Are 20th-century recommendations for growth and height correct? A review

Article *in* South African Journal of Clinical Nutrition · January 2009

DOI: 10.1080/16070658.2009.11734245

Thomas Samaras
Reventropy Associates

Are 20th-century recommendations for growth and height correct? A review

Samaras TT
Reventropy Associates, San Diego, Ca, USA
Correspondence to: Thomas Samaras, e-mail: samarastt@aol.com
Keywords: longevity; nutrition; growth; body height; chronic disease

Abstract

Findings supporting the counter-intuitive thesis that smaller body size promotes greater health and longevity are reviewed. In addition, the risks of promoting further growth through genetic manipulation are explored in relation to chronic disease and longevity. Supporting examples from animal research are also covered. The biological mechanisms that relate to height and longevity conclude the review.

S Afr J Clin Nutr 2009;22(4):171-176

Introduction

The belief that increased height is a healthful by-product of our Western lifestyle and nutrition is widely held.[1–4] This belief is based on the large increase in life expectancy over the past 150 years. However, during the 20th century the increase in longevity has paralleled an explosive growth in chronic diseases.[2,5,6] The fact that people are living longer is the most obvious explanation for this trend. However, India, as well as other countries, has experienced an epidemic of coronary heart disease (CHD) and type 2 diabetes among the young and middle-aged segments of the population.[7] In addition, before the 1970s, over 65% of the world's elderly people lived in developing countries[8] when cardiovascular disease (CVD) and diabetes were rare. The elderly in these countries died from essentially the same causes as young people,[9] for example malnutrition, trauma, infections and lack of medical care.

Over the past few decades, more than 100 researchers (Samaras, unpublished) have questioned whether higher birth weight, rapid growth, early sexual maturation and increased height pose health risks.[1, 2,10–12] Holzenberger[13] also observed that improvements in living conditions over the past 150 years have masked the negative effects of greater growth and height.

Body height and weight have a harmful impact on many physiological parameters.[14] In addition, a world population of taller, leaner humans weighing 25 kg more than previous generations requires a huge increase in energy, resources, water and food. As a result, the environment is negatively impacted, and pollution and global warming threaten human survival. All these issues cannot be explored here and are discussed elsewhere.[14] The focus of this paper is to review the impact of increasing height on chronic diseases and longevity.

Increasing height and weight and the future impact of genetic engineering

The world trend towards increasing height is viewed positively by most growth experts.[3,4] The reasons for this trend have been increased energy and protein intake and reduced childhood illness. However, social bias and increasing life expectancy have propagated the belief that greater height is desirable without a systematic evaluation of the ramifications of increasing body size (height and weight are strongly correlated).

Should we promote a population of 185 cm males and 173 cm women as found in the Netherlands? Or would some other height be better? Based on current public bias favouring tallness, it would appear that if young males averaged 185 cm in 2020, parents would probably favour having their children 2 to 4 cm taller than average, if they had a choice. Advances in genetic engineering offer a mechanism for increasing the average height of future generations through genetic manipulation. Hopefully, growth experts and social scientists can recognise the hazards of this scenario and prevent an undesirable increase in human body size. Some may argue that changes in nutrition to limit growth trends would interfere with nature. However, contemporary eating habits are far from ideal and involve processed and domestic animal foods that were not available during most of human history.

The relation of higher birth weight and rapid growth to obesity and future illness

Higher birth weight has generally been viewed as beneficial to childhood and adult health[3,15] for example, many studies have observed that lighter babies who grow rapidly have higher perinatal mortality and increased adult illness.[15,16] Wells[17] noted that small infants and children exposed to richer nutrition after a more restricted fetal diet also experience harmful health consequences in adulthood. However, a number of recent studies indicate that higher birth weight is not necessary for low perinatal mortality.[15,18,19] In Table I the all-cause and CHD adult mortality of people born during the Dutch famine is shown.[20] While poor fetal nutrition is likely to have a negative health impact, exposure to famine during late gestation had the lowest birth weight and the lowest all-cause mortality.

Rapid growth is also viewed as reflecting good nutrition, and slow growth is viewed as a failure to thrive.[2,3,4] Unfortunately, these views have a number of problems associated with them; for example, DeHeeger,[11] Heude[21] and Freedman[22] have observed that the trend in rapid growth and increasing height has paralleled the increase in the obesity epidemic and has promoted greater chronic disease in adulthood. Mangel and Munch[23] also reported that catch-up growth is related to increased mortality and reduced longevity. These negative trends have been counteracted by improved sanitation and medical care, which have avoided sharp reductions in life expectancy although the elderly suffer from many chronic ailments.

Table I: Decreasing birth weight and mortality from the Dutch famine study[a]

Birth weight (g)	Famine status	All-cause mortality[b]	CHD mortality[b]
3 452 (highest)	Early exposure	6.5%	1.1%
3 418	Conceived after famine	5.9%	1.2%
3 370	Born before famine	9.1%	1.9%
3 215	Mid exposure	6.0%	1.2%
3 135 (lowest)	Late exposure	4.4%	1.2%

[a] One thousand nine hundred and ninety-one people tracked to age 57. Future follow-up findings may change results
[b] Percentage of deaths for each cohort

Studies in recent years have also shown that higher birth weight promotes cancer, type 1 diabetes and obesity.[24–27] In addition, Sorensen[28] found that it did not take much of an increase in birth weight to go from normal to abnormal adult weight as shown in Table II.

Table II: Average birth weight in relation to male adult weight status

Average birth weight (g)	Male adult weight category
3 445	normal
3 545	overweight
3 571	obese

Substantial findings indicate that a rapid increase in height or weight in childhood promotes adult obesity.[24,26,29] Freedman[22] also found that tall children were five times more likely to become obese adults compared to shorter than average children. Recently, Jacobsen[30] found that women who reached puberty before 11 years of age had a 20% higher all-cause mortality compared to those who reached puberty at 17. Soltesz et al [27] also found that type 1 diabetes was related to rapid growth. They reported that increases in birth weight and childhood height and weight correlated with increasing risk of type 1 diabetes.

Cohen and Strum[31] observed that in the recent past, shorter men and women had substantially greater BMIs when compared with taller men and women. However, over the past 40 years, this situation appears to have changed and today taller people are experiencing rising BMIs at a faster rate than seen in shorter people. In the past, taller people were often leaner because lifestyle and standardised food portions provided a relatively lower amount of energy for their body size; for example, a glass of milk or a sandwich provides the same amount of energy to both tall and short people. In addition, taller people expended more energy in moving their larger bodies and were more active years ago.

How body size relates to chronic disease

Many epidemiological studies find that taller people have less CVD.[32] However, a number of studies also find little difference or a positive correlation with height.[29,32,33] Since early in the 20th century, many studies have found that people in southern Europe and developing countries had very little or no CHD and that these people were substantially shorter and leaner than most Western populations (see

Table III: Short (≤ 165 cm) nondeveloped populations with little or no CVD[a]

Population	Population
Congo Pygmies	Okinawa
Papua New Guinea	Vilcabambia (Ecuador)
South African rural blacks	Cook Islands
Tarahumara (Mexico)	Crete (males < 170 cm in1960s)
Fiji (over the past 50 years, CVD has increased substantially)	Vietnam Thailand
Solomon Islands	Kalahari Bushmen
Yanomamo (South America)	Kitava (Trobriand Islands)

[a] Many of these populations are adopting Western nutrition and lifestyle patterns and CVD has increased in recent years

Table IV: Short vs tall male CHD mortality in developed populations[a,b]

Short population	CHD mortality	Tall population	CHD mortality
Japan	35.7	The Netherlands	97.6
Hong Kong	47.6	Germany	125.8
France	49.1	Norway	128.2
Portugal	65.1	Denmark	129.6
Spain	65.5	Sweden	134.4
Italy	74.0	Finland	182.1
Average mortality	**56.2/100K/year**		**132.9/100K/year**

[a] Short average ~ 170 cm and tall ~ 179 cm
[b] Mortality deaths/K = 100 000/year for 1995–1998

Table V: CHD mortality by height in California based on 123 164 male deaths

Ethnic group in order of decreasing height	Male age-standardised mortality rate (age range: 25–84 years)[c]
African American[a]	316
White[a]	302
Latino	175
Asian Indian	258
Chinese[b]	155
Japanese[b]	146

[a] African-American and white males about the same height
[b] Chinese and Japanese males about the same height
[c] Female mortality similar to that of males; stroke declined at a slower rate

Table III).[34] Most of the males averaged less than 165 cm. In addition, examination of 50 developed countries found that in the six countries with the lowest CHD mortality people were relatively short (see Table IV).[34] Table V also shows confirmatory data from California based on 262 333 male and female deaths.

The proponents of the benefits of modern growth trends argue that the developed world cannot be compared to the developing world because different factors are involved and decreased height in developing nations is not the cause of their low CVD. Certainly, lifestyle, diet, stress levels, BMI, medical care and economics all play a role. However, a number of studies indicate that the 20th-century explosive growth of CVD and other Western diseases was mainly due to changes in nutrition,[5,6,12] which led to greater body size.[12] These findings contradict the proposition that abundant protein and energy, along with greater height, promote superior health.

A recent report by the World Cancer Research Fund [6] notes that many chronic diseases common today were rare, even in old age, before the Industrial Revolution. The report also notes that traditional societies that follow a plant-based diet are also relatively free of CVD, diabetes and certain cancers.[6,35,36] Yet, these populations are short and lean.

Ethnicity does not appear to play a role in the low CVD of developing populations.[37] Activity levels can be a factor, but some populations indulge in heavy physical activity (for example Papua New Guinea) while others (Kitava) are not much different in activity terms from Western nations.[38] It should also be borne in mind that dietary practices in developing populations are substantially different in that they focus on plant-based diets and are relatively low in energy intake. These dietary practices generally produce shorter height, low weight and low CVD. Of course, the developing world suffers from much poorer medical care and malnutrition, which affects short- and long-term health; for example, most deaths in rural parts of the developing world are due to the same causes as in the young and elderly.[9]

The question arises, is it diet or body size that sharply reduces CVD in developing populations? Certainly, higher weight or BMI is related to increased CVD for humans[6]; for example, a study of football players found that the largest players had six times the death rate from heart disease as the smallest players.[39] Furthermore, animal studies also find that both energy restriction and smaller body size independently promote longevity,[40] which cannot be readily attained unless CVD is also low. Rollo[41] also reports that smaller body size is more often related to longevity than is energy restriction.

How height affects longevity

Few epidemiological studies that found that tall people live longer actually measured longevity; for example, the average age at death for the entire cohort vs. height was rarely examined. However, Waaler's[42] study tracked mortality over a wide range of ages. He found that male mortality declined with increasing height for ages below 70 years. However, men over 183 cm experienced a substantial increase in mortality from 70 to 85 years and a slight decrease occurred for those aged 86–94 years. Since most people die between the ages of 70 and 85, this can be a more important mortality period than death rates at younger or older ages.

In contrast, studies finding that shorter people live longer than tall ones have involved longevity studies; for example, in these studies deceased cohorts were examined to determine the average ages at death based on height. Over 20 studies have been found that examine longevity or all-cause mortality rates for elderly populations. A summary of 12 studies is shown in Table VI.[34] Many of these studies have shown a 0.5-year drop in average age at death for each centimetre (cm) increase in height.

Table VI: Populations illustrating reduction in longevity with increasing height

Population	Slope (decrease in longevity per centimetre of height [years/cm])
Baseball players (males)	–0.35
Celebrities, study 1 (males)	–0.43
Celebrities, study 2 (males)	–0.41
Elderly Swedes (males and females)	–0.52
Former Finnish athletes (males)	–0.49
Former football players (males)	–0.81
Former Harvard athletes (males)	–0.70
French (19th-century males and females)	–0.51
Males vs females (average based on 21 European countries)	–0.47
Ohio general population (males and females)	–0.47
US males vs females	–0.52
US veterans (males)	–0.47
Average slope	**–0.51years/cm**

In addition, the greater life expectancy of women applies to almost all populations in the world. American males are 9% taller and have a 9% lower life expectancy.[34] When American men and women were compared, male life expectancy dropped by 0.5 years/cm. This pattern was also reported for five different groupings of men and women in 2003.[43] Miller[44] found that males of the same height as females lived somewhat longer. Another study found that dwarf male mice lived 50% longer than their normal size female siblings.[45]

The largest study to date tracked the survival of 1.3 million Spanish males from youth to end of life over a 70-year tracking period.[13] The researchers found a correlation coefficient (r) of $r = -0.6$ between height and longevity ($p < 0.001$). Heights were based on military recruitment records and thus were not confounded by height shrinkage. The researchers also noted that other possible confounders did not affect the results.

More recently, a Sardinian study found that shorter people lived longer.[46] This study involved about 400 males and their heights were based on military records. A subsequent study confirmed the earlier findings (personal correspondence with Salaris, 2008). In addition, Poulain et al[47] showed (see Table VII) that the prevalence of European centenarians increased with decreasing height.

Table VII: Comparative prevalence of centenarians in Europe

Country/region in order of increasing height	Number of centenarians per 100 000 population
Nuoro Province	17.9
Sardinia	16.6
Italy	14.1
Belgium	10.5
Sweden	12.6
Denmark	10.4

A recent evaluation of life expectancy by population showed that the top populations where life expectancy is concerned were relatively short in comparison to the tallest populations in Western Europe (see Table VIII).[34] The shorter populations had an average rank of 3 vs 29 for the tallest populations.

Table VIII: Life expectancy ranking for developed populations (both sexes)

Rank	Shorter states	Life expectancy at birth (years)
1	Andorra (between France and Spain)	83.52
2	Macao (64 km west of Hong Kong)	82.27
3	Japan	82.02
4	San Marino (central Italy)	81.80
4	Singapore	81.80
6	Hong Kong	81.68
	Tallest states (Western Europe)	
7	Sweden	80.63
20	Norway	79.67
28	The Netherlands	79.11
32	Germany	78.95
38	Finland	78.66
47	Denmark	77.96

Centenarians are generally small.[34,48] The highest percentages of centenarians are found in Okinawa and Sardinia; both populations are short people by European standards. The Okinawan centenarian males average 148 cm (152 cm adjusted for shrinkage) and the Sardinians 160 cm. George Francis recently died at 114 years and weighed ~ 45 kg even in his youth.[49] He was exempt from military service during World War I because he was too short. However, there are exceptions. Gavrilov and Gavrilov[50] found that average-height World War I men reached 100 years of age more often than short men. Tall men did better than short but not as well as average-height men. Perhaps the short men, born between 1880 and 1900, were the product of poverty and illness common to urban areas. In contrast, rural men had better nutrition, a healthier environment, grew taller and lived longer.

Since height is related to nutrition, especially protein intake and total energy, it is interesting to note that a World Cancer Research Fund report published found that restricting energy intake postponed the onset of many age-related chronic diseases.[51] It is not surprising that this report urges the public to consume minimal animal protein and lower the intake of high-energy foods. Conflicts with traditional epidemiological findings are probably due to a number of confounders, summarised in Table IX.[4,32,52–55] For example, the impact of socioeconomic status is illustrated by Osika's[56] study that found that higher income tall people had a lower risk of heart disease than higher income short people. However, when the researchers looked at low-income people, they found that tall people had a 40% higher risk than short people. Another example involves the United States where taller upper class people had a higher risk of CVD compared to shorter working class people in the early part of the 20th century, but then the pattern reversed after the 1970s.[57] Since relative height between the classes did not reverse, CVD increased due to a combination of factors, for example greater weight, excess nutrition and lifestyle.

Table IX: Confounders that may explain conflicting height vs CVD findings

- Low-birth weight children experience catch-up growth, remain shorter but also have higher risk of chronic diseases.[4,15,52,53]
- Childhood illness can cause reduced growth and higher mortality in adulthood. However, this decreased height is not the cause of future illness.
- A comparison is made between tall and short people who are not of the same proportions; for example, tall, lean people are compared to short, stocky people.
- Shorter people have more abdominal obesity and the latter increases their CVD risk.[53]
- Taller people are from higher socioeconomic classes[4,10] and have a lower mortality risk due to improved living conditions and higher education.
- Failure to adjust for all three life phases. Early, middle and elderly life phases all play a role in mortality rates. Failure to adjust for these differences can lead to study errors.[54]
- Adjustment for various risk factors is a crude and inexact process that can lead to erroneous results.[55] This adjustment generally reduces the mortality of shorter people.
- If five-year cohorts are used, shorter people in a given age cohort tend to fall at the higher end of the age range while taller people fall closer to the younger age of the cohort. As a result an error of up to 40% can be made in mortality rates due to this age difference; for example, for the age range of 60 to 64 years, tall people may fall near 60 years and short people near 64 years.

Supporting data from animals and trees

Animal studies confirm previously described human longevity findings related to height and body size.[41,58,59] For example, giant mice created through genetic manipulations also have much shorter lives.[41] Bartke[58] also found that smaller mice and rats live substantially longer than larger versions of their species. Several studies have found that smaller and shorter dog breeds live longer than larger ones.[59,60] Smaller horses also live longer than larger ones.[59]

Great Basin bristlecone pines are the world's longest living trees and average only 15 to 30 feet. These trees also grow very slowly. In addition, Eastern white cedar trees growing along the cliffs between

Canada and the US grow extremely slowly and have final heights that are much lower than those of cedars growing on flat terrain. These stunted trees also live much longer than normal-growing white cedars.[61]

Biological changes due to increasing height

There are a number of biological mechanisms that support the ‘smaller lives longer’ thesis. An important one involves cellular proliferation. A larger body requires more cell duplications from birth to final adult height. In addition, maintenance of these cells during a lifetime uses up even more of the body’s ability to create healthy cells in place of defective or cancerous ones. Thus, growth and maintenance processes reduce the potential cell doublings in old age for taller people. Empirical findings indicate that elderly taller people have a lower potential for cell duplications.[62] Women also have more potential cell duplications compared to taller men, although they start life with the same potential as indicated by telomere lengths.

Several other mechanisms exist to support increased longevity of shorter and smaller bodies. These include reduced DNA damage, smaller left ventricular mass, lower exposure to viruses and parasites, and relatively larger organs (see Table X).[30,34,63–71] Energy restriction has been found to be the most effective approach for extending longevity.[58,59,72,73] Thus, a diet that slows growth and avoids excessive body size is more likely to improve longevity, as was found in Hawaii and Okinawa.[73]

Conclusions

Modern developments, such as improvements in working environments and medical care, may have masked the negative effects of excessive nutrition and increased height and weight.[13,59] However, human and animal findings indicate that we can improve human health by emphasising plant-based diets,[6,12] reducing energy intake, slowing growth and attaining shorter height and lower weight.[10,34,72]

The findings in this paper are based on a wide variety of investigations, including prospective, cross-sectional, descriptive and ecological studies. The populations in these studies vary from a few hundred to millions of people, and a variety of ethnic groups and geographical areas are included. The timeline goes back to the 19th century, and historical data[5,6,34] show that Western diseases, the primary cause of later-life mortality, correlate with changes in nutrition, lifestyle and increasing body size.

While substantial evidence supports the health and longevity advantage of low energy, protein and meat intake[6,12,73] and smaller height and body weight, the role of body height per se represents a relatively small slice of the longevity pie (r^2 = 0.13 or 13%). Living standards, medical care, stress, smoking, diet, BMI and satisfaction with one’s life have a greater total impact. That is why some centenarians are not short and many tall people reach advanced ages; for example, John Kenneth Galbraith (203 cm) lived to 98 years of age. However, promoting widespread increases in height and weight through overnutrition will have a major impact on future health costs that could adversely affect most economies of the world.

Table X: Biological mechanisms related to longevity and body size

1. A shorter person has a greater potential cell duplication capability available to replace cells lost during aging.[62]
2. A short person of the same proportions as a tall one may have 40 trillion fewer cells that are exposed to potential carcinogens and is thus less likely to develop cancer.[34]
3. Giovannelli[64] found that taller people have disproportionately more DNA damage than shorter people. A possible explanation is that the higher metabolic rates/energy needs of taller, bigger people produce more free radicals that damage DNA and cell structure, cell components, proteins and lipids.
4. Short people of the same proportions as tall ones have lower BMIs, which reduces insulin, glucose, IGF-1, CRP, homocysteine, and lipid levels, which is less damaging to mortality.[62,63]
5. Most organs are relatively larger for shorter people. Thus they have a greater functional capacity at later ages. The heart and lungs are proportional to body mass in taller people. However, in the case of the heart, this correlates with higher mortality.[65,66]
6. Although their hearts are smaller, shorter, leaner people are at less risk of heart failure because pumping efficiency is greater[65,66]; for example, Baron[49] found that retired big football players had six times the CVD mortality of the smallest. In addition, atrial fibrillation decreases with decreased height.[67]
7. Among nonagenarians, reduced insulin and IGF-1 levels are correlated with shorter height and superior old age survival.[62]
8. Taller people of similar body types as short people have higher blood pressure (BP), and BP is a function of height, weight and BMI.[32] (However, tall, thin people may have lower BP compared to short, stocky people.)
9. Reduction in solid and liquid intake of shorter people decreases exposure to bacteria, viruses, parasites and toxins.[68]
10. Shorter people tend to have slightly lower body temperatures,[69] and this is associated with decreased generation of free radicals. More studies are needed to compare the body temperatures of tall and short people with the same body proportions.
11. The second law of thermodynamics provides a fundamental ageing mechanism. This law says that the disorder of a spontaneous system is a function of its mass and energy. More mass and energy mean more possible states a system can be in. Thus, since larger people have more mass and energy, their bodies have a greater potential for increased disorder of their cells, tissues and organs. Unfortunately, repair is imperfect and disorder accumulates, resulting in aging. Environment, exercise, nutrition and smoking can retard or accelerate the aging process.[70,71]

References

1. Walker ARP, Walker BF, Glatthaar I, Vorster HH. Maximal genetic potential for adult stature: is this aim desirable? Nutri Rev 1994;52:208–15.
2. Cannon G. The fate of nations. Cornwall, UK: Caroline Walker Trust; 2003.
3. Cannon G. Out of the box. Public Health Nutrition 2007;10:1210–3.
4. Wells, JCK. Human body size and the laws of scaling: physiological, performance, growth, longevity and ecological ramifications. In: Samaras TT (ed). New York: Nova Science Publications Inc.; 2007 (Book review). Econ Hum Biol 2008;6:489–91.

5. Temple, NJ, Burkitt, D. Western diseases – their dietary prevention and reversibility. Totowa, NJ: Humana Press; 1994.
6. 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. Bavdekar A, Chittaranjan S, Yajnik CS, et al. Insulin resistance syndrome in 8-year-old Indian children. Diabetes 1999;48:2422–9.
8. Brown, LR, Renner M, Flavin C. Vital signs 1997 (Worldwatch Institute). New York: W. W. Norton & Co; 1997.
9. Drury RAB. The mortality of elderly Ugandan Africans. Trop Geogr Med 1972;24:385–92.
10. Marini E, Maldonado-Contreras AL, Cabras S, et al. Helicobacter pylori and intestinal parasites are not detrimental to the nutritional status of Amerindians. Am J Trop Med Hyg 2007;76:534–40.
11. DeHeeger M, Rolland-Cachera MF. Longitudinal study of anthropometric measurements in Parisian children aged ten months to 18 years. Arch Pediatr 2004;11:1139–44 (in French).
12. Campbell TC, Campbell TM. The China study. Dallas, TX: Benbella Books; 2006.
13. Holzenberger M, Martin-Crespo RM, Vicent D, Ruiz-Torres A. 1991. Decelerated growth and longevity in men. Arch Gerontol Geriatr 1991;13:89–101.
14. Samaras TT, ed. Human body size and the laws of scaling: physiological, performance, growth, longevity and ecological ramifications. New York: Nova Science Publishers; 2007.
15. Samaras TT. The obesity epidemic, birth weight, rapid growth and superior nutrition. 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.
16. Barker DJ, Osmond C, Forsen TJ, Kjantie E, Eriksson JG. Trajectories of growth among children who have coronary heart events as adults. N Engl J Med 2005;27:1802–9.
17. Wells JC. The thrifty phenotype as an adaptive maternal effect. Biol Rev Camb Philos Soc 2007;82:143–72.
18. Vangen S, Stoltenberg C, Skjaerven R, Magnus P, Harris JR, Stray-Pedersen B. The heavier the better? Birth weight and perinatal mortality in different ethnic groups. Int J Epidemiol 2002;31:654–60.
19. Ulizzi L. Absence of selection on birth weight in modern Italy. Hum Biol 2002;74:525–32.
20. Painter RC, Roseboom TJ, Bossuyt PMM, Osmond C, Barker DJP, Bleker OP. Adult mortality at age 57 after prenatal exposure to the Dutch famine. Euro J Epidemiol 2005;20:673–6.
21. Heude B, Lafay L, Borys JM, et al. Time trend in height, weight, and obesity prevalence in school children from Northern France, 1992–2000. Diabetes Metabol 2003;29:235–40.
22. Freedman DS, Khan LK, Mei Z, Dietz WH, Srinivasan SR, Berenson GS. Relation of childhood height to obesity among adults: the Bogalusa heart study. Pediatrics 2002; 109. Available from http://www.pediatrics.org/cgi/content/full/109/2/e23
23. Mangel M, Munch SR. A life-history perspective on short-and long-term consequences of compensatory growth. Am Nat 2005;166:E155–E176.
24. Parsons TJ, Power C, Logan S. Summerbell CD. Childhood predictors of adult obesity: a systematic review. Int J Obesity 1999;23(suppl 8):S-S107.
25. Andersson SW, Bengtsson C, Hallberg L, et al. Cancer risk in Swedish women: the relation to size at birth. Br J Cancer 2001;84:1193–8.
26. Kimm SYS, Obarzanek E. Childhood obesity: a new pandemic of the new millennium. Pediatrics 2002;110:1003–7.
27. Soltesz G, Patterson CC, Dahlquist G. Worldwide childhood type 1 diabetes incidence – what can we learn from epidemiology? Ped Diab 2007;8(suppl 6):6–14.
28. Sorensen HT, Sabroe S, Rothman KJ, Gillman M, Fischer P, Sorensen TIA. Relation between weight and length at birth and body mass index in young adulthood: cohort study. BMJ 1997;315:1137–41.
29. Samaras TT. Longevity in specific populations. In: Heggenhougen K, Quah S, eds. International encyclopedia of public health, vol 4. San Diego: Academic Press; 2008:142–7.
30. Jacobsen BK, Heuch I, Kvale G. Association of low age at menarche with increased all-cause mortality: a 37-year follow-up of 61,319 Norwegian women. Am J Epidemiol 2007;166:431–7.
31. Cohen DA, Sturm R. Body index is increasing faster among taller persons. Am J Clin Nutr 2008;87:445–8.
32. Samaras TT. Body height and its relation to chronic disease and longevity. In: Samaras TT, ed. Human body size and the laws of scaling: physiological, performance, growth, longevity and ecological ramifications. New York: Nova Science Publishers;2007:63–112.
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;158:479–85.
34. Samaras TT. Should we be concerned over increasing body height and weight? Exp Gerontol 2009;44:83–92.
35. 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;23:313–42.
36. Batty GD, Shipley MJ, Langenberg C, Marmot MG, Davey Smith G. Adult height in relation to mortality from 14 cancer sites in men in London (UK): evidence from the original Whitehall study. Ann Oncol 2006;17:157–66.
37. Shaper AG. Cardiovascular disease in the tropics-IV: coronary heart disease. Brit Med J 1974;4:32–5.
38. Lindeberg S, Nilsson-Ehle P, Terent A, Vessby B, Schersten B. Cardiovascular risk factors in a Melanesian population apparently free from stroke and ischaemic heart disease: the Kitava study. J Int Med 1994;236:331–40.
39. The Audible. NIOSH study refutes myth of early death. 1994:17;1–2.
40. Wang C, Weindruch R, Fernandez JR, Coffey CS, Patel P, Allison DB. Caloric restriction and body weight independently affect longevity in Wistar rats. Int J Obesity 2004;28:357–62.
41. Rollo, CD. Overview of research on giant transgenic mice with emphasis on the brain and aging. In: Samaras TT, ed. Human body size and the laws of scaling: physiological, performance, growth, longevity and ecological ramifications. New York: Nova Science Publishers; 2007:235–60.
42. Waaler HT. Height, weight and mortality; the Norwegian experience. Acta Med Scan Supplement 1983;679:1–50.
43. Samaras TT, Elrick H, Storms LH. Is height related to longevity? Life Sci 2003;72:1781–802.
44. Miller DD. Economies of scale. Challenge 1990;33:58–61.
45. Brown-Borg HM, Borg KE, Meilska CJ, Bartke A. 1996. Dwarf mice and the ageing process. Nature 1996;384:33.
46. Salaris L, Poulain M, Piras OS, et al. Height and longevity among males born in Villagrande Strisaili (1866–1915). Conference Proceedings XLIII, Riunione Scientifica SIS, Torino, Italy 14–16 June 2006:649–52.
47. Poulain M, Mario Pes G, Grasland C, et al. Identification of a geographic area characterized by extreme longevity in the Sardinian island: the AKEA study. Exp Gerontol 2004;39:1423–9.
48. Roth GS, Ingram DK, Lang MA. 1995. Slowing ageing by caloric restriction. Nat Med 1995;1:414–5.
49. Dearen J. Oldest man in the US dies in Sacramento at 112. Associated Press, Sun, Dec. 28, 2008, 11:48 pm ET.
50. Gavrilov LA, Gavrilov NS. Biodemography of human longevity: new findings and ideas. Center of Aging Norc and the University of Chicago, Chicago, Ill. Available from http://longevity-science.org
51. 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.
52. Singhal, A, Fewtrell, M, Cole, TJ, Lucas, A. Low nutrient intake and early growth for later insulin resistance in adolescents born preterm. Lancet 2003;361:1089–97.
53. Wells JCK, Treleaven P, Cole TJ. BMI compared with 3D body shape: the UK national sizing survey. Am J Clin Nutr 2007;85:419–25.
54. Davey Smith G, Hart C, Blane D, Gillis C, Hawthorne V. Lifetime socioeconomic position and mortality: prospective observational study. BMJ 1997;314:547–52.
55. Wannamethee, SG, Shaper AG, Whincup PH, Waller M. Migration within Great Britain and cardiovascular disease: early life and adult environmental factors. Int J Epidemiol 2002;31:1054–60.
56. 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.
57. Lasker JN, Egolf BP, Wolf S. Community social change and mortality. Soc Sci Med 1994;39:53–62.
58. 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.
59. 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 ed. San Diego: Academic Press; 2006:512–33.
60. Greer KA, Canterberry SC, Murphy KE. Statistical analysis regarding the effects of height and weight on lifespan of the domestic dog. Res Vet Sci 2007;82:208–14.
61. Wheeler M. The vertical forest. Discover 1996;17:76–81.
62. Maier AB, Van Heemst D, Westendorp RGJ. Relation between body height and replicative capacity of human fibroblasts in nonagenarians. J Gerontol Biol Sci 2008;63:43–5.
63. Samaras TT. BMI and weight: their relation to diabetes, CVD, cancer and all-cause mortality. In: Samaras TT, ed. Human body size and the laws of scaling: physiological, performance, growth, longevity and ecological ramifications. New York: Nova Science Publishers; 2007:112–46.
64. Giovannelli L, Saieva C, Masala G, et al. Nutritional and lifestyle determinants of DNA oxidative damage: a study in a Mediterranean population. Carcinogenesis 2002;23:1483–9.
65. De Simone G, Verdecchia P, Pede S, Gorini M, Maggioni AD. Prognosis of inappropriate left ventricular mass in hypertension: the MAVI study. Hypertension 2002;40:470–6.
66. Dekkers C, Treiber FA, Kapuku G, Van den Oord EJCG, Snieder H. Growth of left ventricular mass in African-American and European-American youth. Hypertension 2002;39:943–51.
67. Hanna IR, Heeke B, Bush H, et al. The relationship between stature and the prevalence of atrial fibrillation in patients with left ventricular dysfunction. J Am Coll Cardiol 2006;18:1683–8.
68. Bartosz G, 2005. Food toxicity contributes to the beneficial effects of calorie restriction. J Behav Biol 2005;233:451–2.
69. Hrdlicka A. The old Americans. Baltimore: The Williams & Wilkins Co.; 1925.
70. Roth GS. Entropy theories of aging revisited. Aging Clin Exp Res 1994;6:139–40.
71. Samaras TT. The law of entropy and the aging process. Human Development 1974;17:314–20.
72. Frame LT, Hart RW, Leakey JEA. Caloric restriction as a mechanism mediating resistance to environmental disease. Environ Health Persp 1998;106(suppl 1):313–24.
73. Willcox BJ, Yano K, Chen R, et al. How much should we eat? The association between energy intake and mortality in a 36-year follow-up study of Japanese-American men. J Gerontol Biol Sci 2004;59A:789–95.