The relationship between adult stature and longevity: tall men are unlikely to outlive their short peers – evidence from a study of all adult deaths in Poland in the years 2004–2008

Piotr Chmielewski

Department of Anatomy, Faculty of Medicine, Wroclaw Medical University, Poland

ABSTRACT: Early epidemiological studies demonstrated that short stature is associated with cardiovascular disease (CVD), diabetes, lower energy intake or food deprivation during growth, poor health, and increased all-cause mortality. Nevertheless, the links between adult height and longevity become tenuous if certain confounders (e.g. BMI, SES, educational attainment, etc.) are allowed for. Furthermore, numerous studies have found that like excess weight, tallness is costly in terms of longevity in late ontogeny, and shorter people tend to outlive their taller peers, especially if they are slim and maintain a healthy diet and lifestyle. Therefore, there is currently a lack of agreement in the literature as to whether and how body height and lifespan are linked. The objective of this study was to explore the relationship between adult stature and longevity on the basis of a large sample from a population-based cohort study. Data on declared height and exact dates of birth and death were available from 480,493 men and 364,666 women who died in the years 2004–2008 in Poland. To control for secular changes, the sample was divided into fifteen birth cohorts and each group was subsequently split into five height categories using pentiles, separately for both sexes. The analysis has revealed an inverse relationship between height and lifespan in men and women. However, after controlling for secular changes in height, the relationship turned out to be very weak and linear in men, and inverted U-shaped in women. In general, taller individuals had lower age at death compared to shorter ones, and this relationship was more pronounced and consistent in men. To sum up, these findings do not comport with the traditional belief that taller individuals live longer. The role of several possible biological mechanisms pertinent to enhanced longevity in smaller individuals was emphasized, and these biological factors were discussed.

KEY WORDS: aging, body height, life expectancy, lifespan, longevity, mortality, predictor

Introduction

A multitude of studies have shown that short stature is associated with lower energy intake during growth and development, inadequate diet and nutrition, poor health, low socioeconomic status (SES), and increased risk of cardiovascular disease (CVD), diabetes mellitus, and premature death (for a review, see...
Moreover, anthropological investigations have revealed that body height correlates positively with social mobility, SES, remuneration, educational attainment, health, physical attractiveness in men, and their reproductive success, at least in Western societies (Kemkes-Grottenthaler 2005; Sear 2006; Pawłowski 2012). For several decades, researchers have been trying to ascertain whether tallness is costly in later life in terms of longevity, which would act as some type of compensation, according to the handicap principle. The final results remain mixed because the relationship between adult height and longevity is weak and tenuous and can be modified by numerous factors, such as body mass index (BMI), body fat percentage (BFP), SES, causes of short stature, illness, etc.

Thus, in general, being tall and slim has long been considered a superior configuration for humans in terms of healthspan and longevity, but it turns out that data supporting this view come from mortality studies that do not track the entire cohort to death. As regards mortality, a host of studies have demonstrated that taller individuals outlive their shorter peers (for a review, see Austad 2010; Özaltin 2012; Perkins et al. 2016), especially when certain confounding factors, such as BMI, SES, and educational attainment, are not controlled in the analysis. Nonetheless, after controlling for such types of confounders, adult stature fails to predict CVD morbidity and mortality in some populations (Liao et al. 1996; Murray 1997; Sear 2006), and after allowing for some CVD risk factors, short stature alone is not linked to elevated risk of all-cause or cardiovascular mortality in either sex, though it is related to increased risk of myocardial infarction in women but not in men (Kannam et al. 1994). Moreover, it should be noted that deaths before age 65 or 70 are premature, and thus such analyses do not provide insights into what is beneficial in terms of longevity.

Likewise, the results of comparative studies of the association between body size and longevity in animals can be misleading. Although large mammals (e.g. whales, elephants) live significantly longer than small ones (e.g. pygmy shrews, mice), numerous studies have found that within the species smaller individuals have greater longevity, and smaller mice, dogs, cows etc. live significantly longer than bigger ones (Miller et al. 2002; Rollo 2002; Miller and Austad 2006; Bartke 2012). To date, the inverse relationship between body size and longevity was confirmed in many different mammalian species. Furthermore, more recent studies suggest that higher rates of growth and greater body size may be costly in terms of longevity in humans (Bartke 2012; He et al. 2014; Samaras 2014) despite the fact that during progressive ontogeny they can be used as reliable proxies for environmental conditions, standard of living, SES, lifestyle, and general health status. The present study aims to investigate whether lifespan depends on adult stature within the Polish population, and whether the association between these two life history parameters is positive or negative.

**Materials and methods**

To evaluate the relationship between adult height and longevity in the Polish population, data on adult stature and exact dates of birth and death from 845,159 individuals, including 480,493 men and 364,666 women, were analyzed. The
study data were obtained from two electronic databases at the Ministry of Internal Affairs and Administration in Warsaw, Poland, i.e. the Universal Electronic System for Registration of the Population (sex, dates of birth and death), and from the State Register of Issued and Invalidated Identity Cards (declared body height), and concerned all adult deaths in the years 2004–2008 in Poland.

The collected data have a number of advantages. First, the study sample is large and representative for the whole studied population, which is crucial and indispensable for the analysis of the studied relationship as gathering a large sample that is representative for the whole population of the country is a sine qua non prerequisite in such studies. This is necessary because body height is an ecologically sensitive feature, determined by many biological and social factors, while the variation in lifespan as well as in body size in every human population is large and significant. Furthermore, there are numerous defined or unknown selection factors which can easily influence the results of such investigations. Second, the study sample is heterogeneous and concerns typical causes of death, i.e. the causes of death in the study sample are the same as in the total population (Chmielewski and Borysławski 2015). Even randomly chosen data sets concerning several thousand subjects are described by some researchers as insufficiently random and large as various local factors, mainly the level of pollution, smog, radiation, and climatic factors, and above all different social and biological factors can easily distort the image of the investigated relationship. Despite these reservations many epidemiological and anthropological studies involve data from small geographical areas, i.e. cities or districts where the residents are affected by specific local factors (e.g. smog), obtained in a short period of time, or concerning individuals who died of a specific disease (CVD, myocardial infarction, cancer, etc.), which is justified for practical reasons but has a negative effect on the value of research and validity of such conclusions. Third, an important advantage of the analyzed study material is the high reliability of data on dates of birth and death, which were proven by relevant documents. According to many authors, the use of the declared body height is justified, or at least acceptable, in a situation when measurements cannot be taken. This is because in young and older adults, there is a high and statistically significant correlation between the self-reported and measured values of body height, especially if height is given for official purposes, and not for matrimonial ones. On the other hand, some respondents tend to overestimate their

Table 1. Number of subjects in the consecutive birth cohorts for both sexes.

<table>
<thead>
<tr>
<th>Birth cohort</th>
<th>Men</th>
<th>Women</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>1896–1910</td>
<td>1762</td>
<td>5017</td>
<td>6779</td>
</tr>
<tr>
<td>1911–1915</td>
<td>8023</td>
<td>18557</td>
<td>26580</td>
</tr>
<tr>
<td>1916–1920</td>
<td>19989</td>
<td>34861</td>
<td>54850</td>
</tr>
<tr>
<td>1921–1925</td>
<td>51588</td>
<td>71157</td>
<td>122745</td>
</tr>
<tr>
<td>1926–1930</td>
<td>73138</td>
<td>70924</td>
<td>144062</td>
</tr>
<tr>
<td>1931–1935</td>
<td>73350</td>
<td>51055</td>
<td>124405</td>
</tr>
<tr>
<td>1936–1940</td>
<td>59876</td>
<td>33827</td>
<td>93703</td>
</tr>
<tr>
<td>1941–1945</td>
<td>41720</td>
<td>21094</td>
<td>62814</td>
</tr>
<tr>
<td>1946–1950</td>
<td>49156</td>
<td>22940</td>
<td>72096</td>
</tr>
<tr>
<td>1951–1955</td>
<td>40661</td>
<td>16150</td>
<td>56811</td>
</tr>
<tr>
<td>1956–1960</td>
<td>27135</td>
<td>9431</td>
<td>36566</td>
</tr>
<tr>
<td>1961–1965</td>
<td>13890</td>
<td>4299</td>
<td>18189</td>
</tr>
<tr>
<td>1966–1970</td>
<td>8634</td>
<td>2344</td>
<td>10978</td>
</tr>
<tr>
<td>1971–1975</td>
<td>6510</td>
<td>1684</td>
<td>8194</td>
</tr>
<tr>
<td>1976–1980</td>
<td>5061</td>
<td>1326</td>
<td>6387</td>
</tr>
<tr>
<td>Total</td>
<td>480493</td>
<td>364666</td>
<td>845159</td>
</tr>
</tbody>
</table>
body height (usually by 1-2 cm), and this more frequently happens with young men as well as with short individuals, while very tall subjects usually indicate slightly lower values of height than actual (Brener et al. 2003; Sherry et al. 2007; Danubio et al. 2008; Krul et al. 2010; Bowring et al. 2012). There is another salient criterion that should be included in such investigations and that is met by the study material, namely the research should be based solely on data gathered specifically to clarify the effect of body size, i.e. body height, on lifespan, and not on the secondary use of material collected previously for another purposes, especially if the material is cross-sectional and there is a high risk of the cohort effect (Kościński et al. 2009). A disadvantage of the collected data is that the analysis

<table>
<thead>
<tr>
<th>Sex</th>
<th>Birth cohort</th>
<th>N</th>
<th>Centiles (cm)</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td>20</td>
<td>40</td>
<td>60</td>
<td>80</td>
<td></td>
</tr>
<tr>
<td>Men</td>
<td>1896–1910</td>
<td>1762</td>
<td>160</td>
<td>165</td>
<td>170</td>
<td>173</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1911–1915</td>
<td>8023</td>
<td>162</td>
<td>165</td>
<td>170</td>
<td>174</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1916–1920</td>
<td>19989</td>
<td>164</td>
<td>168</td>
<td>170</td>
<td>175</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1921–1925</td>
<td>51588</td>
<td>164</td>
<td>168</td>
<td>171</td>
<td>175</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1926–1930</td>
<td>73138</td>
<td>165</td>
<td>170</td>
<td>172</td>
<td>175</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1931–1935</td>
<td>73350</td>
<td>165</td>
<td>170</td>
<td>172</td>
<td>176</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1936–1940</td>
<td>59876</td>
<td>165</td>
<td>170</td>
<td>172</td>
<td>176</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1941–1945</td>
<td>41720</td>
<td>167</td>
<td>170</td>
<td>174</td>
<td>176</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1946–1950</td>
<td>49156</td>
<td>168</td>
<td>170</td>
<td>175</td>
<td>177</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1951–1955</td>
<td>40661</td>
<td>168</td>
<td>172</td>
<td>176</td>
<td>178</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1956–1960</td>
<td>27135</td>
<td>170</td>
<td>173</td>
<td>176</td>
<td>180</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1961–1965</td>
<td>13890</td>
<td>170</td>
<td>174</td>
<td>176</td>
<td>180</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1966–1970</td>
<td>8634</td>
<td>170</td>
<td>175</td>
<td>177</td>
<td>181</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1971–1975</td>
<td>6510</td>
<td>172</td>
<td>176</td>
<td>178</td>
<td>182</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1976–1980</td>
<td>5061</td>
<td>172</td>
<td>176</td>
<td>180</td>
<td>183</td>
<td></td>
</tr>
<tr>
<td>Women</td>
<td>1896–1910</td>
<td>5017</td>
<td>150</td>
<td>155</td>
<td>158</td>
<td>160</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1911–1915</td>
<td>18557</td>
<td>150</td>
<td>155</td>
<td>158</td>
<td>161</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1916–1920</td>
<td>34861</td>
<td>152</td>
<td>156</td>
<td>160</td>
<td>163</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1921–1925</td>
<td>71157</td>
<td>154</td>
<td>158</td>
<td>160</td>
<td>164</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1926–1930</td>
<td>70924</td>
<td>155</td>
<td>158</td>
<td>160</td>
<td>164</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1931–1935</td>
<td>51055</td>
<td>155</td>
<td>159</td>
<td>161</td>
<td>165</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1936–1940</td>
<td>33827</td>
<td>156</td>
<td>160</td>
<td>162</td>
<td>165</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1941–1945</td>
<td>21094</td>
<td>156</td>
<td>160</td>
<td>163</td>
<td>165</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1946–1950</td>
<td>22940</td>
<td>158</td>
<td>160</td>
<td>164</td>
<td>165</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1951–1955</td>
<td>16150</td>
<td>158</td>
<td>160</td>
<td>164</td>
<td>165</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1956–1960</td>
<td>9431</td>
<td>158</td>
<td>160</td>
<td>164</td>
<td>167</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1961–1965</td>
<td>4299</td>
<td>158</td>
<td>162</td>
<td>164</td>
<td>168</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1966–1970</td>
<td>2344</td>
<td>159</td>
<td>163</td>
<td>165</td>
<td>168</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1971–1975</td>
<td>1684</td>
<td>160</td>
<td>164</td>
<td>167</td>
<td>170</td>
<td></td>
</tr>
<tr>
<td></td>
<td>1976–1980</td>
<td>1326</td>
<td>160</td>
<td>164</td>
<td>167</td>
<td>170</td>
<td></td>
</tr>
</tbody>
</table>
did not consider potentially significant confounding factors which can affect the investigated relationships and may modify them in various ways. Unfortunately, it was not possible to compare people of the same body weight or proportions because no such data (e.g. on somatotype, BMI, body fat, etc.) were available for the study sample. Likewise, the study did not allow for health status, diet, nutrition, SES, income, specific causes of death, aging-associated alterations in height, proximal causative factors proposed in the literature, e.g. hormonal profile, growth rate during progressive ontogeny, detrimental effects of catch-up growth, the place of residence (urban/rural), and so forth. Nonetheless, it seems that the relationship between adult height and lifespan can be assessed based on the study sample used for the analysis.

To control for the secular changes in body height and the cohort effect, the whole sample was divided into fifteen birth cohorts (Table 1), and subsequently each group was split into five height categories using centiles (pentiles): very short (0–20), short (21–40), average (41–60), tall (61–80), very tall (81–100) (Table 2). Statistical analysis was performed separately for both sexes. Correlation analysis and ANOVA, including the method of remnant variation, along with Fisher’s Least Significant Difference (LSD) test were run. The normality of the data distribution was tested with the goodness-of-fit test for a normal distribution.

**Results**

In the study sample, both height and lifespan were normally distributed. In general, men were taller than women (171.6±6.6 cm vs. 159.6±6.2 cm; \( F=1.14; p<0.001 \)) and lived significantly shorter (67.9±13.8 years vs. 75.0±12.7 years; \( F=1.19; p<0.001 \)).

In the first cohort, i.e. the group concerning subjects born in the years 1896–1910 (1,762 men and 5,017 women), the differences in lifespan between height categories were on the border of statistical significance in men (\( F=2.30; p=0.057 \)) and statistically significant in women (\( F=3.61; p=0.006 \)). In both sexes, very short subjects had the highest age at death, while tall subjects had the lowest age at death (Fig. 1A).

In the next birth cohort (subjects born in the years 1911–1915, 8,023 men and 18,557 women), the differences in lifespan were statistically significant in men (\( F=4.41; p=0.001 \)) but not in women (\( F=1.57; p=0.179 \)). In men, very short and short individuals lived significantly longer than the rest, while tall subjects had the lowest age at death (Fig. 1B). In women, on the other hand, no relationship between height and lifespan was observed.

In the birth cohort 1916–1920 (19,989 men and 34,861 women), a clear inverse relationship between adult height and lifespan was observed in men (\( F=17.39; p<0.001 \)) and women (\( F=20.07; p<0.001 \)). In both sexes, very short and short subjects had the highest age at death, whereas the lowest age at death was found for very tall men and tall women (Fig. 1C). The differences between tall and very tall subjects were nonsignificant.

In the birth cohort 1921–1925 (51,588 men and 71,157 women), lifespan of both sexes depends on adult height, and the differences were statistically significant (\( F=9.66; p<0.001 \) for men; \( F=14.76; p<0.001 \) for women). The highest age at death was found for the categories of
very short stature, while the lowest age at death occurred in the categories of tall stature in both sexes (Fig. 1D).

The same pattern occurred in the birth cohort 1926–1930 (73,138 men and 70,924 women) and, thus, the differences were statistically significant ($F=10.90; p<0.001; F=16.32; p<0.001$, respectively), and very short individuals lived, on average, longer than subjects from other height categories (Fig. 2A).

Likewise, in the cohort 1931–1935 (73,350 men and 51,055 women), the highest age at death had the shortest subjects, while the lowest age at death had the tallest men and tall women (Fig. 2B). In both sexes, the differences between means were statistically significant ($F=11.22; p<0.001$ for men; $F=19.91; p<0.001$ for women).

Also, an inverse and steady relationship between adult stature and longevity was observed in subjects born in the years 1936–1940 (59,876 men and 33,827 women; Fig. 2C), with the differences being statistically significant in men ($F=2.90; p=0.020$) and women ($F=3.24; p=0.011$).

The same pattern was found for the birth cohort 1941–1945 (41,720 men and 21,094 women). Thus, the arithmetic means differed significantly in men ($F=10.32; p<0.001$) and women ($F=4.33; p=0.002$), and the smallest subjects had the greatest longevity (Fig. 2D).

![Fig. 1. The association between adult height and lifespan in men and women born in the years 1896–1925 and categorized into five height groups using pentiles; data for first four birth cohorts (A–D) are shown](image-url)
Similarly, the relationship between adult height and longevity was negative and linear in men from the birth cohort 1946–1950 (N=49,156), with the differences being statistically significant in both sexes (F=8.01; p<0.001; F=3.46; p=0.008, respectively). However, unexpectedly, tall women had the highest age at death, while women with short and average stature had the lowest age at death (Fig. 3A).

In the cohort 1951–1955 (40,661 men and 16,150 women), as in the previous birth cohort, the association between height and lifespan was linear and inverse in men but tall women had the highest age at death (Fig. 3B). The analysis has revealed that the differences in mean lifespan were significant in men (F=20.74; p<0.001) and women (F=3.46; p=0.008).

Once again, the typical pattern was observed in the birth cohort 1956–1960 (27,135 men and 9,431 women) and, thus, the smallest individuals of both sexes had the greatest longevity (Fig. 3C), and the differences were statistically significant in men (F=19.44; p<0.001) as well as in women (F=15.31; p<0.001).

Also, in the birth cohort 1961–1965 (13,890 men and 4,299 women) the differences were significant (F=9.03; p<0.001; F=4.26; p=0.002, respectively), and the shortest men, as well as short women, had the highest age at death (Fig. 3D), albeit the differences in lifespan
between the neighboring categories of height in women were nonsignificant.

Likewise, an inverse relationship was found for men who were born in the years 1966–1970 (N=8,634), while there were no statistically significant differences in lifespan of women from five categories, except for the tallest women who lived significantly shorter compared with the other groups (Fig. 4A). In general, the arithmetic means were significantly differentiated in men (F=2.91; p=0.020) and women (F=3.23; p=0.012).

In the cohort 1971–1975 (6,510 men and 1,684 women), ANOVA has revealed statistically significant differences in men (F=11.12; p<0.001) but not in women (F=0.44; p=0.777). The highest age at death had the shortest men, whereas the tallest ones had the lowest age at death. In women, however, no directional association between adult height and lifespan was found (Fig. 4B).

In the last cohort, i.e. the group concerning the individuals born in the years 1976–1980 (5,061 men and 1,326 women), the arithmetic means are differentiated in men (F=3.63; p=0.006) and women (F=3.28; p=0.011). The highest age at death was found for short men and women, while the lowest age at death occurred in the categories of average stature in both sexes (Fig. 4C).

Fig. 3. The association between adult height and lifespan in men and women born in the years 1946–1965 and categorized into five height groups using pentiles; data for next four birth cohorts (A–D) are shown.
As regards the analysis concerning the association between stature and longevity in the oldest old subjects, i.e. 85 years of age and older, tallness was negatively associated with lifespan in both sexes. In men ($N=39,191$), the correlation between adult height and longevity was very weak and negative ($r=-0.1; \ p<0.001$), and after the elimination of secular changes in body height it was $-0.03$ and $p<0.001$, respectively (Fig. 5A and 6A). In women ($N=75,405$), the relationship turned out to be inverse and very weak ($r=-0.13; \ p<0.001$), and after adjusting for secular trends in height it changed into $-0.06$ and $p<0.001$, respectively (Fig. 5B and 6B).

**Discussion**

The science of aging seems to be fraught with misinterpretations. For example, the well-known dualism of stochastic and programmed theories of aging is in fact obsolete and discarded (Rattan 2006). Like the processes of graying hair and wrinkle formation, lifespan does not measure the rate of aging (Kowald 2002; Heward 2010). The rates and measures of survival, such as all-cause mortality, life expectancy, and longevity, do not correspond to each other and provide different information (Chmielewski et al. 2016a). Likewise, healthspan does not correspond to lifespan. It is well established that men are physically stronger.
Fig. 5. The relationship between adult height and longevity in men (A) and women (B) from the oldest old group (85 years of age and older)
Fig. 6. The relationship between adult height and longevity in men (A) and women (B) from the oldest old group (85 years of age and older) after eliminating the secular changes in body height which interfered with the observed relationship between adult stature and lifespan.
than women and have relatively longer healthspan (Eskes and Haanen 2007; Møller et al. 2009), but women remain less susceptible to many diseases and significantly less vulnerable to environmental factors compared with men (Stini 1969; 1978; Stinson 1985). The genetic and developmental stability in women is increased presumably due to the homogametic state (Austad 2006). In addition, estrogens play an important protective role, and in women mitochondria produce less reactive oxygen species (ROS). Consequently, women tend to live longer than men despite the fact that their healthspan is relatively shorter (Stindl 2004; Austad 2006; Eskes and Haanen 2007; Møller et al. 2009; Caruso et al. 2013; Chmielewski et al. 2016c).

Similarly, the well-known inverse relationship between adult height and mortality, which has been demonstrated in an abundance of studies, may be in fact spurious, or at least confined to populations with low SES and high mortality rates, because final adult stature strongly depends on health, diet, nutrition, energy intake, and SES during growth and development. It is noteworthy that excess weight, which is not infrequently associated with lower mortality in such populations, also seems to be a predictor of longer life expectancy (Flegal et al. 2005). However, it is unlikely that overweight people have a longevity advantage over slim people. Thus, excess weight as a predictor of longevity is most likely an artifact that result from the fact that slimmer individuals in some populations, and especially in those with low SES and high mortality rates, tend to be undernourished and ill (e.g. emaciation and marasums due to extremely low food energy consumption, cachexia due to chronic diseases, etc.). It is noteworthy that most studies did not compare tall and short individuals of similar body proportions, and thus it is possible that many findings result from different types of artifacts. Likewise, adjusting for risk factors can be a crude and inexact process. Poor health or factors like smoking can easily affect the final results. Year of birth is often an important confounding factor because younger birth cohorts are taller and have longer life expectancy compared to older cohorts. Another confounder can be the fact that those subjects who have spent their entire lives in the upper class are taller and have lower mortality than upper class men who were born in lower classes and worked up to the upper class. They are taller than those who remained in the lower classes all their lives. Some insurance studies have found that taller men have a lower mortality that shorter men. However, the shorter subjects were more overweight than the taller ones. Wilhelmsen et al. 2011 tracked a group of 67-year old Swedish men to 90 years of age, and this investigation has revealed that men who were shorter at baseline were more likely to reach 90 years of age compared to taller men.

It has previously been established that body height is a good and reliable proxy for health status at both progressive and regressive stages of ontogeny, and tallness is believed to be associated with better health, higher SES, reduced all-cause mortality, and longer life expectancy (Waaler 1984; Nyström Peck and Vågerö 1987; Holl et al. 1991; Allebeck and Bergh 1992; Elo and Preston 1992; Herbert et al. 1993; Power and Matthews 1997; Silventoinen et al. 1999; Cave-laars et al. 2000; Jousilahti et al. 2000; Lawlor et al. 2002; 2004; Engeland et al. 2003; Kemkes-Grottenthaler 2005; Song
and Sung 2008; Austad 2010; Kirkwood 2010; Öztalı 2012; Chmielewski et al. 2015a; 2015b; 2016a; 2016b; Perkins et al. 2016). The better health status, nutrition, environmental conditions, medical care, and prophylaxis during the progressive development, the taller the individuals are in a given population. For example, a recent study of children in Tanzania showed that children who lost a father before the age of 15 tended to be significantly shorter and lived shorter than their peers who had a father (Kirkwood 2010). Interestingly, those children who lost a mother were even shorter and lived shorter than their peers who had a mother and than those who lost a father. Probably until late adulthood, height can be used as a reliable and genuine proxy for health. Moreover, adult stature is positively associated with socioeconomic status (SES), social mobility, educational attainment, remuneration, and physical attractiveness, especially in men in Western countries, while it is inversely related to mortality and risk of many diseases (Sear 2006; Öztalı 2012; Pawłowski 2012). Furthermore, tallness may confer protection against some types of cancer, including mouth, esophagus, and gastric (Leoncini et al. 2014; Perkins et al. 2016), which is consistent with many previous investigations showing that short stature reflects childhood illness, low energy intake, low SES, and poor health outcomes in later life (Davey Smith et al. 2000; Öztalı 2012; Perkins et al. 2016). Therefore, height has long been used as a crude indicator of the nutritional health and disease exposure in numerous human populations by epidemiologists, demographers, anthropologists, physicians, and economists. As a result, the majority of researchers and scholars still believe that taller individuals are generally healthier and less prone to many diseases than their shorter peers (Austad 2010). Other epidemiological, biodemographic, and bioarcheological studies have demonstrated that shorter people have increased mortality, and considerably lower age at death compared to taller ones (Peck and Vågerö 1989; Läära and Rantakallio 1996; Lawlor et al. 2002; 2004; Engeland et al. 2003; Kemkes-Grottenthaler 2005; Song and Sung 2008; Öztalı 2012), which seems to be a coincidental rather than a causal relationship, which means that in some samples tallness is coincidentally linked to better health and survival, but shortness alone does not predict shorter life expectancy, especially when BMI, SES, and educational attainment are allowed for (Sear 2006; Samaras 2014).

Although early epidemiological studies have shown that short stature is associated with cardiovascular disease (CVD), diabetes, lower energy intake or food deprivation during growth, poor health, and increased all-cause mortality, the link between adult height and longevity becomes tenuous when certain confounding factors, such as BMI, SES, and educational attainment, are controlled for. Furthermore, more recent studies have found that like excess weight, tallness is costly in terms of longevity in late ontogeny (Gavrilov and Gavrilova 2008), and shorter people tend to outlive their taller peers, especially if they are slim and maintain a healthy diet and lifestyle (Samaras 2014). Therefore, there is currently a lack of agreement in the literature as to whether and how body height and lifespan are linked. Numerous recent studies have challenged the traditional view that tallness predicts greater longevity. It is now well established that the association between
adult height and mortality is in fact heterogeneous and complicated, and several large studies have found a positive association between height and, for instance, some types of cancer (Dieckemann et al. 2008; Cairns and Green 2013; Kabat et al. 2013a; 2013b; Wirén et al. 2014; Jiang et al. 2015). Furthermore, dwarfism and significantly reduced levels of hormones (e.g. GH and IGF-1) and other biological factors that stimulate growth are associated with extended longevity in model organisms such as mice (Bartke 2012). Interestingly, diet-induced alterations in the concentration of these factors can confer survival advantage in both animals and humans, and many studies have found that smaller individuals outlive their taller peers, especially if they remain slim, and the BMI and SES of compared individuals are commensurate (Samaras 2014). It was also suggested that smaller body size can offer advantages in terms of healthy aging and longevity in late ontogeny (Wilhelmsen et al. 2011; Salaris et al. 2012; He et al. 2014; Samaras 2014). Therefore, although historical demographers argue that smaller individuals have increased mortality and shorter life expectancy, biologists and gerontologists contend that small body size may be preferable for longevity, if short stature does not result from illness associated with retarded growth during progressive ontogeny but from the type of physical constitution. Gavrilova and Gavrilov (2008) assert that “biologists are firmly convinced that a small body size is preferable for longevity”. Moreover, this finding makes perfect sense in the light of the hyperfunction theory of aging (Blagosklonny 2013), as well as some other molecular mechanisms related to aging and longevity (Bartke et al. 2013; He et al. 2014; Samaras 2014).

Notwithstanding these findings, which seem compelling, many authors do not agree that the traditional view, according to which taller people are healthier and live longer, is in fact invalid or at least challenged. Austad (2010) states explicitly that: “Economists and historians often use height as an indicator of the nutritional health and disease exposure of human populations and as such an indicator in dozens of studies, greater height correlates with longer life. Samaras, a strident advocate of the opposite view that shorter humans live longer than taller ones – reaches his conclusion by comparing heights of different sexes or different countries or ethnic groups within a country with one another. Due to variation in hormonal milieu, diet, lifestyle, and multiple other factors in his analyses, it is difficult to evaluate these claims in the face of a mountain of opposing epidemiological evidence”. Also, other researchers maintain that a multitude of studies have shown that adult height correlates positively with health, wealth, SES, social mobility, and life expectancy, but if one compares different populations (like e.g. Samaras et al. 2003), the results can be opposite to the results from a relatively homogenous population. For example, shorter, on average, Japanese people tend to live longer than relatively taller Europeans. This approach seems inappropriate as it does not allow for genetic differences associated with various adaptations to different conditions of living. An assessment of men’s health within a given ethnic group would be a much better approach. It should be remembered that body height may be a very indirect cause of differences in incidence of different diseases and there are many
more important factors, such as diet, nutrition, and lifestyle, which have a significantly greater influence on, for instance, coronary heart disease (CHD) than body height alone (Pawłowski 2009).

For comparative purposes, mean lifespan of subjects who differed in body height, and who were thus classified into five height categories, was used in the present study. The relationship between adult stature and longevity was evaluated within one population, and therefore the results are unlikely to be encumbered with effects of genetic or ethnic differences. The analysis concerning longevity was confined to subjects aged 85 and above. These two approaches may seem straightforward but are useful, and they have been employed in numerous previous studies. In the first type of analysis, lifespan of subjects who differed in height was compared only within consecutive birth cohorts, where the differences in lifespan were limited. Thus, the well-known problem of considerable variability in human lifespan, which is often raised, was curbed. The results of this analysis unambiguously show that shorter men tend to live longer, but the relationship between adult stature and lifespan in women is tenuous. In the studied population, the negative association between height and lifespan is more pronounced in men than in women, which may be attributable to greater ecosensitivity of the male sex (Stini 1969; 1978; Stinson 1985). Interestingly, the links between height and lifespan are more tenuous in individuals under the age of 85 compared with individuals aged 85 and over. This observation lends credence to the hypothesis that longevity depends on adult height and shorter individuals tend to live longer than taller ones as the negative association between height and lifespan is young and middle-aged people is unanticipated. Thus, in young and middle-aged people, lifespan usually does not depend on adult stature, mainly due to the fact that deaths before age 65 are premature. By contrast, deaths after age 85 are closely related to the ravages of the aging process, and longevity may favor smaller and slimmer individuals (Bartke 2012).

There are at least two causes of the observed inverse correlation between height and lifespan, which could explain the findings and which do not pertain to biological mechanisms responsible for greater longevity of smaller individuals. First, individuals born earlier were significantly shorter than those who were born later (the secular changes in height), and the latter lived significantly shorter than the former (the cohort effect) because the lifetime of each subject lasted until the fixed limit of the years 2004–2008. Second, long-lived subjects are also shorter, on average, than their short-lived peers as they experience the regressive changes in body height for a relatively longer time (Sorkin et al. 1999; Chmielewski et al. 2015a; 2015b; 2016b). Although only the first effect was controlled in the present study, the second one is unlikely to blur the longevity picture. The age-related changes in height consist in decrease and not increase in height and therefore such aging-associated changes can enhance the studied association, i.e. shorter stature in older individuals, but cannot distort it.

There are several possible biological mechanisms pertinent to enhanced longevity in smaller individuals (Table 3). Smaller individuals have reduced DNA damage as molecular damage increases with taller stature (Giovannelli et al. 2002). Some studies have found that
body size is inversely related to telomere length, and shorter people tend to have longer telomeres (Maier et al. 2008). Presumably longer telomeres and a slower rate of shortening in telomere length, as well as lower level of oxidative damage are associated with increased longevity and decreased risk of some aging-associated diseases such as CVD (Samaras 2014). Interestingly, hypertension is associated with increased oxidative stress (Baradaran et al. 2014), and is also a major risk factor of atherosclerosis, stroke, chronic kidney failure (CKF), coronary heart disease (CHD), heart failure, and myocardial infarction. Thus, higher arterial blood pressure is a strong predictor of a shortened life expectancy (Chobanian et al. 2003; Franco et al. 2005). Some authors claim that lower blood pressure, which is associated with decreased risk of CVD, CHD, myocardial infarction, stroke, and premature death, can be attributed to biological mechanisms that are responsible for greater longevity in smaller individuals (Samaras 2014). Shorter and slimmer people have lower insulin and IGF-1 levels, which are related to greater longevity. Furthermore, increased insulin, insulin-like growth factor 1 (IGF-1), growth hormone (GH), mechanistic target of rapamycin (mTOR), adiponectin, C-reactive protein (CRP), some binding proteins such as IGFBP-1, and sex hormone binding globulin (SHBG) have a negative impact on longevity in humans and tend to increase with body size, i.e. body height, weight, BMI, or with all three. As regards cellular aspects of senescence, some studies have suggested that shorter and slimmer individuals have significantly reduced free

<table>
<thead>
<tr>
<th>Biological factors</th>
<th>Explanation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reduced molecular damage and lower level of oxidative stress due to lower ROS generation</td>
<td>Lower risk of many diseases, including CVD and cancer. For example, hypertension is strongly associated with high level of oxidative damage. Increased generation of ROS and high level of molecular damage are closely related to higher risk of cancer and premature death.</td>
</tr>
<tr>
<td>Longer telomeres and slower rate of telomere attrition</td>
<td>It has been established that telomere length, which can be affected by various lifestyle factors, can affect the pace of aging as well as the onset of aging-associated diseases. In general, shorter telomeres and faster process of telomere shortening are associated with poor health and shorter life expectancy.</td>
</tr>
<tr>
<td>Lower total number of cells and a greater mitotic potential of cells</td>
<td>Reduced cell replication and telomere attrition due to lower life-long maintenance requirements; presumably lower risk of cancer at different anatomic sites. Moreover, lower blood glucose level, insulin, and IGF-1; presumably reduced oxidative damage and improved survival. CR is associated with lower blood pressure and lower risk of many age-related diseases, including CVD and cancer.</td>
</tr>
<tr>
<td>Lower blood pressure</td>
<td>Longer life expectancy and lower risk of renal failure, left ventricular hypertrophy, and other consequences of high blood pressure.</td>
</tr>
<tr>
<td>Relatively lower BMI</td>
<td>Increasing BMI promotes some chronic diseases, including diabetes, CVD, and cancer.</td>
</tr>
<tr>
<td>Lower intake of toxins</td>
<td>Shorter and slimmer people tend to have lower food energy intake and water consumption.</td>
</tr>
<tr>
<td>Lower levels of insulin, GH, IGF-1, SHBG, CRP, mTOR</td>
<td>They increase with greater cell growth and taller stature at the organismal level and are also related to shorter life expectancy.</td>
</tr>
</tbody>
</table>
radical generation. Moreover, lower cell number in shorter subjects, relatively lower need for new cells of the shorter organism compared to taller one, and lower level of ROS generation is associated with slower aging and decreased risk of cancer and other aging-associated diseases such as CVD. In shorter individuals, the size of some internal organs, such as the liver, kidneys, and brain, is relatively larger, and larger organs have greater functional capacity. There can be many other biological factors that favor smaller individuals in terms of healthspan and longevity. For example, He et al. 2014 found that shorter American men of Japanese ancestry have a higher expression of FOXO3 gene, which is related to improved survival and greater longevity.

In addition to the supports provided by biological factors for the greater longevity of shorter people, evidence from eight different types of studies supports the advantages of smaller body size. These include (Samaras 2014): (1) extensive research involving animal and human subjects shows that within a species, the smaller individuals tend to live longer. (2) Hundreds of animal studies show that caloric restriction reduces body size and increases longevity; it seems that both caloric restriction and smaller body size contribute independently to greater longevity. (3) Females are, on average, smaller than males and have enhanced longevity; e.g. U.S. males are 9% taller than females and have a 9% shorter life expectancy. In this study, men averaged 8% taller and had a 10% shorter life expectancy. Interestingly, other studies have also found that among nonhuman species, larger females do not live as long as smaller males within the same species, and therefore sexual dimorphism in lifespan can be associated with the differences in body size of both sexes and other related factors (e.g. the rate of growth and development, GH, the insulin/IGF-1 signaling pathway, mTOR, etc.) within a given species. (4) Ethnic groups within the U.S. show a progressive increase in age-adjusted mortality with the increase in average height of each ethnic group, and these include results based on about 18 million deaths over a 15-year period. This advantage is strongest in the first generation of Latinos and declines, or even disappears, with subsequent generations since they are often taller and heavier than the first generation. (5) Old age survival studies show that shorter people are more likely to reach advanced ages compared to taller people (e.g. Wilhelmsen et al. 2011). (6) The relationship between shorter height and greater longevity has been found among various races and ethnic groups, independent of the nation’s economic status. (7) In general, life expectancy is greater for shorter nations when both taller (e.g. the Dutch people, Norwegians, and Finns) and shorter nations (e.g. the mainland Japanese people, Okinawans, the people of Andorra, and so forth) are developed. (8) By and large, a host of studies have shown that centenarians and supercentenarians tend to be short and light.

Conclusions

A growing body of evidence suggests that shorter and slimmer individuals live longer than taller ones, if they maintain healthful diet, nutrition, and lifestyle. Although body height is definitely not a major factor for exceptional longevity (height is probably only 5–10% of the longevity picture, and genetic inheritance along with lifestyle are the most important factors which constitute the rest
of the longevity picture), more recent studies have challenged the traditional belief that taller people have a longevity advantage over shorter people. There are several plausible biological mechanisms that are responsible for enhanced longevity in smaller individuals, such as reduced DNA damage, longer telomeres and slower rate of telomere attrition, greater functional capacity of some organs, lower levels of insulin, IGF-1, GH, mTOR, SHBG, CRP, and so forth. Although earlier studies demonstrated that taller individuals have lower mortality, higher SES, better health, and longer life expectancy, evidence from novel data indicates now that smaller body size is an advantage under prosperous and similar environmental conditions, lifestyles, and medical care.

Acknowledgements

The author would like to thank the anonymous reviewers for very helpful and insightful suggestions for improving the paper.

Conflict of interest

The author declares that there is no conflict of interest.

Corresponding author

Piotr Chmielewski, Department of Anatomy, Faculty of Medicine, Wroclaw Medical University, ul. T. Chałubińskiego 6a, 50-368 Wroclaw, Poland.
e-mail address: piotr.chmielewski@umed.wroc.pl

References

Blagosklonny MV. 2013. Big mice die young but large animals live longer. Aging 5:227–33.
Caruso C, Accardi G, Virruso C, Candore G. 2013. Sex, gender and immunosenescence: a key to understand the different
Short stature is related to greater longevity


Short stature is related to greater longevity


