Review

Longevity, mortality and body weight

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Abstract

The purpose of this study was to analyze the relation of total body weight to longevity and mortality. The MEDLINE database was searched for data that allow analysis of the relationship between absolute body weight and longevity or mortality. Additional data were used involving US veterans and baseball players. Trend lines of age at death versus body weight are presented. Findings show absolute body size is negatively related to longevity and life expectancy and positively to mortality. Trend lines show an average age at death versus weight slope of -0.4 years/kg. We also found that gender differences in longevity may be due to differences in body size. Animal research is consistent with the findings presented. Biological mechanisms are also presented to explain why increased body mass may reduce longevity. Life expectancy has increased dramatically through improved public health measures and medical care and reduced malnutrition. However, overnourishment and increased body size have promoted an epidemic of chronic disease and reduced our potential longevity. In addition, both excess lean body mass and fat mass may promote chronic disease.
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1. Introduction

In the US, obesity has increased by 30% over the last half century (Willett et al., 1999). American males are now about 25 kg heavier than during World War I. Increasing body fat has been targeted as the primary cause of this trend and efforts have focused on weight reduction methods through diet and exercise. However, obese people also have much higher amounts of lean body mass (LBM) compared with normal weight people (Knittle et al.,

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1979). LBM is subject to increased free radical damage, increased cancer risk and requires more maintenance (Albanes, 1987; Fossel, 1996; Samaras, 1996). western scientists have viewed larger body size as a desirable byproduct of superior nutrition and a higher standard of living. On the other hand, some Asian countries averaging smaller body size have the greatest longevity in the world despite harmful health practices (smoking, drinking, pollution, congestion, and high salt intake). In spite of many of these negative factors, the smaller mainland Japanese, Okinawan Japanese, Shanghai Chinese and Hong Kong Chinese have excellent longevity, with Okinawa exhibiting the highest proportion of centenarians (34/100,000) in the world (Samaras and Elrick, 1999; Willcox et al., 2001). In addition, smaller southern Europeans, such as the long-living Cretans have 1/2 the all-cause and <1/20 coronary heart disease (CHD) mortality of larger northern Europeans (Keys et al., 1984).

Based on research conducted over the last 25 years, we have concluded that larger body size, not just percent body fat, is a health problem. This proposed risk factor has been ignored in the past because larger body size correlates with higher life expectancy and standard of living, improved public health measures, and advances in medical technology and treatment. Increasing obesity also correlates with these advances but presents a more easily identified cause of poor health in middle and advanced ages. We believe that the following longevity findings raise doubts about the benefits of promoting greater body size.

2. Material and methods

The MEDLINE data base was used to conduct a literature search on the relation between body size and longevity. Data obtained from the Veterans Administration in San Diego and other studies published by the authors were used to evaluate the relation between body weight and longevity. Data from various sources were converted into trend plots of body weight versus longevity and mortality.

3. Results

This section includes findings relating to longevity and weight for baseball and football players, Finnish athletes, veterans, centenarians, Chinese rural residents, six ethnic groups in California, and southern Europeans.

3.1. Baseball player longevity versus weight

Weight and longevity data on 1278 deceased professional baseball players were analyzed (Samaras and Storms, 1992). Height and body mass index (BMI, kg/m^2) were used to compute weight using the formula: weight (kg) $= BMI \times height^2$ (m^2). Fig. 1 shows the trend line for average age at death versus body weight. A slope of -0.40 years/kg was produced. The correlation coefficient (r) was -0.22, $P < 0.025$. The BMIs for the heaviest players did not show any significant increase compared with the smallest players (see Fig. 1).
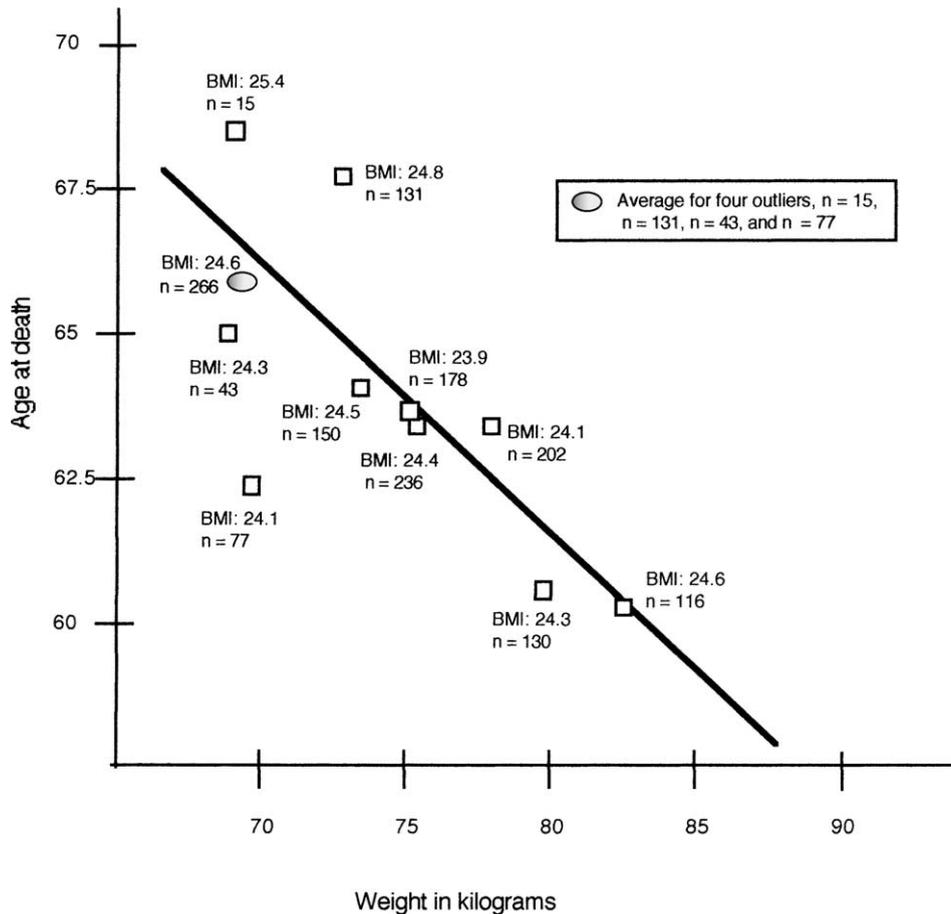
Fig. 1. Decrease in average life span of baseball players with increasing absolute body weight.

Because changes in body weight with age were not given in our source, we used weight during youth as a predictor of a shorter life span in middle age and beyond.

3.2. Football player longevity versus weight

Weight and longevity data for 199 deceased professional football players were analyzed (Maher and Gill, 1997). Weights were grouped into quartiles and a trend line (not shown) through each weight quartile revealed a linear decline in age at death with increasing body weight with a negative slope of -0.55 years/kg. The correlation coefficient between age at death and weight was -0.33, $P < 0.005$. The biggest players had an average BMI = 31.9 versus the lightest players who averaged 24.9. Since changes in weight after retirement were not given, the findings apply only to the relationship between youthful weight and future longevity.

3.3. Finnish elite athletes longevity versus weight

Evaluation of data on elite Finnish athletes revealed a declining life expectancy with increasing body weight. Weight data were provided when the athletes were in their 50s. The subjects included 10 categories grouped under three general athletic categories: endurance, team, and power. A regression analysis based on data by Sarna et al. (1993) and e-mail correspondence (4/6/99) revealed an $r = -0.51$ for body weight versus average life expectancy. However, the result was non-significant (NS). The BMIs varied from 24 to 28; a regression analysis of BMI versus average life expectancy provided an $r = -0.49$, NS.

3.4. Veterans longevity versus weight

Weight and longevity data for 373 male veterans provided a negative trend line (not shown) between age at death for increasing weight quartiles. The slope was -0.22 years/kg. The correlation coefficient was -0.203, $P < 0.001$. The deceased men ranged in age from 29 to 97 years. The data were collected at the San Diego Veterans Administration in San Diego, CA (Samaras and Storms, 1992).

3.5. Centenarian body size versus longevity

Roth reported that centenarians in the past have been small (Roth, 1994). He also observed that individuals with extreme physical development do not live very long. For example, football players, body builders, and Sumo wrestlers have shorter lives than the average person. A study of Hungarian centenarians found them to be very small (Eiben and Bodzsar, 1990): men averaged 153.6 cm and 55.6 kg and females 142.2 cm and 46.3 kg. The BMI for men was 23.4 and for women, 22.4. Another study found that Italian centenarian males were 61.4 kg and females 55.5 kg (Paolisso et al., 1995). Based on a study of centenarians in Los Angeles, CA, Klatt and Meyer (1987) reported that males averaged 53 kg and females 43 kg.

The Okinawans are noted for their high percentage (34/100,000) of centenarians and are small compared with mainland Japanese and westerners. Okanawan and mainland Japanese centenarians males average 44.1 kg and females 36.7 kg (Kagawa, 1978). Most Okinawan centenarians do not have any health problems during most of their lives based on numerous studies (Willcox et al., 2001; Kagawa, 1978). In contrast, British nobility have generally been taller and heavier than the working class population but nobility produced only one centenarian over a period of 1000 years. Queen Elizabeth is a rare exception at 100-year-old and was relatively small during her youth.

3.6. China study mortality by weight

In a study of 65 rural Chinese counties and 130 villages involving 6500 people, Chen et al. (1990) found that larger adult body size was significantly correlated with mortality due to degenerative diseases, such as heart disease and cancer, but inversely related to communicable diseases. They reported a weight-mortality $r = 0.39$, $P < 0.01$ for a combined myocardial infarction and CHD (both sexes). The weight-mortality correlation for all types of cancer was $r = 0.47$, $P < 0.001$ for both sexes.
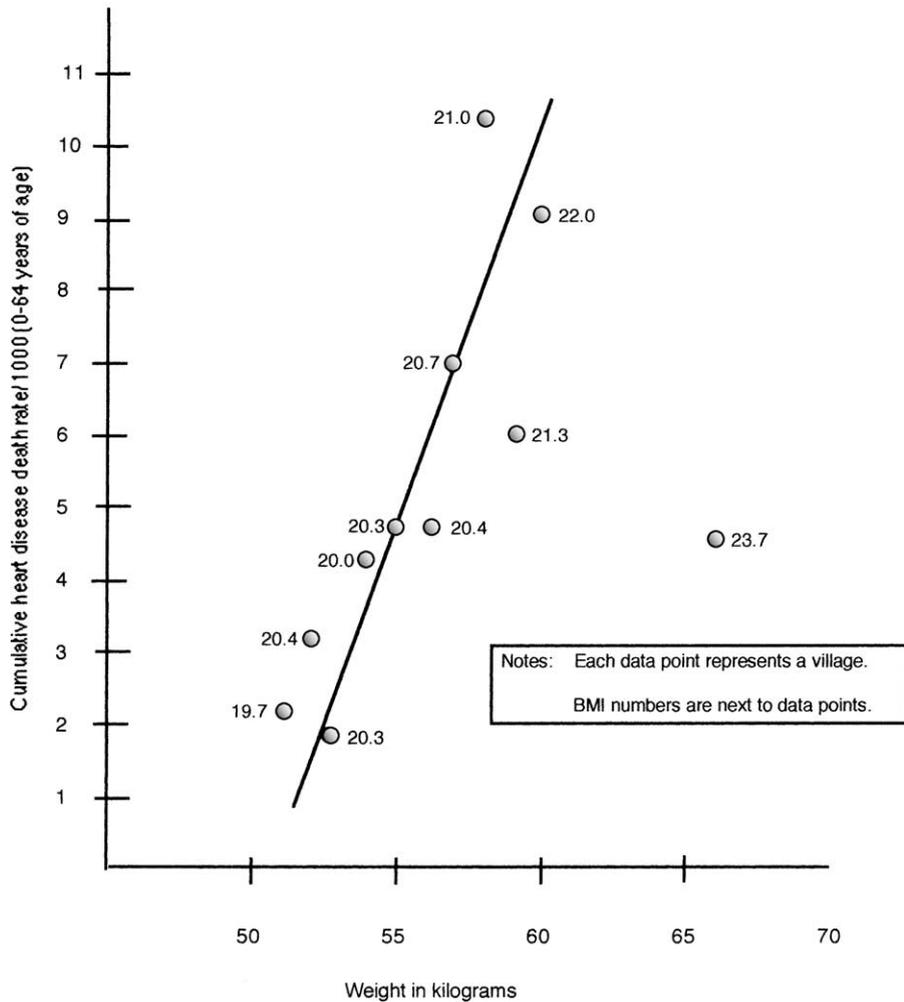
Fig. 2. Chinese male heart disease mortality with increasing weight ($n = 1125$).

Chen et al. (1990) provided the weights of men and women against cumulative heart and cancer disease for the age group 0–64 years of age. We plotted the death rate against male body weight for heart and cancer disease. Fig. 2 shows the trend line for heart disease only; the cancer trend line was similar. Inspection of Fig. 2 indicates that BMI does not confound the impact of increasing weight on heart disease.

3.7. California ethnic group mortality by weight

Wild et al. (1995) provided data on 1 million deaths in California between 1985 and 1990. Six ethnic groups aged 25–84 years were studied and death rates for all causes, CHD,

stroke, and other causes were determined. Weight data were obtained from the California Department of Health (Igra et al., 1982); however, East Indian height and weight were not available for California; therefore, other sources were used (Singh et al., 1997; Kamath et al., 1999). Fig. 3 shows the trend line for all-cause age standardized death rates versus male weight. The BMI's for each group are also shown next to each data point in Fig. 3. While the highest BMI's have the highest all-cause death rates, the difference with the lowest death rates is only about two points. Since mortality increases linearly with BMI (Willett et al., 1999; Samaras and Elrick, 1999), the sharp mortality increase in whites and African Americans does not appear to be due only to increasing BMI. A BMI and weight comparison of Hispanics versus Indians, Chinese and Japanese combined showed Hispanics with a 13% increase in BMI and a 19% increase in weight versus a 20% higher death rate. Thus, the increase in mortality followed the increase in weight more closely than the increase in BMI.

Some argue that migrants to developed countries are healthier than those remaining in their homeland. This belief originated from observations that migrants often have lower mortality compared with the general population of host countries. However, researchers have not found the "healthy migrant effect" to be true for Vietnamese migrants to England (Swerdlow, 1991) or for Latino migrants to the US (Abraido-Lanza et al., 1999). While migrants to the US often have low mortality, Micozzi (1993) reported that Mexican and Japanese Americans show increasing rates of chronic diseases over succeeding generations in parallel with increasing childhood and adult body size in contrast to their parents relative freedom from these diseases.

3.8. Singapore ethnic weight and mortality

Singapore consists of three principal ethnic groups: Chinese, Malay, and East Indian. Mortality data were obtained from Hughes et al. (1990). Weight data were provided by C.E. Tan (personal correspondence, 3/1/2000). An age-adjusted mortality comparison of the three groups (50–59 years) by weight is shown in Fig. 4. As with the previous trend patterns, mortality increased with increasing body weight. BMIs were quite similar varying between 23 and 24 for males and 22 and 25 for females. Note that when Malay and Indian females were about the same weight, their mortalities were almost the same. When Malay and Chinese males weighed the same, their mortalities were almost the same indicating that weight rather than ethnicity was the dominant factor.

3.9. Southern European weight versus mortality

The seven countries study (Menotti et al., 1999; Keys, 1970) has been tracking the mortality of males in various countries for about 40 years. Fig. 5 shows the change in all-cause and CHD mortality with weight for two Greek and two Italian communities over a 25-year tracking period. Weight was computed using height and BMI data for the original cohort of 40–59-year-old men. Thus, Fig. 5 shows the mortality trend at 25 years follow up based on the initial heights, weights, and BMIs. (These parameters were not provided for the 25-year follow-up). While this chart shows that mortality increases for both BMI and weight it should be noted that the average height (166 cm/65 in.) for the two Italian communities is a 0.5 cm greater than for the two Greek communities (Crete and Corfu).
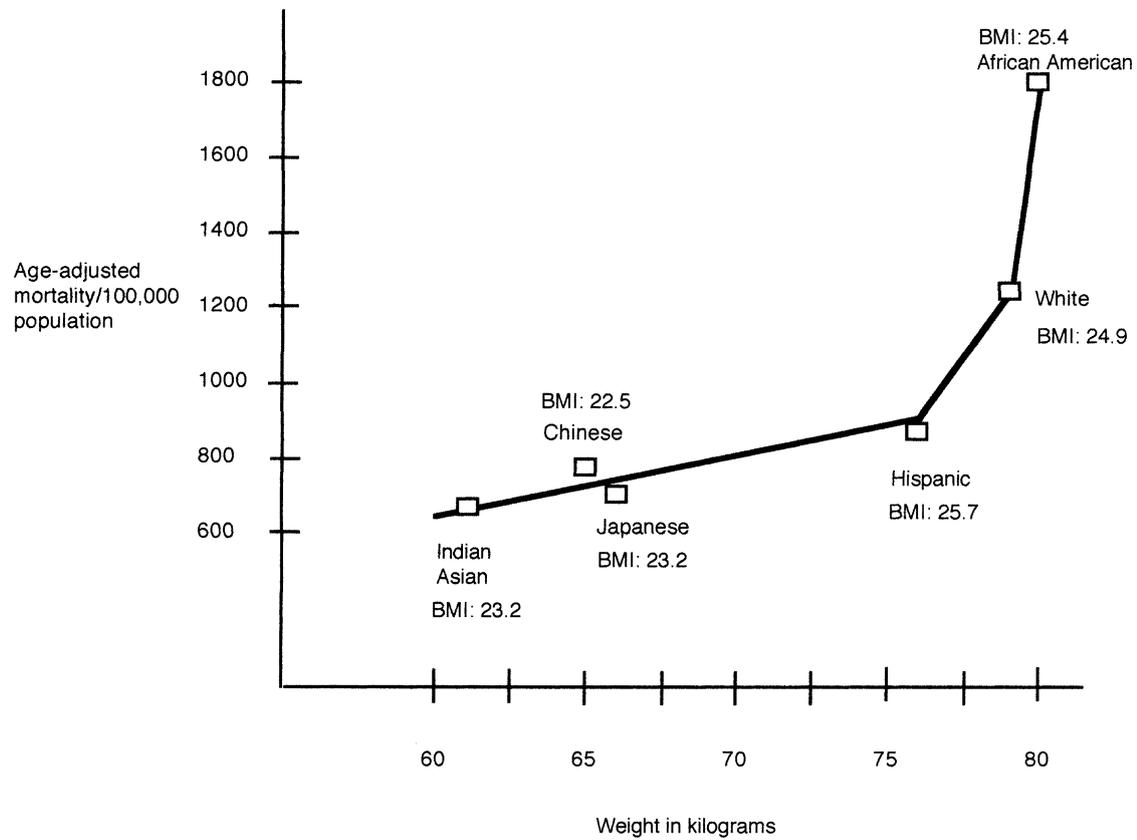
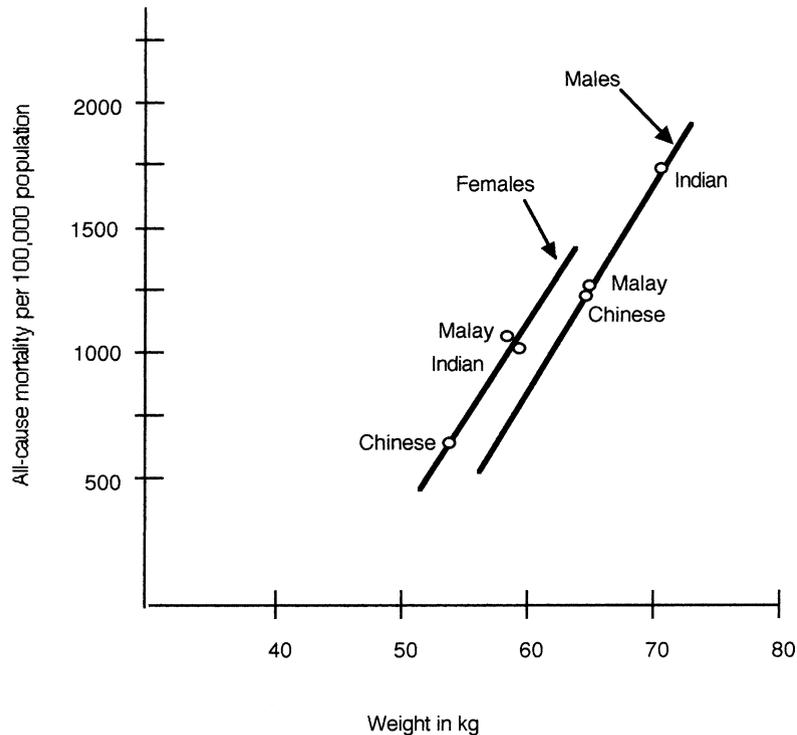Fig. 3. Mortality vs. weight of men in six ethnic groups in California.

Fig. 4. Singapore ethnic all-cause cardiovascular disease mortality, 50–59 years of age.

Since the heights are almost the same, BMI must increase in step with increasing weight (BMI $=$ weight/height^2 with height^2 a constant). Thus, both increasing weight and BMI appear to predict increased mortality.

3.10. Congestive heart failure and absolute body weight

Eriksson et al. (1989) found absolute body weight was independently related to the incidence of congestive heart failure (CHF) for men 50–67 years of age. Based on the 17-year follow-up of a population of 973 men, the researchers found CHF incidence increased with body weight for all ages. The relative risk of having heart failure at 67 years of age with increasing body weight was 1.5.

3.11. A population of small people with no CVD

A study of Kitavans found no evidence of heart disease or stroke deaths and little cancer. Lindeberg et al. (1994) conducted an evaluation of this Melanesian island population which was a traditional horticultural society uncontaminated by western dietary practices. They found no evidence of stroke and heart disease and cancer appeared to be rare. The Kitavan
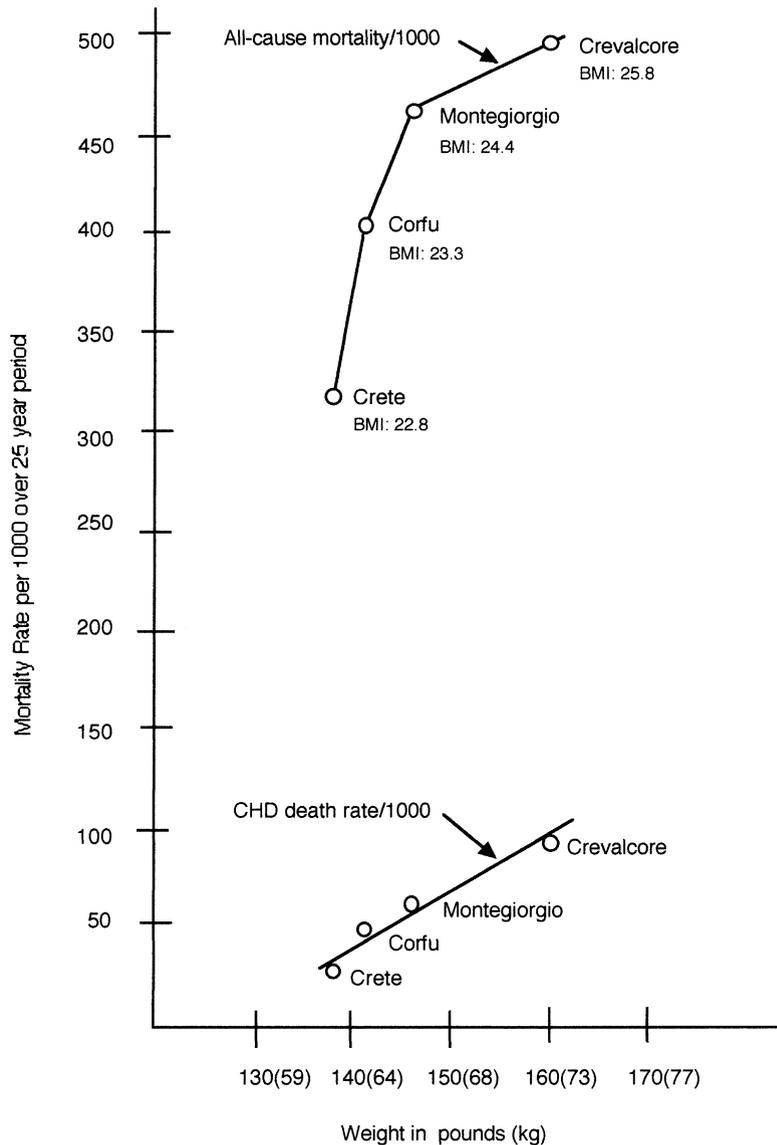
Fig. 5. Mortality increase with increasing body weight of men in Greece and Italy.

males averaged 57 kg (125 lb) between 20 and 39 years of age and 49 kg (107 lb) between 60 and 86 years of age. Women were also very small. Male BMIs (kg/m^2) declined with age from 22 to 19 for males and from 20 to 17 for women. Although about 80% of the men and women smoked, this population was in excellent health. Their diet consisted primarily of fish, tubers, beans, fruit and coconut. Chicken, pork and eggs were eaten less than once a week. Systolic blood pressure was low but rose slightly with age. Thus, in spite of the

known risk of smoking, their hearts apparently handled their daily activities without any signs of failure into old age.

3.12. Body weight decline with age

The American Cancer Society conducted a mortality study of 750,000 men and women drawn from the general population (Lew and Garfinkel, 1979). The report provided mean body weights for various heights and ages and showed a progressive decrease in body weight for each 5-year age increment from 50 years to >85 years of age. This decrease in weight was seen for each height increment ranging from 163 to 183 cm. Thus, among short and tall elderly, a small weight difference of 11 kg was found for a height difference of 20 cm.

Men in the >85 years age group were young adults before or around 1917 when military recruits were 171 cm and 64 kg (Karpinos, 1958). Assuming a 3.8 cm decline in height due to shrinkage with age, these elderly and shorter (168 cm) men averaged 67 kg or only 2.8 kg more than World War I recruits. Thus, long-living people in this study maintained a fairly low body weight in comparison to World War I recruits.

4. Discussion

The following is an assessment of the validity of the data presented, male–female differences, increasing body fat with age, animal findings related to body size and longevity and confounding factors.

4.1. Validity of findings

The negative association between body size and longevity appears valid in view of the variety of population samples examined and the consistency of trend lines. Many of the population samples were relatively homogeneous, including the Chinese who were predominantly of the Han ethnic group.

Only a few examples of long-living or low mortality populations of small people was covered in this report. However, many more exist, such as mainland Greece, Cyprus, Hong Kong, Shanghai, black rural South Africans, Jewish and Italian migrants to New York City, Vilcabambians, and Abkhasians. In addition, we found (unpublished data) over 65 populations of low body weight which have very little CVD and diet-related cancer compared with northern Europeans and North Americans. Most of these populations smoke, have less sophisticated medical care systems and have poor living conditions. Unfortunately, as these societies have deviated from their traditional lifestyles and diets, body size has increased and health has deteriorated.

It is interesting to note that Hu et al. (2000) found the incidence of hypertension rose with BMI starting with 18 and rising to 24. Cholesterol also increased with BMI. Even for this relatively low BMI range, the increase in hypertension was approximately linear. Thus, this finding indicates that the risk of hypertension and its health consequences does not have a threshold value for undesirable CVD risk factors, even for very lean people. In addition, Jousilahti et al. (1996) found that serum cholesterol increased linearly with BMI starting at

<20 and increasing to ≥32.5 in seven steps. Blood pressure also increased linearly starting with a BMI range of 20–22.4. Based on 16,113 middle-aged men and women tracked for 16 years, an increase in body weight of ~1 kg increased risk of CHD mortality by 1 to 1.5% starting at a BMI ~ 22. More recently, Field et al. (2001) found that the risk of developing common chronic disease rose in a dose–response relationship with increasing BMI starting with a BMI of only 18.5–21.9. The study was based on a population of 123,750 male and female health professionals and risk for diabetes, hypertension, colon cancer and heart disease increased with increasing BMI up to ≥35. The researchers concluded that "men and women in the upper half of the healthy weight category (i.e. BMI between 22 and 24.9) are significantly more likely than their leaner peers to develop health problems . . . " (p. 1586).

Stamler et al. (2000) found higher levels of serum cholesterol related to increased levels of CVD and all cause deaths. The findings were based on three on-going studies involving >80,000 men tracked up to 34 years. The lowest death rates and greatest longevity were in men with <160 mg/dl of cholesterol. As mentioned earlier, cholesterol levels increase approximately linearly with increasing BMI. Thus, if geometrically similar short and tall people are compared as described by Samaras and Storms (2002), taller people will have higher BMIs and consequently higher cholesterol levels with reduced lifespan. Taller people of the same proportions as shorter ones would also have higher health risks since CHD and all-cause mortality are linearly related to BMI. It is interesting to note that recent findings (Freedman et al., 2002) suggest that the biggest (weight or height) children had the highest risk for high BMIs in adulthood and the smallest had the lowest risk.

In spite of wide differences in population backgrounds and average ages, the slopes shown in Fig. 6 are very close. The male–female slopes are based on California weight and life expectancy data (Igra et al., 1982; Chan and Oreglia, 1993). It was also found that larger Harvard athletes had reduced longevity compared with smaller athletes based on 1644 athletes (Fig. 6; Polednak, 1979). In a 17-year follow-up study of 973 middle aged men, Eriksson found that body weight was an independent risk factor for congestive heart failure. The average slope for veterans, baseball players and football players presented in this report was −0.39 years/kg. It is unlikely that male–female slopes would be almost same as the slopes of light versus tall heavy athletes unless there is a real relationship between body size and longevity.

The age loss for weight slope (not shown) for California white males versus Asian males was −0.63 years/kg (Samaras and Elrick, 1999). This steeper slope may be due to the combination of low weight and low BMI for Asians. The average slope of −0.39 years/kg for athletes appears high in view of the veterans value of −0.22 years/kg. In addition, the slope for Polednak's Harvard athletes is −0.31 years/kg, which is based on 1644 athletes.

Another factor indicating the validity of the findings is the similarity of BMIs (except for football players) in spite of differences in average life span and the greater longevity of some groups with greater obesity but smaller body weight.

The findings are consistent with the Nurses' Health Study where middle-aged, non-smoking women with a BMI < 19 had the lowest mortality (Manson et al., 1995). At an average height of 162 cm, this is equal to a body weight of only 50.2 kg. It has also reported that body size is positively related to cancer incidence (Albanes and Winick, 1988). In addition, compared with the US and northern Europeans, the relatively smaller southern
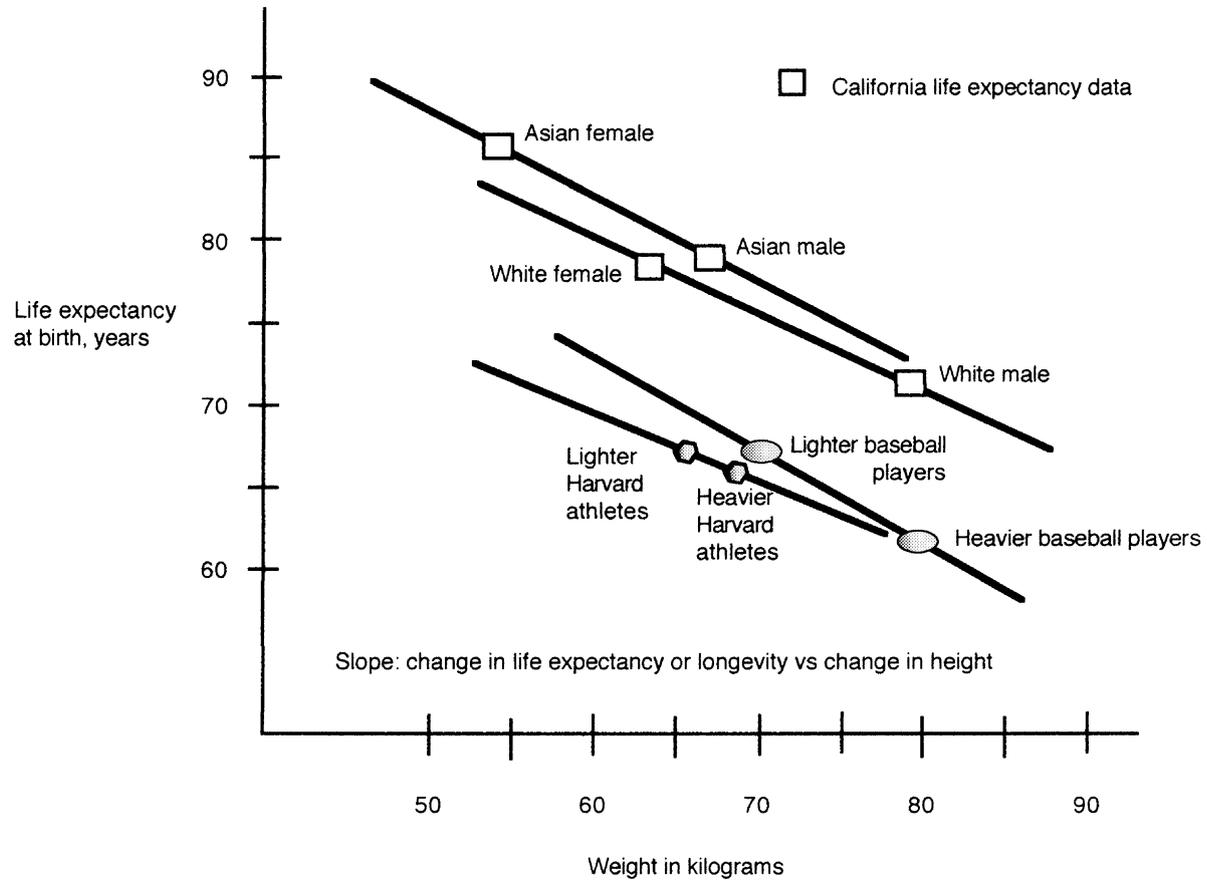
Fig. 6. White and Asian male–female life expectancy vs. adult weight slopes compared with longevity of deceased athletes of different body weights.

Europeans were found to have substantially lower all-cause and CHD death rates (Keys et al., 1984).

4.2. Male–female longevity differences

Women live longer than men. Based on our findings, it appears that their smaller body size may be a substantial factor. In addition, smaller male bats and hamsters live longer than larger females, and smaller male dogs live longer than larger female dogs of different breeds. Within a mammalian species, smaller males live longer than larger females (Promislow, 1992). Promislow (1992) concluded that "... dimorphism in mammals is associated with a viability cost to the larger of the two sexes among adults. The greater the (sexual size) dimorphism, the greater the cost." (p. 207). Bartke (2000) also reported that genetically small male mice live substantially longer than normal size females. Rollo (2002) found that male–female longevity differences among rodents disappeared when adjusted for weight differences. A study of almost 1700 deceased men and women found that men and women of the same height had the same average age at death (Miller, 1990).

4.3. Support from animal studies

Larger species of animals generally live longer than smaller species, such as the long-living elephant and short-living mouse. The greater longevity of larger animals appears to be related to lower resting metabolism, superior free-radical defenses, and improved DNA repair processes as well as greater cell duplication potential. However, within a species these evolutionary benefits do not appear to exist, except possibly for lower metabolism. A brief review of intra-species findings relating smaller body size and longevity follows.

In the 1930s, McCay demonstrated that caloric restriction (CR) produced smaller and longer living mice. Hundreds of these experiments have been conducted since then with the same results (Samaras and Elrick, 1999; Roth et al., 1999). In fact, CR and small body size within a species have been the only proven methods for extending longevity. Bartke (2000) has shown that ad libitum transgenic dwarf mice live much longer than normal size mice. And Rollo et al. (1996) have shown genetically large mice live shorter lives than normal size mice. Comfort (1961) reported many years ago that within a mammalian species, the smaller lives longer. Extensive studies of dogs have demonstrated that smaller dogs live longer than larger ones (Samaras and Elrick, 1999; Samaras and Storms, 2002; Li et al., 1996).

Studies with CR monkeys have been in progress since 1987 by the National Institute on Aging, Universities of Wisconsin and Maryland, and Wake Forest University School of Medicine. Early findings support other CR studies with mice, rats, fish, etc., e.g. lower body temperature, smaller body size and lower levels of glucose and insulin in blood (Roth et al., 1999).

The most recent findings are based on a global evaluation of rodent weight-longevity studies (Rollo, 2002). Rollo evaluated 796 studies of mice and rates conducted during the 20th century and found a correlation coefficient for longevity and body mass of -0.358 with $P < 0.00001$ for rats and -0.454 for mice with $P < 0.00001$. In addition, when he adjusted for weight differences between males and females due to sexual dimorphism, the greater longevity of females disappeared. The role of fat versus lean mass was not clear

and Rollo pointed out that ad libitum fed dwarf mice are prone to obesity but still have exceptional longevity compared with normal size mice.

4.4. Entropy theory and biological mechanisms

A number of mechanisms can explain the harmful ramifications of increased body size within a species, such as humans. They include the entropy theory, increased cell number, reduced cell duplications due to larger size, and smaller organs in comparison to body weight.

4.4.1. Entropy theory

The entropy theory, based on The Second Law of Thermodynamics, states that there is a high probability that an organized system will spontaneously deteriorate with time (Roth, 1994; Samaras, 1974). This theory also states that complexity, large size and the energy content of a system promote disorder. Thus, applying it to humans, larger humans have more cells and more energy intake, and thus, tend to become disorganized more rapidly assuming other factors are the same.

4.4.2. Increased cell number exposed to damage and transmutation

As humans grow in size from conception, the number of body cells increases. Upon completion of growth, a large body may have 40 trillion more cells than a small one. The additional cells are exposed to toxins, cosmic rays, ultra violet light, free radicals, etc. Although most damaged cells are repaired, more cells are subjected to permanent damage or converted into carcinogenic cells because the total number of cells is greater (Albanes and Winick, 1988). In addition, a higher rate of mitosis due to excess cell growth, damage, and overnourishment increases cancer risk. Considerable evidence indicates that taller (bigger) people are at higher risk for cancer (Samaras and Elrick, 1999).

4.4.3. Fewer cell duplications left in adults of larger body size

Upon conception, the process of human cell doubling begins in order to create the adult human body. However, Hayflick reported that the cell is limited to roughly 50 doublings before dying. Since most of the cell duplication potential is used up in attaining full body size (Austad, 1997), a larger body uses up more doublings, leaving fewer potential doublings during the rest of its life.

4.4.4. Organs scaled down in size

While the heart and lungs are nearly the same proportion as body mass, many organs (e.g. liver, kidneys, and brain) are relatively smaller in larger individuals (Schmidt-Nielsen, 1984). Thus, while these organs may keep up with the demands of the body during youth, cell loss due to aging or injury may cause a greater reduction in functional capability in larger bodies in later life.

4.4.5. Lean body mass

LBM is not completely free of problems Leccia et al. (1999) reported that systolic blood pressure correlates with LBM, while percent body fat does not have a strong effect on blood

pressure. A 20-year study (Andersson et al., 1997) involving 135,006 Swedish construction workers found LBM was positively correlated with prostate cancer. LBM is also a separate risk factor for coronary artery disease (Garn, 1996). In rats, both LBM and fat mass were correlated with tumors but LBM was more important than percent body fat (Albanes, 1987).

4.4.6. Greater work load on heart

Promislow (1992) suggested that the small body size could favor longevity by increasing the efficiency of the cardiovascular system. A study comparing small to large professional football players found that the largest football players had six times the death rate from heart disease compared with the smallest (The New Audible, 1994). It was reported: "Although obesity has been linked to cardiovascular disease ... the NIOSH (National Institute of Occupational Safety and Health) study found that one of the strongest associations to date is between body size and death." (The New Audible, 1994, p. 2). The Framingham Off-Spring Study also found a rapid increase in CVD risk when BMI rose above 20 (Manson et al., 1995). Thus, to minimize CVD for a male of average height, the ideal weight should be <63 kg, well below the current weight of 85 kg.

Andersen (1994) evaluated the blood pressure of 13,810 Danish adolescents with a mean age of 17.1 years. He found that a positive relationship existed between blood pressure and weight for both sexes. Fig. 7 is based on his data but only shows the mid and two extreme data points (10 data points per gender provided by Anderson). Note that the BMI at the 50 percentile is 20.4 kg/m^2. Thus, relative weight does not appear to be the major factor in the increase in blood pressure although it can play a role, especially in mature adults. Since high blood pressue is correlated with heart problems, it appears that increasing body weight has a negative impact even for relatively low body weights.

4.5. Confounding factors

Studies relating body size to longevity are subject to several cofounders. For example, higher socioeconomic status, larger body size, and beneficial environmental and health benefits tend to be positively correlated. Diet affects body size and susceptibility to infections and chronic diseases. Illness can result in low body weight. Smoking and BMI can also confound results, e.g. studies have found that smaller people tend to be more obese and smokers tend to be lighter.

Most epidemiological studies are biased toward tall people because they use BMI to compare shorter, broader people against taller people with more linear builds (Samaras and Elrick, 1999). To provide a common baseline, epidemiologists should compare short and tall people of the same geometric configurations (Samaras and Storms, 2002).

The epidemic of chronic disease has been tied to recent western dietary practices (Samaras and Elrick, 1999; Nutrition Reviews, 1991) which paradoxically parallels increased life expectancy and larger body size. Yet, rural poor blacks on low calorie and protein diets in South Africa are relatively free of CHD and diet-related cancer (Walker et al., 1995). These cofounders may explain why larger corporate executives have lower mortalities than their shorter subordinates and why some large athletes (>86 kg) live to 98 years of age (height and weight are positively correlated).
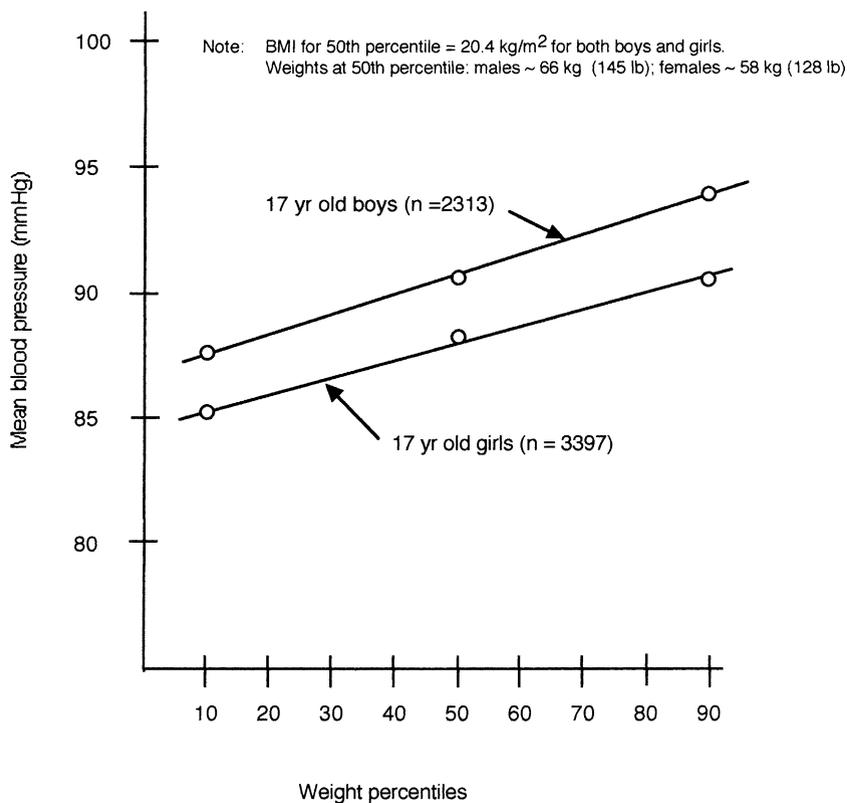
Fig. 7. Increase in mean blood pressure with increasing weight for a population of Danish 17-year-olds.

5. Conclusions

A broad range of evidence was presented showing an inverse relationship between body size and longevity. This evidence involved human and animal research. In addition, several biological mechanisms were reviewed to explain this inverse relation.

It is suggested that the longevity differences between males and females are primarily related to differences in body size and the relatively smaller size of male organs, such as kidneys, the brain and liver.

A modest correlation coefficient of -0.203 indicates that weight is only one risk factor that affects an individuals' longevity (Samaras and Storms, 1992). Longevity is more strongly affected by other traditional risk factors, such as socioeconomic status, BMI, body type, dietary practices, life style (e.g. smoking, drinking and drugs), exercise, hygiene, genetics, and the quality and availability of medical care. Therefore, larger bodied people can live a long time when other risk factors are minimized. However, it appears that dietary changes and increased secular growth during the 20th century have had some harmful effects on our longevity potential.

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