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Should we be concerned over increasing body height and weight?

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A B S T R A C T
Based on 36 years of research, this paper explores the impact of human height on chronic disease, life expectancy, and longevity. The findings presented challenge the common belief that promoting rapid growth and maximum height attainment is a desirable goal. Also presented, are the biological mechanisms related to height and longevity. This paper concludes with a review of paradoxes that face the traditional belief that taller is healthier.

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1. Introduction and background

One of the least examined areas of human health has been the trend towards increasing height and associated body mass while body geometry or proportions are maintained. Yet, an almost universal belief is that being taller with greater lean body mass is a superior configuration for humans. However, Holzenberger et al. (1991), the Gavrilovs (2007) and others have warned that rapid growth and increasing height have detrimental effects on longevity. Holzenberger et al. also noted that these detrimental effects are more than offset by improved living conditions of recent years. DeHeeger and Rolland-Cachera (2004) also reported that over the last 30 years, our health has deteriorated in that children are growing faster, leg length has risen, and androidal fat and overweight have increased along with increases in cancer and cardiovascular disease. In addition, Singhal et al. (2003), reported that promoting rapid childhood growth needs to be reevaluated because it increases coronary heart disease (CHD), diabetes, and obesity in adulthood. In addition, Rollo (2007a) found that rapid growth and early maturation reduces longevity. Bartke (2007) also reported that transgenic and growth hormone knockout receptor mice are smaller and live longer due to the reduced action of the somatotropic (GH-IGF-1) axis.

In addition, since weight increases as a function of the cube of the increase in height, relatively small increases in height produce larger increases in weight. For example, as described by Samaras (2007a), a 10% increase in height results in a 33% increase in weight for geometrically similar bodies. The equation for calculating the weight of a taller person is as follows: Weight of taller = (Height of taller/Height of shorter)³ × Weight of shorter. This cubic increase is seen when children are tracked from ~9 to 20 years of age (Samaras, 2007a,b). Five examples are given in Samaras and Storns (2002) where one study involved almost 500,000 Swedish males and showed weight increased slightly in excess of the cube in height. In addition, Samaras found about 80 populations where weight increases as the cube of the increase in height (unpublished). Note that other researchers have found that weight increases as the square of height. This discrepancy is probably due to differences in lifestyle, dietary habits and socioeconomic status (SES). In addition, tall people can be lighter for their height compared to short ones due to the lower intake of calories since food portions are often standardized; e.g., size of beverage, restaurant meal, etc. provide a lower percentage of calories to the larger person on a weight for weight basis. However, the total consumption of food is still greater in taller people for the same activity levels.

1.1. Background

Over the last 36 years (y), the author has questioned the health and environmental benefits tied to increased height and associated
body weight. The well-established second law of thermodynamics provides a hypothesis which explains two key aspects in the aging process. The author's interpretation of this law is: the tendency for similar bodies to become disordered with time is based on their mass and energy content. For example, increasing the number of cells and the energy required by these cells increases the opportunity for the human system to become disordered due to the increase in potential cellular states; e.g., increased metabolic processes and associated increased body temperature produce more free radicals which alter or damage DNA and other cell elements, and the more cells there are, the more damage occurs. The term “increasing disorder” is interpreted to mean increased aging of the body, and an organized system, such as the human body, has many more options to move into disorder than into a new ordered state when subjected to forces or energy that affect its equilibrium. Roth (1994) and others have forwarded the entropy theory as a basis for explaining the aging process.

Subsequently, published data were collected detailing the longevity of selected groups of individuals; e.g., baseball players, football players, celebrities, and 19th Century French men and women. All these samples provided a negative correlation between height and longevity. Additional investigations involved the research done by others. Collaborations with H. Elrick, L. Storms and other investigations are summarized in Samaras (2007). Despite skepticism of the hypothesis and findings, our work was published in multiple journals and books, including the Encyclopedia of Public Health (Samaras, in press).

The following contains previously published and new findings on cancer, heart disease, all-cause mortality, life expectancy, and longevity.

## 2. Cancer and height

Many studies have found a positive correlation between height and various types of cancer. In 2001, Gunnell et al. (2001) published a systematic review of 300 studies and concluded that taller people had a 20–60% higher cancer risk. Subsequent studies have confirmed their findings.

Table 1 is based on data provided by Suzuki et al. (2001) and provides additional support for the hypothesis that the taller people have more cancer. Note that the populations shown are listed in order of increasing height. As can be seen, four major cancer sites show an increase in cancer mortality with increasing height of the population. Note that the table compares the two shortest populations to the two tallest. Depending on the site, risk increased by ~70% to ~200%.

In a long-term study of 47,690 male health professionals, Giovannucci et al. (2004) found a 74% higher risk for Western type cancers; e.g., prostate, colon, and kidney, and concluded that maximum growth during childhood and adult hyperinsulinemia promote cancer in Western populations.

### 3. Relation of height to heart disease and all-cause mortality

The following sections describe conflicting data on the relationship between height and CHD, stroke, and all-cause mortality. The findings are based on various types of studies, very large population samples, and diverse ethnic groups.

#### 3.1. Increased CHD, stroke and all-cause mortality with shorter height – a controversial topic

Most epidemiologists believe that taller people are healthier and live longer. This belief is supported by studies over the last 40 years which have found that shorter people have higher levels of CHD, stroke and all-cause mortality (Samaras, 2007c). Another reason for this belief is that taller people in the developed world have much longer life expectancies at birth compared to shorter ones in developing nations. However, this can be easily explained by the facts that developed countries have much lower infant mortality, superior sanitation, lower infections, much less starvation, and comprehensive and rapid medical treatment of heart attacks, strokes, traumas, and infections.

In contrast, researchers have studied traditional and developing populations for many years. They found non-westernized populations had very low or nonexistent CHD, and virtually all these populations were short and lean due to low calorie, plant-based diets (Samaras, 2007c).

Table 2 shows five studies that found shorter people had higher risk for CHD and stroke. Within each study, shorter people were compared to taller people and generally showed an increasing risk with decreasing height. There about 20 such studies (Samaras, 2007c). Note that the CHD and stroke risk ranges from 1% to 32% greater for shorter people (~19% average). The largest study involving almost 400,000 South Korean males found only a small increase in risk for shorter height for both CHD and stroke. Song et al. (2003) concluded that the lack of a correlation between short height and CHD indicates that other factors must be involved for short height to translate into greater CHD risk. For example, short height may increase CHD risk “only in the presence of a diet that generates high cholesterol levels and obesity” (McCarron et al., 2002).

Sections 3.2–3.8 review a variety of findings showing that shorter people have lower CHD and all-cause mortality. Section 4...
reviews longevity data, and Sections 5–8 cover other topics including female height and life expectancy and centenarian body size.

3.2. Lower heart disease and stroke mortality for shorter people in US population

Table 3 shows the age-adjusted heart disease (CHD and other heart problems) mortality (Eberhardt et al., 2001) for various ethnic groups in the US population. The table lists the tallest ethnic groups (~175 cm/5’9” males) first and the shortest last (Asian males ~168 cm/5’6”) (Samaras et al., 2003). As can be seen, the tallest males and females have roughly twice the heart disease mortality as the shortest. The pattern for stroke is not as clear although there is decline with shorter height. Note that while the studies finding taller people have lower mortality provide about a 20% advantage (Table 2), the US data approach a 100% greater risk compared to the countries with the lowest CHD risk. The individuals in the lower risk countries were also shorter. Similar to Table 3, the countries with taller individuals had over 100% greater risk compared to the countries with shorter individuals (both genders). This is over 5 times larger than the ~20% greater risk shown in Table 2. Stroke risk was moderately higher in shorter people (data not shown).

Note that males from Japan, Hong Kong, and France have lower CHD mortality than women in the taller states, except for females in The Netherlands. This unusual finding may be due to dietary differences and a higher BMI for women in the tall countries vs males in the shorter ones.

3.4. California ethnic data and CHD

Fig. 1 shows the regression lines for males and females of five ethnic groups in California (Samaras et al., 2004). As with the previous chart, age-adjusted CHD increased progressively with increasing height of the group with the tallest people (Blacks and Whites) having about 100% higher risk than the shortest (Japanese). Note that the slopes for males and females are fairly close, and the findings are based on mortality data of >205,000 men and women.
3.5. European CHD

Another comparison of height and age-adjusted CHD mortality involves 11 Western European countries (males) shown in Fig. 2 (Samaras et al., 2003). Based on mortality rates and the total populations of these countries, the number of deaths for this sample was estimated to be about 300,000. The regression line is similar to that of Californians (Fig. 1) with Scandinavians having twice the death rate from CHD than the shorter Spanish and Portuguese.

It is interesting to note that the two tallest and shortest groups in Europe and California had similar mortality rates and were about the same heights. As can be seen in Fig. 2, the average height of Scandinavians was roughly the same as California Whites and Blacks and the average height of Spaniards and Portuguese was similar to the average height of the California Chinese and Japanese. It is remarkable that the mortalities of these diverse groups appeared more strongly related to their heights than to differences in their diets, ethnicity, climate, lifestyle, and geography.

3.6. Shorter populations with low to nonexistent CHD

As mentioned, researchers have found that people in traditional societies have much lower rates of certain chronic diseases compared to developed populations including Saweri (2001) and Temple and Burkitt (1994). Virtually all of these populations are much shorter than northern Europeans. Yet, Papua and Kitava New Guinea have had no CHD and stroke before Westernization; e.g., Kitava was studied by Lindeberg et al. (1994) for 10 years, and they found no evidence of CHD or stroke in the age range of 20–87 y. Kitavan males have averaged 163 cm (~5’4”) and females 155 cm (~5’1”) for several generations. A partial list (Samaras, 2007c) of shorter populations with little or no CHD or stroke includes: rural South African Blacks, Kalahari bushmen, Congo pygmies, Solomon islanders, Vietnamese, Tarahumara indians (Mexico), Cretans in 1960s (1/10 the CHD of Northern Europeans), Arctic Inuits, Chinese, Vilcabambans, and Hunzas.

Blacks in the US had substantially lower heart disease compared to Whites before the 1930s (Wald et al., 1995). They were also shorter than Whites in the 1930s. (Young Blacks caught up in height to Whites around the 1940s.) In addition, as the Japanese migrated from Japan to Hawaii to California, their CHD rose along with increasing height (Samaras et al., 2004).

3.7. Height and all-cause mortality in California

We have examined the relation between height and all-cause mortality and these findings are not repeated here (see Samaras and Heigh, 1996; Samaras et al., 2003; Samaras et al., 2004). However, one example is shown in Table 5 (Samaras et al., 2003). This California study provided age-standardized all-cause mortality for different ethnic groups and involved ~1 million deaths. As can be seen, the tallest groups (Whites and Blacks) had over twice the mortality as the two shortest groups and this observation applied to both genders.

3.8. Confounders

The findings highlighted in Sections 3.2–3.7 obviously conflict with the findings in Section 3.1. However, the studies showing shorter people have lower CHD and all-cause mortality rates are based on much larger numbers of deaths and the mortality rates are much higher for taller vs shorter populations in Sections 3.2–3.7. Thus, what explains the differences between height-mortality studies with opposite results? Several possible factors that may confound results are reviewed next.

Failure to adjust BMI upward for taller cohorts to maintain the same geometry can favor taller people and provide incorrect conclusions as described by Samaras (2007a); e.g., using the same BMI for short and tall cohorts results in a lower risk comparison between leaner, tall subjects and stockier, short cohorts. Since a 10% increase in BMI can increase mortality by 10%, failure to increase the taller cohort’s BMI by 10% would favor the taller cohort with an unwarranted 10% lower risk.

Childhood illness, trauma or malnutrition stunts growth and carries both a health liability and shortness forward to adulthood. Catch-up growth of low birthweight infants correlates with increased adult mortality from obesity, CHD, stroke, and diabetes (Samaras, 2007d; Cameron and Demerath, 2002). In spite of
catch-up growth, low birthweight infants generally do not attain their maximum genetic height potential and tend to be shorter adults compared to normal or heavier weight infants. Lower SES has a strong impact on adult mortality. In some studies, a lower SES person may experience 4 times the mortality as an upper SES person. In addition, in Western countries lower SES populations tend to be substantially shorter and fatter than upper SES cohorts. As a result, risk adjustment almost always reduces the mortality rates of shorter cohorts in the study.

Most studies are restricted to a specific age range and don’t track cohorts into advanced ages. As can be seen in Table 2 the baseline range is 30–65 y. A cohort born in the same year will have about 70% of its members alive at 65 y of age. Thus, while it can be assumed that mortality risk at younger ages will stay the same for advanced ages, this may not be true.

The use of multiethnic or multinational data has a potential flaw in that it is hard to know whether an unknown factor is confounding the results, such as health care systems and dietary practices (Grant, 1998). Other factors include air pollution, smoking, and alcoholic intake.

Note that while researchers usually make risk adjustments for smoking, SES, and BMI, these adjustments are generally crude and inexact (Wannamethee et al., 2002). Davey Smith et al. (1997) also noted that adjustments for SES need to be based on all three phases of life – (1) childhood and adolescence, (2) young adulthood, and (3) mature adulthood (Samaras, 2007c). Otherwise the impact of SES confounding will reduce the validity of the findings.

4. Height and lifespan

The next sections review findings correlating height with longevity. They include examples from Spain, US, France, Sweden, and Finland.

4.1. Spanish findings

Holzenberger et al. (1991) conducted a large study of Spanish male survival based on height over a 70-y period. The study included 1.3 million males whose mortality was tracked from 1860

---

**Table 5**

<table>
<thead>
<tr>
<th>Ethnic group in order of increasing height (25–85 y)</th>
<th>Age-standardized death rates/100,000/y</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>males</td>
</tr>
<tr>
<td>Japanese&lt;sup&gt;a&lt;/sup&gt;</td>
<td>693</td>
</tr>
<tr>
<td>Chinese&lt;sup&gt;a&lt;/sup&gt;</td>
<td>773</td>
</tr>
<tr>
<td>Asian Indian (females taller vs Latino females)</td>
<td>668</td>
</tr>
<tr>
<td>Hispanic/Latino</td>
<td>856</td>
</tr>
<tr>
<td>African American&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1800</td>
</tr>
<tr>
<td>White&lt;sup&gt;b&lt;/sup&gt;</td>
<td>1243</td>
</tr>
</tbody>
</table>

<sup>a</sup> Japanese and Chinese males about the same height.

<sup>b</sup> African American and Whites are the same height.

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Fig. 2. CHD vs height for 45–65-year-old males in 11 European countries and six 25–84-year-old male and two female ethnic groups in California. (See above mentioned references for further information).
to 1930 using census data. Heights (<159.8 to >162.7 cm) were obtained from military records when the subjects were 18-years-old. The researchers found that taller men had poorer survival with a correlation coefficient \( r \) of \( r = -0.33 \) for men over 60 years of age and \( r = -0.58 \) for men over 70 years of age. The findings were significant with \( p < 0.001 \) for both coefficients. Their results were not confounded by migration, selection, epidemics, or census errors.

Holzenberger et al. (1991) concluded that “...the smaller the mean height at 18 years in a province, the higher the chance for people living there to reach high chronological ages.” p. 89. They also observed that the benefits of shorter height are masked by advances in the modern living conditions which improve longevity faster than the decline in life span due to rapid growth.

4.2. Professional baseball players

Fig. 3 shows the average life span regression lines for two samplings of US professional baseball players (Samaras, 2007c). The lower regression line was based on >3100 deceased players over the period of 1910 through 1975. The higher line involving 454 deaths is based on a more recent period (1976–1992). The difference in the two regression lines is due to the increasing life expectancy (~2.5 y/decade) for people born in more recent times. Similar decreases in average lifespan with greater height were found for football and basketball players (Samaras and Elrick, 1999). While no adjustments for SES were made, baseball players represent a fairly homogeneous cohort and as mentioned, adjusting for SES

Fig. 3. Height and longevity of professional baseball players.

Fig. 4. Nineteenth century French male longevity with increasing height.
and other confounders would further improve the longevity of the shorter athletes.

4.3. Mid 19th Century French

Fig. 4 shows a trend line for French men who died around the 1850s. The slope for women (not shown) was similar (Samaras and Heigh, 1996). The male and female populations were divided in half by height and the average ages at death computed. The taller half (≥ 168 cm) of males averaged 48.6 years and the shorter half (≤ 167 cm) 54.6 years. Female differences were similar at 60.8 years (taller) vs 66.6 years (shorter).

4.4. Years lost with increasing height

A number of studies have shown that the average age at death for a population declines by ~.5 year per centimeter (cm). Table 6 is a partial summary of various studies with their associated slopes (y/cm). The left-hand slopes are based on data collected by Samaras and associates over the years. This list is representative and does not include all findings. Studies in which the original average age vs height data were collected by other researchers are provided on the right-hand side of the table. Some of the data in Table 6 were provided by Miller (1990) and Krakauer (personal communication, 2002), while others are from Samaras and Elrick (1999). Rounded to the nearest tenth, the slope for both sides of Table 6 are the same at ~.5 y/cm.

5. How do women's height and life expectancy compare to men

Women throughout the world are shorter than men. In the industrialized world men average ~10 to15 cm (4” to 6”) taller than women due to genetic factors.


The reduction in male LE is ~.5 y/cm (7.2 y/14.39 cm) and is very close to the values in Table 6. In addition, males averaged 8.7% taller and had a 9.2% lower life expectancy compared to females born in the same year. When rounded to the nearest whole integer, they were identical at 9%. In addition, five similar findings on differences in male-female life expectancy were previously reported by Samaras et al. (2003).

A few additional points should be considered. Brown-Borg et al. (1996) reported that genetically smaller male mice lived 50% longer than larger normal size females. Miller (Samaras, 2007) also noted that human males of the same height as females had a longevity advantage. Rollo (2007b) found that when he adjusted for differences in body mass between male and female rodents, their longevity was the same. Thus, he concluded that sexual size dimorphism accounted for the longer lifespan of females. Stindl (2004) also noted that male–female differences in life expectancy may be due to their height differences. A final point about female longevity comes from Cameron and Demerath (2002) who reported that telomere lengths are greater in women compared to men, and this fact is consistent with the greater longevity of women. Female telomereres would be longer due to the fewer cell replications needed to achieve and maintain their smaller body size.

6. Life expectancy of developed states

As mentioned, the LE of taller developed regions/countries are substantially higher than that of shorter developing states. However, if shorter and taller developed populations are compared, the shorter populations often rank the highest for life expectancy (World Factbook, 2007). Table 7 lists the six highest ranking populations for life expectancy at birth (males and females combined). Andorra is located between Spain and France and consists of Catalonian, Spanish, Portuguese and French people. San Marino is situated in the central part of Italy and its population is primarily Italians. Thus, the heights of people from Andorra and San Marino are probably consistent with the heights of the Spanish, French, and Italians who are shorter than Northern Europeans.

The right side of Table 7 lists six of the tallest populations in Europe. Sweden ranks 7th, Norway 20th, and Denmark 47th in LE, respectively. The average LE ranking for the six tallest populations is 29 vs 3 for the six shorter populations.

<table>
<thead>
<tr>
<th>Rank</th>
<th>Top six states (all relatively short)</th>
<th>LE (y)</th>
<th>Rank</th>
<th>Tallest States (Western Europe)</th>
<th>LE(y)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Andorra (between France and Spain)</td>
<td>83.52</td>
<td>7</td>
<td>Sweden</td>
<td>80.63</td>
</tr>
<tr>
<td>2</td>
<td>Macau (40 mi west of Hong Kong)</td>
<td>82.27</td>
<td>20</td>
<td>Norway</td>
<td>79.67</td>
</tr>
<tr>
<td>3</td>
<td>Japan</td>
<td>82.02</td>
<td>28</td>
<td>The Netherlands</td>
<td>79.11</td>
</tr>
<tr>
<td>4</td>
<td>San Marino (central Italy)</td>
<td>81.80</td>
<td>32</td>
<td>Germany</td>
<td>78.95</td>
</tr>
<tr>
<td>5</td>
<td>Singapore</td>
<td>81.80</td>
<td>38</td>
<td>Finland</td>
<td>78.66</td>
</tr>
<tr>
<td>6</td>
<td>Hong Kong</td>
<td>81.68</td>
<td>47</td>
<td>Denmark</td>
<td>77.96</td>
</tr>
</tbody>
</table>
7. Life expectancy of American WW II twins

Zaretsky (2003) reported on the longevity data of \(\sim 27,000\) twins born between 1917 and 1927 from the veterans registry. The results showed that the LE of identical twins was 82.0 years and fraternal twins 80.5 years.

The birthweight of twins is generally 1 kg lighter than singletons, and fraternal twins average \(\sim 150\) g heavier than identical twins. A strong positive correlation exists between birthweight and adult height and weight (Samaras, 2007a; Loos et al., 2001). In addition, Oken and Gillman (2003) reported that differences in birthweight within identical twins correlate with differences in adult height and weight. Thus, identical twins are shorter and lighter than fraternal twins. Consequently, the greater longevity of identical twins supports the hypothesis that their smaller size is an advantage.

Data collection for the aforementioned study of twin longevity began when the twins were between 40 and 50 y of age. The LE of same age white US singletons was 72 y and 73 y, respectively, well below that of the twins (i.e., LE of identical twins was 82.0 y and fraternal twins was 80.5 y).

The preceding findings are consistent with an earlier study of this same group of twins conducted between 1946 and 1978 (Hruby and Neel, 1981). Identical twins had an all-cause mortality of 10.2% vs 11.4% for fraternal twins for the age range of 51–61 y. Similar findings were reported for the longevity of larger body size. These include reduced resting metabolic rate, lower heart rate, and lower arterial stiffness. For example, in Western societies shortness is often associated with congenital problems, catch-up growth, childhood health problems, malnutrition, trauma, and psychological factors which are also related to lower SES. In addition, the Gavrilov study group was born in the late 1800s when urban conditions were not especially compatible with good health and increased height among lower SES people.

9. Smaller animals and trees live longer

It is well known that smaller animal species do not live as long as larger ones. The classic example is a comparison between the long-lived elephant and the short-lived mouse. However, within the same species, smaller usually lasts longer, e.g.; the smaller Asian elephant lives substantially longer than the African elephant. Although not considered the same species, crossbreeding has occurred, but it is rare because elephants generally do not mate in captivity.

The greater longevity of smaller animals within the same species became widely known when it was found that mice fed low calorie diets grew to be smaller than normal but had extended longevity. In addition, genetically smaller rodents, fed ad libitum, also lived much longer. The lifespan of giant transgenic mice were also found to be much lower than normal size mice. These findings are described in more detail by Bartke (2007) and Rollo (2007b). In addition, Ozanne and Hales (2004) found that retarding the growth of suckling rats increased their longevity and reduced their risk of becoming obese due to a rich diet later in life.

Extensive research has also shown that smaller dogs live longer than medium and large dogs [Miller and Austad (2006)]. Recently, Greer et al. (2007) found both height and weight were independently correlated with longevity in dogs. They found a reduction in lifespan with a height correlation of \(r = \sim 0.60\) and a weight \(r = \sim 0.68\). Note that the height coefficient is very close to Holzenberger’s \(r = \sim 0.58\) for Spanish males. Samaras and Storms (2002) also found that taller dogs had reduced longevity.

The greater longevity of smaller size was also found for eastern white cedar trees. Wheeler (1996) reported that these trees growing on cliffs grew very slowly due water run off and reduced mineral content of the soil. These smaller, nutritionally deprived trees were noted as one of the slowest growing trees in the world reaching only a fraction of the height (usually \(<10\) or \(\sim 3.3\) m) that normal growth (50’ or \(\sim 15\) m) white cedar trees reached on flat terrain. In addition, the branches and roots of Bonsai trees are pruned on a regular basis to keep them small. They survive for hundreds of years and equal or exceed the longevity of full-sized trees of the same species (Roth, 1994).

Willcox et al. (2004) (Samaras, 2007c) found Japanese males in Hawaii increased their survival as caloric intake declined down to 975 kcal. (Lower caloric intake also correlated with reduced height.) Below 975 kcal, mortality started increasing. This major study involved tracking the cohort for 36 y and covered a final age range of 80–104 y. Note that for taller Europeans, the lower threshold of caloric intake may need to be raised from 975 to \(\sim 1200\) to 1400 kcal. Okinawans have also been found to experience reduced caloric intake for part of their lives and this may also explain why they live longer than mainland Japanese.

10. Biological mechanisms that relate body size with longevity

There are certain biological factors or mechanisms that support the longevity of larger body size. These include reduced resting metabolic rate, lower heart rate, and lower arterial stiffness. For some tall people, slower than normal growth may have led to later sexual maturity and lower BMI. These benefits can lead to lower blood pressure, cancer, diabetes, and CHD. Slow growth and later sexual maturity promote greater longevity (Rollo, 2007a). Taller people also have larger diameter blood vessels which are less likely to clog up with fatty deposits from a typical Western diet. Shorter and geometrically similar humans, however, have a much larger
In contrast, studies that found shorter individuals have lower CHD and all-cause mortality, along with increased longevity, are based on much larger populations; e.g., the combined deaths for California and Spain exceed 2 million.

In addition, the relative absence of tall individuals in populations of centenarians contradicts the belief that taller people live longer. As an example, Okinawans, with the highest percentage (500/million) of centenarians, are 10–13 cm shorter than Scandinavians. However, with improved medical technology and care, it is expected that more tall people will reach centenarian status.

The advent of genetic engineering holds out false promises that an increase in height can lead to a more happy life. On the other hand, this paper suggests that the increase in height along with weight correlates with an increase in obesity and other chronic diseases. A high birthweight (>4 kg), accelerated growth in height and weight during childhood, and even a moderately higher BMI within the normal range poses risks for maintaining good health during middle and advanced ages. Thus, promoting the attainment of one’s maximum potential for height through nutrition or genetic manipulation has undesirable ramifications.

While the non-medical related threats of a worldwide increase in the average size of humans have not been discussed in this paper, the ramifications in terms of increased demands for natural resources, food, water, and energy can be enormous as described by Samaras and Storms (2002) and Samaras (2007e). The fiscal and environmental impact of a future world population which is substantially taller than at present will place a greater burden on resources and increasing air, water, and food pollution and pose a threat to the health and survival of future generations.

Height is an index of early dietary practices, rapid growth, and body weight. This paper has identified height as an independent health risk. However, height might not be a strong negative factor if childhood growth is slowed so that final height is not attained until after 20 y of age instead of around 18 y of age. For example, it is well known that slow growing species live longer.

While the previous findings favor the longevity potential of shorter people, tall individuals can certainly reach advanced ages. However, tall people need to keep their weight down, avoid smoking, exercise regularly and eat wholesome diets to improve their chances of attaining advanced ages. While we cannot change our heights, we CAN eat fewer animal products and processed foods, and replace them with whole grains, legumes, vegetables, nuts, and fruits while avoiding excess caloric intake.

The thesis that shorter, smaller body size offers health and other advantages is counter-intuitive. However, the previously described evidence seems strong. In addition, based on the findings of an exceptionally comprehensive study on human health and diet, Campbell and Campbell, 2006 observed that animal protein produces taller and heavier people and bigger size comes with a high price in terms of excessive levels of heart disease, cancer, and diabetes.

We have all been subjected to the “tall is superior” thesis since childhood. As scientists, it’s time to evaluate the worldwide increase in body height and weight objectively based on scientific research which reveals the real advantages and disadvantages of increased body size.

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