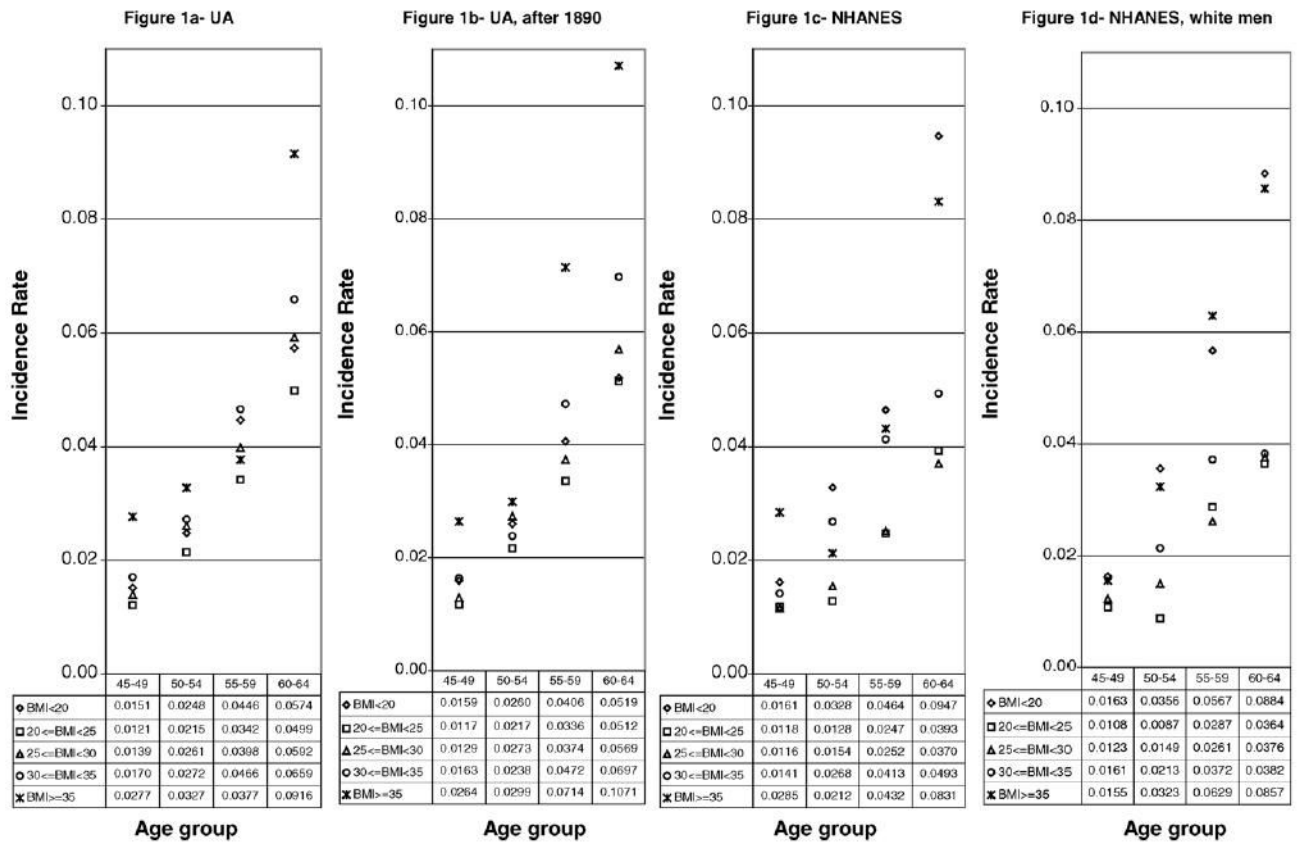


Fig. 1. Incidence rate of death for UA and NHANES I men by BMI and age groups (censored 20 years after medical examination). Note: The incidence rate was calculated as the number of deaths/person-years.

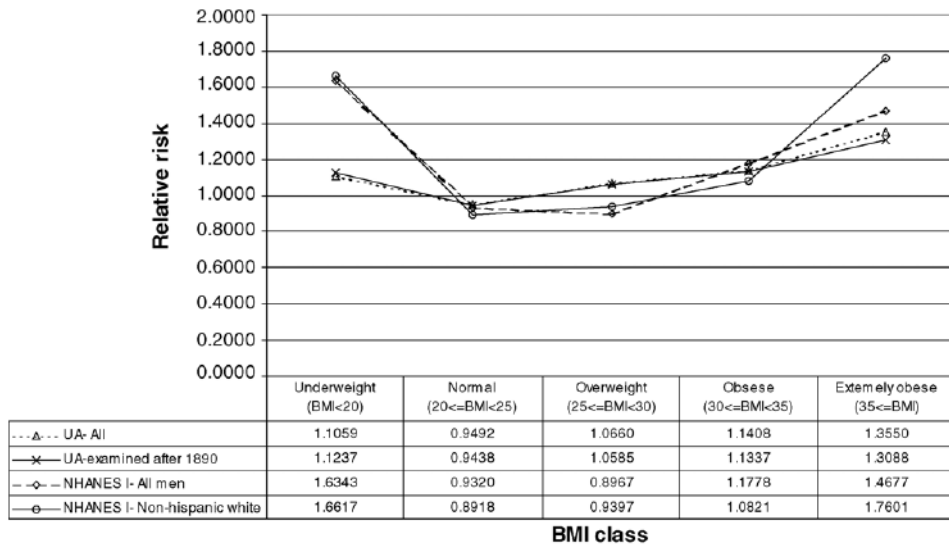


Fig. 2. Relative risk of death within 20 years of examination for men ages 45–64, 1870–1992.

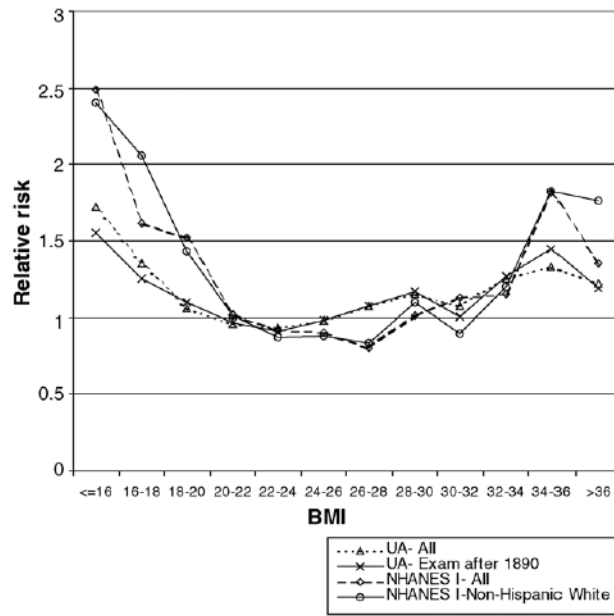


Fig. 3. Relative risk of death within 20 years of exam by BMI for men ages 45–64.

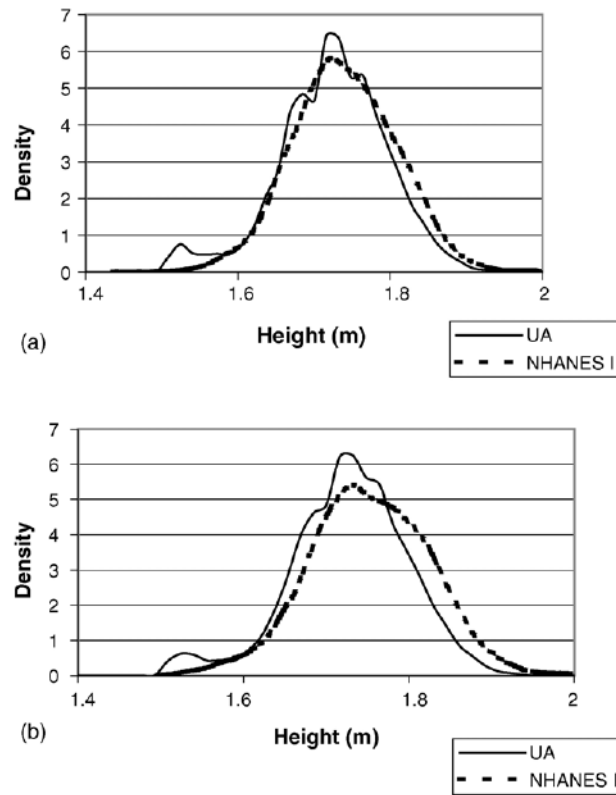


Fig. 4. (a) Kernel density functions of height in the UA and NHANES I for men ages 45–64. (b) Kernel density functions of height in the UA and NHANES I for men ages 45–49.

Table 1

Classification of desirable weight levels as assessed by the BMI according to selected expert panels

Country/ region	Panel	Gender	Group	Date	BMI recommendations		
					Normal	Overweight	Obese
Asia	IOTF/IASO/ WHO (a)	Both	Adults	2000	18.5–22.9	23–24.9	≥25
Australia	NHMRC (b)	Both	Adults	1997		25–30	≥30
Canada	Health and Welfare (c)	Both	Adults	1988	20–25	25–27	≥27
Canada	Health and Welfare (d)	Both	Adults	2003	18.5–24.9	25–29.9	≥30
Mexico	SSA (e)	Female	Adults with height ≥ 1.5 m	1998		25–27	≥27
		Female	Adults with height < 1.5 m			23–25	≥25
		Male	Adults with height ≥ 1.6 m			25–27	≥27
		Male	Adults with height < 1.6 m			23–25	≥25
United States	NIH/NHLBI/ NASSO (f)	Both	Adults	2000	18.5–24.9	25–29.9	≥30
United States	National Research Council (g)	Both	Ages 19–34	1989	19–25		
			Ages 35+ Adults		21–27 18.5–25	25–30	≥30
United States	USDA/ DHHS (h)	Both	Adults	2000	18.5–24.9	25–29.9	≥30
United States	CDC (i)	Both	Adults	2004	18.5–24.9	25–29.9	≥30
World	WHO (j)	Both	Adults	1998	18.5–24.9	25–29.9	≥30

Sources: (a) IOTF (2000); (b) NHMRC (1997); (c) Health and Welfare Canada (1988); (d) Health and Welfare Canada (2003); (e) Secretaría de Salubridad y Asistencia (1998); (f) NIH (2000); (g) NRC (1989); (h) USDA/USDH (2000); (i) CDC (2004); and (j) WHO (1998).

Table A1

Age distribution in UA and NHANES I samples

Age group	UA (<i>n</i> = 9509) (%)	NHANES I (<i>n</i> = 1912) (%)
45–49	38.4	28.4
50–54	29.3	26.6
55–59	19.7	25.8
60–64	12.6	19.2

Table 2

NIH recommendations for weight classification

Type	BMI
Underweight	Below 18.5 (to 20 following Garrow)
Normal	18.5–24.9
Overweight	25–29.9
Obesity class I (high)	30–34.9
Obesity class II (very high)	35–39.9
Obesity class III (extremely high)	40 and above

Source: NIH, 2004.

Table A2

Descriptive statistics of age and BMI at measurement for UA veterans by sample

Variable	All (<i>n</i> = 9509)		With recorded cause of death within 20 years after exam (<i>n</i> = 6345)	
	Mean	S.D.	Mean	S.D.
Age at measurement	52.2	5.3	51.2	4.9
BMI	23.3	3.3	23.2	3.2

Table 3
Cox Proportional Hazard Model- Output variable: years lived at first examination (censored at 20 years)

Covariates	UA- All		UA- Exam after 1890		NHANES I- All		NHANES I-Non-Hispanic white	
	Haz. Ratio	$P > z $	Haz. Ratio	$P > z $	Haz. Ratio	$P > z $	Haz. Ratio	$P > z $
Age	1.0892	0.0000	1.0893	0.0000	1.0936	0.0000	1.0938	0.0000
BMI dummies = 1 if								
BMI < 20	1.2021	0.0000	1.1688	0.0210	2.1304	0.0000	2.3955	0.0000
25 ≤ BMI	1.1454	0.0000	1.1332	0.0100	1.0385	0.7210	1.1287	0.3490
< 30								
30 ≤ BMI	1.2635	0.0020	1.2656	0.0100	1.6035	0.0020	1.4890	0.0370
< 35								
35 ≤ BMI	1.6629	0.0010	1.8372	0.0000	1.9807	0.0030	2.5657	0.0000
n	9444		4817		1871		1219	
Prob > χ^2	0.0000		0.0000		0.0000		0.0000	

Note: $P > |z|$ calculated using robust standard errors. Omitted category $20 \leq \text{BMI} < 25$.

Table A3

Birthplace distribution of UA veterans examined between the ages of 45 to 64 by sample

Birthplace	All (n = 9509) (%)	With recorded cause of death within 20 years after exam (n = 6345) (%)
United States	78.2	80.7
Canada	2.7	2.8
British	3.0	2.7
Irish	4.9	4.0
European other	9.2	8.4
Foreign other	0.1	0.1
Missing	1.9	1.3

Table 4a

Cox proportional hazard model on ua veterans—output variable: years lived after first examination (censored at 20 years)

Covariate	All				Examined after 1890			
	Ages 45–54		Ages 55–64		Ages 45–54		Ages 55–64	
	Haz. ratio	$P > z $	Haz. ratio	$P > z $	Haz. ratio	$P > z $	Haz. ratio	$P > z $
Age	1.1017	0.0000	1.0783	0.0000	1.1086	0.0000	1.0791	0.0000
BMI dummies = 1 if:								
BMI < 20	1.1887	0.0060	1.2168	0.0020	1.2536	0.0200	1.1004	0.3020
25 ≤ BMI < 30	1.1507	0.0050	1.1416	0.0130	1.1641	0.0250	1.1027	0.1560
30 ≤ BMI < 35	1.2420	0.0260	1.2952	0.0230	1.1778	0.2180	1.3582	0.0180
35 ≤ BMI	1.7714	0.0020	1.5355	0.0370	1.6681	0.0120	2.3045	0.0020
n	6404		3040		3128		1689	
Prob > χ^2	0.0000		0.0000		0.0000		0.0000	

Note: $P > |z|$ calculated using robust standard errors. Omitted category $20 \leq \text{BMI} < 25$.

Table 4b

Cox proportional hazard model on NHANES I examinees—output variable: years lived after first examination (censored at 20 years)

	All				Non-Hispanic white			
	Ages 45 to 54		Ages 55 to 64		Ages 45 to 54		Ages 55 to 64	
	Haz. ratio	$P > z $	Haz. ratio	$P > z $	Haz. ratio	$P > z $	Haz. ratio	$P > z $
Age	1.0465	0.1000	1.0915	0.0000	1.0309	0.3890	1.0674	0.0070
BMI dummies = 1 if	1.9569	0.0090	2.2985	0.0000	2.2585	0.0220	2.3479	0.0000
BMI < 20								
25 ≤	1.0965	0.6050	0.9741	0.8450	1.3999	0.1400	0.9729	0.8650
BMI < 30								
30 ≤	1.6866	0.0280	1.5498	0.0340	1.9366	0.0300	1.2380	0.3850
BMI < 35								
35 ≤ BMI	1.9080	0.0970	2.1125	0.0090	2.7338	0.0200	2.6932	0.0010
n	1035		836		674		545	
Prob > χ^2	0.0174		0.0000		0.0633		0.0000	

Note: $P > |z|$ calculated using robust standard errors. Omitted category $20 \leq \text{BMI} < 25$.

Table A4
 Absolute mean risk of death within 20 years of examination by BMI class for men ages 45–64

BMI class	UA						NHANES I					
	95% CI			Examined after 1890			All			Non-Hispanic white		
	Mean	LB	UB	Mean	LB	UB	Mean	LB	UB	Mean	LB	UB
Underweight (BMI < 20)	0.5401	0.5109	0.5693	0.5614	0.5176	0.6051	0.5920	0.4754	0.7087	0.5874	0.4513	0.7235
Normal (20 ≥ BMI < 25)	0.4636	0.4511	0.4760	0.4715	0.4537	0.4893	0.3376	0.2908	0.3844	0.3153	0.2568	0.3737
Overweight (25 ≥ BMI < 30)	0.5206	0.4976	0.5436	0.5288	0.4987	0.5589	0.3248	0.2755	0.3742	0.3322	0.2836	0.3808
Obese (30 ≥ BMI < 35)	0.5571	0.5048	0.6094	0.5664	0.5013	0.6315	0.4266	0.3346	0.5187	0.3825	0.2890	0.4761
Extremely obese (35 ≥ BMI)	0.6618	0.5464	0.7771	0.6538	0.5201	0.7876	0.5317	0.3561	0.7072	0.6222	0.3921	0.8523
Sample mean	0.4884	0.4783	0.4984	0.4996	0.4855	0.5137	0.3622	0.3288	0.3957	0.3535	0.3179	0.3891

Table 5b
 d competing risk model on NHANES I examinees—output variable: years lived after first examination (censored at 20 years)

Case	Infectious			Cancer			CVD			Other								
	Non-Hispanic white			All			Non-Hispanic white			All								
	<i>P</i> > <i>z</i>	Haz. Ratio	<i>P</i> > <i>z</i>	Haz. Ratio	<i>P</i> > <i>z</i>	Haz. Ratio	<i>P</i> > <i>z</i>	Haz. Ratio	<i>P</i> > <i>z</i>	Haz. Ratio	<i>P</i> > <i>z</i>	Haz. Ratio						
0.0000	1.0933	0.0000	1.1127	0.0810	1.0509	0.4030	1.0800	0.0000	1.0855	0.0000	1.0809	0.0000	1.0875	0.0000	1.1442	0.0000	1.1261	0.0000
0.0000	2.4410	0.0000	1.6478	0.5720	2.3918	0.3520	1.7288	0.0960	1.5552	0.3360	1.1770	0.6410	1.7232	0.1810	4.6021	0.0000	4.7269	0.0000
0.7500	1.1266	0.3710	0.8003	0.7400	0.8529	0.8300	0.9786	0.9120	1.0343	0.8850	1.3935	0.0430	1.6883	0.0110	0.5725	0.0210	0.5444	0.0430
0.0020	1.5225	0.0300	1.1492	0.9020	6.1E-17	0.0000	1.6716	0.1160	1.2426	0.5390	2.0670	0.0010	2.6608	0.0000	0.9312	0.8630	0.5704	0.4180
0.0020	2.6775	0.0000	1.6E-15	0.0000	5.8E-17	0.0000	1.8234	0.1970	3.0379	0.0170	3.3098	0.0000	4.0046	0.0000	0.5452	0.3370	0.7450	0.6830
1197	1197	1197	1837	1197	1197	1197	1837	1837	1197	1197	1837	1837	1197	1197	1837	1837	1197	1197
0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000	0.0000

ed using robust standard errors. Omitted category 20 ≤ BMI < 25.

Table A5

Cox proportional hazard model—output variable: years lived after first examination (censored at 20 years)

Covariates	UA—all		UA—exam after 1890		NHANES I—all		NHANES I—non-Hispanic white	
	Haz. ratio	$P > z $	Haz. ratio	$P > z $	Haz. ratio	$P > z $	Haz. ratio	$P > z $
Age	1.0890	0.0000	1.0893	0.0000	1.0938	0.0000	1.0957	0.0000
BMI dummies = 1 if:								
BMI < 16	2.9114	0.0000	1.9354	0.4470	6.8610	0.0000	7.8163	0.0000
16 ≥ BMI	1.4999	0.0000	1.2942	0.1590	2.4380	0.0090	3.7987	0.0000
< 18								
18 ≥ BMI	1.1236	0.0460	1.1404	0.1090	1.7489	0.0020	1.9055	0.0060
< 20								
20 ≥ BMI	1.0004	0.9930	1.0239	0.7090	1.1353	0.4970	1.3552	0.1740
< 22								
22 ≥ BMI	0.9645	0.4380	0.9583	0.4920	0.9595	0.7890	1.0045	0.9820
< 24								
26 ≥ BMI	1.1606	0.0160	1.1901	0.0250	0.8818	0.4000	1.0222	0.9040
< 28								
28 ≥ BMI	1.2784	0.0020	1.3262	0.0030	1.3456	0.0690	1.6023	0.0160
< 30								
30 ≥ BMI	1.1666	0.1390	1.1073	0.4310	1.5368	0.0520	1.2412	0.4000
< 32								
32 ≥ BMI	1.3918	0.0110	1.4020	0.0390	1.5657	0.0590	1.8426	0.0420
< 34								
34 ≥ BMI	1.4546	0.0210	1.8195	0.0000	2.2698	0.0040	2.6554	0.0130
< 36								
36 ≥ BMI	1.5300	0.0370	1.7722	0.0130	1.9229	0.0170	2.9268	0.0000
n	9444		4817		1871		1219	
Prob > χ^2	0.0000		0.0000		0.0000		0.0000	

Note: $P > |z|$ calculated using robust standard errors. Omitted category 24 ≥ BMI < 26.

Table 6a
Risk of death within 20 years of examination (UA—all) men ages 45–64

Height (m)	BMI (kg/m ²) class					Total
	BMI < 20	20 ≤ BMI < 25	25 ≤ BMI < 30	30 ≤ BMI < 35	35 ≤ BMI	
<1.50		1.000	0.000	0.000	1.000	0.571 1.170
	0	3	1	2	1	7
1.5	0.545	0.582	0.516	0.495	0.458	0.525
	1.117	1.191	1.057	1.014	0.938	1.076
	11	110	184	103	24	432
1.6	0.609	0.506	0.557	0.719	0.750	0.532
	1.247	1.036	1.139	1.472	1.536	1.090
	248	1692	469	64	12	2485
1.7	0.524	0.445	0.511	0.524	0.786	0.471
	1.072	0.911	1.046	1.073	1.609	0.963
	634	3451	953	145	28	5211
1.8	0.533	0.440	0.481	0.636	0.667	0.467
	1.091	0.901	0.984	1.303	1.365	0.956
	214	879	206	33	3	1335
1.9	0.231	0.400	0.833	0.333		0.405
	0.473	0.819	1.706	0.683		0.830
	13	15	6	3	0	37
≥2	0.000					0.000
	0.000					0.000
	2	0	0	0	0	2
Total	0.540	0.464	0.521	0.557	0.662	0.488
	1.106	0.949	1.066	1.141	1.355	1.000
	1122	6150	1819	350	68	9509

Note: Each cell gives the 20-year relative risk of mortality, 20-year (absolute) mortality risk, and the number of observations.

Table 6b
Risk of death within twenty years of examination (NHANES I)- men ages 45–64

Height (m)	BMI (kg/m ²) class					Total
	BMI < 20	20 ≤ BMI < 25	25 ≤ BMI < 30	30 ≤ BMI < 35	35 ≤ BMI	
<1.50	1.000	1.000				1.000
	2.761	2.761				
1.5	1	1	0	0	0	2
	0.648	0.043	0.550	0.424		0.442
1.6	1.790	0.119	1.518	1.171	0.000	1.221
	6	8	17	8	1	40
1.7	0.716	0.429	0.353	0.341	0.000	0.396
	1.975	1.185	0.974	0.943	0.000	1.094
1.8	32	150	227	61	11	481
	0.545	0.328	0.326	0.518	0.336	0.370
1.9	1.506	0.904	0.900	1.430	0.927	1.022
	64	369	446	120	28	1027
≥2	0.497	0.286	0.281	0.265	0.567	0.298
	1.373	0.790	0.775	0.731	1.566	0.822
Total	22	129	143	42	7	343
	0.000	0.187	0.000	0.166	0.855	0.091
Total	0.000	0.516	0.000	0.459	2.359	0.251
	1	9	6	3	0	19
Total	0	0	0	0	0	0
	0.592	0.338	0.325	0.427	0.532	0.362
Total	1.634	0.932	0.897	1.178	1.468	1.000
	126	666	839	234	47	1912

Note: Each cell gives the 20-year relative risk of mortality, 20-year (absolute) mortality risk, and the number of observations.

Table 7

Mean height (m) and weight (kg) by BMI class

BMI class	UA				NHANES I			
	All		After 1890		All		Non-Hispanic white	
	Height	Weight	Height	Weight	Height	Weight	Height	Weight
BMI < 20	1.742	57.587	1.738	57.381	1.723	54.713	1.721	54.690
20 ≤ BMI < 30	1.729	66.885	1.726	66.891	1.750	70.330	1.753	70.657
25 ≤ BMI < 30	1.710	78.322	1.715	78.928	1.742	82.627	1.744	82.896
30 ≤ BMI < 35	1.673	89.628	1.679	90.317	1.734	96.046	1.736	96.119
35 ≤ BMI	1.653	103.853	1.644	102.800	1.732	117.304	1.734	115.201