Is short height really a risk factor for coronary heart disease and stroke mortality? A review

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Summary

A number of studies have reported an inverse relationship between height and cardiovascular disease (CVD). Most of these studies have involved a relatively small number of deceased people and may have been confounded by socioeconomic and other factors. In contrast, many studies have found short populations in traditional and western societies have very low CVD compared to taller Western populations. The purpose of this study was to evaluate the impact of short height on coronary heart disease (CHD) and stroke incidence or mortality based on a variety of inter-ethnic and intra-ethnic groups involving much larger deceased populations compared to previous studies. The results of this study indicate that shorter people have substantially lower rates of CHD mortality and moderately lower levels of stroke mortality. For example, shorter southern Europeans had about half the CHD mortality rate of northern Europeans. In addition, shorter ethnic groups vs taller groups in California had substantially lower mortality rates. Native American, Japanese, Indian, and Pakistani studies also showed shorter people had lower CHD and stroke incidence or mortality compared to taller people within each group. The rate of increase in CHD mortality with increasing height was similar for shorter females vs taller males and for shorter males vs taller males.

key words: coronary heart disease • stroke • mortality • longevity • height • body size • body mass index • nutrition • low birthweight • catch-up growth

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BACKGROUND

For over 50 years, a number of Western researchers have found shorter people are at higher risk for coronary heart disease (CHD) or stroke, such as Paffenbarger et al. [1], Leon et al. [2], Rich Edwards et al. [3], Mc Carron et al. [4], Kannam et al. [5], Davey Smith et al. [6], Hebert et al. [7], Eriksson et al. [8], Forsen et al. [9], Wannamethee et al. [10], Jousilahti et al. [11], and Goldbourt and Tanne [12]. For example, a large 22-year follow-up study by Jousilahti et al. [11], based on 31,199 middle-aged men and women, found CHD mortality decreased 4–7% [nonsignificant (NS)] with every 5 cm increase in height. Stroke mortality was 16% (p<0.001) lower for men and 10% lower (NS) for women for a height increase of 5 cm. Goldbourt and Tanne [12] found an increase in stroke of 13% between the shortest and tallest quartiles. Forsen et al. [9] found a 33% increased cardiovascular (CVD) risk (p=0.006) for short men. Table 1 summarizes findings from several recent studies [4,10,13-15].

The lower CVD mortality or incidence of taller people has been attributed to superior intrauterine and childhood nutrition and growth, higher socioeconomic status (SES), less smoking, lower relative weight, and larger diameter blood vessels. This picture seems convincing if we only focus on studies that have found a negative correlation between height and CHD/stroke, and some researchers, such as Forsen et al. [9], concluded that short height is an independent risk factor for heart disease. However, if we evaluate the numerous studies or reports that have provided conflicting results, a different CVD model evolves. For example, many Western researchers such as, Allebeck and Bergh [16], Blumchen and Jette [17], Rich-Edwards et al. [3], Leon et al. [2], Kannam et al. [5], Liao et al. [18], Davenport and Love [19], Cox et al. [20], Eriksson et al. [8], Mendall [21], Hebert et al. [7], and Polednak [22] did not find an inverse relation between height and CHD or stroke risk or described the relation as ‘trivial.’ For example, based on 1098 deaths from CHD, Goldbourt et al. [12] found ‘excess risk was eliminated entirely’ between height and CHD when adjusted for age. And more recently, a study involving 386,627 Korean males (40–64 years of age) found no age-adjusted association between height and CHD [13].

In addition to the abundance of conflicting or neutral studies, a number of paradoxes exist; for example, shorter Japanese and Southern Europeans have substantially lower CHD compared to taller northern Europeans. And women, who average ~13 cm shorter than men, have much less CHD throughout most of their lives. Furthermore, early in the 20th century, Europeans and Americans experienced much lower rates of CHD [23–25] even though they were substantially shorter than today. When we expand our view beyond the Western world, we find a massive amount of evidence that shorter, smaller people have substantially lower CHD than in the West; e.g., Diehl [26] reported that CHD was rare to non-existent in peasant populations in Asia, Africa, and Central America. Likewise, China and India had very low CHD before they adopted more Western dietary practices and increased in height.

Although a few African populations are tall, Elrick studied vigorous and long-living people throughout the world and found that most populations following traditional diets and lifestyles are much shorter, lighter, and have lower CVD than people in Western industrialized countries [27–29]. It is the purpose of this paper to summarize our findings showing that shorter populations have lower CHD and stroke than taller populations.

MATERIAL AND METHODS

The authors of this paper have been studying the relation between height and health and longevity for about 30 years. Elrick has led teams of researchers to study vigorous and healthy people in eight countries. Storms and Samaras have studied records of deceased veterans at the San Diego Veterans Administration Hospital, and have analyzed longevity data based on populations of high achievers, baseball and football players, and 19th century French men and women [30–51]. For this review, numerous papers and reports were found that present CHD or stroke data on various populations in the world. In addition, Medline and PubMed were used to search for papers on CVD, all-cause mortality, longevity, and height. References in these papers have also been used as well as books on longevity and nutrition. During our research, we have collected thousands of research papers/government reports that have data on height, longevity, cancer, and heart disease or stroke incidence/mortality. Many of our findings were presented in other papers [30–57]. The new findings for this paper include graphical trend lines or Pearson statistical correlations based on our analyses of data presented in the papers discussed in this report. Two-tail tests are used for statistical significance.

RESULTS

The following material on height and CVD is based on populations in Europe, the US, Japan, China, Papua New Guinea, India, and Pakistan. The findings involve millions of deaths and are grouped under two headings: inter-ethnic and intra-ethnic.

Inter-ethnic findings

European CHD vs height

Analysis of European data on CHD mortality and height provided a strong positive correlation coefficient (r =0.87, p<0.01) based on mortality rates for each country. Figure 1 shows a trend or regression line of the data from two sources for men in the age group of 45–65 years of age in 1985 [38,39]. Height data were based on military conscripts in 1960 and annual mortality data reported by Thom [39] were based on statistics from the World Health Organization. The annual CHD mortality slope for taller vs shorter males is 18.2/100,000/ cm (calculated from source data). Since height data were not given for females, we assumed a height difference of 12.8 cm which was used to calculate a slope of 14.14 deaths/100,000/ cm for females vs 18.2 deaths/100,000/ cm for males. (It should be noted that Northern
Europe has had substantially higher CHD mortality rates compared to the south for over 40 years [40]).

Seven countries study and CHD

The following findings are based on a 25-year follow up of 12,763 men who were enrolled in 1958–64 at the age of 40–59 years. This study, known as the Seven Countries Study, provided height and CHD data for 16 population samples from the US, Europe, and Japan [40, 41]. Analysis of height and CHD data resulted in a positive correlation between height and CHD mortality of r=0.47, p<0.1 and was consistent with European data shown in Figure 1. Figure 2 shows a mortality slope of 27/100,000/cm (calculated from source data).

Mortality data were converted from deaths per thousand over a 25-year tracking period to an average annual death rate/100,000/cm. The lowest CHD mortality was found for men from Crete, Greece, Tanushimaru, Japan, and Ushibuka Japan, who were among the shortest men in the study. Crete had <1/10 the death rate of East Finland.

East Finland had the highest CHD of all the populations; e.g., its males had a CHD mortality of 268 deaths/1000 over a 25-year period compared to the next highest, West Finland with 180 deaths/1000. Recently, East Finland was noted as having the highest serum cholesterol of all the populations and had a high intake of mercury from lake fish, which may explain its outlier status [42]. If East Finland is omitted, a reanalysis results in r=0.67 with p<0.01 and the mortality slope increases to 28.6 deaths /100,000/cm. Figure 2 also shows the average CHD mortality for each of three groups (tertiles) consisting of five populations of similar height.

California ethnic groups, CHD, stroke, and height

Two trend lines for six ethnic groups in California are shown in Figure 3. Wild et al. [43] provided mortality data for 1 million deaths (males and females). CHD and stroke mortality for each group (262,333 deaths) was based on age-standardized mortality for 25–84 year olds. (All-cause mortality followed a pattern similar to the CHD mortality trend shown in Figure 3.) Height data were obtained from another report [35]. The trend lines show a consistent increase in CHD mortality with increasing height. Correlation coefficient (r)=0.85, p<0.05 for males and 0.93, p<0.01 for females. For

<table>
<thead>
<tr>
<th>Study</th>
<th>Subjects</th>
<th>Duration yr</th>
<th>Age at baseline</th>
<th>Increase in CHD stroke</th>
<th>S/NS/NA*</th>
<th>Remarks**</th>
</tr>
</thead>
<tbody>
<tr>
<td>Song et al., 2003 [13]</td>
<td>386,627 males</td>
<td>6</td>
<td>40–65</td>
<td>1.01 1.08</td>
<td>NA</td>
<td>Increase in relative per 5 cm decrease in height</td>
</tr>
<tr>
<td>Wannamethee et al. 19998 [10]</td>
<td>7735 males</td>
<td>16.8</td>
<td>40–59</td>
<td>1.22 1.15</td>
<td>S,NS</td>
<td>Relative risk for shortest vs tallest categories***</td>
</tr>
</tbody>
</table>

* NS – non significant, S – significant; NA – significance data not available;
** adjusted for various risk factors;
*** original data converted to reciprocal to provide consistent comparisons in this summary

![Figure 1. CHD mortality vs height for 11 European countries for males 45–65 years of age.](image-url)

Slope calculations (mortality difference/high difference)

Taller vs short male slope = 18.2 deaths/100k/cm
(calculated from source data)

172.5 cm males vs 159.7 cm females: (230–49)/12.8 cm
= 14.14 deaths/100k/cm

Table 1. Studies showing increased mortality or incidence of CHD and stroke with decreasing height.
males, the height vs stroke correlation coefficient was $r=0.66$ (NS) and for females $r=0.74$, $p<0.1$. The calculated increase in CHD mortality with height for men was 15.2 deaths/100,000/cm vs 18.2 deaths/100,000/cm for the previous European study.

Male California white and black Californians averaged the same height and almost the same CHD mortality rate. Similarly, male Chinese and Japanese Californians had about the same mortality and height. Stroke mortality was substantially lower for Hispanics, Asian Indians, and Japanese males compared to taller whites and blacks. The Chinese males had about the same stroke rate as white male Californians. Inter-ethnic female stroke differences were small, except for blacks who had about twice the stroke mortality as other ethnic groups.

A comparison between the findings in California (Figure 3) and Europe (Figure 1) indicate that the tallest and shortest groups in each male population had about the same CHD mortality rates in spite of substantial differences in geography, climate, diet, lifestyle and ethnicity. For example, northern European males over 175 cm in height had an annual mortality of 319 deaths/100,000 vs 309 deaths/100,000 for California whites and blacks who were over 175 cm. The shortest (<167 cm) male Europeans (Portugal and Spain) had a mortality of 140 deaths/100,000 vs 150 for the shortest (<170 cm) Californians (Chinese and Japanese males).

It should be noted that the findings presented here are not unique to California. Nationwide, Asians, Hispanics, and native Americans and Alaskan natives also have age-adjusted CHD and stroke death rates below that of taller whites and blacks [44].

CVD between taller Belfast residents and shorter Toulouse residents

The World Health Organization Monica project has been studying the CVD mortality rates of 45–64 year old males living in Belfast, northern Ireland and Toulouse, southern France [45]. Based on 800 subjects tracked for 2 years, the taller Irish had substantially higher mortality from ischemic heart disease (IHD), stroke, and other CVD compared to the shorter French. For example, in the age range 55–64, the Belfast males experienced an IHD mortality of 761 vs 175/100,000 for Toulouse males. The annual stroke mortality was 130 vs 61/100,000 for Belfast and Toulouse males respectively. The pattern for these diseases was similar for the younger 45–54 year old cohort. Belfast males averaged 172.2 cm vs 170.0 cm for Toulouse males (correspondence from E.E. McCrum, Belfast Monica Project, 4/11/2000). Total energy and fat intake were almost the same in both cohorts. In addition, neither classic risk factors nor dietary differences explained the large differences in IHD, stroke, and other cardiovascular disease.

Toulouse males (170 cm) had a similar annual mortality rate of 130–175 deaths/100,000 as California Chinese and Japanese males (169 cm) who had 155 and 146 deaths/100,000 respectively. This close relationship existed in spite of substantial differences in lifestyle, diet, and ethnicity.

CHD between taller Indian Hindus and shorter Muslims

A study of 1415 male and 797 female Hindus and Muslims evaluated the incidence of CHD and hypertension in these two Indian populations [46]. Based on ECG data and clinical history, taller Hindu males had a CHD prevalence of 7.1% vs 1.8% for Muslim males.
Hindu and Muslim females had a smaller difference in prevalence of 10.4% vs 6.6%. Hypertension was also substantially higher among Hindu males and females, especially for ages ≥50 years. The Hindu males averaged 2.1 cm taller than Muslim men and Hindu females were 0.8 cm taller than Muslim women. However, BMI and obesity were not significantly different. Smoking rates were also similar in Hindu and Muslim men but higher in Hindu women. Thus, the prevalence of CHD and hypertension was substantially higher in taller Hindus compared to shorter Muslims.

**Intraethnic findings**

**Native American CVD vs height**

Figure 4 shows the increase of CVD mortality with increasing height for three tribes of Indians based on data from the Strong Heart Study [47,48]. A virtually linear increase in CVD mortality occurred when mortality was plotted against height for a population of 1623 men and women aged 65–74 years. A similar pattern was obtained for all-cause mortality but is not shown. This trend was positive ($r=0.98$, $p<0.05$) for older Native Americans but not evident for the age range 45–64. The CVD mortality slope was 120/100,000/cm, which was much higher than the inter-ethnic studies reported. Stroke showed no clear trend with height.

**Japanese CHD vs height**

Evaluation of CHD mortality vs height for three Japanese male populations [49] is shown in Figure 5. The three groups ($n=8093$) of Japanese include mainland Japan, Hawaii, and California. For the age group 60–64 years of age, a linear increase in mortality with height was observed ($r=0.999$, $p<0.02$). The CHD mortality slope was 87 deaths/100,000/cm. A trend line (not shown) for 50–64 year olds also showed a progressive increase in CHD with height ($r=0.58$, $p<0.1$). Stroke mortality for ages 55–65 years was substantially higher in Japan vs Hawaii and California. However, taller California Japanese had 2.3 times the stroke mortality of shorter Hawaiian Japanese for ages 60–64.

**Chinese CVD vs height**

A joint US, UK, and Chinese Government study found that CHD mortality was positively correlated with increasing height ($r=0.33$, $p<0.05$) [50]. The CHD mortality slope was 63 deaths/100,000/cm. Stroke mortality also increased with height ($r=0.20$) but was non-significant. The study was based on 65 counties throughout China. Most of these counties were rural and consisted mostly of the Han ethnic group.

Campbell et al. [51] reported that Sichuan and Guizhou counties had extremely low CHD. The study involved >400,000 men and women tracked over 3 years. For the age group 0–64 years, no one died from coronary artery disease. The populations in these two counties were next to the shortest of the 65 counties being studied and well below northern European heights.

**CVD in Kitava**

Lindeberg et al. [52] have studied the Kitavan population for over 10 years. These Melanesian people live on an island near Papua New Guinea and follow a traditional horticulturalist lifestyle almost entirely free of Western culture and diet. In addition, a secular trend in growth does not appear to be evident in Kitava.

After studying 220 randomly selected subjects aged 14 to 87 years of age, they found no evidence of IHD or stroke. This population was short with males averaging under 162 cm and females 150 cm. Not all aspects of their lives were healthful. Almost 80% of the men and women smoked and 100% were betel nut chewers. In addition, physical activity was only slightly higher than sedentary Western populations. Height declined only 1 cm from 20–39 to 60–86 years of age, and weight declined by 8 kg for the 60–86 year age range for both men and women.

**CVD in Okinawa**

Okinawa is an island off mainland Japan. Okinawans have been studied extensively for 25 years because they...
have healthy longevity and an exceptional number of centenarians [53,54]. They are also short and have the smallest physique of all Japanese. Death rates from stroke and heart disease are over 40% lower than the taller mainland. The annual death rate per 100,000 from CHD is 18 vs 102 for Sweden and 100 for the US [54]. Stroke mortality is lower than that of Sweden, Italy, and Greece but 25% higher than for the US. However, Willcox et al. [54] reported that studies of the elderly Okinawans found that they have exceptionally young arteries and low blood homocysteine and cholesterol levels compared to Westerners.

The heights of the elderly (75 to <100 years) average 158.5 cm for males and 144.8 cm for females [55]. However, centenarian males average 148.3 cm and females, 138.6 cm. When Okinawans suffer heart attacks, they are more than twice as likely to survive than Americans. Willcox et al. [54] observed that Okinawan heart health is not due to genetics because younger Okinawans experience substantial increases in heart disease associated with changes in diet and lifestyle. Younger people have also seen a secular increase in body size as well [55].

**CVD between taller and shorter Pakistanis**

A Pakistan study compared the incidence of IHD of two urban populations consisting affluent and poor people [56]. The study included 4232 male and female adults. Based on weight and BMI data, we calculated the heights for the two cohorts. Compared to the short cohort, affluent males were 2 cm taller and females 4 cm taller. The taller affluent population had a relative risk for IHD 3 times that of the shorter cohort. This risk was higher for all age groups ranging from 25 to >65 years of age. In addition, the shorter cohort averaged 5 to 4 years older than the taller cohort, and thus the IHD risk of the taller cohort was underestimated because the incidence of heart disease increases rapidly with age.

**CVD and height in Great Britain**

A long-term British study of regional differences in heart disease of 7735 men has been in progress since 1978 [57]. Based on a recent 22-year follow-up of men averaging 60 to 82 years of age, taller non-smoking men in the more affluent south were found to have a 24% lower age-adjusted CVD mortality rate compared to shorter non-smoking men in other parts of Great Britain. However, shorter men (~1 cm) who migrated to the south from other parts of Great Britain had an 18% lower age-adjusted risk (NS) of heart disease incidence than the taller males born and living in the south. In addition, CVD mortality risk declined 16% (NS) for shorter migrants to the south. Paradoxically, taller men born in the south who moved to other parts of Great Britain had a 32% higher risk (NS) of CVD mortality vs shorter migrants living in the south.

**Female and male CHD differences**

Females have substantially lower CHD mortality within ethnic groups throughout most of the world. As can be seen in Figure 3, the slope for women is similar to that of men except that absolute annual mortality is lower by about 100 deaths/100,000 population. Remarkably, the average of mortality slopes for European males and females and US white females and males (calculated from data in Eberhardt et al. [44]) is 18.4/100,000/cm vs 18.2/100,000/cm for European short and tall males. Thus, the similarities in slopes appear to be relatively independent of gender and provide additional support for a real positive relationship between height and CHD mortality. It should be noted that these findings are consistent with research by Miller [58] who found that women and men of the same height had about the same average longevity. Rollo [59] also reported that the difference in male-female longevity of rodents was due to sexual size dimorphism or greater size.

**Childhood height and adult CVD risk factors and mortality**

The Minneapolis Children’s Blood Pressure Study [60] tracked the CVD risk factors of 679 individual males and females from 7.7 yrs to 23.6 years of age. The results of this study indicated that increased height at 7.7 yrs of age was positively correlated with increased adult levels of fasting insulin (r=0.12), triglycerides (r=0.13), and systolic BP (r=0.21). Greater height at 7.7 yrs was also negatively correlated with HDL levels (r=–0.08) in adulthood. All correlation coefficients were highly significant (p=0.0001). It was also found that height in childhood tracked height into young adulthood. The results were nearly identical for male, female, black, and white subjects. The study also produced similar results (although the correlation coefficients were larger) when the separate effects of absolute weight and BMI on CVD risk factors were evaluated.

A longitudinal Finnish study found CHD hazard ratios (HR) in adulthood increase with childhood height [8]. Based on 3048 males, adults who were shortest at 11 years of age had a 43% lower HR for CHD mortality compared to the tallest. The mean age at death was 58 years (range: 38 to 71 years).

**DISCUSSION**

The findings of this report are based on heterogeneous and homogeneous populations and reflect much larger populations than studies that have found short people have higher CVD risk factors, incidence, or mortality. Our data conflict with the previous findings of other researchers. We have not presented all our findings here but Eaton et al. [25] reported that clinical and post-mortem studies found little or no CHD in Bushmen, Congo pygmies, Navajo Indians, arctic Eskimos, New Guinean highland natives, Masai pastoralists, Australian Aborigines and Solomon Islanders. (Developing populations historically have experienced low CVD [61]). These populations are generally shorter than Western populations. In Sweden, Allebeck and Bergh [16] also found the tallest men had 1.9 times the mortality risk from CVD and respiratory disorders vs the shortest men in a longitudinal study spanning 20 years. In addition, Walker et al. [62] reported that rural black South
African children were fed about half the calories compared to white South African children and were 5% shorter and had virtually no CHD in adulthood. One of the authors of this paper led a team of health professionals to study the Tarahumara Indians in Mexico. The team found no evidence of heart disease among the 16 men who averaged 162.6 cm [28]. (Other larger studies have also confirmed these findings [63,64]). These preceding groups were short except for the Masai who average about 172 cm but weigh only 64 kg. With changing diets and lifestyles, these findings may no longer be valid. For example, Cretans had extremely low CHD in the 1960s. By 1988, they had grown taller (but not heavier), and CHD mortality increased by 14% and stroke mortality by 24% [65].

In spite of their low CHD levels, the Japanese have higher stroke levels compared to some European countries and the US [54]. A large US study of 121,700 female nurses tracked them over a 14-year period and found no relation between height and stroke. However, some short groups such as US Hispanics and US Japanese have substantially lower stroke rate than taller whites, and Kitavans have virtually no stroke. At older ages, Native Americans also have a substantially lower stroke rate than the general population. Shorter ethnic groups in California also have substantially lower stroke mortality based on 56,449 deaths [43], and shorter men from Toulouse had about half the stroke rate of taller Belfast men [45]. Another factor related to developing countries is that infectious disease is an important cause of stroke whereas diet [66], weight, and lifestyle risk factors are the major factors in developed countries. Thus, poverty and poor sanitation can lead to higher stroke levels in developing countries which have nothing to do with height.

The Japanese present a paradox in that they have seen substantial increases in height, cholesterol levels, hypertension, and diabetes over the last 40 years but CHD mortality has not changed much. However, obesity is still low, smoking and salt intake have declined, and medical care has improved. Sekikawa et al. [67] observed that, based on dietary and lifestyle changes, the Japanese CHD rates should be similar to that of the US. They suggest that the level of Japanese CHD may be substantially higher than statistical data indicate due to the artifact of incorrect classification of causes of death.

In the US and most of Western Europe, we have seen a 50-year trend of declining CVD mortality which has paralleled secular increases in height and obesity. This paradox is probably correlated with changes in diet and improved medical practices involving prevention and treatment of heart problems.

Short height per se does not appear be a risk factor based on extensive data showing rapid increases in CVD and diabetes as societies experience temporal increases in height and weight. For example, S. Korea has seen a sharp increase in CVD mortality over the last 40-years [68]. This increase in mortality paralleled a secular increase in body height and weight. Singapore has also experienced an increase of ~400% in CVD over a 30-year period which has seen parallel changes in diet and body size. China has also seen rapid rises in CHD concurrent with rapid increases in height over the last 50 years [50].

Magnitude of findings
As can be seen from Table 1, studies showing shorter people have greater risk of CVD incidence or mortality find relatively low differences in risk; e.g., shorter people experience 1% to 32% higher risk than taller people. In contrast, the studies presented in this paper show taller people often have 100% to over 400% greater CHD/IHD mortality as shown in Figures 1, 2, 3, 5. In addition, Keys et al. [69] findings show the tallest group had >300% higher mortality compared to the shortest, and the Dutch had a 576% higher CHD mortality compared to men from Crete who were 9 cm shorter [40]. While our stroke findings are not as strong as CHD findings, some of the studies in this paper have shown taller people have over 100% higher stroke rates compared to shorter groups [43,45]. This is substantially higher than the less than 30% increased stroke risk of shorter people found in recent conflicting studies (Table 1).

Our findings are also based on much larger population samples. For example, studies identified in Table 1 showing shorter people have higher CVD are based on about 11,000 deaths. This is a small sample compared to findings in the Results showing lower CVD for shorter people based on millions of deaths.

Impact of risk adjustments on results
Most of the findings presented in this paper do not involve risk adjustments. Therefore, a certain amount of error may apply to these findings. For example, studies finding taller people have lower CVD mortality than shorter people usually adjust for a variety of factors, such as SES, smoking, BMI, alcohol intake, hypertension, and physical activity. However, the magnitudes of these adjustments are relatively small, and they reduce the CVD mortality risk of shorter people by 2% to 17% (based on studies in Table 1 and [9]). If we assume that the advantage of risk adjustment for this study is reversed from previously published studies and favors tall people, the impact on the results of our study would be negligible. For example, a 2% to 17% reduction in risk would not neutralize the excess mortality of 100% to over 400% for tall people reported in this report.

Consistency with related findings
The lower CHD and stroke rates of shorter people are consistent with lower cancer death rates and longer longevity of women and shorter people described in previous reports [30–37,58,70]. Most recently, Krakauer (personal communication 7/8/02) found in a 10-year study of 116 Swedish men and women (50–74 years of age) that height was negatively correlated with age at death (r=-0.26, p=0.006). In addition, the age at death due to CVD decreased with height at the rate of ~0.4 yr/cm. Another study of 1 million recruits during WW I
found tall men were ‘especially prone’ to cardiac disorders [19].

While California and Europe have substantial differences in lifestyle, race, ethnicity, climate, and geography, the studies reported in the Results are highly consistent; i.e., California Chinese and Japanese men had about the same height and CHD mortality as Spanish and Portuguese men. Likewise, California blacks and whites had about the same height and CHD mortality as northern Europeans. Thus, in spite of many differences in ethnicity, SES and life style, height seems to be a more powerful predictor of CHD mortality.

The same increase in CHD mortality rates between taller and shorter European men and between taller men and shorter women is consistent with the difference in life expectancy between men and women compared to their difference in height. For example, in 1992, we found that men had an 7.5% lower life expectancy compared to women and 7.3% taller [30]. Similar findings were found recently for 5 other populations; e.g., based on 21 European countries, males were found to be 7.7% taller and had an 8.0% lower life expectancy compared to females [36]. While this remarkable inverse correlation between life expectancy and height may not be causally related, Table 2 shows lifespan reduction for increasing height (slope) for five populations is highly consistent. The slopes (years/cm) for a mix of male-female, female only, male and female, and men only are very close ranging from −0.35 to −0.82 yr/cm. The slope (−0.47 yr/cm in Table 2) for 21 European countries is based on differences in average life expectancy and height between males and females. These slopes are also consistent with previous findings described in 1992 and 2003 reports [30,36]. Krakauer et al. (personal communication, 7/8/02) found a height-age slope of −0.52 yr/cm which is identical to our 1992 findings and very close (−0.50 yr/cm) to our 1999 findings and −0.52 yr/cm which is consistent with our 1992 findings [30,31].

While a number of confounding factors could explain some of our findings, it interesting to note that the Belfast-Toulouse study researchers concluded that ‘Neither the classic risk factor scores nor the similarity in major nutrient intake adequately explain the large differences in IHD and other causes mortality between the centres [45.]’ Shaper [71] also observed that ethnic, racial, climatic and geographical factors are ‘completely irrelevant’ to the incidence of CHD except when they determine social class and economic status. Certainly, the findings are consistent across ethnic differences as found in California or among homogeneous populations, such as in China and Kitava [43,50,52]. In spite of these observations, other factors certainly play a role and are discussed in the following sections.

### Impact of BMI on findings

Since increasing BMI is normally associated with increasing CVD, BMI may have confounded the results of this study if taller people had higher BMIs than shorter people. However, evaluation of BMI differences and CHD/stroke incidence or mortality does not indicate a major impact on our findings. For example, the shortest half of cohorts in the Chinese study (Results section) had an average BMI of 20.7 kg/m² vs 20.9 kg/m² for the tallest. Thus, this BMI difference of about 1% should not have confounded the results. The BMIs of Belfast and Toulouse cohorts differed by only 0.3 kg/m² and this small difference cannot account for the large CVD mortality found. In contrast, the three cohorts of Native Americans were all overweight or obese. Paradoxically, CVD mortality declined with decreasing height and increasing BMI. We found a decrease of 2.6 kg/m² from the shortest to the tallest cohort of Native Americans in parallel with a substantial increase in mortality by height.

No BMI data were provided for the Seven Countries Study (Figure 2). However, relative body weight (RBW) (average weight for each ethnic group/standard table weight for height) was given for 14 of the 16 cohorts. Excluding the outlier East Finland, analysis of these data indicated that r = 0.56 p < 0.05 for RBW and mortality [40]. Comparison of the three tallest and three shortest cohorts indicated that the tallest cohort had a 7.3% higher RBW. This small a difference cannot explain the 204% excess mortality for the three tallest populations.

The BMIs for the three Japanese cohorts (Figure 5) were consistent with increasing CVD mortality with the lowest mortality cohort having a BMI = 21.2 and the highest mortality cohort a BMI of 24.4. Based on another Japanese study [72], a 3.2 kg/m² increase in BMI should result in a 37% increase in annual CHD mortality. Subtracting this percentage to compensate for the higher BMI would reduce the taller cohort’s excess mortality from 133% to 96%. The California study (Figure 3) involves Japanese and Chinese men who have a 2.1 kg/m² lower BMI and >100% lower mortality compared to whites. Thus, the lower Chinese and Japanese BMI could explain part of the difference in mortality (25%) but doesn’t account for the remaining 75% excess mortality. California Hispanics also had about 70% lower CHD mortality compared to white

<table>
<thead>
<tr>
<th>Study population</th>
<th>Sex</th>
<th>Slope (yr/cm)</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>21 European countries</td>
<td>Male vs female</td>
<td>−0.47</td>
<td>Average slope for University students (slope range: 0.23 to 0.61 yr/cm)</td>
</tr>
<tr>
<td>19th century French</td>
<td>Female</td>
<td>−0.43</td>
<td></td>
</tr>
<tr>
<td>Cleveland Ohio</td>
<td>Both</td>
<td>−0.47</td>
<td>Average slope for both sexes combined</td>
</tr>
<tr>
<td>Male to male populations</td>
<td>Male</td>
<td>−0.52</td>
<td>Slope for various male to male comparisons (slope range: 0.35 to 0.82 yr/cm)</td>
</tr>
<tr>
<td>Swedish study</td>
<td>Male &amp; female</td>
<td>−0.52</td>
<td>Krakauer, J (personal communication, 2002)</td>
</tr>
</tbody>
</table>

* slopes for seven populations (based on average ages at death for each population)
Table 3. Heart disease and stroke risk factors correlated with increased height.

<table>
<thead>
<tr>
<th>Risk factor</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>Larger left ventricle mass &amp; left ventricle hypertropy [78,79,81]</td>
<td>Both are independent CVD risk factors</td>
</tr>
<tr>
<td>Higher blood pressure [60,80,82,83]</td>
<td>BP strongly r with height</td>
</tr>
<tr>
<td>Higher BMI/obesity [32,60,84,85,86]</td>
<td>Over 80 population samples show weight increases as the cube of the increase in height (or BMI increases linearly with height)</td>
</tr>
<tr>
<td>Higher C-reactive protein (C-RP) [21]</td>
<td>Still controversial as to whether C-RP is the cause or a symptom of CVD disease</td>
</tr>
<tr>
<td>Lower HDL [60,87,88]</td>
<td></td>
</tr>
<tr>
<td>Lower SHBG [50,89,90]</td>
<td>Lower SHBG correlates with higher CHD mortality</td>
</tr>
<tr>
<td>Higher insulin [60,88,90]</td>
<td></td>
</tr>
<tr>
<td>Higher cholesterol or triglycerides</td>
<td>Also co-related with increasing BMI [60,88]</td>
</tr>
</tbody>
</table>

Californians but their average BMI was slightly higher. In conclusion, it does not appear that the relatively small variations in BMI account for the large increases in mortality found with increasing height.

**Height, body size, and nutrition**

Height and nutrition are related. Abundant protein and calories lead to taller height and heavier bodies [50]. Amador et al. [73] reported that the same factors that make us fatter make us taller and increase our lean body mass. So the question arises whether height per se promotes CHD and stroke or a diet that promotes body growth. It appears that both apply. Based on a study of 27,862 men and women, Trichopoulou et al. [74] found that protein was responsible for obesity in adulthood rather than fat or carbohydrates when adjustment for their energy contribution was made. McCarty [75] also reported that high protein in combination with high carbohydrate intake potentiates insulin response and promotes increased adiposity. In the 7 Country Study, animal (except fish) products, butter, milk, and pastries were significantly and positively correlated with CHD mortality [41].

Although human observational studies cannot prove the thesis that greater height increases mortality, experimental animal studies are consistent in showing that smaller size (shorter length and lower weight) is related to greater longevity independent of diet as in the case of Barke’s dwarf mice and Rollo’s giant mice [76,77]. In a comprehensive review of rodent body size vs longevity, Rollo [59] found a substantial negative correlation between weight and longevity based on 760 studies (r = -0.4, p < 0.0001). Since it is unlikely that greater longevity can be obtained without improved heart function and durability, it is likely that heart disease is reduced in shorter, lighter bodies as in the case of Cretans, Kitavans, and Okinawans.

**Biological considerations**

A number of biological risk factors are related to increased height and CVD. These include higher left ventricle mass (LVM), left ventricle hypertropy (LVH), blood pressure (BP), BMI and others shown in Table 3 [21,32,50,60,78-90]. Both LVM and LVH are independent risk factors for CVD. Cordain et al. [90] has also attributed increases in height and many modern diseases to the increased consumption of high glycemic foods which have raised levels of insulin and insulin like growth factor-1 (IGF-1) over the last two centuries. They have pointed out that increased levels of insulin and IGF-1 promote faster growth, greater height and obesity, earlier puberty, and metabolic syndrome (which is related to heart disease, hypertension, and dyslipidemia).

While taller, proportionately heavier bodies may be at higher CHD risk, thin, tall bodies may have lower CVD due to a heart scaled for a bigger body which has a relatively smaller functional load. Thus, the relative size of the heart may be a factor in CVD risk. In addition, greater heart growth (hyperplasia) during childhood may improve functional capability while hypertrophy during adulthood may reflect a response to greater height, higher blood pressure, higher BMI, or other pathological conditions.

Almost all studies have found that higher birthweights are correlated with higher adult BMI [68]. Height is also positively correlated with birthweight (37). Typically, BMI increases 0.5 to 0.7 kg/m² for each kilogram increment in birthweight; subsequent accelerated childhood growth also contributes further to increases in BMI. Thus, increasing birthweight, macrosomia, and rapid growth have a direct effect on adult BMI and explain at least part of the modern trend towards overweight and obesity [37]. This increase in BMI can thus lead to increased CVD and diabetes (an independent risk factor for CVD). However, low birthweight coupled with childhood catch up growth can also lead to increased adult BMI and CVD.

**Why the conflict between findings?**

Why do our findings conflict with previous studies? First, height is only one part of the total CVD picture. Other risk factors can overwhelm the influence of height on CVD mortality. These include socioeconomic status (SES), relative weight, lifestyle, lipid profile, diet, birthweight, medical care, rate of growth during childhood, and genetics. For example, Stamler et al. [91] found that three large, long-term studies were consistent in showing that lower serum total cholesterol (TC) was correlated with lower mortality from CHD and stroke with a TC of <160 mg/dL being optimal. And Bavdakar et al. [88] reported that there is a clear positive association between increasing height and obesity in developing countries.

**Low birthweight and catch-up growth**

A possible reason for the conflict between our findings and those of others may be related to childhood growth.
Forsen et al. [92] reported that small birth size in combination with improved postnatal nutrition and catch up growth increases adult CHD for both sexes. Thus, attempts to compensate for small childhood body size may result in catch up growth due to overnutrition and this greater growth may result in excessive weight or other harmful health impacts resulting in adult CHD. Analysis of data from Sorensen et al. [93] has shown that adult height is linearly tied to birthweight (BW). Thus, even with extra nutrition, many low birthweight children probably attain shorter adult height and above average BMI. Low birthweight per se does not appear to be the driving force for higher CVD because Bradley [94] and others [37] have reported that less developed populations produce substantially lower BW children who have almost no CHD in adulthood. Thus, in developed countries, shorter adults may experience a disproportionately greater childhood catch up growth and adult CHD compared to higher BW, slower growing children.

Socioeconomic status

Higher SES has been shown to have a substantial positive effect on health and a negative effect on mortality [6,32]. In addition, there are more tall people in higher social classes compared to lower ones [11], and lower SES people in Western society tend to be fatter, less active during non-working time, smoke more, and generally have poorer diets and medical care. While some studies have adjusted for SES and found that the inverse relation between height and mortality holds, others have not [18]. Measurement of SES risk factors is imprecise and crude and even after adjustment, substantial residual confounding is likely to remain [57,95]. For example, the process of adjusting for SES may not identify all factors, and when the factors identified are assessed, they may not be correct in magnitude. Poor self image, behavioral problems, hostility, depression, and dissatisfaction with life in lower SES people may be powerful influences on health and are difficult to accurately quantify. In addition, Davey Smith et al. [6] pointed out that studies of the impact of socioeconomic factors on health and mortality risk are inadequate unless all three stages of life are evaluated in terms of their impact on adult mortality.

Age of subject population

CHD mortality represents a relatively high percentage of all deaths for males in their 40s. However, the total death rate is still quite low for ages below 65 years of age and over 75% of a birth cohort is still alive at 65 years of age. Therefore, a low age range of a study population can fail to show the positive relation presented in this report as was reported previously for younger and older Native Americans. Jousilahti et al. [11] also found the 56–64 age group had virtually no difference in CVD risk between tall and short people.

Shorter people are more likely to be overweight

Since food portions tend to be standardized, short people tend to consume more food and calories in relation to their size than taller people, especially if they are of lower SES. Thus, a glass of milk, a hamburger, steak or potato, or complete meal is served without regard to one’s adolescent or adult size, especially when eating out. As little as 60 extra kilocalories a day can result in a 20 kg weight increase in 10 years. The impact of this excess calorie intake is probably greatest during middle and older ages when physical activity and metabolism tend to decline.

Western diet, smaller coronary artery vessels, body size, and CHD

Smaller people have smaller blood vessels which when combined with a Western diet rich in saturated fat, sugar and protein promotes increased weight and serum homocysteine which in turn promote damage and clogging of arteries [4,31,32]. Mendall et al. [21] found that taller people had higher levels of C-reactive protein although it was only significantly related to all-cause mortality and was not significant for CHD mortality. The fact that immigrants from countries with low heart disease maintain this condition in the US and other developed countries suggests that early childhood dietary practices have a long-term protective effect which may extend for 20 years or more after immigration [96]. McCarron et al. [97] observed that short height may promote increased CHD risk only when combined with high cholesterol and obesity.

The atherogenic risks of smaller blood vessels combined with a fat-rich diet and obesity appears logical, but women with their smaller vessels present a paradox because they appear to be at lower risk of CHD compared to bigger men with similar diets. A possible explanation is that smaller bodies within a species may result in more efficient cardiac function [31,98]. Smaller bodies also have lower BP, especially in a low salt environment as found in the Intersalt study [98]; e.g., out of 52 populations studied, the lowest BP was found in four short populations and increased with increasing height of this group but not with age. The Yanomamo Indians (152.5 cm for males and 142.1 cm for females) had the lowest BP with a male systolic BP a little over 100 mmHg which declined with age. Voors et al. [99] also observed that BP increased linearly with height and weight. This increase was related to greater blood volume and peripheral resistance, which are related to total body mass with lean body mass having a greater effect than fat mass.

Shorter height may be due to childhood illness

A certain percentage of shorter adults may have long-standing health impact due to congenital or childhood illness. About 50 types of illnesses can cause growth stunting and some can have a subtle long-term impact on health [100]. In a large study of short school children, 18% were found to have an underlying medical problem [101]. In some cases, the impact on health may not be evident until many years later. Thus, studies, such as described in Table 1, may be detecting increased mortality of shorter people which is due to early illness.
that caused the shorter stature (reverse causation). Some examples of diseases that can retard linear growth include rheumatic fever, asthma, H. pylori infection, and intestinal infections.

Small age differences between cohorts can introduce errors

If age cohorts are used in a study, shorter people can have a higher mortality simply because they are older. For example, in one study the shortest men were found to be about 4.5 years older than the tallest [5]. Thus, for a 5-year cohort of 60–65 years, most short men will average closer to 65 years and most tall men will average closer to 60 years. If CHD mortality increases by 80% from 60 to 65 years, the shortest men could have a 40% higher mortality simply because they are 2 or 3 years older. Thus, researchers need to adjust for even small age differences to correct this source of error.

Taller people tend to be younger and benefit from the increasing life expectancy over the last 100 or more years. For example, a taller person born 10 years after an older shorter person also has a 2.5 year longer life expectancy due to improved living conditions and medical care. Thus, a shorter person born in 1950 who had a non-fatal CVD event at 50 years (1980) would have a poorer mortality prognosis than a taller person born in 1940 who experienced a non-fatal CVD event at 50 years of age (1990).

Smaller size in animals, CVD, and longevity

We are not aware of studies comparing heart disease in animals with animal height or body size. However, smaller animals within a species generally live longer and have delayed development of CVD and other degenerative diseases [31,76]. We recently analyzed height vs longevity data for a sample of about 13,000 deceased dogs and found a substantial increase in longevity with decreasing height [35]. Although our source data did not provide information on CVD death rates, it is unlikely that the smallest dog breeds had CVD problems. In addition, Patronek et al. [102] reported that larger dogs have earlier onset of cancer and other degenerative disease, which presumably included CVD. Height differences in humans can differ by about 20% but dogs differ up to 600%. If short height was correlated with increased mortality, this huge height difference would result in a large increase in CVD morbidity or mortality with decreasing height among dogs. Therefore, it is highly unlikely that small dogs would outlive large ones; e.g., over 9% of the shortest breeds and 3% of middle breeds were still alive after 15 years of age but none of the tall dog breeds were alive [35].

Using the same BMI gives taller people an advantage

A number of height vs CHD studies have compared shorter and taller population samples with approximately the same Body Mass Index (BMI), which is a measure of relative weight for height and an index for classifying people as underweight, normal weight, overweight, or obese. Although not perfect, it is an excellent index for predicting health problems in adolescents and adults. The healthy normal range of this index is generally 22.0–24.9 kg/m² but a large study of men and women found an optimum range of 18.5 to 21.9 [103]. Another 30-year study of 6040 healthy men (45–68 years at baseline), found men with a BMI <20 and the strongest grip strength had the lowest mortality [104].

The BMI (kg/m²) is a quadratic equation in which height is a squared function. The Ponderal Index (PI), however, is an index number like BMI but it is a function of the cube of the increase in height and its units are kg/m³. Since BMI has been found to correlate well with skinfold thickness and total percent body fat, epidemiologists prefer its use over the ponderal index (PI), which was more commonly used in earlier work. However, this practice of using BMI results in comparing tall people of linear body build to short people of more stocky build. Thus, to provide an even playing field for both tall and short people, the PI should be used because body proportions would be more closely aligned since biologists have found that as animals increase in body size their body weight increases in accordance with cube increase in height or length, not the square as in the BMI [105].

While some studies have found a quadratic relation between height and weight, we found >80 diverse populations which increased in accordance with the PI rule. Some of these population comparisons were based on comparing two birth cohorts born 10 to 25 years apart and height and weight differences reflected secular growth trend. For example, in a comparison of about 500,000 male Swedish 18 year olds, the actual weight exceeded the calculated weight by 6% using the PI formula. A Harvard study also found that four generations of Harvard male students followed the PI rule [32]. A sample calculation follows for Harvard male entrants in 1958-59 and the 1930s [32]:

1958–59: height (ht): 178.5 cm weight (wt): 73.7 kg;
1930s: height (ht): 173.8 cm weight (wt): 66 kg.

Calculation of the predicted weight using the PI equation follows: wt taller = (ht taller/ht shorter)² × wt shorter = (178.5 cm/173.8 cm)² × 66 kg = 71.5 kg (predicted) vs 73.7 kg (actual) Thus, the actual weight increase for height exceeds the PI prediction by 3%.

The health of shorter and taller people should be compared based on the same body build or proportions to provide a fair comparison. Thus, a 1.52 cm male weighing 50 kg should be compared to a 1.83 cm male weight of 86.4 kg. The shorter male BMI would be 21.5 and the taller male 25.8 kg/m². Since CHD and all-cause mortality rises in step with increasing BMI up to 75 years of age [106], the taller person would have a higher mortality compared to a shorter person with the same proportions. Consequently, the use of the same BMI for tall and short people subjects short people to an inherently biased health test.
Publication bias

The popularity of the paradigm that taller people have lower CVD may impede publication of research which shows the opposite trend. For example, a large Kaiser Permanente study [87] found that taller people had higher CVD risk factors. The paper was presented at the annual conference on CVD epidemiology and prevention sponsored by the American Heart Association. This prospective study of 5115 young male and female adults tracked for 10 years found that tall individuals were at increased risk for CVD, which included higher BP, lower HDL levels, and higher insulin levels. While these findings, reported in 2000, strongly opposed the popular paradigm, it seems that a paper on these contrary findings would have been a valuable contribution to the literature. However, we could not find evidence that it was published in any medical journal. Whether this particular paper remained unpublished because of bias is unknown; however, publication bias may be a possible reason for the dearth of findings showing taller height may promote CVD.

CONCLUSIONS

Women [107], the Japanese, the Chinese, and most short populations within traditional cultures experience much less CHD than taller populations in Western countries. Some shorter groups such as Hispanics and Kitavans, have both very low CHD and stroke. While some of our findings do not show as strong a positive relation between stroke and height, the California ethnic study based on 56,449 stroke deaths shows a substantial relation [43]. Also, Kitavans, who are substantially shorter than Europeans, were stroke free. The Okinawans also had substantially less stroke than taller mainland Japanese.

The results showing that shorter people have less CHD than taller people are not confounded by SES, climate, life style, ethnicity, race, geography, because short Europeans (Spaniards, Portuguese, and Toulouse Frenchmen) have the same low CHD mortality as short Californians (Chinese and Japanese). In addition, tall Europeans (Scandinavians) have almost the same high CHD as tall Californians (Chinese and Japanese). In former college students: Methods of study and observations on mortality from coronary heart disease. Am J Pub Health, 1966; 56: 962-71

As mentioned, traditional societies generally have much lower levels of CHD, stroke, cancer and diabetes. Since these societies are characterized by lower birthweight and shorter height, it is illogical to attribute increased CVD to shorter height. A more rational explanation would be that in overfed Western societies [108,109], a certain percentage of the people who grow up shorter have nutritional or other health issues during pregnancy or childhood that reduce final height and promote health problems in adulthood.

REFERENCES:


