

## Role of height in cancer and cardiovascular disease

Thomas T. Samaras

[ **Abstract** ] Findings are presented showing how height relates to cancer and cardiovascular disease (CVD). The preponderance of evidence shows that height is positively correlated with cancer. However, studies on height and CVD are not consistent, and the causes for this conflict are explored. The role of nutrition in promoting rapid growth, greater height and chronic diseases is also discussed. The interrelationships among increasing birth weight, rapid growth in height and weight, obesity, cancer and CVD are also reviewed. Biological mechanisms are presented to explain how height is related to cancer and CVD.

[ **Key words** ] height; cancer; cardiovascular disease; nutrition; biological mechanisms; telomeres

### INTRODUCTION

Many health professionals believe that taller height is correlated with better health and longevity. This belief is based on the parallel increase in height and life expectancy over the last 150 years. In addition, a number of studies found that taller people have lower all-cause mortality and cardiovascular disease (CVD). However, numerous exceptions indicate that shorter height and lower body weight promote lower CVD and cancer and greater longevity<sup>[1-5]</sup>.

Most studies that find taller people have lower all-cause and CVD mortality are based on industrialized societies. However, scores of studies involving developed and developing populations have found shorter people have the same or lower all-cause and CVD mortality as taller people<sup>[1,6]</sup>. In addition, many of the studies showing shorter people have lower cancer and CVD are based on much larger population samples than those showing the opposite trend. Few studies show that tall people actually live longer than shorter people. In fact, most centenarians are short even after adjusting for shrinkage with age<sup>[7]</sup>. For example, male centenarians in Okinawa, Italy, and Cuba average less than 155 cm

after adjustment for shrinkage<sup>[9-10]</sup>. It is unlikely that these centenarians could have reached 100 years of age if they had heart problems or cancer in old age.

Although tall people have substantial health advantages due to socioeconomic and medical benefits, no tall Western populations are free of CVD (coronary heart disease, circulation problems and stroke). In contrast, a number of short traditional populations appear to be free of CVD. These include Kitava, Cook and Solomon Islands, New Guinea, Congo pygmies, Kalahari bushmen, Yanomamo indians, and Tarahumara indians<sup>[1,3,6,11]</sup>.

The reasons for these conflicting findings involve socioeconomic, education, health care and life style differences. In addition, early childhood health problems retard childhood growth, undermine adult health and increase later mortality. Early malnutrition and illness also stunt growth giving the impression that shorter height is an intrinsic negative health factor. However, researchers are well-aware that modern Western dietary practices are related to increased height and CVD<sup>[12-14]</sup>. As populations experience improved economic status, their dietary practices change and they grow taller and heavier.

Increased chronic disease then follows this growth pattern. In fact, before the industrial revolution, the chronic diseases of today were rare even among the elderly, and people were shorter and lighter<sup>[15]</sup>.

After finding that shorter people live longer based on a study of 1.3 million young men tracked over a 70-year period, Holzenberger, *et al.*<sup>[4]</sup> concluded that the increase in life expectancy over recent years has masked the negative health aspects of increased height.

The following summarizes various findings related to height, cancer and CVD morbidity and mortality. The relationship between height and longevity is not covered in this paper. However, it is intimately related to greater cancer and CVD pathology. For information on longevity and body size, refer to other sources<sup>[3,4,5,11]</sup>.

## MATERIALS AND METHODS

Over the last 20 years, literature searches were conducted on Medline and PubMed to find papers that specifically addressed the relation between height and cancer or CVD. These papers were evaluated to determine whether increasing height affected the incidence or mortality due to cancer or CVD. Government and non-governmental reports were also reviewed.

## RESULTS AND DISCUSSION

The following sections discuss findings on height and its relation to cancer and CVD morbidity and mortality. The section concludes with how birth weight, obesity and childhood growth affect increased risk of cancer and CVD in adulthood.

### Results

In the recent past, the elderly in traditional societies were almost free of chronic disease, including coronary heart disease (CHD) and many cancers<sup>[12,16]</sup>. However, the following focuses on CVD and certain major cancers, which are generally viewed as modern Western diseases.

### Cancer and height

Many studies have found a positive correlation between

height and cancer<sup>[3]</sup>.

Miller and Austad<sup>[17]</sup> reported that their findings “present a strong case” for increased longevity of smaller body size in mice, dogs and humans through reduced mortality from various types of cancer. Recently, an extensive review by the World Cancer Research Fund/American Institute for Cancer Research (WCRF/AICR) concluded that there was “strong, consistent and impressive” evidence that height was positively correlated with a number of cancers<sup>[18]</sup>. The WCRF/AICR report was prepared over a 5-year period and involved the review and evaluation of thousands of studies, including studies related to obesity and other chronic diseases.

Zuccolo, *et al.*<sup>[19]</sup> conducted a meta-analysis of 58 studies involving prostate cancer and found that greater height was tied to greater risk of prostate cancer. There was also stronger evidence that height was tied to high-grade prostate cancer and this was mainly associated with longer legs. Engeland, *et al.*<sup>[20]</sup> conducted a 21-year follow-up study of 950,000 Norwegian men and found that the tallest men had a 72% higher risk of prostate cancer compared to the shortest men. During the study, 33,300 cases of prostate cancer were identified.

Gunnell and associates<sup>[21]</sup> conducted a review of ~300 studies on height and cancer. They found a large body of literature which indicates that taller people have a 20% ~60% increased risk of a variety of cancers, such as breast, prostate, colorectal, thyroid, hematopoietic, and pancreatic. They also reported that some studies indicate that leg length is a better predictor of cancer than overall height.

Few studies have found that taller people have lower cancer risk than shorter people. However, stomach cancer is often related to shorter height<sup>[21]</sup>. This cancer is thought to be caused by *H-pylori* bacteria and is most often found in low income or less developed and shorter populations.

The WCRF/AICR<sup>[12]</sup> reported that a number of cancers are rare in parts of the world where traditional dietary patterns are plant-based. These populations are

shorter and leaner than populations in developed countries<sup>[15]</sup>. Other researchers have noted that maximal growth and high insulin levels during adulthood appear to explain the greater risk of some cancers<sup>[22]</sup>. The WCRF<sup>[23]</sup> also reported that increased levels of insulin, leptin, IGF-1 and sex hormones promote cancer. Many of these factors are also correlated with increased height and body size<sup>[24]</sup>.

Egenvall, *et al.*<sup>[25]</sup> found that mortality from cancer was lowest in the smallest dogs. For example, Poodles had a mortality rate of 0.3 *vs.* 4.6 for a Great Dane. Large dogs had rates from 1.7 to 17. Miniature Dachshunds had a mortality rate of 0.3 compared to 0.4 for standard size Dachshunds. As with humans, heavier dogs are generally taller than lighter ones.

The biological mechanisms that may explain the relationship between height and cancer are covered in the Discussion section.

### Cardiovascular disease and height

Both CVD and height are correlated with the Western diet<sup>[13,14]</sup>. The WCRF/AICR<sup>[26]</sup> reported that before industrialization, CVD and other chronic diseases were rare, even among elderly people. Burkitt<sup>[27]</sup> also provided extensive evidence that the increase in CVD during the 20th century was driven by Western dietary practices. In addition, Willett<sup>[28]</sup> reported that CVD was very rare in the US during the early part of the 20th century. In all these examples, people were shorter and lighter than today.

Contemporary findings relating CVD and height present less clear results compared to the previously discussed relation between cancer and height. For example, there are substantial findings showing taller people have lower CVD, but many findings show no relation between height and CVD. In addition, a large number of national and international studies show that shorter people have very low CVD. These findings are summarized next.

### Studies showing taller people have lower CVD

Many studies have found taller people have a lower risk

of CVD incidence or mortality<sup>[3]</sup>. A few sample studies are described next.

A 33-year study of 17,139 male British civil servants 40 to 69 years of age found that taller men had a lower CVD hazard ratio (0.87) than 15 cm shorter males<sup>[29]</sup>. The risk for shorter men was greater for CHD than for stroke. Risk due to lower height was most obvious in high employment grades but was weaker and non significant for middle (0.96) and low grades (1.05). Within high employment grades, shorter men were found to be upwardly mobile and were at higher CVD risk due to less favorable childhood backgrounds.

Bhargava<sup>[30]</sup> evaluated the CHD events of 5,124 individuals involved with the Framingham Offspring Study. Data were collected 8, 12, 16 and 20 years following baseline. Taller people had lower risk of CHD but the finding was non significant.

A study of Glasgow University students found height was inversely correlated with CVD mortality<sup>[31]</sup>. The study involved 8,361 male students who were followed-up for 41.3 years. The hazard ratio for each 10 cm increase in height was 0.78. Stroke was inversely related to height but was not significant. Aortic aneurysm increased with height but was also not significant.

Fogel<sup>[3]</sup> also found Civil War veterans had substantially higher mortality from heart disease compared to WW II veterans. WW II veterans were a few centimeters taller than Civil War veterans. The possible reasons for these findings are covered in the Discussion section.

### Studies showing CVD is independent of height

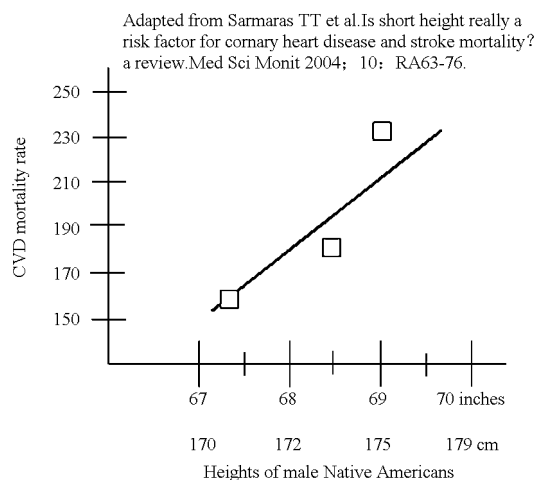
About 20 studies found virtually no relation between height and CVD<sup>[3]</sup>. For example, Song and Sung<sup>[32]</sup> and Song, *et al.*<sup>[33]</sup> evaluated 600,000 South Korean men and women and found almost no relation between CHD mortality and height. Hosegood and Campbell<sup>[34]</sup> also found no trend in CHD vs height among women in a 19-year longitudinal study. Eriksson, *et al.*<sup>[35]</sup> evaluated 3,048 adult men and found no trend pattern between CHD and height. However,

males who were the shortest (136.8 cm) at 11 years of age had a 33% lower CHD risk in adulthood compared to the tallest (140.6 cm) at 11 years old. Silventoinen<sup>[36]</sup> also reported that in the absence of childhood environmental variables, short height is not related to CHD.

### Studies showing shorter height is related to lower CVD

Many researchers have found shorter people have lower CVD within relatively homogeneous groups<sup>[1-3,6,37]</sup>. In addition, a review of CVD studies found shorter people in traditional societies have very little CVD<sup>[6]</sup> compared to taller Western populations. As mentioned, traditional societies are shorter than Western populations. In addition, comparisons among interethnic populations living within the same country found shorter ethnic groups have substantially less CHD. Women in most of the world have lower risk of CVD and are shorter than male counterparts. However, exceptions exist, such as in Australia when leaner Greek men have less CVD than heavier Greek women<sup>[38]</sup>. Comparisons between taller and shorter Native Americans (Figure 1)<sup>[1]</sup>, Japanese, Indians and Pakistanis also found that the shorter individuals had lower CHD<sup>[3]</sup>.

Table 1 shows CHD mortality declined substantially with decreasing height for six ethnic groups in



**Figure 1** Native American major CVD vs. height for age range of 65 ~ 74 years

California<sup>[39]</sup>. Similar findings were based on data from Western Europe. Table 2 shows that taller Western Europeans had 72% higher mortality compared to shorter Western Europeans<sup>[39,40]</sup>. Table 3 shows a similar pattern based on US national data.

A CHD mortality comparison of Scandinavians to Whites and Blacks in California found that their heights and CHD mortality were roughly the same<sup>[40]</sup>. When the shortest groups in California and Europe were compared, California Chinese and Japanese were almost the same height as Spanish and Portuguese males and had almost the same mortality.

A much higher (~ 100% greater) CHD mortality was found for the six tallest countries in Europe vs. the six countries with the lowest mortality in the developed world<sup>[40]</sup>. The countries with the lowest CHD were also relatively short.

**Table 1** Age-adjusted mortality rates for seven taller and seven shorter Western European countries (1995-1998)

Tallest Countries		Shorter Countries	
Country	Mortality Rate	Country	Mortality Rate
Norway (tallest)	370	Switzerland (tallest)	180
Sweden	320	Belgium	225
Netherlands	290	France	125
Denmark	340	Italy	200
Germany	260	Spain	145
Switzerland (shortest)	180	Portugal (shortest)	145
Average Mortality	293	Average Mortality	170

**Table 2** Age-adjusted mortality rate by height for California male ethnic groups (1985 ~ 1990)

Ethnic Group Listed in Order of Decreasing Height	Mortality Rate per 100,000 men/year
Blacks (~ same height as Whites)	316
Whites	302
Latinos	175
Asian Indians	258
Chinese	155
Japanese	146

**Table 3 Age-adjusted coronary heart disease (CHD) mortality vs. height per 100,000 subjects/year (US National Data)**

Ethnic group listed in order of declining height	CHD mortality rate	
	Male	Female
Blacks ( ~ same height as Whites )	408	292
Whites	333	218
Native Americans	220	138
Latino	214	146
Asian	198	121
% increase in mortality for Blacks & Whites vs. Asians	87%	110 %

A comparison of CHD mortality rates for the US, Japan and Okinawa is shown in Table 4 [41]. US males have 6 times the mortality of Okinawans and almost 4 times that of the Japanese. US females have 12 times the CHD mortality of Okinawan females and over 4 times that of Japanese women. Americans are taller than mainland Japanese and mainland Japanese are taller than Okinawans.

Davey Smith, *et al.* [3] found taller higher class people in the UK have lower CVD compared to shorter working class people. However, in developing countries the reverse is true. Peasants and working class people have lower CVD than taller and heavier upper class people [3]. Singh, *et al.* [42] also reported that large studies based on developing populations found CVD was more prevalent among wealthier communities. This higher CVD was attributed to dietary and lifestyle differences between upper and lower classes. The WCRF/AICR report [26] found that CHD and other chronic diseases were rare before urbanization. The report also stated that traditional societies have

**Table 4 Comparison of CHD and relative height for three populations**

Population in order of increasing height	Age-Adjusted CHD mortality	
	( Males )	( Females )
Okinawa	33 [ baseline ( BL ) ]	15 ( BL )
Japan	51 ( ~ 1.5 × BL )	43 ( ~ 3 × BL )
US	193 ( ~ 6 × BL )	177 ( ~ 12 × BL )

very little CVD and other chronic diseases until they adopt Western diets and lifestyles. Both these populations were shorter than today’s developed populations. Examples of populations free of CHD and or stroke included the Solomon and Cook Islands, Congo pygmies, Tarahumara Indians, Kitavans, Papua New Guinea, Yanomamo indians and the Fijii Islands [3]. These populations ranged from about 152 to 163 cm, and generally followed a plant-based diet with relatively low intake of red meat and processed foods. WCRF/AICR [43] and Campbell and Campbell [44] recommend a plant-based diet for reducing the risk of CVD and other chronic disease. ( Studies indicate that centenarians followed plant-based diets most of their lives [45] ). However, when these populations increase their meat, fat and sugar intake, rapid increases in CHD and stroke occur; e. g. Fijians have altered their diets and grown taller and heavier along with substantial increases in CHD. Shaper [3] also reported that racial, ethnic, climatic and geographical facts were not relevant to these findings.

During the 1940 s, South Korea had low rates of CVD. However, with economic development, their CHD mortality has increased by ~ 2,800% [3]. Cancer and diabetes have increased by similar amounts. This trend has occurred in Singapore and India in parallel with economic development, increasing consumption of protein, animal products and energy, and growth in height and weight. Bavdekar [46] reported that young and middle aged people were experiencing an epidemic of CVD in India.

**The roles of birth weight, obesity and rapid growth in promoting adult height, CVD and cancer**

A number of childhood factors are indirectly related to increased CVD and cancer risk in adulthood. These factors include birth weight, catch-up growth, and early overweight or obesity. A brief review follows.

Higher birth weight and rapid growth in weight and height are correlated with increased risk of adult CVD and cancer. Recent studies and reviews on childhood growth indicate that rapid weight gain during in-

fancy is often connected to obesity in childhood and adulthood and greater height in childhood<sup>[47]</sup>. Studies also found that rapid infant weight gain due to greater nutritional intake was tied to higher insulin-like growth factor-1 (IGF-I) which led to taller childhood height and earlier puberty. It was also reported that rapid weight gain from birth to 3 years of age promoted insulin resistance at 8 years of age. Parsons<sup>[48]</sup> reported children that reached a higher percentage of their adult height by 7 years were heavier at birth and had a greater risk of obesity at 33 years of age. Cohen *et al.*<sup>[49]</sup> found that taller Americans have reversed a previous trend and are now increasing in obesity at a faster rate than shorter people. Thus, the ramifications of greater nutrition and growth in childhood promote CVD and cancer in adulthood through increased obesity and insulin resistance. In addition, early maturation correlates with increased body size in terms of height, weight and fatness<sup>[50]</sup>.

Early biological maturation is a product of nutrition, including high intakes of protein, fat and sugar. Several studies have found a correlation between later fatness and the percentage of energy from protein and fat<sup>[47,50,51]</sup>. While this is not universally accepted, studies of lean long-lived non-developed populations show that they have low intakes of animal and total protein, fat and energy. In addition, the global trend in accelerated growth is correlated with the greater prevalence of obesity<sup>[52]</sup>. Studies have also shown that height at 2 years of age is a strong predictor of adult height<sup>[53]</sup>.

The following indicates that birth weight and child growth indirectly drive increases in CVD and cancer due to higher growth hormone levels, adult height and obesity. Note that birth weight and body size track from childhood to adulthood<sup>[47]</sup>. In addition, numerous studies have shown that birth weight correlates with height, weight and BMI in childhood, adolescence and adulthood<sup>[53,54]</sup>.

Ramifications of maternal height and weight

- *Birth weight increases with the mother's height, pre-pregnancy BMI and weight gain during pregnancy*<sup>[47]</sup>.

- *Independent of birth weight, children born to heavier mothers were taller and heavier at 7 years and gained weight more rapidly after 7 years of age*<sup>[55]</sup>.

Ramifications of higher birth weight and neonatal growth

- *Birth weight is strongly and linearly related to later childhood obesity*<sup>[54]</sup>.

- *Myocardial infarction increased progressively (P = 0.05) with increasing birth weight in a study of 855 men tracked from 50 to 80 years of age*<sup>[56]</sup>.

- *Accelerated neonatal growth in weight or height predicts later CVD independent of birth weight*<sup>[57]</sup>.

- *Among normal birth weight (3 to 4 kg) children, rapid growth in weight during the first 5 months following birth results in the greatest proportion of overweight 4.5 year old children. (Parental over weight and smoking also increased risk of childhood excess weight.)*<sup>[58]</sup>.

- *Taller height in infancy after rapid postnatal growth in weight correlates with overweight at 3 years of age*<sup>[59]</sup>.

Ramifications of rapid childhood or adolescent growth in height

- *At ~ 8 years of age, the tallest children were found to have 5 times the risk of adult obesity compared to those who were shorter than average*<sup>[47]</sup>.

- *The tallest children at 11 year had the most risk for adult CHD*<sup>[35]</sup>.

- *Stroke in adulthood was related to above average growth rate in height between 7 and 15 years of age*<sup>[60]</sup>.

- *Rapid growth in height correlates with obesity in young adulthood*<sup>[50]</sup>.

- *Excess weight during early childhood is related to increased adult CHD and cancer mortality. In addition, even small increases in weight for an entire population can have a major impact on public health*<sup>[61]</sup>.

- *Although pulse wave velocity (PWV) has been reported to be lower in taller people, Kumaran<sup>[62]</sup> found that PWV was higher in taller men. Higher PWV is associated with increased risk of heart/circulatory problems.*

*The reason for conflicting studies may be due to comparing leaner, taller people to shorter, stockier cohorts.*

Ramifications of Increased protein and growth hormone levels

- *Increased protein levels promote growth in weight and height and higher levels of growth factors, such as growth hormone (GH) and IGF-1<sup>[47]</sup>.*

- *Growth hormone and total IGF-1 levels are elevated on high energy or high (30%) protein diets and promote growth<sup>[24,47,63]</sup>.*

- *Elevated growth hormone has been found to increase the risk of prostate, breast and colon cancer. IGF-1 is also related to cancer and both IGF-1 and GH are tied to reduced longevity<sup>[64]</sup>.*

A lower calorie and protein diet for children may be considered questionable based on earlier beliefs that heavier and fatter infants are potentially healthier. However, The Okinawan experience has shown that lower than normal energy diets during childhood result in lower CHD and cancer compared to populations on higher-energy diets<sup>[3,41]</sup>. Fontana, *et al.*<sup>[65]</sup> recently found that calorie restricted (CR) diets improved virtually all CVD risk factors, including total cholesterol, triglycerides, high density lipoprotein, blood pressure, glucose, and insulin. Long-term CR provided strong protection against atherosclerosis and increased thickness of the carotid artery intima media.

The preceding factors play an important role in promoting bigger adults with higher risks for cancer and CVD. While taller height is correlated with obesity, CVD and cancer, it should be noted that slow growing children that don't reach their final height until late in adolescence may be relatively tall and lean with a lower risk for these diseases in adulthood<sup>[66]</sup>.

## DISCUSSION

There are certainly contradictory findings in relation to height, cancer and CVD. However, it appears that there is wide acceptance of the fact that height is positively correlated with cancer risk. In contrast, taller height is still generally believed to be related to lower CVD. This

conflict is due to a number of confounders. For example, socioeconomic status is a major factor because people with higher earning power and education have a large advantage in avoiding CVD or having it effectively treated when discovered. In addition, they are generally taller than lower economic groups. Smoking, low physical activity, stress, early catch-up growth for low birth weight, higher unemployment, and higher intakes of fast food or highly processed nutrition are also important socioeconomic-related factors that can affect study results.

A major potential for error in epidemiological studies is comparing short and tall cohorts with the same BMI. When this procedure is followed, the researchers effectively compare taller, leaner people to shorter, stockier people. To avoid this error, tall and short cohorts with the same body types or proportions should be compared. To do this, taller cohorts need to be selected with higher BMIs because the body weight of people with the same body type/proportions increases as the cube of height not as the square as occurs when the BMI is used. (This phenomenon is discussed in detail in Human Body Size and the Laws of Scaling<sup>[67]</sup>.) Thus the Ponderal Index (weight/height<sup>3</sup>) facilitates a closer approximation of similar body builds and is a preferred method for comparison. However, if use of the BMI is preferred, then taller people need to be selected with BMIs that are in proportion to their percent taller height. For example, if a study cohort is 10% taller than the shorter cohort, then its BMI should be 10% greater than the shorter cohort. The need for this procedure should be clear by comparing a 1 cm tall cube to a 2 cm tall cube. The taller cube has 8 times ( $2 \times 2 \times 2$ ) the weight although it is only twice as tall. Thus, if the 1 cm cube weighs 1 gm, the 2 cm cube will weigh 8 gm. Using the formula  $BMI = \text{weight}/\text{height}^2$ , the BMI of the shorter cube is 1 and the taller cube is 2; thus in this example, the BMI is 100% greater for the taller cube. For humans of the same body type, a 10% taller cohort will have a 2.3 point higher BMI (25.3) compared to a shorter cohort with a BMI of

23. Based on the Kenchaiah, *et al.* [68] findings, the taller cohort should have a 25% higher risk of heart failure due to a higher BMI. The Kenchaiah *et al.* findings indicate that for every 1 point increase in BMI, the risk of heart failure increases linearly by 11%.

There is no doubt that abundant food and an improved standard of living have promoted increased height and body weight. This increased body size has also correlated with a substantial increase in life expectancy among developed countries. However, over nutrition has also become a dominant factor in promoting chronic disease. Campbell and Campbell [13] observed that while animal protein produces taller, bigger bodies, it also increases chronic diseases and reduces longevity. Siventoinen [36] also reported that the Western diet promotes both greater height and CVD. Marini, *et al.* [8] observed that lower energy intake, slower growth, and reduced height doesn't necessarily indicate impaired development and may have health advantages in adult life. Cannon [70] noted that: "Bigger and bigger is not better and better. To the contrary; the principle that accelerated growth means health, and that for this reason animal protein is the master nutrient, has proved disastrous." He also stated that energy-dense foods that are high in animal protein and saturated fats increase the risk of heart disease and cancer. Willett [71] also reported that beef and other red meats were associated with CHD.

Many believe that increasing prosperity and growth is always a positive factor in improved health. However, during WW II, Europeans enjoyed a substantial drop in mortality from various illnesses. Height also declined during the war. Granados and Roux [72] studied the effects of the Great Depression on public health. Contrary to what they expected, mortality from most causes declined substantially. Suicides, however, rose. Infant mortality declined and life expectancy rose by 6.3 years over this 4-year depression. This was much larger than the usual increase of about 2 to 2.5 years per decade. The decreased mortality was attributed to less smoking, improved dietary practices, reduced pollution

and decreased driving and industrial injuries. In Norway, it was found that mortality from heart disease declined sharply for all age groups (especially among the youngest) during WW II [73]. This reduced mortality was attributed to food shortages that led to substantially reduced energy and fat intake. Heart disease rose after the war ended with the return to pre-WW II dietary practices. Reduced mortality wasn't limited to Norway; e. g., Europe experienced a large drop in mortality during WW II [74]. These findings conflict with the nutritional scientists goals to promote rapid growth, early sexual growth, and a big and tall population [24,54,75].

Previously, it was reported that Civil War veterans had a substantially higher mortality compared to WW II veterans. A number of factors could have affected these findings. For example, a much higher percentage of Civil War veterans saw combat compared to WW II veterans and as result a much higher number died. In addition, most WW II veterans did not experience physical trauma because they performed non-combat activities, such as logistics, medical functions, ordinance, electronics, intelligence, and administration. During the Civil war, veterans were subjected to much higher infections due to polluted water and food. Modern antibiotics were not available, and minor wounds often got infected requiring limb removal. Thus, malnutrition, increased infections and trauma promoted later CVD.

While many researchers are convinced that greater body height and associated weight reflects optimum health, the findings covered in the Results section provide a mixed picture. A major reason for conflicting findings is the fact that height by itself is only 10% ~ 15% of many factors that are related to cancer and CVD. As mentioned, socioeconomic status, early childhood health, BMI, medical care, smoking and other lifestyle factors as a group have a greater impact on chronic disease and health than height alone. However, the preponderance of evidence supports the "shorter" is healthier thesis. Many of the studies have found that taller people have about 100% greater risk of CHD for a 10 cm height difference. Some of these studies are



based on millions of deaths and involve different ethnic groups<sup>[40]</sup>. It is interesting to note that other studies have found centenarians to be short and light, even when adjusted for shrinkage<sup>[3]</sup>. For example, centenarian studies from Cuba and Italy found that 100-year old males averaged about 153 ~ 156 cm after adjustment for shrinkage with age<sup>[7]</sup>. Okinawan centenarian males were 148 cm<sup>[8]</sup>. When adjusted for shrinkage, they were probably 153 cm in their youth. Without strong hearts and low cancer, it is unlikely that these small centenarians would have reached such an advanced age.

It is interesting to note that a number of short traditional populations have virtually no CHD or stroke<sup>[3,6]</sup>. None of the tallest populations in industrialized nations approaches such a low level of heart disease and cancer. Certainly, differences in diet, BMI and lifestyle between traditional and developed populations can affect CVD and cancer incidence or mortality. However, a number of biological factors indicate that shorter bodies have inherent advantages in terms of lower risk of CVD and cancer. These include lower blood pressure, smaller left ventricles, greater pumping efficiency<sup>[76]</sup>, lower atrial fibrillation<sup>[77]</sup>, and less frequent left ventricular hypertrophy<sup>[3]</sup>. Their hearts have a lower work load because less blood is pumped a shorter distance through the body. In addition, data from animal studies show that smaller animals within the same species have lower cancer and CVD. For example, larger dogs have 6 times the heart disease as small dogs<sup>[25]</sup>. Small dogs have a mortality risk of 0.1 for Dachshunds and, for the tallest dogs, 21 for Great Danes and 27 for Irish Wolfhounds. Miniature Dachshunds and Poodles have rates of 0.3 and 0.4 respectively. A similar pattern was found for cancer.

Additional discussion of biological mechanisms related to CVD and cancer mortality follows.

### Biological mechanisms related to height

Taller people have a few advantages in term of heart health. They have lower resting heart rates and greater

arterial compliance due to their larger diameter blood vessels. In addition, larger blood vessels may take longer to accumulate plaques on their walls which constrict blood flow and increase the risk of heart attacks. However, the hearts of shorter people of similar body proportions and life style also have characteristics that may explain why they can avoid CVD and cancer under the right conditions. These characteristics are identified below<sup>[11,40]</sup>.

- *Trillions of fewer cells with resulting lower cancer risk*
- *Fewer cell replications over a life time leave less opportunity for cell errors that can lead to cancer*
- *Lower blood pressure*
- *Smaller left ventricular mass and lower left ventricular hypertrophy*
- *Higher heart pumping efficiency*
- *Less work for heart per stroke*
- *Fewer free radicals and lower DNA damage*
- *Lower BMI for same body proportions (discussed before)*
- *Greater lung surface area for weight*
- *Lower atrial fibrillation*
- *Lower Oxidative stress produces less damage to heart muscle*

When short and tall people of the same body proportions are compared, a number of biological parameters are related to shorter, lighter bodies. Note that according to the laws of scaling and empirical data, taller people of the same proportions as shorter ones have higher BMIs and there is a linear correlation between BMI and CVD mortality<sup>[24]</sup>. These parameters are shown in Table 5<sup>[24]</sup> and undesirable changes are associated with increased risk of CVD or cancer, or both.

Note that lower SHBG levels are related to increased risk of CVD, cancer and all-cause mortality<sup>[24]</sup>. Lower SHBG levels are correlated with increased height and BMI. McQueen, *et al.*<sup>[78]</sup> found that the ratio of non-fasting Apolipoprotein B (Apo B)/Apolipoprotein A (Apo A1) was a superior method for predicting the risk of acute myocardial infarction in all

ethnic groups, both sexes, and all ages. Apo B is positively correlated with BMI and Apo A is negatively correlated with BMI<sup>[24]</sup>.

Smaller hearts are more efficient because as they grow larger, their pumping efficiency declines in accordance with body mass raised to the 0.71 power<sup>[76]</sup>. Another benefit is lower risk of atrial fibrillation<sup>[77]</sup>. As mentioned, bigger people eat more and have higher total metabolisms which generate more free radicals. Misra, *et al.*<sup>[79]</sup> reported that free radicals are generated within and external to cells and produce a toxic impact on vascular cells and promote CVD, including atherosclerosis. Free radicals can damage nucleic acids (DNA and RNA), lipids, protein and cell membranes in the heart muscle.

**Table 5 Undesirable increases and decreases in biological parameters for taller people of the same body proportions as shorter people**

Undesirable increases	Undesirable decreases
Insulin	High-density lipoprotein
Insulin-like growth factor-1 (IGF-1)	Sex hormone binding globulin
Blood pressure	IGF binding protein-1
Glucose	Adiponectin
Cholesterol, triglycerides, and low-density lipoprotein	Apolipoprotein A Maximum oxygen uptake (ml/kg/min)
Cystatin C	Heart pumping efficiency
Creatinine	
C-reactive protein	
Homocysteine	
Fibrinogen	
Apolipoprotein B	
Lipoprotein (a)	
Left ventricular mass	
Left ventricular hypertrophy	

Larger hearts have been identified as being susceptible to CVD among the general population. Yet, endurance athletes tend to have larger hearts and appear to have fewer heart problems later in life. The explanation for this paradox may lie in the nature of the greater heart size in the two groups. Average people tend to develop larger heart muscles because they are taller and

heavier or because they have higher blood pressure and atherosclerosis. This causes the heart muscle to become thicker and stiffer but the chamber volumes don't increase to provide greater stroke volume and lower heart rates. In contrast, athletic hearts increase in both muscle size and chamber size and have improved blood and oxygen supply. This results in a more efficient heart, with greater stroke volume and lower resting heart rate. In addition, endurance athletes tend to have shorter and leaner bodies compared to sports like football and basketball. After retirement, the largest football players were found to have six times the CHD mortality as the smallest players<sup>[3]</sup>.

## CONCLUSION

The data showing that a plant-based diet and shorter height results in lower cancer is well established. The data showing shorter people have lower CVD is also convincing because of the following reasons:

- *Only short populations are free of CHD or CVD*
- *CVD was rare before industrialization when people were lighter and shorter*
- *Both international and intra-national data support the thesis that shorter people have lower CVD.*
- *Data showing shorter people have less CVD include studies that were based on millions of deaths while conflicting studies are based on much lower population samples.*
- *Much lower CVD was found in shorter, pre-industrialized populations.*
- *A number of biological mechanisms explain why shorter height can be an advantage if not counteracted by overweight, socioeconomic factors, and accelerated growth in childhood.*
- *Confounding factors explain why a number of studies found taller people had lower CVD. These include socioeconomic status, lifestyle and catch-up growth. Another confounder is failure to account for the impact of socioeconomic level on mortality rates for all life phases. Thus, focusing on middle age alone fails to account for factors in childhood and adolescence. Another factor is*

failure to adjust BMI upward for tall cohorts thus providing a handicap to taller people at the expense of shorter people (see Discussion section).

The height findings in this paper identify future problem areas for humanity in terms of health, longevity and medical costs. Failure to adopt a new approach to what constitutes the optimum human configuration presages increased cancer and CVD and potential disaster for civilization. Increasing birth weight, rapid growth in height and weight, earlier sexual maturation and obesity will promote chronic diseases, overstress the medical establishment and raise costs to the point of possible economic failure of developed countries. Large body size and current nutritional practices will also place great stresses on the food and water supply systems. The findings in this paper should provide further support for nutritionists and health experts who favor plant-based and lower energy diets.

Education of the public on the benefits of smaller body size, whether due to shorter height or lower weight, will help defuse the present belief that “bigger” is better and will hopefully avoid the use of genetic engineering to produce taller and heavier youth in future generations. While avoiding the progressive increase of humans will have substantial health benefits, smaller body size of 7 billion people will conserve huge amounts of energy, food, water, raw materials and fiscal expenditures. These topics were not discussed here but are covered elsewhere<sup>[80,81]</sup>.

The future of taller individuals in terms of cancer and CVD is not bleak. With modern developments in medicine and surgery, many tall people with cancer and heart problems will survive to advanced ages. However, regardless of medical advances, low energy, plant-based diets, low BMIs and regular exercise should be the preferred approach to life extension for short, average and tall people.

## REFERENCES

1. Samaras TT, Elrick H, Storms LH. Is short height really a risk factor for coronary heart disease and stroke mortality? a review. *Med Sci Monit*, 2004, 10: RA63 – 76.
2. Mendall MA, Strachan DP, Butland K, Ballam L, Morris J, Sweetnam PM, Elwood PC. C-reactive protein: relation to total mortality, cardiovascular mortality and cardiovascular risk factors in men. *Eur Heart J*, 2000, 21: 1584 – 1590.
3. Samaras TT. Body height and its relation to chronic disease and longevity. In: Samaras, T. (Ed.), *Human Body Size and The Laws of Scaling: Physiological, Performance, Growth, Longevity and Ecological Ramifications*. New York: Nova Science Publishers, 2007, 63 – 112.
4. Holzenberger M, Martin-Crespo RM, Vicent D, Ruiz-Torres A. Decelerated growth and longevity in men. *Arch Gerontol Geriatr*, 1991, 13: 89 – 101.
5. Salaris L, Poulain M, Piras IS, Ghiani ME, Inghes S, Vona G, Calo CM. Height and longevity among males born in Villigrande Strisaili (1866 ~ 1915). *Conference Proceedings XLIII, Riunione Scientifica SIS, Torino, Italy, 14 – 16 June 2006*: 649 – 652.
6. Bruhn JG, Wolf S. Studies reporting “low rates” of ischemic heart disease: a critical review. *AJPH*, 1970, 60: 1477 – 1495.
7. Roth G. S. Entropy theories of aging revisited. *Aging Clin Exp Res*, 1994, 6: 139 – 140.
8. Chan Y-C, Suzuki M, Yamamoto S. Dietary, anthropometric, hematological and biochemical assessment of the nutritional status of centenarians and elderly people in Okinawa, Japan. *Am College Nutr*, 1997, 16: 229 – 235.
9. Ravaglia G, Morini P, Forti P, Maioli F, Boschi F, Bernardi M, et al. Anthropometric characteristics of healthy Italian nonagenarians and centenarians. *Brit J Nutr*, 1997, 77: 9 – 17.
10. Martinez CP, Calzadilla EC, Fonseca MG, Castellano MB, Seco AF. Somatophysiological and nutritional characterization of the Cuban centenarian population of Villa Clara province: reproductive patterns and gender perspectives. *Int J Cuban Studies*, 2009, 2: 1 – 12.
11. Samaras TT. Are recommendations for growth and height correct? a review. *S Afr J Clin Nutr*, 2009, 22 (4): 171 – 176.
12. World Cancer Research Fund/American Institute for Cancer Research. *Food, Nutrition, Physical Activity and the Prevention of Cancer: a Global Perspective*. Washington, DC: AICR, 2007, 192.
13. Campbell, TC, Campbell, TM. *The China Study*. Dallas, Texas: Benbella Books, 2006, 102.
14. Wells JCK. Book Review: *Human Body Size and the Laws of Scaling: Physiological, Performance, Growth, Longevity and Ecological Ramifications*, T. T. Samaras (Ed.). *Eco Hum Biol*, 2008, 6: 489 – 491.
15. World Cancer Research Fund/American Institute for Cancer Research. *Food, Nutrition, Physical Activity and the Prevention of Cancer: a Global Perspective*. Washington, DC: AICR, 2007, 7 – 9, 352 – 353.
16. Drury RAB. The mortality of elderly Ugandan Africans. *Trop Geogr Med*, 1972, 24: 385 – 392.
17. Miller RA, Austad SN. Growth and aging: why do big dogs die young. In: Masoro EJ, Austad SN (Eds). *Handbook of the Biology of Aging*.

- 6th edition. San Diego, Ca: Academic Press, 2006, 512 – 533.
18. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity and the Prevention of Cancer: a Global Perspective. Washington, DC: AICR, 2007, 229.
  19. Zuccolo L, Harris R, Gunnell D, Oliver S, Lane JA, Davis M et al. Height and prostate cancer risk: a large nested case-control (Protect) and meta-analysis. *Cancer Epidemiol Biomarkers Prev*, 2008, 17: 2325 – 36
  20. Engeland A, Tretli S, Bjorge T. Height, body mass index and prostate cancer: a follow-up of 950,000 Norwegian men. *Brit J Cancer*, 2003, 89: 1237 – 1242.
  21. Gunnell D, Okasha M, Davey Smith G, Oliver SE, Sandhu J, Holly JMP. Height, leg length, and cancer risk: a systematic review. *Epidemiol Rev*, 2001, 23: 313 – 342,
  22. Giovannucci E, Rimm EB, Willett WC. Height, predictors of C-peptide and cancer risk in men. *Int J Epidemiol*, 2004, 33: 217 – 225.
  23. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity and the Prevention of Cancer: a Global Perspective. Washington, DC: AICR, 2007, 43.
  24. Samaras TT. BMI and weight: their relation to diabetes, CVD, cancer and all-cause mortality. In: Samaras, T. (Ed.), *Human Body Size and The Laws of Scaling: Physiological, Performance, Growth, Longevity and Ecological Ramifications*. New York: Nova Science Publishers, 2007, 113 – 146.
  25. Egenvall A, Bonnett BN, Hedhammar A, Olson P. Mortality in over 350,000 insured Swedish dogs from 1995 ~ 2000: breed-specific age and survival patterns and relative risk for causes of death. *Acta Vet Scand*, 2005, 46: 121 – 136.
  26. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity and the Prevention of Cancer: a Global Perspective. Washington, DC: AICR, 2007, 5 – 10, 352 – 3.
  27. Burkitt DP. Western diseases and what they encompass. In: Temple NJ, Burkitt DP (Eds). *Western Diseases-Their Prevention and Reversibility*. Humana Press, Totowa, New Jersey: Humana Press, 1994, 15 – 27.
  28. Willett WC. Nutritional epidemiology issues in chronic disease at the turn of the century. *Epidemiol Rev*, 2000, 22: 82 – 86.
  29. Langenberg C, Shipley MJ, Batty GD, Marmot MG. Adult socioeconomic position and the association between height and coronary heart disease mortality: findings from 33 years of follow-up in the Whitehall Study. *Am J Public Health*, 2005, 95: 628 – 632.
  30. Bhargava A. A longitudinal analysis of the risk factors for diabetes and coronary heart disease in the Framingham Offspring Study. *Pop Health Metrics*, 2003, 1; <http://www.pophealthmetrics.com/content/1/1/3>.
  31. McCarron P, Okasha M, McEwin J, Davey Smith, G. Height in young adulthood and risk of death from cardiorespiratory disease: a prospective study of male former students of Glasgow University, Scotland. *Am J Epidemiol*, 2002, 155: 683 – 687.
  32. Song Y-M, Sung J. Adult height and the risk of mortality in South Korean women. *Am J Epidemiol*, 2008, 168: 497 – 505.
  33. Song, Y-M., Davey Smith, G., Sung, J. Adult height and cause – specific mortality: a large prospective study of South Korean men. *Am J Epidemiol*, 2003, 158: 479 – 485.
  34. Hosegood V, Campbell OMR. Body mass index, height, weight, arm circumference and mortality in rural Bangladeshi women: a 19-y longitudinal study. *Am J Clin Nutr*, 2003, 77: 341 – 347.
  35. Eriksson JG, Forsen T, Tuomilehto J, Winter PD, Osmond C and Barker DJP. Catch-up growth in childhood and death from coronary heart disease: longitudinal study. *BMJ*, 1999, 38: 427 – 431.
  36. Silventoinen AJE. The first author replies. 2006; Doi: 10.1093/aje/kwk096.
  37. Osika W, Montgomery SM. Economic disadvantage modifies the association of height with low mood in the US, 2004: the disappointment paradox. *Econ Hum Biol*, 2008, 6: 95 – 107.
  38. Kouris-Blazos, Wahlqvist ML, Trichopoulou A, Polychronopoulos E, Trichopoulos D. Health and nutritional status of elderly Greek migrants to Melbourne, Australia. *Age Ageing*, 1996, 25: 177 – 189.
  39. Samaras TT, Elrick H., Storms LH. Is height related to longevity? *Life Sci*, 2003, 72: 1781 – 1802.
  40. Samaras TT. Should we be concerned over increasing body height and weight? *Exp Gerontol*, 2009, 44: 83 – 92.
  41. Willcox BJ, Willcox DC, Todoriki H, Fujiyoshi A, Yano K, He Q, et al. Caloric restriction, the traditional Okinawan diet, and healthy aging. *Ann NY Aca Sci*, 2007, 1114: 434 – 455.
  42. Singh RB, Sharma JP, Rastogi V, Niaz MA, Ghosh S, Beegom, Janus ED. Social class and coronary disease in a rural population of north India. *Eur Heart J*, 1997, 18: 588 – 595.
  43. World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity and the Prevention of Cancer: a Global Perspective. Washington, DC: AICR, 2007, 380 – 383.
  44. Campbell, TC, Campbell, TM. *The China Study*. Dallas, Texas: Benbella Books, 2006, 348 – 350.
  45. Buettner D. *The Blue Zones*. Washington, DC: National Geographic Society, 2009.
  46. Bavdekar A, Yjnik CS, Falli CHD, Bapat S, Pandit AN, Deshpande V, et al. Insulin resistance syndrome in 8-year old Indian children. *Diabetes*, 1999, 48: 2422 – 2429.
  47. Samaras TT. The obesity epidemic, birthweight, rapid growth and superior nutrition. In: Samaras, T. (Ed.), *Human Body Size and The Laws of Scaling: Physiological, Performance, Growth, Longevity and Ecological Ramifications*. New York: Nova Science Publishers, 2007, 147 – 190.
  48. Parsons TJ, Powr C, Manor O. Fetal and early life growth and body mass index from birth to early adulthood in 1958 British cohort: longitudinal study. *BMJ*, 2001, 323: 1331 – 1335.
  49. Cohen DA, Strum R. Body mass index is increasing faster among tal-

- ler persons. *Am J Clin Nutr*, 2008, 87: 445 – 448.
50. Parsons TJ, Power C, Logan S, Summerbell CD. Childhood predictors of adult obesity: a systematic review. *Int J Obesity*, 1999, 23 Suppl 8: S1 – S107.
51. Metges C. Does dietary protein in early life affect the development of adiposity in mammals? *J Nutr*, 2001, 131: 2062 – 2066.
52. Heude B, Lafay L, Borys JM, Thibault N, Romon M, Ducimetiere P, Charles MA. Time trend in height, weight, and obesity prevalence in school children from Northern France, 1992 – 2000. *Diab Metab*, 2003, 29: 235 – 240.
53. Samaras TT. Why the study of human size is important. In Samaras, T. (Ed.), *Human Body Size and The Laws of Scaling: Physiological, Performance, Growth, Longevity and Ecological Ramifications*. New York: Nova Science Publishers, 2007, 1 – 16.
54. Mardones F, Villarroel L, Karzulovic L, Barja S, Arnaiz P, Taibo M, et al. Association of perinatal factors and obesity in 6- to 8-year old Chilean children. *Int J Epidemiol*, 2008, 37: 902 – 910.
55. Forsen T, Eriksson J, Tuomilehto J, Reunanen A, Osmond C, Barker D. The fetal and childhood growth of persons who develop type 2 diabetes. *Ann Intern Med*, 2000, 133: 176 – 182.
56. Eriksson M, Tibblin G, Cnattingius S. Low birthweight and ischaemic heart disease. *The Lancet*, 1994, 343: 731 – 732.
57. Singhal A, Cle TJ, Fewtrell M, Deanfield J, Lucas A. Is slower early growth beneficial for long-term cardiovascular health? *Circulation*, 2004, 109: 1106 – 1113.
58. Dubois L, Girard M. Early determinants of overweight at 4.5 years in a population-based longitudinal study. *Int J Obesity*, 2006, 30: 610 – 617.
59. Akaboshi I, Haraguchi Y, Mizumoto Y, Kitano A, Kan H. Taller stature after postnatal rapid weight gain in early infancy predicts overweight status at age 3. *Acta Paediatr* 2009; 97: 1460 – 1464.
60. Eriksson JG, Forsen T, Tuomilehto M, Osmond C, Barker DJP. Early growth, adult income, and risk of stroke. *Stroke*, 2000, 31: 869 – 874.
61. Stein CJ, Colditz GA. The epidemic of obesity. *J Clin Endocrinol Metab*, 2004, 89: 2522 – 2525.
62. Kumaran K, Fall CHD, Martyn CN, Vijayakumar M, Stein C, Shier R. Blood pressure, arterial compliance, and left ventricular mass: no relation to small size at birth in south Indian adults. *Heart*, 2000, 63: 272 – 277.
63. Nuttall FQ, Gannon MC, Saeed A, Jordan K, Hoove H. The metabolic response of subjects with type 2 diabetes to a high protein, weight-maintenance diet. *J Clin Endocrinol Metab*, 2003, 88: 3577 – 3583.
64. Bartke A. Long-lived mutant, gene knockout and transgenic mice. In: Samaras, TT (Ed.), *Human Body Size and The Laws of Scaling: Physiological, Performance, Growth, Longevity and Ecological Ramifications*. New York: Nova Science Publishers, 2007, 191 – 212.
65. Fontana L, Meyer TE, Klein S, Holloszy JO. Long-term calorie restriction is highly effective in reducing the risk for atherosclerosis in humans. *PNAS*, 2004, 101: 6659 – 6663.
66. Rollo CD. In: Samaras, T. (Ed.), *Human Body Size and The Laws of Scaling: Physiological, Performance, Growth, Longevity and Ecological Ramifications*. New York: Nova Science Publishers, 2007, 235 – 260.
67. Samaras TT. In: Samaras, T. (Ed.), *Human Body Size and The Laws of Scaling: Physiological, Performance, Growth, Longevity and Ecological Ramifications*. New York: Nova Science Publishers, 2007, 17 – 32.
68. Kenchaiah S, Sesso HD, Gaziano JM. Body-mass index and vigorous physical activity and the risk of heart failure among men. *Circulation*, 2009, 6: 44 – 52.
69. Marini E, Maldonado-Contreras AL, Cabras S, Hidalgo G, Buffa R, et al. *Helicobacter Pylori* and intestinal parasites are not detrimental to the nutritional status of Amerindians. *Am J Trop Med Hyg*, 2007, 76: 534 – 540.
70. Cannon G. *The Fate of Nations: Food and Nutrition Policy in the New World*. Cornwall, UK: Caroline Walker Trust, 2003, 33.
71. Willett W. Lessons from dietary studies in Adventists and questions for the future. *Am J Clin Nutr*, 2003, 78 (suppl): 539 – 549S.
72. Granado JAT, Roux AVD. Life and death during the Great Depression. *Proc Natl Acad Sci USA*, 2009, 106: 17290 – 17295.
73. Strom A, Jensen RA. *The Lancet*, 1951, 1: 126 – 129.
74. Diehl H in Temple NJ, Burkitt DP (Eds). *Western Diseases-Their Prevention and Reversibility*. Humana Press, Totowa, New Jersey: Humana Press, 1994, 237 – 316.
75. Cannon G. *The Fate of Nations: Food and Nutrition Policy in the New World*. Cornwall, UK: Caroline Walker Trust, 2003, 11 – 14, 29 – 39, 54.
76. de Simone G, Devereux RB, Daniels SR, Mureddu GF, Roman MJ, Kimball TR, et al. Stroke volume and cardiac output in normotensive children and adults. *Circulation*, 1997, 95: 1837 – 1843.
77. Hanna IR, Heeke B, Bush H, Brosius L, King-Hageman D, Beshai JF, Langeberg JJ. The relationship between stature and the prevalence of atrial fibrillation in patients with left ventricular dysfunction. *J Am Coll Cardiol*, 2006, 47: 1683 – 168.
78. McQueen MJ, Hawken S, Wang X, Ounpuu S, Sniderman A, Probstfield J et al. Lipids, lipoproteins, and apolipoproteins as risk markers of myocardial infarction in 52 countries (the Interheart Study): a case-control study. *The Lancet*, 2008, 372: 224 – 233.
79. Misra MK, Sarwat M, Bhakuni P, Tuteja R, Tuteja N. Oxidative stress and ischemic myocardial syndromes. *Med Sci Monit*, 2009, 15: RA209 – 219.
80. Samaras, TT. (Ed.), *Human Body Size and The Laws of Scaling: Physiological, Performance, Growth, Longevity and Ecological Ramifications*. New York: Nova Science Publishers, 2007, 147 – 190.
81. Marson SM. Book Review: *How big should we be? A Herculean task accomplished*. *Pub Health Nutri*, 2009, 12: 1299 – 1300.