Published in final edited form as:

Am J Hum Biol. 2011 ; 23(3): 412–416. doi:10.1002/ajhb.21165.

How Boys Grow Determines How Long They Live

DAVID J.P. BARKER^{1,2,3,*}, EERO KAJANTIE^{4,5}, CLIVE OSMOND¹, KENT L. THORNBURG²,
and JOHAN G. ERIKSSON^{4,6,7,8,9}

¹MRC Lifecourse Epidemiology Unit, University of Southampton, Southampton General Hospital, Southampton SO16 6YD, United Kingdom ²Heart Research Center, Oregon Health and Science University, Portland, Oregon 97201-3098 ³Chair of Fetal Programming, Zoology Department, College of Science, King Saud University, Saudi Arabia ⁴Department of Chronic Disease Prevention, National Institute for Health and Welfare, Helsinki, Finland ⁵Hospital for Children and Adolescents, Helsinki University Central Hospital, Helsinki, Finland ⁶University of Helsinki, Department of General Practice and Primary Health Care, Helsingin yliopisto, Finland ⁷Vasa Central Hospital, Sandviksgatan 2-4, 65130 Vasa, Finland ⁸Folkhälsan Research Centre, Helsinki, PB 63, 00014 Helsingfors Universitet, Finland ⁹Unit of General Practice, Helsinki University Central Hospital, Finland

Abstract

Objectives—Increase in height in modern societies has been accompanied by an increase in lifespan. The longer lives of taller people suggest that good nutrition during childhood, together with freedom from recurrent minor infection, prolong human life. There is, however, a caveat. Tall adult stature may be the result of rapid “compensatory” growth following a setback. Compensatory growth is known to reduce the lifespan of animals, possibly because it is disorganized.

Methods—We analyzed lifespan among 6,975 men born in Helsinki, Finland, during 1934–44. Their early growth was recorded.

Results—Boys who were tallest at seven years of age had lower all cause mortality, the hazard ratio being 0.79(95%CI 0.70 to 0.89, $P < 0.0001$) per 10 cm increase in height. There was, however, a group of boys among whom being tall was associated with increased all cause mortality, the hazard ratio being 1.32(1.00 to 1.75, $P = 0.05$). These boys were taller at seven years than their birthweight and length at birth predicted. After they were excluded from the analysis, boys who were more than 126 cm in height at seven lived for eight years longer than those who were 114 cm or less. This increase in lifespan was similar to the effect of high socio-economic status in adult life.

Conclusions—Rapid growth in childhood height usually predicts a longer life. But tallness among men may be a misleading indicator of wellbeing and longer life expectancy in populations where compensatory growth is widespread. African Americans may be an example.

During the last century the lifespan of people in industrialized countries has increased by about 30 years (Crews, 2003; Kinsella, 1992). The change is too rapid to be explained by alterations in the genotype. It is usually attributed to better living conditions and health care. Studies of elderly people show, however, that these only partly explain why one person lives longer than another (Crews, 2003). In modern societies, increase in life expectancy has been

accompanied by more rapid childhood growth and an increase in adult stature (Floud et al., 1990; Fogel, 2004; Tanner, 1981). Taller people have lower mortality rates, the strongest effects being in the age range 50–75 years (Hebert et al., 1993; Davey-Smith et al., 2000; Langenberg et al., 2005; McCarron et al., 2002; Sunder, 2005; Waaler, 1984). Tall stature is the product of good nutrition in utero and during childhood combined with the absence of recurrent infection, which diverts nutrients away from growth (Tanner, 1989). The longer lives of taller people therefore suggest that good nutrition during development prolongs human lifespan. There is, however, a caveat. Tall adult stature may be the result of rapid “compensatory” growth following a period of adversity during childhood (Golden, 1998). In domestic animals compensatory growth is known to have long term costs that include reduced lifespan (Metcalf and Monaghan, 2001). Little is known about the effects of compensatory growth on lifespan in humans. We have examined the relation between early growth and longevity within a cohort of 6,975 men born in Helsinki, Finland.

Consistent with many other studies analyses within the Helsinki Birth Cohort have shown that small body size at birth is associated with increased rates of cardiovascular disease, and the disorders related to it, in later life (Barker et al., 2005). We have found that the effects of childhood growth on later disease are modified by size at birth (Eriksson et al., 2001; Barker et al., 2009). In the present analyses we therefore sub-divided the men according to their birthweight. We have also found that the effects of birth size on later disease are modified by maternal stature (Barker et al., 2010). This may reflect the link between maternal stature and rates of fetal growth. A small baby born to a tall mother may have had a different path of fetal growth than a baby of the same size born to a short mother. In the present analyses we therefore examined the effects of childhood growth among men with short or tall mothers. We defined compensatory growth by the height attained at seven years exceeding that predicted by size at birth.

METHODS

The Helsinki Birth Cohort includes 6,975 men who were born in the city during 1934–44 and went to child welfare clinics. They were born in either the University Central Hospital or in the Maternity Hospital. Details of the birth records have been described (Barker et al., 2005). They include the mother’s height and weight in late pregnancy and the date of her last menstrual period. The weight and length of the baby were recorded. The child welfare clinics recorded growth from birth to seven years. Each boy had an average of 11 measurements of height and weight before the age of two years. On school entry, which in Finland occurs at seven years, height and weight were again recorded.

We used the father’s occupation to define the mother’s social class. On the basis of a classification from Statistics Finland fathers were grouped into upper and lower middle class and manual workers (Barker et al., 2001). The men’s own occupations, recorded at successive five-year censuses from 1970 to 2000, were obtained from Statistics Finland, who grouped them into four categories—higher official, lower official, self-employed, and manual worker. We used the highest category attained. Using the personal identification number assigned to each Finnish citizen we identified all deaths among the men during 1971–2007. All deaths in Finland are recorded in the national mortality register.

Statistical methods

The end point for our survival analysis was death. Men were censored in the analysis when they migrated from Finland or survived to the end of 2007. We used a Cox proportional hazards model to calculate the hazard ratios for death stratified by year of birth. To determine the statistical significance of trends measurements were analyzed as continuous variables although they are presented in the tables as groups. We used a linear regression

analysis to predict height at age seven years from length at birth. The residuals from this regression measure the extent to which height at age seven years exceeds that predicted from length at birth. We used the residuals as a measure of compensatory growth in height.

RESULTS

Totally, 1,570 of the men had died. Table 1 shows the causes of death. There were 520 deaths from cardiovascular disease and 1,050 deaths from non-cardiovascular causes. Table 2 shows the trends in mortality among all men in relation to their height at seven years. Mortality fell with increasing height. There were strong trends with both cardiovascular mortality, for which the hazard ratio for each 10 cm increase in height at 7 was 0.78 (95% CI 0.64–0.96), and for non-cardiovascular mortality, hazard ratio 0.78 (95% CI 0.66–0.92). Across the range of height there was a 4.9-year difference in life expectancy. The boy's body mass index at seven years did not predict all-cause mortality.

In Table 2 the men are divided according to whether their birth weights were above or below the median, 3,470 g. In men with birth weights below the median, all cause mortality fell with increasing height at seven years. There was no similar trend in men with birth weights above the median. There was a statistically significant interaction between the effects of height at seven years and birth weight.

In Table 3 men with birth weights above the median are divided according to whether their mother's heights were below or above the median, 160 cm. In men whose mother's height was below the median, all cause mortality fell with increasing height at seven years. In men whose mother's height was above the median, mortality rose with increasing height at seven years. There was a statistically significant interaction between the effects of height at seven years and mother's height.

Figure 1 shows the hazard ratios for all cause mortality according to a 10 cm increase in childhood height among all the men, subdivided around their median birth weight and mother's height. In the group for whom both measurements were above the median, mortality increased with increasing height. This contrasts with the trends in the other three groups. In Table 4 these three groups are combined, and among them men who were short at age seven years lost eight years of life compared with those who were shortest at age seven years lost eight years of life compared with those who were tallest.

We examined the separate effects of growth in height during infancy (birth to two years) and early childhood (two to seven years). Within the combined group (Table 4) rapid growth during both infancy and early childhood was associated with reduced mortality (P for trend < 0.001 for both). Among men with birth weights and mother's heights above the median, rapid growth in height during infancy was associated with reduced lifespan (P for trend = 0.007), but there was no similar trend with rapid growth during childhood (P for trend = 0.4).

Compensatory growth

Among men with birth weights and mother's heights above the median, reduced life expectancy was associated with low birth weight and short length at birth. In a simultaneous regression, mortality increased with lower birth weight ($P = 0.04$) and with greater height at seven years ($P = 0.03$). Birth weight was correlated with mother's body mass index in late pregnancy ($r = 0.25$). When this was added to the regression the findings were $P = 0.007$ for birth weight and $P = 0.03$ for height at 7. When birth weight was replaced by birth length the P -values were 0.01 for length and 0.02 for height at 7. These associations were little changed by adjusting for gestation. We examined the correlates of growth in height among

these men. Rapid growth during infancy was associated with tall maternal stature ($P < 0.001$) but not with maternal weight or body mass index.

We examined which causes of death within this group of men were predicted by being tall at seven years. For each 10 cm increase in height at 7, the hazard ratio for cardiovascular death was 1.24 (95% CI 0.76–2.01) and for noncardiovascular death 1.45 (95% CI 0.97–2.15). Of the individual causes of death in Table 1, only stroke was predicted by height at 7, with a hazard ratio of 4.26 (95% CI 1.32–13.75).

Social class

All cause mortality was increased in men whose fathers had low occupational status. The hazard ratio among the sons of manual workers was 1.59 (95% CI 1.31–1.92, $P < 0.001$) compared with the sons of middle class men. The men's own occupational status was also associated with mortality. The hazard ratio among men who were manual workers was 2.13 (95% CI 1.83–2.47, $P < 0.0001$) compared with men who were higher officials. Manual workers lost 8.1 years of life compared with higher officials. In a simultaneous regression both low father's occupational status and the men's own low occupational status predicted increased mortality. The hazard ratios were 1.46 (95% CI 1.18–1.80) and 1.98 (1.69–2.31).

In a regression restricted to the combined group (Table 4) both low father's occupational status and short stature were associated with increased mortality. The hazard ratios were 1.48 (95% CI 1.08–2.01) for low occupational status, and 1.42 (1.22–1.65) for a 10 cm reduction in stature. Similarly among men with birthweight and mother's height above the median both low father's occupational status and tall stature were associated with increased mortality. The hazard ratios were 1.75 (95% CI 1.09–2.81) for low occupational status, and 1.33 (95% CI 1.00–1.78) for a 10 cm increase in stature.

DISCUSSION

In a cohort of boys born in Finland around 70 years ago those who were tallest when they entered school at seven years of age had longer lives. The effect of short stature on years of life lost was comparable to the effect of low socioeconomic status in adulthood. This demonstrates the importance of early development in determining lifespan.

In most modern societies tall people live longer than short people (Davey-Smith et al., 2000; Hebert et al., 1993; Langenberg et al., 2005; McCarron et al., 2002; Sunder, 2005; Waaler, 1984). Waaler (1984) has used extensive Norwegian data to define contours of mortality as a function of height. Among the common causes of death, cardiovascular disease is most strongly related to height (Davey-Smith et al., 2000; Hebert et al., 1993; Langenberg et al., 2005; McCarron et al., 2002; Sunder, 2005). In the US NHANES study each additional inch of height reduced the risk of death from cardiovascular disease by 3.5% in men and by 5.2% in women (Sunder, 2005). Like the other processes that accompany development, growth is plastic and responds to the environment, although it is also influenced by genetic inheritance (Tanner, 1989). The longer lives of taller people indicate that influences that affect lifespan also affect growth. Height is the product of two biological processes, the ability to grow and an adequate supply of nutrients. The ability to grow depends on hormonal signals and metabolic processes that are programmed during life in utero and in infancy (Harding, 2001; Jackson, 2000).

It is a common observation that after a child's growth has slowed because of malnutrition it may, on recovery, grow more rapidly than usual so that the child returns to the size it would have been had it not been malnourished (Golden, 1998). The ability to mount rapid compensatory growth is common in animals, and familiar to farmers (Metcalf and

Monaghan, 2001). If energy is allocated to rapid growth, the allocation to some other developmental activity must be reduced. Compensatory growth has a wide range of physiological and metabolic costs that include premature death and failure of the immune system to develop (Metcalf and Monaghan, 2001). For that reason farmers do not force young domestic animals to grow as fast as they could.

In our study there was a group of boys among whom being tall at seven was associated with a shorter life. They belonged to a group of boys who had birth weights and maternal heights above the median. We suggest that their tallness was the result of compensatory postnatal growth. While they had above median birth weights they were lighter and shorter at birth than their mothers body mass index predicted. This suggests that their growth slowed at some point during gestation. We do not know the cause of this but low father's occupational status, which predicted reduced lifespan, is one possibility. There were food scarcities in Finland during the time when the boys were born (Pesonen et al., 2007). These would have been more severe among people of low socioeconomic status. After birth the boys grew rapidly to become taller than their birth weights and birth lengths predicted. This rapid growth was associated with their mothers' tall stature. The processes that underlie this are unclear.

Among the men for whom tallness at seven years was detrimental greater height predicted stroke. This is consistent with the association between rapid postnatal growth and raised blood pressure, the main risk factor for stroke. Children and adolescents who are growing rapidly have high blood pressure for their age (Lever and Harrap, 1992). Because blood pressure "tracks" through childhood this leads to higher blood pressure in early adult life. In a study of Swedish men the highest blood pressures occurred in those who had had low birthweight but were currently tall (Leon et al., 1996). Small size at birth is known to be associated with reduced nephron number (Brenner and Chertow, 1993; Hinchcliffe et al., 1992). Large body size in adult life increases the excretory load on this reduced functional capacity, and this is thought to lead to premature nephron death and hypertension (Barker et al., 2006). Another possible link between compensatory growth and stroke is that, while normal growth is highly organized, compensatory growth may be disorganized so that the balance of growth of brain tissue and blood vessels in the brain is disturbed. We have no evidence for this.

Compensatory growth may be common among African Americans. Their mean birth weight is 250 g below European Americans, which has been suggested to be an intergenerational legacy of slavery (Jasienska, 2009). After birth, they tend to grow tall and they have high rates of stroke, hypertension, and renal failure (Barker and Lackland, 2003; Lackland et al., 2000).

CONCLUSION

Tall stature is widely used as an indicator of good health and affluence in a population (Steckel, 2009). Our findings show, however, that it may be misleading in populations where compensatory growth is widespread. African Americans may be an example.

Acknowledgments

Contract grant sponsors: National Institute of Aging, The Academy of Finland, British Heart Foundation, Finnish Medical Society Duodecim, Finska Läkaresällskapet, Foundation for Pediatric Research, Jalmari and Rauha Ahokas Foundation, Juho Vainio Foundation, Paivikki, and Sakari Sohlberg Foundation, Signe and Ane Gyllenberg Foundation, Yrjö Jahnsson Foundation and the Edwards Endowment.

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Fig. 1.

Hazard ratios for all cause mortality according to increasing childhood height in men subdivided around their median birth weight and mother's height.

TABLE 1

Numbers of deaths by cause

	ICD codes (9th; 10th rev)	Number of deaths	Percent
All causes		1570	100
Cardiovascular disease	390–459; I00–I99	520	33
Coronary heart disease	410–414; I21–I25	315	20
Stroke	430–438; I60–I69	83	5
Non-cardiovascular disease		1050	67
Cancer	140–209; C00–C99	337	21
Lung	161–162; C32–C34	99	6
Gastrointestinal system	150–157; C15–C26	104	7
Prostate	185; C61	13	1
Respiratory disease	460–519; J00–J99	64	4
Gastrointestinal disease	520–579; K00–K99	151	10
Non-natural causes	800–999; W00–X99	84	5

TABLE 2

Hazard ratios for all causes of death among men according to height at age seven years and birthweight

	All men			Birth weight (g)		
	HR (95% CI)	Years of life lost	Deaths/men	HR (95% CI)	Deaths/men	HR (95% CI)
Height at age 7 years (cm)						
114	1.6 (1.3–2.0)	4.9	98/298	1.4 (1.1–1.9)	45/147	1.3 (0.9–1.9)
–117	1.2 (1.0–1.5)	2.0	117/440	1.1 (0.9–1.5)	64/276	0.9 (0.7–1.3)
–120	1.1 (0.9–1.3)	0.8	148/616	1.0 (0.8–1.3)	112/520	0.9 (0.7–1.1)
–123	1.0 (0.9–1.3)	0.5	127/607	0.9 (0.7–1.2)	139/628	0.9 (0.7–1.2)
–126	1.0 (0.8–1.3)	0.2	74/359	0.9 (0.7–1.2)	119/574	0.9 (0.7–1.2)
>126	1.0 (baseline)	0.0	37/242	0.6 (0.4–0.9)	107/476	1.0 (baseline)
HR per 10 cm	0.79 (0.70–0.89)			0.64 (0.54–0.76)		0.97 (0.82–1.16)
<i>P</i> for trend	<0.0001			<0.0001		0.8
<i>P</i> for interaction					0.001	

HR, hazard ratio; CI, confidence interval.

Hazard ratios for all causes of death according to height at age seven years and mother's height among men with birthweights above the median

TABLE 3

Height at age 7 (cm)	Mother's height (cm)			
	160		>160	
	Deaths/men	HR (95% CI)	Deaths/men	HR (95% CI)
114	40/114	1.4 (1.0–2.1)	3/23	0.6 (0.2–1.8)
–117	43/189	0.9 (0.6–1.3)	17/70	0.9 (0.5–1.5)
–120	65/283	0.9 (0.7–1.3)	38/203	0.7 (0.5–1.1)
–123	65/290	0.9 (0.7–1.3)	61/288	0.8 (0.6–1.2)
–126	40/195	0.9 (0.6–1.3)	69/325	0.9 (0.6–1.2)
>126	24/125	0.8 (0.5–1.3)	72/308	1.0 (baseline)
HR per 10 cm		0.77 (0.59–1.01)		1.32 (1.00–1.75)
P for trend		0.06		0.05
P for interaction			0.007	

HR, hazard ratio; CI, confidence interval.

TABLE 4

Hazard ratios for all causes of death according to height at age seven years after excluding men with birthweight and mother's height above the median

Height at age 7 (cm)	HR (95%CI)	Years of life lost
114	2.1 (1.5–2.9)	8.0
–117	1.5 (1.1–2.1)	4.7
–120	1.4 (1.1–1.9)	4.0
–123	1.4 (1.0–1.8)	3.2
–126	1.3 (0.9–1.8)	2.7
>126	1.0 (baseline)	0.0
HR per 10 cm	0.67 (0.58–0.78)	
<i>P</i> for trend	<0.0001	

HR, hazard ratio; CI, confidence interval.