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DETERMINANTS OF VARIATION IN ADULT BODY HEIGHT

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Summary. Final body height is achieved as the result of a combination of genetic and environmental factors. The aim of this article is to review past studies on body height that have followed different scientific traditions. In modern Western societies, about 20% of variation in body height is due to environmental variation. In poorer environments, this proportion is probably larger, with lower heritability of body height as well as larger socioeconomic body height differences. The role of childhood environment is seen in the increase in body height during the 20th century simultaneously with the increase in the standard of living. The most important non-genetic factors affecting growth and adult body height are nutrition and diseases. Short stature is associated with poorer education and lower social position in adulthood. This is mainly due to family background, but other environmental factors in childhood also contribute to this association. Body height is a good indicator of childhood living conditions, not only in developing countries but also in modern Western societies. Future studies combining different scientific traditions in auxology are needed to create a more holistic view of body height.

Introduction

When studying variation in adult body height, both systematic and unsystematic components are apparent, with most of the variation appearing to be unsystematic. Because short and tall people exist in all age and social groups, it is impossible to conjecture anything on an individual level based on stature. At a population level, however, clear systematic variation can be found. For example, today's adults are taller than their parents. As genetic factors are unlikely to explain this difference, the contribution of environmental factors needs to be explored. Body height may, in turn, yield information about the communities being studied and can be used as an indicator of early life experiences, including fetal conditions as well as living conditions in childhood (Nyström Peck & Lundberg, 1995; Silventoinen, 2000).

A specific feature of auxology is that it comprises several traditions that have limited interaction. The main underlying difference between these traditions is a

scientific background in social or natural sciences. However, the traditions have also differences in whether they stress systematic or unsystematic variation in body height and whether the scope of the research is at an individual or a population level. At least three traditions can be distinguished. First, an enormous number of studies exist where body height differences have been examined by socioeconomic factors. The theoretical background of this tradition is mainly in social sciences, and it stresses the systematic variation of body height. Typical of this tradition is that it largely ignores the interpretation of these differences, i.e. specific factors explaining the differences are not examined. Second, as body height is a good example of polygenetically heritable characteristics, it has been studied since the beginning of the 20th century in population genetic studies (Pearson & Lee, 1903). Many twin and family study designs have been used to estimate the relative proportions of genetic and environmental variation in body height. These studies have examined the unsystematic variation of body height but have usually not speculated on how the environmental part of the variation could explain the systematic differences found in society. Third, studies exist where the effects of specific environmental factors, such as nutrition and diseases, on growth have been investigated. These studies have mainly followed the biomedical tradition, disregarding all social factors. Contrary to the first and second traditions, the scope of this third tradition is at the individual level.

The aim of this review is to systematize current knowledge based on previous studies following the above three traditions and form a more coherent picture of the factors underlying the variation in body height. First, the kinds of systematic body height differences that exist in societies are reviewed. Then, earlier twin and family studies to estimate the relative proportions in the variation of body height due to genetic and environmental factors are reviewed. The most important factors explaining the environmental part of the variation and systematic body height differences in society, and how genetic factors may modify the effect of the environment on final body height, are then examined. Finally, how current knowledge of the effects of genetic and environmental factors helps us to understand variation in body height is summarized, and guidelines as to how these three traditions could be integrated in future studies are provided.

Systematic variation in body height

The most universal of the systematic features are a secular trend towards increased body height and socioeconomic variation in body height. During the 20th century average body height has increased throughout industrialized populations, including the Nordic countries (Brundtland, Liestøl & Wallø, 1975, 1980; Nyström Peck & Vågerö, 1987; Proos, 1993; Liestøl & Rosenberg, 1995; Silventoinen, 2000), other European countries (Terrenato & Ulizzi, 1983; Kuh, Power & Rodgers, 1991; Tanner, 1992; Weber *et al*., 1995; Hauspie, Vercauteren & Susanne, 1997; Hughes *et al*., 1997; Castro *et al*., 1998; Bielicki & Szklarska, 1999; Padez & Johnston, 1999), North America (Gordon-Larsen, Zemel & Johnston, 1997; Hoppa & Garlie, 1998; Freedman *et al*., 2000) and Japan (Murata & Hibi, 1992; Kouchi, 1996). A similar secular trend is also seen in poorer countries and populations such as South Africa, which includes people of both European and African ancestry (Henneberg & Berg, 1990), Brazil

(Monteiro, Benicio & Gouveia, 1994) and Canadian Inuits (Shephard & Rode, 1995). This increase has been continuous and regular throughout the 20th century, with the exception of a decreasing trend during the two World Wars (Tanner, 1992; Liestøl & Rosenberg, 1995).

Besides differences between birth cohorts, socioeconomic differences in body height are substantial; people in higher socioeconomic positions tend to be taller than those in lower positions. Marmot (1995) reported that among male and female London civil servants the average body height in the lowest position was about 5 cm shorter than among those in the highest position. Walker, Shaper & Wannamethee (1988), in turn, found a 6 cm difference between working-class and professional middle-aged British men. Similar associations have also been identified for body height and education in the Netherlands (Mackenbach, 1992), Norway (Meyer & Selmer, 1999), Finland (Silventoinen, Lahelma & Rahkonen, 1999) and Poland (Pawlowski, Dunbar & Lipowicz, 2000).

The socioeconomic differences in adulthood are probably mainly due to social background. Socioeconomic position of the family has been identified as being positively associated with the stature of children. The most frequently used indicator is father's social position, but the association is also found for father's and mother's education and family income in studies in developing (Singh & Harrison, 1997; Bégi, Frongillo & Delisle, 1998) as well as developed (Goldstein, 1971; Rona, Swan & Altman, 1978; Kuh & Wadsworth, 1989; Gulliford, Chinn & Rona, 1991; Cernerud & Elfving, 1995) countries. The social position of the family is likely to be associated with nutrition, especially when the average nutritional state of the population is poor. Mothers' education may also be associated with maternal care, as educated mothers are more aware of improved methods of care and better treatment of diseases. A finding in two studies in developing countries (Vella *et al.*, 1994; Liu, Jalil & Karlberg, 1998) indicates that mother's low education is a better predictor of stunted growth of a child than father's low education.

However, body height has also been found to be associated with social mobility, i.e. the difference between father's social class in childhood and one's own social class in adulthood. Several studies (Illsley, 1955; Bielicki & Charzewski, 1983; Bielicki & Waliszko, 1992; Nyström Peck, 1992; Bielicki & Szklarska, 1999; Blane, Davey Smith & Hart, 1999) have shown that tall people have a higher probability of being upwardly mobile. Similar associations have been found for education (Cernerud, 1995; Bielicki & Szklarska, 2000). Silventoinen, Kaprio & Lahelma (2000a) found that the association between body height and education in Finland is mainly due to factors associated with family background, but other environmental factors also have an effect. Interestingly, a positive correlation has also been demonstrated between body height and intelligence (Humphreys, Davey & Park, 1985; Teasdale, Owen & Sørensen, 1991) as well as cognitive function (Abbott *et al.*, 1998).

Differences in childhood living conditions probably explain most of the differences found between birth cohorts and social classes. The role of the increasing standard of living behind the secular trend in body height is strongly supported by it being closely aligned with an increasing trend in national gross product (Floud, 1985; Fogel, 1993; Harris, 1994; Komlos, 1995; Steckel, 1995), as well as a decreasing trend in infant mortality (Schmidt, Jørgensen & Michaelsen, 1995) and menarche age (Hauspie,

Vercauteren & Sussanne, 1996). Increased genetic mixing (van Wieringen, 1986) and imprinting during fetal life (Pembrey, 1996) have also been suggested to contribute to the secular trend in body height. However, little empirical evidence is available about the importance of these factors. Nevertheless, it seems obvious that the secular trend in body height is largely due to the dramatic improvement in the standard of living during the last century.

Further, differences in childhood conditions are likely to explain most of the socioeconomic differences in body height. Path analyses of Sudanese infants (Brush, Harrison & Zumrawi, 1993) revealed indirect effects of social factors on growth. Mothers' occupational status, income of the family and weaning age had effects on the health of infants, and thus on growth. Large family size and overcrowding have been found to be associated with short stature in both developed (Topp *et al.*, 1970; Rona *et al.*, 1978; Kuh & Wadsworth, 1989) and developing (Christiansen, Mora & Herrera, 1975; Liu *et al.*, 1998) countries, and these associations may partly explain the socioeconomic variation in body height. Interestingly, body height is reported to increase with parity, with the body height of later-born children being close to that of children in small families (Grant, 1962; Moyes, 1981). Just the birth of subsequent children appears to have a negative impact on the growth of a child. This suggests that in addition to a lower standard of living in large families and a higher rate of infections, maternal care in early childhood may be an important explanatory factor.

One might expect that as the overall standard of living has increased during the 20th century a decrease in environmental variation would occur. Some evidence does exist that body height differences associated with social background have diminished, while average body height has increased. In Denmark, Teasdale, Sørensen & Owen (1989) found that the association between body height and education had weakened after the Second World War with the concomitant rise in the standard of living. The narrowing trend can also be seen in Great Britain, where time series are available on body height differences between upper- and manual-class children from the beginning of the 19th century (Floud, Watcher & Gregory, 1990). The rate of this narrowing is likely to vary between countries. In Sweden, the difference by childhood socioeconomic group has been gradually disappearing since the Second World War (Nyström Peck & Vågerö, 1987), but a marked difference may still exist in other countries (Macintyre, 1988). In Europe, body height differences between countries have remained fairly similar (Schmidt *et al.*, 1995; Cavelaars *et al.*, 2000), although in one study (Silventoinen *et al.*, 2001b), a narrowing in the gap of mean body height between Finland and Sweden has been found.

Heritability of body height

Before specific factors underlying the systematic variation of body height can be examined, the proportion of genetic factors affecting the variation of body height must be studied, i.e. the heritability of body height. It is well known that human growth is a polygenic process. Numerous genes have been found to be associated with growth disorders, and it is likely that these genes also affect final body height at the population level (OMIM, 2001). Moreover, the sex chromosomes are assumed to have a crucial role in growth. The effect of the X chromosome can be observed in the

growth retardation in Turner's syndrome (Ellison *et al.*, 1997). The sex difference in mean body height, as well as the higher stature of XYY boys as compared with XY boys, further suggest that the Y chromosome may also include genes affecting body height (Ogata & Matsuo, 1992; Ratcliffe, Pan & McKie, 1992). A sex-related genetic factor for growth and body height has been found in previous studies (Phillips & Matheny, 1990; Byard, Guo & Roche, 1993), but inconsistent findings have also been reported (Byard, Siervogel & Roche, 1983; Dasgupta, Dasgupta & Daschaudhuri, 1997; Silventoinen *et al.*, 2001a).

Differences in growth curves have been found between ethnic groups, indicating the role of genetic factors (Eveleth & Tanner, 1976; Marshall, 1981; Eveleth, 1986; Rona & Chinn, 1986; Ulijaszek, 1994). African-origin populations in Europe and the USA reach a higher final body height than Caucasian populations when body height is adjusted for social position. In contrast, the mean stature of the Asian population has been found to be lower than that of the Caucasian population, possibly associated with the earlier occurrence of the adolescent growth spurt in the former (Murata & Hibi, 1992). In meta-analyses of European and US growth studies, Hermanussen *et al.* (1998) found no major differences in growth patterns between populations in these countries and that the patterns have remained fairly similar during the 20th century despite a striking increase in mean body height.

The heritability of body height can be estimated by using twin or other suitable family data sets. Table 1 summarizes the results of previous twin studies. The heritability estimates have been re-computed from the correlations by using linear structural equations (Neale & Cardon, 1992). A close review of the previous studies revealed, however, many problems in data collection. Clearly unacceptable data collection, i.e. advertisements in newspapers and on television, was used in two studies (Shields, 1962; Huntley, 1965). In most of the other studies, problems centred around creating a sample frame, and only four studies (Husén, 1959; Stunkard, Foch & Hrubec, 1986; Silventoinen *et al.*, 2000b, 2001a) gave a response rate. The lack of a sample frame does not necessarily mean poor data quality. For instance, if all families in a certain geographical area are taken into account, the sample may not be biased. However, in most of the studies, the sample protocol was not fully described, and thus it is not possible to appreciate the quality of the data. Especially suspicious is that in many studies the number of monozygotic (MZ) twins is larger than the number of dizygotic (DZ) twins. This shows that the response rates have been higher among MZ twins and suggests that the response rates among DZ twins may be biased. If, for example, the most similar of DZ twins responded, the DZ correlation would be higher than it should be, and thus heritability would be underestimated.

The first accurate twin study was done for Swedish conscripts (Husén 1959), and the heritability estimate in this study was 0·60 (95% CI 0·50–0·71). A higher heritability was found for US men (0·80, 95% CI 0·74–0·86) by Stunkard *et al.* (1986) and for Finnish men (0·77, 95% CI 0·73–0·82) and women (0·76, 95% CI 0·71–0·80) by Silventoinen *et al.* (2000b). Heritability estimates were slightly lower in another Finnish study for adolescents 18 years of age (0·76, 95% CI 0·63–0·90 in men; 0·64, 95% CI 0·52–0·77 in women) (Silventoinen *et al.* 2001a). If the results of these four studies are compared with the other studies in Table 1, no systematic differences are present. Heritability estimates in most of the studies have varied between 0·4 and 0·9.

Table 1. Correlations within twin pairs and heritability estimates of body height in twin studies

Country	Year of data collection	Age (years)	Sex/type	*N*	*r*	h^2 (95% CI)
Sweden[a] (Dahlberg, 1926)	1923	3–80	Male MZ	44	0·99	0·16
			Male DZ	37	0·91	(0·09–0·26)
			Female MZ	49	0·99	0·40
			Female DZ	54	0·79	(0·26–0·58)
Great Britain[b] (London; Stocks, 1932)	1925–1927	3–15	MZ	55	0·93	0·88
			DZ[c]	51	0·49	(0·53–1·00)
USA[a] (Chicago; Newman *et al*., 1937)	1926	8–18	MZ	50	0·93	0·56
			DZ[c]	50	0·65	(0·32-0·88)
Sweden (Husén, 1959)	1948–1952	19–21	Male MZ	266	0·89	0·60
			Male DZ	510	0·59	(0·50–0·71)
Great Britain (Shields, 1962)	1954–1958	9–59	Female MZ	29	0·94	0·94
			Female DZ	16	0·44	(0·61–1·00)
Finland (Takkunen, 1964)	1957	37–46	Male MZ	75	0·90	0·40
			Male DZ	74	0·70	(0·22–0·63)
Great Britain (Huntley, 1965)	—	5–15	MZ	85	0·90	0·66
			DZ[c]	135	0·57	(0·47–0·89)
Japan (Furusho, 1969)	1952–1969	17	Male MZ	51	0·92	0·18
			Male DZ	9	0·83	(0·01–0·68)
			Female MZ	51	0·95	0·26
			Female DZ	11	0·82	(0·04–0·67)
USA (NE Georgia; Osborne 1970)	—	11–22	MZ	13	0·88	0·40
			DZ[c]	6	0·68	(0·01–1·00)
Brazil (Da Rocha *et al*. 1972)	—	13–26	Male MZ	21	0·97	0·97
			Male DZ	22	0·42	(0·65–1·00)
			Female MZ	27	0·98	0·70
			Female DZ	29	0·63	(0·37–1·00)
Sweden (Fischbein, 1977)	1966	18	Male MZ	26	0·93	0·78
			Male DZ	39	0·54	0·42–1·00
		16	Female MZ	21	0·94	0·40
			Female DZ	35	0·74	0·18–0·70
Great Britain (Hawk & Brook, 1979)	—	Adults	Male MZ	29	0·94	0·74
			Male DZ	46	0·57	(0·43–1·00)
			Female MZ	20	0·86	0·86
			Female DZ	45	0·37	(0·60–1·00)
India[b] (Punjab; Sharma *et al*. 1984)	—	3–19	MZ	88	0·98	0·78
			DZ	200	0·59	(0·63–0·95)
India[b] (Andra Pradesh; Byard *et al*. 1985)	—	—	MZ	44	0·95	0·54
			DZ	64	0·68	(0·34–0·79)
USA (Stunkard *et al*. 1986)	1974	47–57	Male MZ	1974	0·91	0·80
			Male DZ	2097	0·51	(0·74–0·86)

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Table 1. Continued

Country	Year of data collection	Age (years)	Sex/type	*N*	*r*	h^2 (95% CI)
China (Wang *et al.* 1990)	1989	7–12	MZ	75	0·95	0·58
			DZ	35	0·66	(0·30–0·95)
Great Britain (Hewitt *et al.* 1991)	—	6–24	Male MZ	40	0·82	0·10
			Male DZ	40	0·77	(0·01–0·52)
Norway[b] (Tambs *et al.* 1992)	1984–1986	20+	Male MZ	41	0·90	0·64
			Male DZ	48	0·58	(0·34–1·00)
			Female MZ	42	0·86	0·86
			Female DZ	44	0·34	(0·63–1·00)
USA (Minnesota; Castro, 1993)	—	27–50	MZ	109	0·96	0·34
			DZ[c]	86	0·79	(0·22–0·48)
Japan (Ooki & Asaka, 1993)	1982–1991	11	MZ	220	0·94	0·84
			DZ[c]	57	0·52	(0·53–1·00)
Poland (Hauspie *et al.* 1994)	1967	18	Male MZ	74	0·96	0·54
			Male DZ	74	0·69	(0·36–0·76)
USA (Minnesota; Carmichael & McGue, 1995)	—	18–38	Male MZ	121	0·86	0·86
			Male DZ	84	0·36	(0·70–1·00)
			Female MZ	243	0·92	0·92
			Female DZ	162	0·46	(0·81–1·00)
		39–59	Male MZ	46	0·94	0·94
			Male DZ	42	0·26	(0·71–1·00)
			Female MZ	97	0·90	0·90
			Female DZ	97	0·42	(0·74–1·00)
		60–81	Male MZ	29	0·88	0·88
			Male DZ	35	0·37	(0·61–1·00)
			Female MZ	50	0·80	0·80
			Female DZ	27	0·33	(0·55–1·00)
Finland[a] (Silventoinen *et al.* 2000b)	1981	23+	Male MZ	1561	0·91	0·77
			Male DZ	3592	0·51	(0·73–0·82)
			Female MZ	1905	0·87	0·76
			Female DZ	3858	0·50	(0·71–0·80)
Finland (Silventoinen *et al.* 2001a)	1993–1997	18	Male MZ	303	0·90	0·76
			Male DZ	385	0·52	(0·63–0·90)
			Female MZ	476	0·88	0·64
			Female DZ	399	0·56	(0·52–0·77)

[a]Adjusted for age; [b]adjusted for age and sex; [c]same-sex twins only.

Table 2 summarizes the sibling correlations of body height in previous family studies. The earliest estimates are by Pearson & Lee (1903), who report on family data from Great Britain. They found that brother, sister and brother–sister correlations were around 0·5. The heritability for body height in this data set was later estimated to be 0·79 (Crow & Kimura, 1970). Other correlation estimates for siblings based on relatively large data sets were from the United States (0·34; Garn, Cole & Bailey,

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Table 2. Correlations within siblings and heritability estimates of body height in family studies

Country	Year of data collection	Age (years)	Type	*n*	*r*	h^2
Great Britain (Pearson & Lee, 1903)	—	18+	Brother–brother	328	0·51	0·79
			Sister–sister	473	0·54	
			Brother–sister	1401	0·51	
Great Britain[a] (Hewitt, 1957)	—	1–5	Brother–brother	23	0·37	
			Sister–sister	27	0·72	
			Brother–sister	33	0·70	
France (Schreider, 1961)	—	Adults	Brother–brother	32	0·35	
Great Britain[b] (Wales; Acheson & Fowler, 1967)	1954, 1956	—	Brother–brother	108	0·56	
			Sister–sister	113	0·34	
Malesia (McHenry & Giles, 1971)	1963, 1968	Adults	Sibling–sibling	171	0·35	
United States (Garn *et al*., 1973)	—	10	Brother–brother	60	0·45	
			Sister–sister	40	0·71	
			Brother–sister	90	0·47	
Guatemala[a] (Russell, 1976)	1959–1964	3	Brother–brother	88	0·53	
			Sister–sister	93	0·44	
			Brother–sister	177	0·45	
Columbia (Mueller, 1977)	1973	Adults	Brother–brother	50	0·63	
			Sister–sister	34	0·36	
			Brother–sister	50	0·42	
Africa[b] (West; Roberts *et al*., 1978)	1949–1975	16–19	Brother–brother	114	0·43	
			Sister–sister	115	0·33	
USA (Garn *et al*., 1979)	1969–1970	—	Sibling–sibling	8860	0·34	
Canada (Montreal; Bouchard, 1980)	—	10	Sibling–sibling	208	0·53	
USA (Philadelphia; Mueller & Malina, 1980)	—	6–12	Brother–brother	52	0·52	
			Sister–sister	49	0·49	
			Brother–sister	109	0·46	
India (Kaur & Singh, 1981)	—	18–39	Brother–brother	42	0·24	
			Sister–sister	32	0·24	0·55
			Brother–sister	84	0·23	
Finland (Solomon & Thompson, 1983)	1973–1975	—	Sibling–sibling	477	0·36	
India[b] (Punjab; Sharma *et al*., 1984)	—	3–19	Sibling–sibling	151	0·30	
India[b] (Andra Pradesh; Byard *et al*., 1985)	—	Adults	Sibling–sibling	113	0·46	
Brazil[b] (Province & Rao, 1985)	—	—	Sibling–sibling	2789	0·35	
Caribea[a] (St Vincent Island; Hutchinson & Byard, 1987)	1979, 1982	18+	Brother–brother	22	0·28	
			Sister–sister	82	0·42	
			Brother–sister	82	0·41	
USA (Fels; Byard *et al*., 1988)	1986	18	Brother–brother	60	0·33	
			Sister–sister	128	0·47	
			Brother–sister	128	0·37	

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Table 2. Continued

Country	Year of data collection	Age (years)	Type	*n*	*r*	h^2
Mexico[a] (Little *et al.*, 1990)	1978	6–13	Brother–brother	44	0·36	
			Sister–sister	44	0·34	
			Brother–sister	110	0·11	
Norway[b] (Tambs *et al.*, 1992)	1984–1986	20+	Brother–brother	6016	0·47	
			Sister–sister	3869	0·46	
			Brother–sister	9283	0·44	
Spain[a] (Rebato *et al.*, 1997)	—	15+	Sibling–sibling	238	0·48	

[a]Adjusted for age; [b]adjusted for age and sex.

1979), Brazil (0·35; Province & Rao, 1985) and Norway (about 0·46; Tambs *et al.*, 1992). In Norwegian data, sibling correlations for brother–sister pairs were slightly lower than for same-sex pairs. In other studies, no systematic differences in body height correlations were found between brothers, sisters or opposite-sex pairs. This suggests that no sex-related genetic factors are present in the variation in body height. Thus, the genes in the Y chromosome may only increase the mean body height, while not affecting the variation in body height. Sibling correlations in Table 2 were slightly lower than DZ correlations in Table 1, possibly indicating an inadequate age-adjustment in many familial studies or the greater influence of a shared environment among twins.

Table 3 presents body height correlations in published adoption studies. Correlations between biological relatives were invariably much higher than correlations in foster families, indicating a strong genetic component in body height. However, these studies were based on small data sets, and thus give little information on the heritability of body height.

In sum, previous studies have suggested that the heritability of body height is about 0·8, although lower estimates also exist. However, despite extensive study, only a few twin studies can be found which contain no problems in the data sample.

It is likely that the heritability of body height is not a constant factor. In a meta-analysis, Mueller (1976) compared parent–child correlations for body height in 24 studies. He found that correlations were higher in European than non-European countries, probably due to differences in environmental factors. Lauderdale & Rathouz (1999) found that among brothers mustered into the Union Army of the US Civil War the correlation decreased and variation increased with increasing county population. A negative correlation was found between mean body height and county population, suggesting that environmental variation is larger in poorer environments. The lower heritability of body height in poor environments is also suggested by Silventoinen *et al.* (2000b), who found that the heritability of body height increased in Finland during the first half of the 20th century simultaneously with the improvement in the standard of living. In Tables 1–3, no systematic differences in correlations between birth cohorts or countries were seen. However, this may be due to poor data quality in many of these studies.

Table 3. Familial correlations of body height in adoption studies

Country	Year of data collection	Age (years)	Type	*n*	*r*
USA[a] (Newman *et al.*, 1937)	—	—	MZ apart	19	0·89
USA (Shields, 1962)	1954–1958	8–59	Male MZ apart	15	0·82
			Female MZ apart	29	0·82
USA[a] (Michigan; Garn *et al.*, 1976)	1959–1969	15–18	B father–son	206	0·27
			A father–son	26	−0·18
			B mother–son	238	0·43
			A mother–son	15	0·10
			B father–daughter	203	0·44
			A father–daughter	38	0·39
			B mother–daughter	247	0·49
			A mother–daughter	21	−0·07
Canada[b] (Annest *et al.*, 1983)	1972–1974	1–10	B father–child	127	0·34
			A father–child	334	0·10
			B mother–child	127	0·43
			A mother–child	334	0·08
			A sibling–A sibling	119	0·08
			A sibling–B sibling	54	0·16
			B sibling–B sibling	47	0·37
Denmark (Teasdale & Owen, 1984)	1956–1965	18	Full siblings apart	28	0·27
			Half-siblings apart	64	0·20
			Unrelated reared together	24	0·03
Finland (Langinvainio *et al.*, 1984)	1975, 1979	—	Male MZ apart	13	0·89
			Male DZ apart	33	0·34
			Female MZ apart	16	0·71
			Female DZ apart	54	0·17
Sweden[a] (Pedersen *et al.*, 1984)	1961, 1973	—	MZ apart	111	0·82
			DZ apart	278	0·62

A=adoptee relationship; B=biological relationship.
[a]Adjusted for age.

Effect of environmental factors

Nutrition and diseases in childhood are usually regarded as the main factors affecting body height. The effect of undernutrition on body height may already start during fetal life. In a clinical trial in East Java, pregnant women received either high or low caloric supplementation during the last trimester of pregnancy. Newborns were heavier (Kardjati, Kusin & De With, 1988) among the women with the high caloric supplement than among those with the low caloric supplement, and they were taller by the end of a 5-year follow-up (Kusin *et al.*, 1992). In contrast, Stanner *et al.* (1997) did not find that adult body height of those who had been exposed to intrauterine starvation during the siege of Leningrad in 1941–1944 was any shorter than that of an unexposed control group. In a Dutch Famine Study (Susser & Stein, 1994), an

increasing trend in body height was found for 19 year olds from the cohort born one year before the famine as compared with that born one year after the famine, suggesting that body height is responsive to the postnatal environment. However, no differences were found between those who had been exposed to famine during pregnancy and the others, indicating that the prenatal environment has no obvious effect on adult body height.

The role of postnatal nutrition in growth is well documented: for example, in studies of supplementary programmes in developing countries (Beaton & Ghassemi, 1982). Among single nutrients affecting growth, protein is probably the most important. Many experimental studies have investigated the effect of protein on growth (Zerfas, 1986; Allen, 1994), the first being conducted as early as 1928 (Orr; 1928; Leighton & Clark, 1929), when supplementary milk was found to be associated with higher growth velocity among schoolchildren in England. In developing countries, protein deficiency is recognized to be one of the main contributors to the stunting of growth in infants (Martorell & Habicht, 1986).

Minerals and vitamins A and D may also influence growth. Numerous intervention studies have established that calcium, phosphorus, magnesium, zinc and iron have an effect on human growth (Allen, 1994; Prentice & Bates, 1994). However, in most cases, mineral amounts are so close to biological requirements, even in developing countries, that any supplementary minerals have only a minor impact on growth. Vitamin D is essential for the absorption of calcium, and thus its deficiency does have an effect on the mineralization of bones (Johnson & Kumar, 1994; Wasserman & Fullmer, 1995), an especially difficult problem in northern regions due to a shorter light period in winter-time (Belton, 1986). While the impact of vitamin A on growth based on supplementary studies is unclear, and some negative results have been found, vitamin A deficiency is a major health problem in many developing countries (Humphreys *et al.*, 1985; Allen, 1994; Fawzi *et al.*, 1997; Smith *et al.*, 1999; Hadi *et al.*, 2000).

Besides nutrition, childhood diseases are suggested to have an effect on growth. In many cases, the effect of disease is intertwined with that of nutrition. Disease can prevent food intake, impair nutrient absorption, cause direct nutrient losses, increase metabolic requirements or catabolic losses and impair transport to target issues (Stephensen, 1999). The associations between undernutrition and disease are likely to be bidirectional; thus, diseases not only affect nutrition, but undernutrition also predisposes to diseases (Victora *et al.*, 1990; Walter *et al.*, 1997). Moreover, some medical treatments of diseases, such as corticosteroid use, retard growth (Pedersen, 2001). Inflammatory diseases have also been found to hinder the growth of long bones (Skerry, 1994).

In developing countries, clear evidence exists about the effect of diseases on human growth. While the impact of diarrhoea on slowed growth has frequently been studied, pneumonia has been found to have similar effects on growth (Martorell *et al.*, 1975; Rowland, Cole & Whitehead, 1977; Victora *et al.*, 1990; Brush, Harrison & Waterlow, 1997). Both diseases seem to be associated with nutrition. An interaction is clearly seen in a food supplementation study in Guatemala, where a larger benefit was found among the children with a higher rate of diarrhoea (Ruel *et al.*, 1995). Decreased food intake is found in conjunction with diarrhoea as well

as with other diseases (Martorell *et al.*, 1980; Brown *et al.*, 1990). This decreased food intake may partly be due to apathy, but some traditional treatment methods also encourage a decrease in nutrition during illness, especially in the case of diarrhoea (Mata, 1992).

In developed countries, the association between diseases and growth is not as strong as in developing countries. The prevalence of serious child diseases is much lower than in developing countries and treatment methods are more advanced. Furthermore, nutritional stress is rare and is not associated with diseases, unlike in developing countries, where the poor economic situation and inadequate nutrition are risk factors for disease (Brush, Harrison & Waterlow, 1993). Thus, the synergetic effect of poor nutrition and disease is not as pronounced in developed countries.

In developed countries, the impact of asthma and diabetes on growth has been well studied. Hauspie, Vercauteren & Susanne (1976) found that growth velocity was somewhat slower among asthmatic children, but this gap disappeared in puberty. In recent studies (Power & Manor, 1995; Neville *et al.*, 1996; McCowan *et al.*, 1998), weaker evidence has been found in support of an association between asthma and slowed growth. Some evidence exists that diabetes could be associated with a lower growth rate (Tattersall & Pyke, 1973; Herber & Dunsmore, 1988; Thon *et al.*, 1992; Wise, Kolbe & Sauder *et al.*, 1992). However, other studies have found no growth delay or shorter stature to be present among diabetic children (Birkbeck, 1972; Clarson, Daneman & Ehrlich, 1985; Caju, Rooman & Beeck, 1995).

Chronic diseases, including severe conditions such as congenital heart disease, are likely to have an influence on growth as well as on final body height (Poskitt, 1993). However, the prevalence of such diseases is so low that the effects of single diseases are difficult to study. Kuh & Wadsworth (1989) found in Great Britain that those who had had a serious disease in childhood were shorter than others. The difference was about 2 cm for both men and women. Power & Manor (1995) found a similar 2 cm difference between children who had experienced a serious disease and other children in a longitudinal study in Britain.

To summarize, differences in protein input are important when explaining environmental variation in body height in developing countries. The effect of diseases on growth is also strong in developing countries, where diseases are typically associated with nutritional deprivation. A major factor in the delay of growth is the synergetic effect between poor nutrition and disease. In developed countries, nutritional stress is not common and only the most serious diseases have a clear impact on growth. Asthma and diabetes are not likely to be major factors affecting environmental variation in body height.

Gene–environment interaction

Genetic and environmental factors do not act independently of each other, but rather the genotype determines the response to the environment. This phenomenon is called gene–environment interaction. In practice, gene–environment interaction is seen in certain persons being genetically more sensitive to the effects of environment than others (Plomin, DeFries & McClearn, 1997).

Some evidence has been found for the effect of a sex–environment interaction on growth (Stinson, 1985). Better buffering of women has been reported for stress caused by many different factors such as nutrition (Stini, 1969), climate (Johnston, Borden & MacVean, 1977) and psychosocial stress (Rudolf & Hochberg, 1990). Kuh *et al.* (1991) have also found that body height has increased more rapidly among men than women in England and Wales during the 20th century, which may indicate a stronger impact of environmental factors on men at the beginning of the century. On the other hand, Silventoinen *et al.* (2000b) found that heritability of body height increased in Finland more among women than men during the first half of the 20th century, contradicting the hypothesis of women having better resistance against environmental factors.

The nature of the sex–environment interaction of body height is poorly understood. However, parallel results have been obtained on sex differences in the state of food deprivation. Women are more resistant against starvation, as seen in the reduced loss of proteins and lower mortality. These differences probably result from differences in metabolism. Possibly, the difference in buffering between the sexes is due to different selection mechanisms that have evolved among men and women (Hoyenga & Hoyenga, 1982). Nevertheless, most of the results are based on studies of adults, and it is difficult to see how the sex differences manifest before puberty, when only minor differences in body composition exist between boys and girls (Forbes, 1986).

Concluding remarks

While variation in body height is mainly due to genetic factors, environmental factors also have a substantial effect. The environmental part of the variation is important as it is likely to explain systematic body height differences in society. According to previous studies, about 20% of the variation in body height is due to environmental factors in the United States (Stunkard *et al.*, 1986) and Finland (Silventoinen *et al.*, 2000b) after the Second World War. The proportion of the variation due to genetics seems to be less important when environmental stress is strong. This is supported by socioeconomic body height differences diminishing during the 20th century. However, at least as interesting is that the differences between the proportions of genetic factors are not very large when they are compared with the dramatic increase in mean body height. This suggests that body height is very sensitive to environmental factors, and this effect is not only seen when studying stunted growth but is also evident in the tallest individuals.

Nutrition and diseases are universally the most important environmental factors affecting growth and subsequent adult body height. In developing countries, the influence of nutrition is well documented. The lack of protein is a particularly important factor explaining slow growth rate and is also likely to explain systematic body height differences in final body height. Diseases in developing countries are often associated with nutritional deprivation. A major factor in the delay of growth would thus be the synergetic association between poor nutrition and disease.

In developed countries, the influence of environmental factors on growth is less obvious, as the general standard of living is higher. However, similar to developing countries, the social environment during childhood in developed countries has

contributed to adult body height. This is seen in the relative proportion of environmental variation in the total variation of body height. This suggests that such factors as overcrowding or an imbalanced diet are problems in affluent societies as well. Thus, body height can also be used as an indicator of childhood living conditions in affluent societies. However, environmental factors that are not associated with family background are also present. The results of large American and Finnish twin studies (Stunkard *et al.*, 1986; Silventoinen *et al.*, 2000b) reveal that in addition to family background non-familial environmental factors have substantial effects on body height. A challenge for future studies is to determine which factors underlie this non-familial environmental variation.

Body height is associated with social position in adult life. An obvious explanation is that these associations reflect the role of the childhood environment. However, the associations between body height and adulthood should be interpreted with caution. The association between body height and social position is mainly, although not fully, due to social background. Childhood family circumstances affect both achieved body height and adult social position. However, environmental factors which are not associated with family background, as seen in the association between social mobility and body height, also exist. This suggests that a gene–environment correlation is present in the association between social class and body height. Thus, distinguishing between genetic and environmental factors may be more difficult than previously expected.

Guidelines for future research

Despite extensive study over the last century, the need for further knowledge on determinants of adult body height remains. In a recent review, Rona (2000) emphasized that a large portion of current auxological studies is based on aggregate data, which provide limited evidence for causation. Therefore, more follow-up studies are needed, with specific data on environmental factors such as nutrition. Studies based on good twin data sets are also lacking. However, in addition to better data sets and more sophisticated study designs, there is also a need for theoretically more coherent studies that combine the different traditions.

Potential studies might include examining how differences in specific environmental factors, such as nutrition and diseases, explain socioeconomic variation in body height. While the general conclusion that socioeconomic body height differences describe deprivation in childhood may be correct, it is only marginally relevant for practical interventions. It is also possible that the role of specific factors varies between societies. On the other hand, if the socioeconomic body height differences in a certain region were known to be mainly due to a lack of protein, this information could be used when considering supplementary food programmes.

A second important focal area is the interaction and correlation between genetic and environmental factors. Genetic factors are known to have an effect on human susceptibility to environmental stress. In addition, some studies exist on the different effects of environmental stress on the sexes, but the results are controversial. A future study might focus on the differences in the heritability of body height in different social classes. These studies are needed to analyse gene–environment correlations in the heritability of body height. Adaptation studies are also important, as they would

provide the opportunity to study the interaction between genetic and environmental factors. These studies could shed new light on the interactions between genetic, social and environmental factors.

The current diversity in the traditions of auxology should not, however, be regarded as a problem. Rather, it underlines what a multi-faceted phenomenon body height is. Body height can be approached from a social or a biological point of view and can be studied at either the individual or societal level. In the future, combining of these separate traditions would yield a more holistic view of body height and may also improve dialogue between natural and social sciences.

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References

Abbott, R. D., Ross, W., Petrovitch, H., Masaki, K. H., Snowdon, D. A. & Curb, J. D. (1998) Height as a marker of childhood development and late-life cognitive function: the Honolulu–Asia Aging Study. *Pediatrics* **102**, 602–609.

Acheson, R. M. & Fowler, G. B. (1967) On the inheritance of stature and blood pressure. *J. chronic. Dis.* **20**, 731–745.

Allen, L. H. (1994) Nutritional influences on linear growth: a general review. *Eur. J. clin. Nutr.* **48**, S75–89.

Annest, J. L., Sing, C. F., Biron, P. & Mongeau, J. G. (1983) Familial aggregation of blood pressure and weight in adoptive families. *Am. J. Epidemiol.* **117**, 492–506.

Beaton, G. H. & Ghassemi, H. (1982) Supplementary feeding programs for young children in developed countries. *Am. J. clin. Nutr.* **35**, 864–916.

Bégin, F., Frongillo, E. A. & Delisle, H. (1998) Caregiver behaviors and resources influence child height-for-age in rural *Chad. J. Nutr.* **129**, 680–686.

Belton, N. R. (1986) Rickets – not only the 'English Disease'. *Acta paediatr. scand. suppl.* **323**, 68–75.

Bielicki, T. & Charzewski, J. (1983) Body height and upward social mobility. *Ann. hum. Biol.* **10**, 403–408.

Bielicki, T. & Szklarska, A. (1999) Secular trends in stature in Poland: national and social class-specific. *Ann. hum. Biol.* **26**, 251–258.

Bielicki, T. & Szklarska, A. (2000) Are social-class differences in stature partly genetic? A hypothesis revisited. *Am. J. hum. Biol.* **12**, 97–101.

Bielicki, T. & Waliszko, H. (1992) Stature, upward social mobility and the nature of statural differences between social classes. *Ann. hum. Biol.* **19**, 589–593.

Birkbeck, J. A. (1972) Growth in juvenile diabetes mellitus. *Diabetologia* **8**, 221–224.

Blane, D., Davey Smith, G. & Hart, C. (1999) Some social and physical correlates of intergenerational social mobility: evidence from the West of Scotland Collaborative Study. *Sociology* **33**, 169–183.

Bouchard, C. (1980) Transient environmental effects detected in sibling correlations. *Ann. hum. Biol.* **7**, 89–92.

Brown, K. H., Stallings, R. Y., de Kanashiro, H. C., de Romaña, G. L. & Black, R. E. (1990) Effects of common illnesses on infants' energy intakes from breast milk and other foods during longitudinal community-based studies in Huascar (Lima), Peru. *Am. J. clin. Nutr.* **52**, 1005–1013.

Brundtland, G. H., Liestøl, K. & Walløe, L. (1975) Height and weight of school children and adolescent girls and boys in Oslo 1970. *Acta paediatr. scand.* **64**, 565–573.

Brundtland, G. H., Liestøl, K. & Walløe, L. (1980) Height, weight and menarcheal age of Oslo schoolchildren during the last 60 years. *Ann. hum. Biol.* **7**, 307–322.

Brush, G., Harrison, G. A. & Waterlow, J. C. (1997) Effects of early disease on later growth and early growth on later disease, in Khartoum infants. *Ann. hum. Biol.* **24**, 187–195.

Brush, G., Harrison, G. A. & Zumrawi, F. Y. (1993) A path analysis of some determinants of infant growth in Khartoum. *Ann. hum. Biol.* **20**, 381–387.

Byard, P. J., Guo, S. & Roche, A. F. (1993) Family resemblance for Preece–Baines growth curve parameters in the Fels Longitudinal Growth Study. *Am. J. hum. Biol.* **5**, 151–157.

Byard, P. J., Poosha, D. V. R. & Satyanarayana, M. (1985) Genetic and environmental determinants of height and weight in families from Andhra Pradesh, India. *Hum Biol.* **57**, 621–633.

Byard, P. J., Siervogel, R. M. & Roche, A. F. (1983) Sibling correlations for weight/stature^2 and calf circumference: age changes and possible sex linkage. *Hum. Biol.* **55**, 677–685.

Byard, P. J., Siervogel, R. M. & Roche, A. F. (1988) Age trends in transmissible and non-transmissible components of family resemblance for stature. *Ann. hum. Biol.* **15**, 111–118.

Caju, M. V. L., Rooman, R. P. & Beeck, L. (1995) Longitudinal data on growth and final height in diabetic children. *Pediatr. Res.* **38**, 607–611.

Carmichael, C. M. & McGue, M. (1995) A cross-sectional examination of height, weight and body mass index in adults twins. *J. Gerontol.* **50A**, B237–244.

Castro, J. J., Dias, J. A., Baptista, F., Costa, J. E., Calvão-Teles, A. & Camilo-Alves, A. (1998) Secular trends of weight, height and obesity in cohorts of young Portuguese males in the district of Lisbon: 1969 to 1990. *Eur. J. Epidemiol.* **14**, 299–303.

Castro, J. M. (1993) Genetic influences on daily intake and meal patterns of humans. *Physiol. Behav.* **53**, 777–782.

Cavelaars, A. E. J. M., Kunst, A. E., Geurts, J. J. M., Crialesi, R., *et al.* (2000) Persistent variations in average height between countries and between socio-economic groups: an overview of 10 European countries. *Ann. hum. Biol.* **27**, 407–421.

Cernerud, L. (1995) Height and social mobility. *Scand. J. social Med.* **23**, 28–31.

Cernerud, L. & Elfving, J. (1995) Social inequality in height. *Scand. J. social Med.* **23**, 23–27.

Christiansen, N., Mora, J. O. & Herrera, M. G. (1975) Family social characteristics related to physical growth of young children. *Br. J. prev. soc. Med.* **29**, 121–130.

Clarson, C., Daneman, D. & Ehrlich, R. M. (1985) The relationship of metabolic control to growth and pubertal development in children with insulin-dependent diabetes. *Diabetes Res.* **2**, 237–241.

Crow, J. F. & Kimura, M. (1970) *An Introduction to Population Genetics Theory*. Harper and Row, New York.

Da Rocha, F. J., Salzano, F. M., Pena, H. F. & Callegari, S. M. (1972) New studies on the heritability of anthropometric characteristics as ascertained from twins. *Acta genet. med. gemell.* **21**, 125–134.

Dahlberg, G. (1926) *Twin Births and Twins from a Hereditary Point of View*. Tidens Tryckeri, Stockholm.

Dasgupta, I., Dasgupta, P. & Daschaudhuri, A. B. (1997) Familial resemblance in height and weight in an endogamous Mahisya caste population of rural West Bengal. *Am. J. hum. Biol.* **9**, 7–9.

Ellison, J. W., Wardak, Z., Young, M. F., Robey, P. G., Laig-Webster, M. & Chiong, W. (1997) PHOG, a candidate gene for involvement in the short stature of Turner syndrome. *Hum. mol. Genet.* **6**, 1341–1347.

Eveleth, P. & Tanner, J. (1976) *Worldwide Variation of Human Growth*. Cambridge University Press, Cambridge.

Eveleth, P. B. (1986) Population differences in growth: environmental and genetic factors. In: *Human Growth*, vol. 3, *Methodology, Ecological, Genetic and Nutritional Effects on Growth*, 2nd edn, pp. 221–239. Edited by F. Falkner & J. M. Tanner. Plenum Press, New York.

Fawzi, W. W., Herrera, M. G., Willett, W. C., Nestel, P., Amin, A. E. & Mohamed, K. A. (1997) The effect of vitamin A supplementation on the growth of preschool children in the Sudan. *Am. J. publ Hlth* **87**, 1359–1362.

Fischbein, S. (1977) Intra-pair similarity in physical growth of monozygotic and of dizygotic twins during puberty. *Ann. hum. Biol.* **4**, 417–430.

Floud, R., Wachter, K. & Gregory, A. (1990) *Height, Health and History. Nutritional Status in the United Kingdom, 1750–1980*. Cambridge University Press, Cambridge.

Floud, R. C. (1985) Measuring the transformation of the European economies: income, health and welfare. *Hist. soc. Res.* **33**, 25–41.

Fogel, R. W. (1993) New sources and new techniques for the study of secular trends in nutritional status, health, mortality and the process of aging. *Hist. Meth.* **26**, 5–43.

Forbes, G. B. (1986) Body consumption in adolescence. In: *Human Growth*, vol. 2, *Postnatal Growth, Neurobiology*, 2nd edn, pp. 119–145. Edited by F. Falkner & J. M. Tanner. Plenum Press, New York.

Freedman, D. S., Khan, L. K., Serdula, M. K., Srinivasan, S. R. & Berenson, G. S. (2000) Secular trends in height among children during 2 decades. *Arch. pediatr. adolesc. Med.* **154**, 155–161.

Furusho, T. (1969) On the manifestation of genotypes responsible for stature. *Hum. Biol.* **40**, 437–455.

Garn, S. M., Bailey, S. M. & Cole, P. E. (1976) Similarities between parents and their adopted children. *Am. J. phys. Anthrop.* **45**, 539–544.

Garn, S. M., Cole, P. E. & Bailey, S. M. (1979) Living together as a factor in family-line resemblances. *Hum. Biol.* **51**, 565–587.

Garn, S. M., Hertzog, K. P. & Rohmann, C. G. (1973) Evidence for X-linkage of tibial length and body length. *Am. J. phys. Anthrop.* **31**, 187–190.

Goldstein, H. (1971) Factors influencing the height of seven year old children – results from the national child development study. *Hum. Biol.* **43**, 92–111.

Gordon-Larsen, P., Zemel, B. S. & Johnston, F. E. (1997) Secular changes in stature, weight, fatness, overweight and obesity in urban African American adolescents from the mid-1950's to the mid-1990's. *Am. J. hum. Biol.* **9**, 675–688.

Grant, M. W. (1962) Rate of growth in relation to birth rank and family size. *Br. J. prev. soc. Med.* **18**, 35–42.

Gulliford, M. C., Chinn, S. & Rona, R. J. (1991) Social environment and height: England and Scotland 1987 and 1988. *Arch. Dis. Child* **66**, 235–240.

Hadi, H., Stoltzfus, R. J., Dibley, M. J., Moulton, L. H., West, K. P. Jr, Kjolhede, C. L. & Sadjimin, T. (2000) Vitamin A supplementation selectively improves the linear growth of Indonesian preschool children: results from a randomized controlled trial. *Am. J. clin. Nutr.* **71**, 507–513.

Harris, B. (1994) Health, height and history: an overview of recent developments in anthropometric history. *Social Hist. Med.* **7**, 297–320.

Hauspie, R., Susanne, C. & Alexander, F. (1976) A mixed longitudinal study of the growth in height and weight in asthmatic children. *Hum. Biol.* **48**, 271–283.

Hauspie, R. C., Bergman, P., Bielicki, T. & Susanne, C. (1994) Genetic variance in the pattern of the growth curve for height: a longitudinal analysis of male twins. *Ann. hum. Biol.* **21**, 347–362.

Hauspie, R. C., Vercauteren, M. & Susanne, C. (1996) Secular changes in growth. *Horm. Res.* **45**, 8–17.

Hauspie, R. C., Vercauteren, M. & Susanne, C. (1997) Secular changes in growth and maturation: an update. *Acta paediatr. suppl.* **423**, 20–27.

Hawk, L. J. & Brook, C. G. D. (1979) Family resemblances of height, weight and body fatness. *Arch. Dis. Child* **54**, 877–879.

Henneberg, M. & Berg, E. R. (1990) Test of socioeconomic causation of secular trend: stature changes among favored and oppressed South Africans are parallel. *Am. J. phys. Anthrop.* **83**, 459–465.

Herber, S. M. & Dunsmore, I. R. (1988) Does control affect growth in diabetes mellitus? *Acta paediatr. scand.* **77**, 303–305.

Hermanussen, M., Thiel, C., von Bûren, E., Lama, M. A. R., Romero, A. P., Ruiz, C. A., Burmeister, J. & Tresguerres, J. A. F (1998) Micro and macro perspective in auxology: findings and considerations upon the variability of short term and individual growth and the stability of population derived parameters. *Ann. hum. Biol.* **25**, 359–385.

Hewitt, D. (1957) Some familial correlations in height, weight and skeletal maturity. *Ann. hum. Genet.* **22**, 26–35.

Hewitt, J. K., Stunkard, A. J., Carroll, D., Sims, J. & Turner, J. R. (1991) A twin study approach towards understanding genetic contribution to body size and metabolic rate. *Acta genet. med. gemell.* **40**, 133–146.

Hoppa, R. D. & Garlie, T. N. (1998) Secular changes in the growth of Toronto children during the last century. *Ann. hum. Biol.* **25**, 553–561.

Hoyenga, K. B. & Hoyenga, K. T. (1982) Gender and energy balance: sex differences in adaptations for feast and famine. *Physiol. Behav.* **28**, 545–563.

Hughes, J. M., Li, L., Chinn, S. & Rona, R. J. (1997) Trends in growth in England and Scotland, 1972 to 1994. *Arch. Dis. Child* **76**, 182–189.

Humphreys, L. G., Davey, T. C. & Park, R. K. (1985) Longitudinal correlation analysis of standing height and intelligence. *Child Dev.* **56**, 1465–1478.

Huntley, R. M. C. (1965) Heritability of intelligence. In: *Genetic and Environmental Factors in Human Ability*, pp. 201–218. Edited by J. E. Meade & A. S. Parkes. Oliver and Boyd, Edinburgh.

Husén, T. (1959) *Psychological Twin Research.* Almqwist and Wiksells, Uppsala.

Hutchinson, J. & Byard, P. J. (1987) Family resemblance for anthropometric and blood pressure measurements in black Caribs and Creoles from St Vincent Island. *Am. J. phys. Anthrop.* **73**, 33–39.

Illsley, R. (1955) Social class selection and class differences in relation to stillbirths and infant deaths. *Br. med. J.* **ii**, 1520–1524.

Johnson, J. A. & Kumar, R. (1994) Renal and intestinal calcium transport: roles of vitamin D and vitamin D-dependent calcium binding proteins. *Semin. Nephrol.* **14**, 119–128.

Johnston, F., Borden, M. & MacVean, R. (1977) The effects of genetic and environmental factors upon the growth of children in Guatemala City. In: *Biosocial Interrelations in Population Adaptation*, pp. 377–388. Edited by E. Watts, F. Johnston & G. Lasker. Mouton Publishers, Paris.

Kardjati, S., Kusin, J. A. & De With, C. (1988) Energy supplementation in the last trimester of pregnancy in East Java: I. Effect on birthweight. *Br. J. Obstet. Gynaec.* **95**, 783–794.

Kaur, D. P. & Singh, R. (1981) Parent–adult offspring correlations and heritability of body measurements in a rural Indian population. *Ann. hum. Biol.* **8**, 333–339.

Komlos, J. (1995) *The Biological Standard of Living in Europe and America 1700–1900.* Variorum, Great Yarmouth.

Kouchi, M. (1996) Secular change and socioeconomic difference in height in Japan. *Anthrop. Sci.* **104**, 325–340.

Kuh, D., Power, C. & Rodgers, B. (1991) Secular trends in social class and sex differences in adult height. *Int. J. Epidemiol.* **20**, 1001–1009.

Kuh, D. & Wadsworth, M. (1989) Parental height: childhood environment and subsequent adult height in a national birth cohort. *Int. J. Epidemiol.* **18**, 663–668.

Kusin, J. A., Kardjati, S., Houtkooper, J. M. & Renqvist, U. H. (1992) Energy supplementation during pregnancy and postnatal growth. *Lancet* **340**, 623–626.

Langinvainio, H., Koskenvuo, M., Kaprio, J. & Sistonen, P. (1984) Finnish twins reared apart II: Validation of zygosity, environmental dissimilarity and weight and height. *Acta genet. med. gemell.* **33**, 251–258.

Lauderdale, D. S. & Rathouz, P. J. (1999) Evidence of environment suppression of familial resemblance: height among US Civil War brothers. *Ann. hum. Biol.* **26**, 413–426.

Leighton, G. & Clark, M. L. (1929) Milk consumption and the growth of school-children. *Lancet* **i**, 40–43.

Liestøl, K. & Rosenberg, M. (1995) Height, weight and menarcheal age of schoolgirls in Oslo – an update. *Ann. hum. Biol.* **22**, 199–205.

Little, B. B., Malina, R. M. & Buschang, P. H. (1990) Sibling similarity in annual growth increments in schoolchildren from a rural community in Oaxaca, Mexico. *Ann. hum. Biol.* **17**, 41–47.

Liu, X. Y., Jalil, F. & Karlberg, J. (1998) Risk factors for impaired length growth in early life viewed in terms of the infancy–childhood–puberty (ICP) growth model. *Acta Paediatr.* **87**, 237–243.

Macintyre, S. (1988) A review of the social patterning and significance of measures of height, weight, blood pressure and respiratory function. *Social Sci. Med.* **27**, 327–337.

Mackenbach, J. P. (1992) Socio-economic health differences in the Netherlands: a review of recent empirical findings. *Social Sci. Med.* **34**, 213–226.

Marmot, M. (1995) Social differentials in mortality: the Whitehall Studies. In: *Adult Mortality in Developed Countries: From Description to Explanation*, pp. 243–260. Edited by A. Lopez, G. Caselli & T. Valkonen. Clarendon Press, Oxford.

Marshall, W. A. (1981) Geographical and ethnic variations in human growth. *Br. med. Bull.* **37**, 273–279.

Martorell, R. & Habicht, J-P. (1986) Growth in early childhood in developing countries. In: *Human Growth*, vol. 3, *Methodology, Ecological, Genetic and Nutritional Effects on Growth*, 2nd edn, pp. 241–262. Edited by F. Falkner & J. M. Tanner. Plenum Press, New York.

Martorell, R., Habicht, J-P., Yarbrough, C., Lechtig, A., Klein, R. E. & Western, K. A. (1975) Acute morbidity and physical growth in rural Guatemalan children. *Am. J. Dis. Child* **129**, 1296–1301.

Martorell, R., Yarbrough, C., Yarbrough, S. & Klein, R. E. (1980) The impact of ordinary illnesses on the dietary intakes of malnourished children. *Am. J. clin. Nutr.* **33**, 345–350.

Mata, L. (1992) Diarrheal disease as a cause of malnutrition. *Am. J. trop. Med. Hyg.* **47**, S16–27.

McCowan, C., Neville, R. G., Thomas, G. E., Crombie, I. K., Clark, R. A., Ricketts, I. W., Cairns, A. Y., Warner, F. C., Greene, S. A. & White, E. (1998) Effect of asthma and its treatment on growth: four year follow up of cohort of children from general practices in Tayside, Scotland. *Br. med. J.* **316**, 668–672.

McHenry, H. & Giles, E. (1971) Morphological variation and heritability in three Malesian population: a multivariate approach. *Am. J. phys. Anthrop.* **35**, 241–254.

Meyer, H. E. & Selmer, R. (1999) Income, educational level and body height. *Ann. hum. Biol.* **26**, 219–227.

Monteiro, C. A., Benicio, M. H. D. & Gouveia, N. C. (1994) Secular growth trends in Brazil over three decades. *Ann. hum. Biol.* **21**, 381–390.

Moyes, C. D. (1981) Stature and birth rank. *Arch. Dis. Child.* **56**, 116–120.

Mueller, W. H. (1976) Parent-child correlations for stature and weight among school aged children: a review of 24 studies. *Hum. Biol.* **48**, 379–397.

Mueller, W. H. (1977) Sibling correlations in growth and adult morphology in a rural Columbian population. *Ann. hum. Biol.* **4**, 133–142.

Mueller, W. H. & Malina, R. M. (1980) Genetic and environmental influences on growth of Philadelphia black and white schoolchildren. *Ann. hum. Biol.* **7**, 441–448.

Murata, M. & Hibi, I. (1992) Nutrition and the secular trend of growth. *Horm. Res*. **38**, S89–96.

Neale, M. C. & Cardon, L. R. (1992) *Methodology for Genetic Studies of Twins and Families*. Kluwer Academic Publisher, Dordrecht.

Neville, R. G., McCowan, C., Thomas, G. & Crombie, I. K. (1996) Asthma and growth – cause or concern? *Ann. hum. Biol.* **23**, 323–331.

Newman, H. H., Freeman, F. N. & Holzinger, K. J. (1937) *Twins: A Study of Heredity and Environment.* The University of Chicago Press, Chicago.

Nyström Peck, A. M. (1992) Childhood environment, intergenerational mobility and adult health – evidence from Swedish data. *J. Epidemiol. comm. Hlth* **46**, 71–74.

Nyström Peck, M. & Lundberg, O. (1995) Short stature as an effect of economic and social conditions in childhood. *Social Sci. Med.* **41**, 733–738.

Nyström Peck, M. & Vågerö, D. H. (1987) Adult body height and childhood socioeconomic group in the Swedish population. *J. Epidemiol. comm. Hlth* **41**, 333–337.

Ogata, T. & Matsuo, N. (1992) Comparison of adult height between patients with XX and XY gonadal dysgenesis: support for a Y specific growth gene(s). *J. med. Genet.* **29**, 539–541.

OMIM (2001) *Online Mendelian Inheritance in Man*. http://www.ncbi.nlm.nih.gov/Omim/

Ooki, S. & Asaka, A. (1993) Physical growth of Japanese twins. *Acta genet. med. gemell.* **42**, 275–287.

Orr, J. B. (1928) Milk consumption and the growth of school-children. *Lancet* **i**, 202–203.

Osborne, R. T. (1970) Heritability estimates for the visual evoked response. *Life Sci.* **9**(II), 481–490.

Padez, C. & Johnston, F. (1999) Secular trends in male adult height 1904–1996 in relation to place of residence and parent's educational level in Portugal. *Ann. hum. Biol.* **26**, 287–298.

Pawlowski, B., Dunbar, R. I. M. & Lipowicz, A. (2000) Tall men have more reproductive success. *Nature* **403**, 156–156.

Pearson, K. & Lee, A. (1903) On the laws on inheritance in man. *Biometrika* **2**, 356–462.

Pedersen, N. L., Friberg, L., Floderus-Myrhed, B., McClear, G. E. & Plomin, R. (1984) Swedish early separated twins: identification and characterization. *Acta genet. med. gemell.* **33**, 243–250.

Pedersen, S. (2001) Assessing the effect of intranasal steroids on growth. *J. Allergy clin. Immunol.* **108**, S40–44.

Pembrey, M. (1996) Imprinting and transgenerational modulation of gene expression: human growth as a model. *Acta genet. med. gemell.* **45**, 111–125.

Phillips, K. & Matheny, A. P. Jr (1990) Quantitative genetic analysis of longitudinal trends in height: preliminary results from the Louisville Twin Study. *Acta genet. med. gemell.* **39**, 143–163.

Plomin, R., DeFries, J. C. & McClearn, G. (1997) *Behavioral Genetics*, 3rd edn. W. H. Freeman and Company, New York.

Poskitt, E. M. E. (1993) Failure to thrive in congenital heart disease. *Arch. Dis. Child* **68**, 158–160.

Power, C. & Manor, O. (1995) Asthma, enuresis and chronic illness: long term impact on height. *Arch. Dis. Child* **73**, 298–304.

Prentice, A. & Bates, C. J. (1994) Adequacy of dietary mineral supply for human bone growth and mineralisation. *Eur. J. clin. Nutr.* **48**, S161–177.

Proos, L. A. (1993) Anthropometry in adolescence – secular trends, adoption, ethnic and environmental differences. *Horm. Res.* **39**, 18–24.

Province, M. A. & Rao, D. C. (1985) Path analysis of family resemblance with temporal trends: applications to height, weight and Quetelet Index in Northeastern Brazil. *Am. J. hum. Genet.* **37**, 178–192.

Ratcliffe, S. G., Pan, H. & McKie, M. (1992) Growth during puberty in the XYY boy. *Ann. hum. Biol.* **19**, 579–587.

Rebato, E., Salces, I., Martin, S., Rosique, J., Hauspie, R. & Susanne, C. (1997) Age variations in sibling correlations for height, sitting height and weight. *Ann. hum. Biol.* **24**, 585–592.

Roberts, D. F., Billewicz, W. Z. & McGregor, I. A. (1978) Heritability of stature in a West African population. *Ann. hum. Genet.* **42**, 15–24.

Rona, R. J. (2000) The impact of the environment on height in Europe: conceptual and theoretical considerations. *Ann. hum. Biol.* **27**, 111–126.

Rona, R. J. & Chinn, S. (1986) National study of health and growth: social and biological factors associated with height of children from ethnic groups living in England. *Ann. hum. Biol.* **13**, 453–471.

Rona, R. J., Swan, A. V. & Altman, D. G. (1978) Social factors and height of primary schoolchildren in England and Scotland. *J. Epidemiol. comm. Hlth* **32**, 147–154.

Rowland, M. G. M., Cole, T. J. & Whitehead, R. G. (1977) A quantitative study into the role of infection in determining nutritional status in Gambian village children. *Br. J. Nutr.* **37**, 441–450.

Rudolf, M. C. J. & Hochberg, Z. (1990) Are boys more vulnerable to psychosocial growth retardation? *Dev. Med. Child Neurol.* **32**, 1022–1025.

Ruel, M. T., Rivera, J., Habicht, J. P. & Martorell, R. (1995) Differential response to early nutrition supplementation: long-term effects on height at adolescence. *Int. J. Epidemiol.* **24**, 404–412.

Russell, M. (1976) Parent–child and sibling–sibling correlations of height and weight in a rural Guatemalan population of preschool children. *Hum. Biol.* **48**, 501–515.

Schmidt, I. M., Jørgensen, M. H. & Michaelsen, K. F. (1995) Height of conscripts in Europe: is postneonatal mortality a predictor? *Ann. hum. Biol.* **22**, 57–67.

Schreider, E. (1961) Anthropometric correlations between adult brothers. *Nature* 1311–1311.

Sharma, K., Byard, P. J., Russell, J. M. & Rao, D. C. (1984) A family study of anthropometric traits in a Punjabi community: 1. Introduction and familial correlations. *Am. J. phys. Anthrop.* **63**, 389–395.

Shephard, R. J. & Rode, A. (1995) Growth patterns of Canadian Inuit children. A longitudinal study. *Arctic med. Res.* **54**, 60–68.

Shields, J. (1962) *Monozygotic Twins Brought up Apart and Brought up Together*. Oxford University Press, London.

Silventoinen, K. (2000) *Body-height: Determinants and Associations with Social Position and Adult Health*. Department of Public Health, University of Helsinki, Helsinki.

Silventoinen, K., Kaprio, J. & Lahelma, E. (2000a) Genetic and environmental contributions to the association between body-height and educational attainment. A study of adult Finnish twins. *Behavl Genet.* **30**, 477–485.

Silventoinen, K., Kaprio, J., Lahelma, E. & Koskenvuo, M. (2000b) Relative effect of genetic and environmental factors on body height: differences across birth cohorts among Finnish men and women. *Am. J. publ. Hlth* **90**, 627–630.

Silventoinen, K., Kaprio, J., Lahelma, E., Viken, R. J. & Rose, R. J. (2001a) Sex differences in genetic and environmental factors contributing to body-height. *Twin Res.* **4**, 25–29.

Silventoinen, K., Lahelma, E., Lundberg, O. & Rahkonen, O. (2001b) Body-height, birth cohort and social background in Finland and Sweden. *Eur. J. publ. Hlth* **11**, 124–129.

Silventoinen, K., Lahelma, E. & Rahkonen, O. (1999) Social background, adult body-height and health. *Int. J. Epidemiol.* **28**, 911–918.

Singh, L. P. & Harrison, G. A. (1997) The impact of caste on the growth of male Sikhs in Punjab, India. *Ann. hum. Biol.* **24**, 131–139.

Skerry, T. M. (1994) The effects of the inflammatory response on bone growth. *Eur. J. clin. Nutr.* **48**, S190–198.

Smith, J. C., Makdani, D., Hegar, A., Rao, D. & Douglass, L. W. (1999) Vitamin A and zinc supplementation of preschool children. *J. Am. Coll. Nutr.* **18**, 213–222.

Solomon, P. J. & Thompson, E. A. (1983) The inheritance of height in a Finnish population. *Ann. hum. Biol.* **10**, 247–256.

Stanner, S. A., Bulmer, K. Andrès, C., Lantseva, O. E., Borodina, V., Poteen, V. V. & Yudkin, J. S. (1997) Does malnutrition *in utero* determine diabetes and coronary heart disease in adulthood? Results from the Leningrad siege study, a cross sectional study. *Br. med. J.* **315**, 1342–1349.

Steckel, R. H. (1995) Stature and the standard of living. *J. econ. Lit.* **33**, 1903–1940.

Stephensen, C. B. (1999) Burden of infection on growth failure. *J. Nutr.* **129**, 534–538S.

Stini, W. A. (1969) Nutritional stress and growth: sex difference in adaptive response. *Am. J. phys. Anthrop.* **31**, 417–426.

Stinson, S. (1985) Sex differences in environmental sensitivity during growth and development. *Yearb. phys Anthrop.* **28**, 123–147.

Stocks, P. (1932) A biometric investigation of twins and their brothers and sisters. *Eugenics* **4**, 7–108.

Stunkard, A. J., Foch, T. T. & Hrubec, Z. (1986) A twin study of human obesity. *J. Am. med. Ass.* **256**, 51–54.

Susser, M. & Stein, Z. (1994) Timing in prenatal nutrition: a reprise of the Dutch Famine Study. *Nutr. Rev.* **52**, 84–94.

Takkunen, J. (1964) *Anthropometric, Electrocardiographic and Blood Pressure Studies on Adult Male Twins*. Annales Academiae Scientiarum Fennicae, Helsinki.

Tambs, K., Moum, T., Eaves, L. J., Neale, M. C., Midthjell, K., Lund-Larsen, P. G. & Naess, S. (1992) Genetic and environmental contributions to the variance of body height in a sample of first and second degree relatives. *Am. J. phys. Anthrop.* **88**, 285–294.

Tanner, J. M. (1992) Growth as a measure of the nutritional and hygienic status of a population. *Horm. Res.* **38**, 106–115S.

Tattersall, R. B. & Pyke, D. A. (1973) Growth in diabetic children. Studies in identical twins. *Lancet* **ii**, 1105–1109.

Teasdale, T. W. & Owen, D. R. (1984) Heredity and familial environment in intelligence and educational level – a sibling study. *Nature* **309**, 620–622.

Teasdale, T. W., Owen, D. R. & Sørensen, T. I. A. (1991) Intelligence and educational level in adult males at the extremes of stature. *Hum. Biol.* **63**, 19–30.

Teasdale, T. W., Sørensen, T. I. A. & Owen, D. R. (1989) Fall in association of height with intelligence and educational level. *Br. med. J.* **298**, 1292–1293.

Terrenato, L. & Ulizzi, L. (1983) Genotype–environment relationship: an analysis of stature distribution curves during the last century in Italy. *Ann. hum. Biol.* **10**, 335–346.

Thon, A., Heinze, E., Feilen, K-D., Holl, R. W., Schmidt, H., Koletzko, S., Wendel, U. & Nothjunge, J. (1992) Development of height and weight in children with diabetes mellitus: report on two prospective multicentre studies, one cross-sectional, one longitudinal. *Eur. J. Pediatr.* **151**, 258–262.

Topp, S. G., Cook, J., Holland, W. W. & Elliott, A. (1970) Influence of environmental factors on height and weight of schoolchildren. *Br. J. prev. soc. Med.* **24**, 154–162.

Ulijaszek, S. J. (1994) Between-population variation in pre-adolescent growth. *Eur. J. clin. Nutr.* **48**, S5–14.

van Wieringen, J. C. (1986) Secular growth changes. In: *Human Growth*, vol. 3, *Methodology, Ecological, Genetic and Nutritional Effects on Growth*, 2nd edn, pp. 307–331. Edited by F. Falkner & J. M. Tanner. Plenum Press, New York.

Vella, V., Tomkins, A., Borghesi, A., Migliori, G. B. & Oryem, V. Y. (1994) Determinants of stunting and recovery from stunting in Northwest Uganda. *Int. J. Epidemiol.* **23**, 782–786.

Victora, C. G., Barros, F. C., Kirkwood, B. R. & Vaughan, J. P. (1990) Pneumonia, diarrhea and growth in the first 4 y of life: a longitudinal study of 5914 urban Brazilian children. *Am. J. clin. Nutr.* **52**, 391–396.

Walker, M., Shaper, A. G. & Wannamethee, G. (1988) Height and social class in middle-aged British men. *J. Epidemiol. comm. Hlth* **42**, 299–303.

Walter, T., Olivares, M., Pizarro, F. & Muñoz, C. (1997) Iron, anemia and infection. *Nutr. Rev.* **55**, 111–124.

Wang, Z., Ouyang, Z., Wang, D. & Tang, X. (1990) Heritability of blood pressure in 7- to 12-year-old Chinese twins, with special reference to body size effects. *Genet. Epidemiol.* **7**, 447–452.

Wasserman, R. H. & Fullmer, C. S. (1995) Vitamin D and intestinal calcium transport: facts, speculations and hypotheses. *J. Nutr.* **125**, 1971–1979S.

Weber, G., Seidler, H., Wilfing, H. & Hauser, G. (1995) Secular change in height in Austria: an effect of population stratification? *Ann. hum. Biol.* **22**, 277–288.

Wise, J. E., Kolb, E. L. & Sauder, S. E. (1992) Effect of glycemic control on growth velocity in children with IDDM. *Diabetes Care* **15**, 826–830

Zerfas, A. J. (1986) Epidemiology and nutrition. In: *Human Growth*, vol. 3, *Methodology, Ecological, Genetic and Nutritional Effects on Growth*, 2nd edn, pp. 475–500. Edited by F. Falkner & J. M. Tanner. Plenum Press, New York.