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Long-term physiological and economic consequences of growth retardation in children and adolescents

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The application of a lifespan perspective in human biology in recent years has shown that a number of early environmental factors influencing human growth and development have long-term biological or psycho-social consequences. Human growth is characterized by prolonged infancy, an extended childhood phase and high rates of growth during the adolescent growth spurt. It is unlikely that these characteristics would have evolved without having advantages, and curtailments have the potential for disadvantage. The present paper examines the evidence for long-term physiological and economic consequences of growth retardation in children and adolescents. The emphasis is the biological and economic imperatives of survival, subsistence, reproduction and production rather than aspects of metabolic competence. Many of the consequences of growth retardation are determined by the direct effect on body size, but many other consequences arise from the conditions that cause the growth retardation. Catch up of retarded growth can occur, but does not usually do so because of the continued presence of the retarding agents. Basal metabolism and physical work capacity are usually commensurate with the size of the individual; mechanical efficiency of physical work is unchanged, but falls in activity levels may occur along with a reduction in the pace of activity. Growth retardation in childhood is associated with a higher disease and mortality risk in adulthood, with decreased productivity and employment and promotion prospects. Studies are showing that relative deprivation and the accumulation of socially patterned exposures are important in some societies. Height and growth retardation have proved invaluable in reflecting these factors, but the next generation of studies may require more discriminating indices.

Catch-up growth: Human energetics: Morbidity and mortality: Deprivation

The application of a lifespan perspective in human biology in recent years has shown that a number of early environmental factors influencing human growth and development have long-term biological or psycho-social consequences. Much attention has focused recently on the foetal period and infancy, leading to the belief that most critical periods may have passed by 1 year of age. Less attention has been given to the long-term effects of growth faltering at other ages. The pattern of human growth is characterized by prolonged infancy, an extended childhood phase and high rates of growth during the adolescent growth spurt. It is unlikely that these characteristics would have evolved without having an advantage, and curtailments have the potential for disadvantage. The traditional textbook explanations of childhood have emphasized the extended period for brain development, the acquisition of technical skills and time for socialization. To these factors Bogin (1996) has added its value as a reproductive strategy of

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spaced births for the parents and 'as a mechanism that allows for precise "tracking" of ecological conditions via developmental plasticity'. The present paper examines the evidence for long-term physiological and economic consequences of growth retardation (GR) in children and adolescents. The emphasis is the biological and economic imperatives of survival, subsistence, reproduction and production rather than aspects of metabolic competence.

Challenges to growth rarely occupy precise defined epochs, and even more rarely do they operate in a vacuum. The ecological settings vary in different parts of the world and with time, more so in the last 30–40 years than at any other period. This factor makes it difficult to describe the long-term consequences of GR, and it precludes the enunciation of law-like relationships between GR and its long-term consequences. Moreover, it must be stressed that whereas GR has direct consequences, many operating through reduced body size, its major significance is as an indicator variable. Growth has been described as 'a mirror of the conditions of society' (Tanner, 1986) and height as a 'proxy for health' (Tanner, 1989). Even in the apparently favourable conditions of the so-called developed nations, growth is curtailed in some sections of the population by deprivation, illness, psycho-social stress and family size. These associates and correlates of GR may be the important causative agents of long-term effects rather than the smaller size *per se*.

Growth retardation

Growth, in the biological sense, is a quantitative increase in size or mass. GR is a reduction or delay in growth appropriate for that individual or population. Problems arise immediately. Not all small children or populations are growth retarded, but growth potential is rarely known. Both growth and GR require serial measurements for characterization, but these measurements are rarely available in the nutritional context. Physical linear dimensions such as length or height are the common indices of GR, but weight or muscle mass could be used. GR is usually quantified by comparing dimensions with reference data from children who grew up in overall relatively good environments.

GR of a child or a population in developing countries is said to be most common and marked between 6 and 12 months (Karlberg et al. 1994). This timing is, according to Karlberg et al. (1994), the onset of the childhood phase of growth, and the growth pattern in developing countries is for a delay in onset of this phase. Growth velocities, and hence nutrient needs, are higher than at any other time including adolescence. The frequent and severe infections of this group undergoing the troublesome weaning period are well documented, and the infants are still totally dependent on their caregivers. The points of the intersection of the different phases are regarded as critical times when the system is unusually sensitive to stress or perturbation (Jackson et al. 1996). In the second and third years growth curves are regarded as paralleling those of Western children (Martorell et al. 1994). The Martorell et al. (1994)

suggestion that after 3 years GR children may grow at the same rate as reference data is not true for all ecological settings. Rural South African children are reported to be near the 50th percentile at 5 years, but a growth rate slower than the reference rate and a delayed adolescent growth spurt finds them at or near the 3rd percentile by the onset of adolescence (Cameron, 1992).

The significance of growth retardation

In the context of the long-term physiological and economic consequences of GR, the reduction in size and any carry-over of this factor to later years is the key direct outcome. Possible associated physiological curtailments are altered body composition, decreased basal metabolism, reduced physical activity and varying metabolic and mechanical efficiency. Other associative curtailments such as poor intellectual and behavioural development may have greater economic consequences, but these curtailments do not arise from GR, rather all these curtailments arise from poor deprived environments.

GR can have a variety of causes, but in much of the world undernutrition features as the major cause. Growth faltering is regarded as an early sign and symptom of poor nutrition, and nutritionists rely heavily on anthropometry for indices of nutritional status. However, nowadays there is another use for growth and GR data: 'growth as a mirror of the conditions of society' (Tanner, 1986). Data on physical growth and maturation can provide insight on the conditions of society, of the presence of social inequalities and the changes with time.

Study requirements

There are good accounts of the physiological and economic effects in adulthood of growing up in settings where there is GR in childhood and adolescence, but these accounts have been based mainly on cross-sectional data. In most cases deprivation continued through adolescence into adult life. Cross-sectional studies are only able to provide a low level of evidence of causation. One particular problem in this context is that cross-sectional studies deal with the survivors, which may mean the consequences are under-estimated. Also, it is often difficult to separate out the effects of GR *per se* from other environmental challenges. This factor is important when it comes to tackling the problem. What are needed are longitudinal or serial prospective studies with adequate numbers in age and sex groups, and good baseline data on indices and outcomes. Full description and quantification of intervening and confounding variables and the ecological setting should be performed. There have been few longitudinal studies meeting these criteria that have looked at the long-term consequences of GR in childhood or adolescence. The earlier work of Satyarayana et al. (1980a, 1981, 1989), and the more recent INCAP Follow-up Study (see Martorell et al. 1995) to be described later (see p. 247 and p. 245 respectively) are notable exceptions and all the more valuable.

Physiological consequences of previous growth retardation

The size issue: catch-up growth

As so many of the physiological and economic consequences of GR in developing countries are determined, at least in part, by body size, a key question is: can growth be 'caught up' after GR? This question has received much attention in recent years (Tanner, 1989; Martorell et al. 1994; Golden, 1994, 1996). Opinions range from the view of Martorell et al. (1994) that stunting arises from events early in life and that once present it remains for life, to that of Tanner (1989) that 'the undernourished child slows down and waits for better times'. Tanner (1989) considers that 'in a world where nutrition is never assured, any species unable to regulate its growth in this way would long since have been eliminated'.

The longitudinal studies of Satyanarayana et al. (1980a) provide data on the question of catch up in an Indian setting. The data were collected from 1965 onwards from boys and girls in communities around Hyderabad not experiencing any deliberate intervention. They were classified into four groups of nutritional status at 5 years of age on the basis of deficits in height-for-age according to Boston reference data (Reed & Stuart, 1959). Group 1 had heights down to 2SD below the mean and were categorized as normal, group 2 had deficits of −2SD to −3SD below the mean and were described as mild GR, group 3 had deficits of −3SD to −4SD and were described as moderate, and group 4 had deficits of >4SD below the mean and were described as severe GR. Of this latter group, 20 % had had kwashiorkor or marasmus in early childhood. They had thus experienced severe and chronic undernutrition and GR from early childhood. Group 4 boys had heights which were 0·165 m lower than those of group 1 boys at 5 years of age. Between 5 and 18 years they grew 0·622 m compared with 0·605 m in group 1 boys. At 5 years group 4 girls were 0·142 m shorter than group 1 girls, but gained 0·582 m v. 0·518 m of group 1 girls (Satyanarayana et al. 1981). The shorter severely growth-retarded children remain short at 18 years, but the differences were reduced, particularly in girls. Similar results of catch up in severely stunted girls between school age and late adolescence, but not in boys, were found in a recent study in Senegal, West Africa (Simondon et al. 1998). Golden (1996) has drawn attention to the concordance of the higher Zn requirement of males of all ages to these differing growth patterns in the sexes.

What is notable about the data of Satyanarayana et al. (1980a, 1981) is that the height growths between 5 and 18 years are comparable with those of Western children, and in the case of group 4 girls exceed them (Eveleth & Tanner, 1990). In the most severely growth-retarded boys puberty occurred later than in British boys, by about 2 years, and the intensity, as shown by peak height velocity (PHV), was reduced (Satyanarayana et al. 1980a). A further study showed that such boys gained a similar amount of height to that of British boys during puberty due to an extended puberty (Satyanarayana et al. 1989). Girls too had a delayed and extended pubertal growth spurt (Satyanarayana et al. 1981).

Thus, surviving Indian children who have experienced severe GR by age 5 years and who remain in the same environment that led to this situation, experienced some catch up in height. Between 5 and 18 years they grew as much as Western children and had a similar pubertal growth. Puberty was delayed, and this extended childhood growth phase provided an opportunity for catch up or for achieving the same growth between 5 and 18 years of age as more advantaged children in other parts of the world.

A similar picture was seen at the same time in The Gambia, West Africa. Billewicz & McGregor (1982) found that after the period of growth faltering between 3 months and 3 years, the growth from 3 years to adulthood was the same as that in British boys and girls. The pubertal growth spurts were comparable in magnitude but with different patterns. In The Gambia the beginning of the spurt in height was later and PHV less, but the duration of the spurt was prolonged, leading to similar total increases. In contrast to height, differences in weight increased from 3 years of age to adulthood.

Rural Ladino (Spanish-Amerindian) Guatemalan children and adolescents are growth retarded (Martorell et al. 1995). The median length at birth was 20 mm less than US reference data (Hamill et al. 1977) and equal to the 16th percentile, 50 mm less by 6 months and at the 5th percentile, and 100 mm less at 3 years. In adolescence no further GR and no catch-up occurred, leading to short stature in adults. Thus, growth was markedly retarded only in early childhood.

In summary, GR in early childhood appears to persist into adulthood. The primary factor leading to short adult stature is GR by the start of puberty. This is not to say that catch up cannot occur given the right circumstances, but in most cases children remain in or return to the environment that causes retardation.

Ameliorating and confounding factors

Two factors may ameliorate the effects. Where GR is or has been severe, childhood growth may be prolonged because of delayed puberty. This delay is usually less than 2 years and compensates for only a small proportion of the retardation. Pubertal growth may be increased because of an increased duration, even though PHV may be smaller than that in Western children. However, the common experience is of marked to moderate stunting with only moderate to minor maturational delays.

Other intervening or confounding variables may influence the possibility and achievement of catch up. The effects and consequences of GR are likely to depend on: the age of onset of challenge; its duration, and whether continuous or intermittent (seasonal); its intensity; the complex of challenges experienced, e.g. the coexistence of illness, poverty, etc. Another obvious mediator is whether any subsequent improvements in the environment are experienced. This situation means that no prediction of whether stunting is or is not reversible in an individual or society can be made without full background information.

Improvements in nutrition

Reports of the effects of treatment of children with kwashiorkor, which include short-term improvements in nutrition followed by return to the same environment that caused the GR, provide evidence of catch up. Such studies have been reviewed by Golden (1996). Catch up in older children was notable in a study in South Africa with a 15-year follow-up (Cameron *et al.* 1986), and in Jamaican children followed for 11 years (Richardson, 1975). Catch up here is in relation to other children in the same environment, usually siblings, who are more appropriate controls than the general population. In contrast, little evidence of catch up of discharged malnourished children in Uganda has been reported (MacWilliam & Dean, 1965).

Community-based supplementation studies provide a better idea of what is achievable in the habitual environment. The INCAP Longitudinal Study (1969–77; see Martorell *et al.* 1995) provides some unique data. Supplements were made available to children aged 0–7 years. The amount of supplement varied, which allowed a dose–response approach to the analysis using multiple regression and controlling for confounding factors (Habicht *et al.* 1995; Schroeder *et al.* 1995). Supplementation of 420 kJ/d was associated with additional length gains of 9 mm in the first year, 5 and 4 mm in years 2 and 3, but had no significant impact after age 3 years. Other studies in the literature have been analysed differently, but show similar pictures when a stratified approach has been used. Children from unsupplemented villages were found to weigh less, be shorter and have lower fat-free masses (FFM) than those from supplemented villages at adolescence (Rivera *et al.* 1995). However, differences in height were reduced compared with those at 3 years of age.

Relocation to better environments

Migration and adoption involve considerably greater changes in the environment, both in number and degree, than nutritional intervention alone. Adoption studies have shown accelerated growth rates, but it is not clear if these translate to increased adult stature. General improvements in the whole height distribution of Southeast Asian refugees to USA have been observed (Lien *et al.* 1977). Proos *et al.* (1993) show marked improvements over 2 years in Indian preschool children adopted in Sweden. Accelerated growth may trigger endocrinological responses accelerating maturity and so shortening the growth period and curtailing adult stature. Proos *et al.* (1991) found very early menarche, median 11·6 years, in 107 Indian girl adoptees in Sweden, five of whom experienced menarche before 9 years of age.

Catch up or not, and catch up of what?

Three issues need to be considered in the debate on catch up.

First, the choice of reference data may affect the conclusions. Populations may have different genetic potentials for childhood growth, the adolescent growth spurt or adult size. Little *et al.* (1983) found prepubertal GR of 100–150 mm in the nomadic Turkana pastoralists. By adulthood, heights were similar to US reference data (Hamill *et al.* 1977). This finding might be taken as evidence of catch-up growth, but it is quite possible that these linear Africans have a greater genetic potential than the observed height and that GR persisted, albeit at a reduced level. The question of the appropriate reference population is complicated particularly for adolescence. Little is known about genetic differences in the timing, magnitude and duration of the pubertal spurt.

Second, the phenomenon of catch-up growth in height is not mirrored in increased weight. The severely height-retarded boys of Satyanarayana *et al.* (1980*a*) were 4 kg lighter than boys in the same communities without GR, but by 18 years this difference had increased to 11 kg. The average weight gain of all boys in the study was 29 kg, much less than the 45–50 kg common in Western boys in the 1960s. For girls, the 4 kg difference was maintained throughout the period. This finding has important consequences, as weight and weight-for-height, as seen by BMI, may be more important than height *per se* in terms of functional outcomes. Height may be a better mirror of the conditions of society, but weight and BMI may be better at indexing the functional consequences.

Third, Haas & Habicht (1990) considered that many methodological aspects of growth faltering and catch up have been understudied. They pointed out that if a child grows but maintains a constant absolute height or weight deficit, he (she) will move up the percentiles and appear to show catch-up growth but without any real compensation.

Conclusion

Catch up can occur, but does not usually do so because of the continued presence of the retarding factors. With a change in environment through intervention, emigration or adoption, there is usually definite catch up, although usually not to levels found in Western children. Martorell *et al.* (1994) concluded that the extent of the catch up depends on the degree of maturation delay. However, it appears that maturation is not markedly delayed in most cases of GR.

Maturation

Slow growth during childhood and adolescence resulting from poor living conditions is associated with a late adolescent growth spurt and an extended period of growth (Eveleth & Tanner, 1990). This may arise from suppression of the hypothalamic stimulation of pituitary sex hormone output, as sexual and skeletal maturation are also delayed. The secular trends in growth, menarche and skeletal maturation are a record of the effects of previous living conditions on maturation.

Kwashiorkor at ages between 5 and 52 months in Cape Coloureds of South Africa was not associated with any significant differences in physical growth or age of entry into any of the stages of puberty in the survivors compared with sibling controls (Cameron *et al.* 1986, 1990). Compared with British girls, there were significant delays in the development of pubic hair and menarche but not in breast development or PHV. Compared with British boys, there were significant delays in secondary sexual characteristics but not age of PHV.

The INCAP Follow-up Study found similar ages of menarche in girls who were or were not exposed to supplementation (Khan *et al.* 1995). This finding occurred against a background of a fall in the age of menarche of 0·7 years over the 15-year period of supplementation and follow-up. There were no differences in skeletal maturation in boys and girls after allowing for socio-economic status (Pickett *et al.* 1995). Hence, there was no evidence of marked delays in maturation in the population, and exposure to high-protein, high-energy supplementation had little or no effect. The opportunity for catch-up growth by delayed maturity would seem to be low in this population.

Late maturation can have its advantages. Late maturers tend to be leaner and have a low-risk fat distribution. The longitudinal Leuven Growth Study of Belgian boys between 13 and 18 years with a follow-up at 30 years found that later maturing boys have a less-central distribution of subcutaneous fat in adolescence and adulthood, which is associated with a lower risk of several adult degenerative diseases (Beunen *et al.* 1994). Late maturers in the longitudinal Amsterdam Health and Growth Study were leaner than early maturers at age 27 years (van Lenthe *et al.* 1996*a*). Late maturing girls had lower abdominal fat distribution, based on skinfold thicknesses, than early maturers and these differences persisted during the follow-up period (van Lenthe *et al.* 1996*b*).

Body composition

The lower levels of body fat and FFM seen in GR are commensurate with the lower body size and mass, except in severe GR. The extent of the deficits depends on the duration and intensity of GR.

Studies of the persistence of fatness and fat patterning have tended to concentrate on overweight and obese individuals, and there is a lack of data on such tracking in lean individuals. Events after adolescence seem more important for the development of chronic obesity than events before or during adolescence (Cronk *et al.* 1982). Roche & Baumgartner (1988) summarized the few longitudinal studies on tracking of fat distribution and found little evidence of tracking during infancy or from infancy to puberty. Fat patterns in early life do not seem to be associated with the environment, do not persist in individuals into the later years, and do not appear to be associated with elevated risk of chronic disease in adults (Johnston, 1998).

Schroeder *et al.* (1999) examined the effect of poor growth in early childhood on adult fatness and distribution. Childhood stunting was associated with a lower BMI and percentage body fat in men. BMI were at the 55th percentile of reference data in women but the 20th for men. In both sexes severely stunted children had significantly greater adult abdominal fatness. Migration to urban centres was associated with an even greater waist:hip value in severely stunted females.

In contrast, an increased risk of obesity in stunted children has been described based on an analysis of the prevalence of obesity and stunting in four countries (Popkin *et al.* 1996). The relative risk ranged between 1·7 in Brazil and 7·8 in Russia, with the Republic of South Africa and China being intermediate.

Bone mineralization in Guatemalan adolescents increased according to the calculated childhood energy supplementation but not according to supplement type (Caulfield *et al.* 1995). The effects became non-significant after controlling for weight and stature at adolescence, suggesting they followed improvements in somatic growth rather than maturation.

Energetics

GR can have a variety of causes, but in much of the world undernutrition features as the major cause. Undernutrition may also be responsible for many of the associates and correlates of GR. Energy undernutrition has a variety of effects on metabolism (as shown by a 10 % fall in BMR during energy restriction), on other physiological processes and on behavioural characteristics such as physical activity. These effects have been reviewed in recent years in the debates on adaptation to energy undernutrition (Garby, 1990; Waterlow, 1990; Shetty, 1993, 1999; Norgan & Ferro-Luzzi, 1996). It is important to reiterate that many consequences that may appear alongside or be found several years after GR have their origins in energy undernutrition not in GR *per se*.

BMR. Reductions in BMR have been sought as a key variable in physiological adaptations to undernutrition and GR. Low BMR during and following GR can be attributed mainly to low body weight. However, BMR per kg FFM was found to be 14 % lower in a group of fourteen underweight but otherwise fit and healthy Indian labourers compared with fourteen controls (Shetty, 1984). Measurements in larger samples of chronically undernourished Indian adults with low BMI have not confirmed the existence of such increased metabolic efficiency (Soares & Shetty, 1991; Ferro-Luzzi *et al.* 1997).

Nowadays, it is accepted that ratio scaling, e.g. BMR per kg, is inappropriate to adjust for differences in body size when attempting to uncover differences in energetics variables. It is also necessary to take into account variations in the composition of FFM (Lawrence *et al.* 1988; Shetty, 1999). Garby (1990), in one of the most detailed reviews on metabolic adaptation to restricted energy intake, concluded that the proposal for metabolic adaptation of 15 % BMR in populations of developing countries could not be supported or rejected by the evidence available at that time. A tentative conclusion was a 5–10 % effect (Norgan & Ferro-Luzzi, 1996). Recently, Shetty (1999) has concluded that BMR per kg FFM or active tissue mass 'hitherto considered the definitive indicator of metabolic efficiency does not seem to be altered and there is thus no conclusive evidence of the existence of metabolic adaptation'.

Net mechanical efficiency of work. There has also been a debate as to whether the net mechanical efficiency of work is altered in small adults formerly growth retarded and/or presently undernourished. Waterlow (1990) proposed a mechanism by which this change might come about, i.e. that slow-twitch fibres develop better or are better preserved than fast-twitch fibres. As slow-twitch fibres utilize the energetically more efficient aerobic as opposed to anaerobic

processes, they have a higher efficiency. Coyle *et al.* (1992) found mechanical efficiency in cycling was highly correlated with the proportion of slow-twitch fibres in well-nourished young men, and a selective reduction in fast-twitch anaerobic fibres has been shown to occur in undernutrition (Henriksson, 1992). Ulijaszek (1996) has gone on to propose that low thyroid hormone status occurring in undernourished children may reduce the proportion and size of fast-twitch muscle fibres in skeletal muscle during development, leading to increased efficiency in adulthood.

There has also been a search for energetic differences in everyday activities between the chronically undernourished and growth-retarded subjects and control subjects better favoured. There is some evidence suggesting lower energy costs, but it is difficult to discover what might be the origins (reduced pace of activity, more skilled actions or metabolic efficiency). One factor often overlooked in studies of net mechanical efficiency of work or of everyday activities is that as a given task will be a higher proportion of a smaller individual's work capacity, the task is more stressful and more likely to invoke anaerobic processes. The contribution of such processes is not detected by indirect calorimetry, and these individuals may appear to be more efficient. Norgan & Ferro-Luzzi (1996) reviewed the literature and concluded that there was little evidence of increased mechanical efficiency in chronic energy deficiency or in populations where GR was common, any differences being small and inconsistent. Even in well-nourished subjects there is unexplained variation. Shetty (1999) has emphasized 'real life efficiency' in every day activities where pace may be varied in contrast to the standardized ergometry of laboratory studies.

Physical activity. The behavioural response of a reduction in physical activity is regarded as the key response to energy undernutrition and GR. Often work activities are maintained, albeit at a lower rate, so that working times are extended, but social and other discretionary activities are reduced (Immink, 1988). In children any reduction could affect the learning experiences from play and exploration and social interaction. However, this reduction in physical activity is not well documented, but the findings are concordant (Ferro-Luzzi, 1990; Torun, 1990). In some instances, peer pressure may maintain activity levels, thus preventing deficits in cognitive or motor development, with the result that GR becomes the first line of defence in undernutrition (Spurr & Reina, 1987).

Physical Work Capacity

In much of the world work is minimally mechanized, and productivity and earnings depend on human labour and effort. It is these regions that have the highest prevalences of GR. GR leads to small adult size, which causes a reduced physical work capacity, with consequences for productivity and income in those individuals engaged in moderate or heavy physical work. Size is the strongest correlate of physical work capacity (PWC) in adults. Body size, PWC and work output have been reviewed at regular intervals (Spurr, 1984, 1990; Martorell & Arroyave, 1988; Norgan & Ferro-Luzzi, 1996) and Spurr (1988) has reviewed the implications of marginal malnutrition in childhood for adult PWC and productivity.

In chronically undernourished adults body weight and FFM are low, as is PWC expressed as maximal aerobic capacity (l O_2/min). When expressed per kg body weight, per kg FFM, or per kg muscle mass there may be no difference in PWC unless the undernutrition is severe (Barac-Nieto *et al.* 1978). There is little or no impairment of ability to work at submaximal levels typical of subsistence activities. However, the strain of a given task is greater in smaller individuals. These findings, were replicated in marginally malnourished Colombian boys (Spurr *et al.* 1983).

What of children growth retarded at some stage? There is evidence from cross-sectional studies suggesting that adolescents undernourished as children have low PWC arising from their low body weights and muscle masses. To undernutrition as a causative factor could be added the effects of low physical activity levels and concurrent infection or diseases such as anaemia. Maturation affects PWC independently of size, but the effects of delayed maturation *per se* have not been widely investigated.

Satyaranayana *et al.* (1978, 1979), in a rare prospective study, found that boys growth retarded at 5 years, some of them more than 4SD below Boston reference means (Reed & Stuart, 1959), had significantly lower work capacities at 15 years of age than boys from the same population without GR. This difference arose because the boys remained growth retarded. Differences in PWC disappeared when PWC was expressed per kg body weight, and there was no relationship between height at 5 years of age and PWC per kg body weight at 15 years of age. Values for PWC expressed per kg body weight were similar to those of American and Swedish children at that time. These findings emphasize the importance of size, and show an apparent absence of functional changes.

The INCAP Follow-up Study found lower PWC for adolescents and young adults from villages not receiving supplements than for those from villages receiving supplements (Haas *et al.* 1995). In 18–26-year-old males who would have been exposed to any supplementation between 4 and 7 years of age, the maximum rates of O_2 consumption were 2·77 l/min for males from villages not receiving supplements and 2·98 l/min for those from villages receiving supplements ($P < 0.05$) and remained significantly different after controlling for body weight and FFM. The differences in maximum rates of O_2 consumption for 14–18-year olds, who if exposed to the supplement would have been so from gestation to 3 years, were 0·38 l/min on average. The differences in women were in the same direction but not significant, perhaps a result of the lower activity levels in these adolescent girls. Differences in both sexes were reduced after controlling for FFM, but remained significant in males. The persisting differences in the males, in contrast to the Indian adolescents, may be related to possible effects of early malnutrition and GR on muscle-fibre type and oxidative capacity in later life.

Kulkarni *et al.* (1993) reported a lower maximum rate of O_2 consumption, but not when expressed per kg FFM, in adult Indians who were chronically energy deficient compared with well-nourished controls. Recovery rate of O_2

consumption was lower (147 (SE 9) v. 249 (SE 17) ml/kg FFM), and single as opposed to biphasic. It was suggested that recovery may be an important period for energy saving, but these savings seem rather small. Such a saving can be calculated to be approximately 7 litres O_2 or 160 kJ from a single bout of exhaustive exercise, which could not be repeated often during 1 d.

Size effects on PWC may be more important in static work, such as lifting and handling, than in dynamic work. Vaz *et al*. (1996) found maximum voluntary contraction (grip strength) to be significantly lower in Indian men with low height and weight arising from chronic energy deficiency, compared with men of similar BMI but with heights equal to those of well-nourished controls.

Reproduction and fertility

Poor preconception nutrition, which may go back to the mother's childhood, has long-term effects on pregnancy outcome. Smaller mothers have smaller babies and a higher incidence of low birth weight. The risk of obstetric complications rises when GR has led to the mothers having small pelvic outlets. Fertility and successful pregnancy outcome are related to height even in developed countries. There is also a positive relationship between female height and/or weight and fertility (Eveleth, 1985). Mayan Guatemalan women are short, approximately 1·42 m on average (Martorell *et al*. 1981). However, even in this short population clear differences in fertility and infant survival were found in different height groups. Shorter women (lowest tertile for height) had the greatest parity, but infant mortality was highest in their offspring such that these mothers had the lowest numbers of surviving children.

The interaction of nutrition and other environmental effects and the genome undergoes a crucial phase during pregnancy. Parental environments can affect the degree and timing of the expression of genes in the offspring by what is known as imprinting. This mechanism may explain many interesting observations on nutrition and growth (Golden, 1996). Early events could in this way programme subsequent growth and metabolism. Most of the little information on the role of intergenerational effects on linear growth comes from developed countries. However, in a study from Guatemala, maternal body size was a significant predictor of child's birth size after adjusting for potential confounders (Ramakrishnan *et al*. 1999). Child birth weight increased by 290 g/kg increase in maternal birth weight, twice that reported in developed countries and by 2 mm/10 mm increase in mother's birth length.

Economic consequences of previous growth retardation

Persistent GR has economic consequences for the individual and for the community. For the individual, earning capacity may be low and health and welfare compromised. Increased costs and expenditures for the individual and the community follow. For this reason, morbidity and mortality are included under economic consequences and are described first.

Mortality and morbidity

Waaler (1984) produced data showing that at all ages up to 80 years shorter males and females had, in a 10-year period after measurement, higher mortality than their taller counterparts. This finding is an example of height as a measure of living conditions. A difference of 200 mm in women and 300 mm in men was associated with a mortality risk of 2:1. Few of the Norwegians of Waaler's (1984) study had the extreme low heights associated with markedly raised mortality, but these extreme low heights were present in the past and are present in other parts of the world today.

Barker *et al*. (1989) began their work on early experiences and later outcomes by showing regional associations in England and Wales between high cardio-vascular mortality and comparative shortness of 10-year-olds. Earlier, Forsdahl (1977) had concluded from an inter-county comparison in Norway that '....great poverty in childhood and adolescence followed by prosperity is a risk factor for arteriosclerotic heart disease'. Barker (1994) went on to emphasize the role of the foetal and infant environments. The long-term effects of these factors on disease risk in adulthood have received much attention, but it remains difficult to separate these very early effects from the later childhood and adolescent environments. Subjects aged 2–15 years participating in the Carnegie UK Dietary and Clinical Survey of 1937 into poverty, nutrition and child health (Rowett Research Institute, 1955) have been followed up, and results show that leg length in childhood is associated with mortality in the following 60 years (Gunnell *et al*. 1998). Leg length develops most during childhood as opposed to infancy or puberty, and is expected to be a better index than total height or trunk length. The relative risk of CHD mortality of the shortest male and female quintiles were three to four times that of the upper quintiles for children aged 2–8 years at the time of measurement. There was no leg length effect for cancer mortality in females, but in males the risk was 2·5 times greater in the tallest quintile, supporting the findings of Waaler (1984) on adult height and cancer mortality.

A prospective epidemiological study from Finland found significant negative associations between the risk of hip fracture in middle-aged and elderly individuals and height and weight gain during the school years (Cooper *et al*. 1999). Childhood growth rates more than 1SD below the mean had a fourfold risk of hip fracture. Poor childhood growth was a strong risk factor for later hip fracture.

Questions have been raised as to whether maximum human growth is associated with better health in later years and increased longevity. Animal experiments show that dietary restriction slows growth and delays the onset of diseases in later life, but the extent to which this effect can be transposed to human subjects is not known. The Waaler (1984) data on total mortality suggest the effect cannot be transposed, although some causes of death, e.g. some cancers, may be more common in taller individuals. The evidence strongly suggests that shortness arising from environmental and social causes is associated with increased morbidity and mortality.

Behavioural and cognitive development

There is plentiful evidence that GR is associated with deficits in behavioural and cognitive development. This evidence comes from animal work, cross-sectional population studies correlating growth with psychological test performances, comparisons of formerly malnourished and growth-retarded subjects with controls, and the effects of supplementation on behaviour and test performance. More than any other outcome, the relationship between previous GR and mental development is complicated by the coexistence of GR with poor socio-economic conditions. The functional correlates of GR, such as poor educational performance and other impaired behavioural and social characteristics, are unlikely to be a direct outcome of GR, but instead reflect common origins. The environment that leads to GR is also a poor learning environment.

Motor development in the first 2 years of life in countries with widespread GR is usually satisfactory. Indeed, some African populations show precocity. However, developmental lags arise, affecting motor performance and strength. The importance of physical activity in cognitive, social and motor development of children is well known (Malina, 1984). However, Grantham-McGregor et al. (1991) could find no causal evidence linking poor behavioural development to the low activity levels associated with chronic undernutrition and GR. Activity per se may be less important than what the child does when active.

Pollitt et al. (1995) provide a summary of the findings on nutrition in early life and the fulfilment of intellectual potential from the INCAP Follow-up Study. Supplementation after 2 years of age improved mental development if it was effective, i.e. if growth improved. Until now there has been little evidence of whether the functional capabilities unrelated to size can improve with no change in linear growth. At adolescence consistent differences between the groups were observed on psycho-educational tests, in favour of subjects receiving atole, the high-energy high-protein supplement. Performance was not related to socio-economic status in villages where atole was provided, but was in villages provided with a low-energy low-protein supplement. Play is another important ingredient.

The study demonstrated that there are strong links among malnutrition, human capital formation and poverty which justify investments in health and nutrition as components of economic development strategies (Martorell, 1995). The marked effects of adoption illustrate what can be achieved in terms of behavioural and cognitive development in growth-retarded children (Winick et al. 1975). Poor social environments are associated with reduced intelligence and motivation, which in turn affect work capacity and productivity (Martorell & Arroyave, 1988).

Productivity

Smaller individuals have lower PWC, and in all but the lightest tasks a given job is a greater stress than for a bigger individual. Smallness, in this context, is not a beneficial adaptation. The importance of body size on productivity in strenuous work, and to a lesser extent low-intensity work,

has been reviewed (Spurr, 1984; Ferro-Luzzi, 1985; Martorell & Arroyave, 1985; Immink, 1988; Norgan & Ferro-Luzzi, 1996). Sugar-cane cutting is strenuous work, but lends itself to feasible measurements of productivity. Tall workers have been found to be more productive and to take fewer rest days. Agricultural work output in Africa and India demonstrate that size matters and, in the case of Indian adolescents, that GR influenced earning capacity (Satyanarayana et al. 1980b). Smaller lighter Indians had lower work outputs and earned less incentives in their employment in a light industry (Satyanarayana et al. 1977). This finding was valid for weight controlled for height, but not for height controlled for weight. Martorell (1996) reported that being 3SD below US reference medians (Hamill et al. 1977) in childhood resulted in a FFM which was 6·4 kg less in Guatemalan men and 5·4 kg less in women than in individuals who were less than 1SD below reference medians. These individuals also had had less schooling, 1·8 years in the case of men and 1·0 years in women. In this setting each additional year at school is associated with 6 % higher wages in adults.

GR has effects on earnings, but what of the effects of employment on growth? Satyanarayana et al. (1986) found that child labourers who had been working for 4–8 years suffered significant growth deficits by age 14 years compared with students matched for nutritional status at 5 years of age.

Height has important economic consequences in developed countries too. Adult height is related to social mobility, in particular tall people are promoted more often. However, data from the British 1958 National Child Development Survey showed that unemployment was related more to height at age 7 years than to current height (Montgomery et al. 1996), a finding explained by Wilkinson (1996) in terms of a lower self-esteem and capacity to cope with life's challenges.

Small body size has been considered by some workers to be an appropriate adaptive response to environmental challenges, the 'small but healthy' hypothesis (Seckler, 1982). In the context of natural selection, small size decreases the demand on food resources, with reproduction and physical performance said to be uncompromised, thereby increasing survival. However, to be short means to have a lower PWC, higher disease risk and poor reproductive performance than other taller individuals. The most telling argument against becoming growth retarded or being growth retarded is that it relates to individuals kept in poverty, ill health, undernutrition and low socio-economic status, i.e. denied basic human rights. The stunted are the survivors, and smallness incurs a heavy cost. According to the Human Capital theory (Martorell, 1996) there are major advantages for individuals and for nations to the improvement of human capital, i.e. the productive capacities embodied in all individuals, consisting of both physical and mental abilities. However, it is not easy to show the relationships between body size and PWC and economic indices such as productivity and income. These variables depend on socio-economic, political, educational and other environmental factors as much as on physiological and nutritional factors. Ferro-Luzzi (1985) has discussed some of the non-biological variables that intervene to obscure the

relationships and the methodological difficulties in data collection and synthesis. The section of the population likely to have been growth retarded has little influence on the labour market and the rewards and incentives that could ensue. Studies on children have to emphasize 'potential' rather than 'productivity' because of the age of the participants. Potential can be operationalized by improved growth, body composition, work capacity, intelligence and functional competence. Thus, governments must invest in programmes to prevent the circumstances which lead to GR and, in order to realize the potential from this investment, must provide work opportunities for this potential to be deployed.

What of the future?

Can we expect GR, or the conditions that give rise to it, to lessen? *Per capita* food production is being maintained or is improving in most regions of the world, except much of Africa. However, growth is curtailed and health inequalities exist even in the apparently favourable conditions of developed nations. The experience of one affluent member of the G7 Group of Industrialized Nations is salutary. Within the UK over the last 15–20 years, mortality differentials and socio-economic differentials, particularly incomes, have widened (Davey Smith & Brunner, 1997). Davey Smith *et al.* (1998) have found that adverse socio-economic circumstances in childhood have an influence on mortality from stroke and stomach cancer that is independent of the continuity of disadvantage throughout life. Deprivation in childhood also influences CHD mortality, but with an added influence of adulthood circumstances. The Whitehall Study of British Civil Servants II (Marmot & Davey Smith, 1997) has demonstrated that the gradient in morbidity between job grades is due neither to health selection nor to differences in lifestyle. It points to the importance of rather subtler concepts of relative deprivation and the 'accumulation of socially-patterned exposures over the life course' (Marmot & Davey Smith, 1997). Other workers have emphasized the psycho-social quality of life (Wilkinson, 1996). Egalitarian socially cohesive societies tend to be healthier societies. Among developed countries, the richest are not the healthiest.

Thus, there is a recognition of the importance of a life-course perspective on variations in health. How people grow, live and work contains the operation of the factors. How to quantify the cumulative effects is a problem. Until now height and GR have proved invaluable, but the next generation of studies may require more discriminating indicators.

References

Barac-Nieto M, Spur GB, Maksud MG & Lotero H (1978) Aerobic work capacity in chronically undernourished adult males. *Journal of Applied Physiology* **44**, 209–215.

Barker DJP (1994) *Mothers, Babies and Disease in Later Life*. London: BMJ Publications.

Barker DJP, Osmond C, Golding J & Kuh D (1989) Growth in utero, blood pressure in childhood and adult life, and mortality from cardiovascular disease. *British Medical Journal* **298**, 564–567.

Beunen G, Malina RM, Lefevre J, Claessens AL, Renson R, Simons J, Maes H, Vanreusel B & Lysens E (1994) Size, fatness and relative fat distribution of males of contrasting maturity status during adolescence and as adults. *International Journal of Obesity* **18**, 670–678.

Billewicz WZ & McGregor IAM (1982) A birth-to-maturity longitudinal study of heights and weights in two West African (Gambian) villages, 1951–1975. *Annals of Human Biology* **9**, 309–320.

Bogin B (1996) Human growth and development from an evolutionary perspective. In *Long Term Consequences of Early Environment: Growth, Development and the Lifespan Developmental Perspective*, pp. 7–43 [CJK Henry and SJ Ulijaszek, editors]. Cambridge: Cambridge University Press.

Cameron N (1992) The monitoring of growth and nutritional status in South Africa. *American Journal of Human Biology* **4**, 223–234.

Cameron N, Jones PRM, Moodie A, Mitchell J, Bowie MD, Mann MD & Hansen JDL (1986) Timing and magnitude of adolescent growth in height and weight in Cape Coloured children after kwashiorkor. *Journal of Pediatrics* **109**, 548–555.

Cameron N, Mitchell J, Meyer D, Moodie A, Bowie MD, Mann MD & Hansen JDL (1990) Secondary sexual characteristics of Cape Coloured boys following kwashiorkor. *Annals of Human Biology* **17**, 217–228.

Caulfield LE, Himes JH & Rivera JA (1995) Nutritional supplementation during early childhood and bone mineralisation during adolescence. *Journal of Nutrition* **125**, 1104S–1110S.

Cooper C, Eriksson JG, Forsen T, Osmond C, Tuomilehto J & Barker DJP (1999) Childhood growth trajectory is a strong determinant of later risk of hip fracture: a prospective study. *Calcified Tissue International* S38Abstr.

Coyle EF, Sidossis LS, Horowitz JF & Beltz JD (1992) Cycling efficiency is related to the percentage of Type 1 muscle fibres. *Medicine and Science in Sports and Exercise* **24**, 782–788.

Cronk C, Roche AF, Chumlea W & Kent R (1982) Longitudinal trends of weight/stature^2 in childhood in relation to adult body fat measures. *Human Biology* **54**, 751–764.

Davey Smith G & Brunner E (1997) Socio-economic differentials in health: the role of nutrition. *Proceedings of the Nutrition Society* **56**, 75–90.

Davey Smith GD, Hart C & Blane D (1998) Adverse socio-economic conditions in childhood and cause specific adult mortality: prospective observational study. *British Medical Journal* **316**, 1631–1635.

Eveleth PB (1985) Nutritional implications of differences in adolescent growth and maturation and in adult body size. In *Nutritonal Adaptation in Man*, pp. 31–43 [K Blaxter and JC Waterlow, editors]. London: John Libbey.

Eveleth PB & Tanner JM (1990) *Worldwide Variation in Human Growth*. Cambridge: Cambridge University Press.

Ferro-Luzzi A (1985) Work capacity and productivity in long-term adaptation to low energy intakes. In *Nutritional Adaptation in Man*, pp. 61–68 [K Blaxter and JC Waterlow, editors]. London: John Libbey.

Ferro-Luzzi A (1990) Social and public health issues in adaptation to low energy intakes. *American Journal of Clinical Nutrition* **51**, 309–315.

Ferro-Luzzi A, Petrachi C, Juriyan R & Kurpad AV (1997) Basal metabolism of weight stable chronically undernourished men and women, lack of metabolic adaptation and ethnic differences. *American Journal of Clinical Nutrition* **66**, 1086–1093.

Forsdahl A (1977) Are poor living conditions in childhood and adolescence an important risk factor for arteriosclerotic heart

disease? *British Journal of Preventive and Social Medicine* **31**, 91–97.

Garby L (1990) Metabolic adaptations to decreases in energy intake due to changes in the energy cost of low energy expenditure regimen. *World Review of Nutrition and Dietetics* **61**, 173–208.

Golden MHN (1994) Is catch-up possible for stunted malnourished children? *European Journal of Clinical Nutrition* **48**, S58–S71.

Golden MHN (1996) The effect of early nutrition on later growth. In *Long Term Consequences of Early Environment: Growth, Development and the Lifespan Developmental Perspective*, pp. 91–108 [CJK Henry and SJ Ulijaszek, editors]. Cambridge: Cambridge University Press.

Grantham-McGregor S, Meeks Gardner JM, Walker S & Powell C (1991) The relationship between undernutrition, activity levels and development in young children. In *Activity, Energy Expenditure and Energy Requirements of Infants and Children*, pp. 361–383 [B Schurch and NS Scrimshaw, editors]. Lausanne, Switzerland: IDECG.

Gunnell DJ, Davey Smith G, Frankel S, Nanchahal K, Braddon FEM, Pemberton J & Peters TJ (1998) Childhood leg length and adult mortality: follow up of the Carnegie (Boyd Orr) Survey of Diet and Health in pre-war Britain. *Journal of Epidemiology and Community Health* **52**, 142–152.

Haas JD & Habicht J-P (1990) Growth and growth charts in the assessment of preschool nutritional status. In *Diet and Disease in Traditional and Developing Societies*, pp. 160–183 [GA Harrison and JC Waterlow, editors]. Cambridge: Cambridge University Press.

Haas JD, Martinez EJ, Murdoch S, Conlisk E, Rivera JA & Martorell R (1995) Nutritional supplementation during the preschool years and physical work capacity in adolescent and young adult Guatemalans. *Journal of Nutrition* **125**, 1078S–1089S.

Habicht J-P, Martorell R & Rivera JA (1995) Nutritional impact of supplementation in the INCAP Longitudinal Study: analytical strategies and inferences. *Journal of Nutrition* **125**, 1042S–1050S.

Hamill PVV, Drizd TA, Johnson CL, Reed RB & Roche AF (1977) *NCHS Growth Curves for Children from Birth to 18 years. US Department of Health, Education and Welfare Publication* no. (PHS) 78-1650. Series 11, no. 165. Hyattsville, MD: US Department of Health, Education and Welfare.

Henriksson J (1992) Energy metabolism in muscle: its possible role in the adaptation to energy deficiency. In *Energy Metabolism: Tissue Determinants and Cellular Corollaries*, pp. 345–363 [JM McKinney and HN Tucker, editors]. New York: Raven Press.

Immink MDC (1988) Economic effects of chronic energy deficiency. In *Chronic Energy Deficiency: Causes and Consequences*, pp. 153–174 [B Schurch and NS Scrimshaw, editors]. Lausanne, Switzerland: IDECG.

Jackson AA, Langley-Evans SC & McCarthy HD (1996) Nutritional influences in early life upon obesity and body proportions. In *The Origins and Consequences of Obesity*, pp. 118–137 [DJ Chadwick and G Cardew, editors]. Chichester, West Sussex: John Wiley & Sons.

Johnston FE (1998) Fat patterning. In *The Cambridge Encyclopedia of Human Growth and Development*, p. 419 [SJ Ulijaszek, FE Johnston and MA Preece, editors]. Cambridge: Cambridge University Press.

Karlberg J, Jalil F, Lam B, Low L & Yeung C (1994) Linear growth retardation in relation to the three phases of growth. *European Journal of Clinical Nutrition* **48**, S25–S44.

Khan AD, Schroeder DG, Martorell R & Rivera JA (1995) Age at menarche and nutritional supplementation. *Journal of Nutrition* **125**, 1090S–1096S.

Kulkarni RN, Kurpad AV & Shetty PS (1993) Reduced post-exercise recovery oxygen consumptions: An adaptive response in chronic energy deficiency. *Metabolism* **42**, 544–547.

Lawrence M, Thongprasert K & Durnin JVGA (1988) Between-group differences in basal metabolic rates: an analysis of data collected in Scotland, The Gambia and Thailand. *European Journal of Clinical Nutrition* **42**, 877–891.

Lien NM, Meyer KK & Winick M (1977) Early malnutrition and 'late' adoption: a study of their effects on the development of Korean orphans adopted into American families. *American Journal of Clinical Nutrition* **30**, 1734–1739.

Little MA, Galvin K & Leslie PW (1983) Cross-sectional growth of nomadic Turkana pastoralists. *Human Biology* **55**, 811–830.

MacWilliam KM & Dean RFA (1965) The growth of malnourished children (with kwashiorkor) after hospital treatment. *East African Medical Journal* **42**, 297–304.

Malina RM (1984) Physical activity and motor development/performance in populations nutritionally at risk. In *Energy Intake and Activity*, pp. 285–302 [E Pollitt and P Amante, editors]. New York: AR Liss.

Marmot MG & Davey Smith G (1997) Socio-economic differentials in health. The contribution of the Whitehall study. *Journal of Health Psychology* **2**, 283–296.

Martorell R (1995) Results and implications of the INCAP Follow-up Study. *Journal of Nutrition* **125**, 1127S–1138S.

Martorell R (1996) The role of nutrition in economic development. *Nutrition Reviews* **54**, 566–571.

Martorell R & Arroyave G (1988) Malnutrition, work output and energy needs. In *Capacity for Work in the Tropics*, pp. 57–75 [KJ Collins and DF Roberts, editors]. Cambridge: Cambridge University Press.

Martorell R, Delgado HL, Valverde V & Klein RE (1981) Maternal stature, fertility and infant mortality. *Human Nutrition: Clinical Nutrition* **53**, 302–312.

Martorell R, Habicht J-P & Rivera JA (1995) History and design of the INCAP Longitudinal Study (1969–77) and its Follow-Up (1988–89). *Journal of Nutrition* **125**, 1027S–1041S.

Martorell R, Kettel Khan L & Schroeder DG (1994) Reversibility of stunting: epidemiological findings in children from developing countries. *European Journal of Clinical Nutrition* **48**, S45–S57.

Martorell R, Schroeder DG, Rivera JA & Kaplowitz HJ (1995) Patterns of linear growth in rural Guatemalan adolescents and children. *Journal of Nutrition* **125**, 1060S–1067S.

Montgomery SM, Bartley MJ, Cook DG & Wadsworth MEJ (1996) Health and social precursors of unemployment in young men. *Journal of Epidemiology and Community Health* **50**, 415–422.

Norgan NG & Ferro-Luzzi A (1996) Human adaptation to energy undernutrition. In *Handbook of Physiology*. section 4. *Environmental Physiology*, pp. 1391–1409. [MJ Fregly and CM Blatteis, editors]. New York: Oxford University Press.

Pickett KE, Haas JD, Murdoch SM, Rivera JA & Martorell R (1995) Early nutritional supplementation and skeletal maturation in Guatemalan adolescents. *Journal of Nutrition* **125**, 1097S–1103S.

Pollitt E, Gorman KS, Engle PL, Rivera JA & Martorell R (1995) Nutrition in early life and fulfillment of intellectual potential. *Journal of Nutrition* **125**, 1111S–1118S.

Popkin BM, Richards MK & Montiero CA (1996) Stunting is associated with overweight children of four nations that are undergoing the nutrition transition. *Journal of Nutrition* **126**, 3009–3016.

Proos LA, Hofvander Y & Tuvemo T (1991) Menarcheal age and growth pattern of Indian girls adopted in Sweden. I. Menarcheal age. *Acta Paediatrica Scandinavica* **80**, 852–858.

Proos LA, Karlberg J, Hofvander Y & Tuvemo T (1993) Pubertal linear growth of Indian girls adopted in Sweden. *Acta Paediatrica* **82**, 641–644.

Ramakrishnan U, Martorell R, Schroeder DG & Flores R (1999) Role of intergenerational effects of linear growth. *Journal of Nutrition* **129**, 544S–549S.

Reed RB & Stuart HC (1959) Patterns of growth in height and weight from birth to eighteen years of age. *Pediatrics* **24**, 904–921.

Richardson SA (1975) Physical growth of Jamaican school children who were severely malnourished before 2 years of age. *Journal of Biosocial Science* **7**, 445–462.

Rivera JA, Martorell R, Ruel MT, Habicht J-P & Haas JD (1995) Nutritional supplementation during the preschool years influences body size and composition of Guatemalan adolescents. *Journal of Nutrition* **125**, 1068S–1077S.

Roche AF & Baumgartner RN (1988) Sex differences in fat patterning in children and youths. In *Fat Distribution during Growth and Later Health Outcomes*, pp. 85–102 [C Bouchard and FE Johnston, editors]. New York: Liss.

Rowett Research Institute (1955) *Family Diet and Health in Pre-war Britain*. Dunfermline: Carnegie United Kingdom Trust.

Satyanarayana K, Nadamuni Naidu A, Chatterjee B & Narasinga Rao BS (1977) Body size and work output. *American Journal of Clinical Nutrition* **30**, 322–325.

Satyanarayana K, Nadamuni Naidu A & Narasinga Rao BS (1978) Nutrition, physical work capacity and work output. *Indian Journal of Medical Research* **68**, Suppl., 88–93.

Satyanarayana K, Nadamuni Naidu A & Narasinga Rao BS (1979) Nutritional deprivation in childhood and the body size, activity, physical work capacity of young boys. *American Journal of Clinical Nutrition* **32**, 1769–1775.

Satyanarayana K, Nadamuni Naidu A & Narasinga Rao BS (1980*a*) Adolescent growth spurt among rural Indian boys in relation to their nutritional status in early childhood. *Annals of Human Biology* **7**, 359–365.

Satyanarayana K, Nadamuni Naidu A & Narasinga Rao BS (1980*b*) Agricultural employment, wage earnings and nutritional status of teenage rural Hyderabad boys. *Indian Journal of Nutrition and Dietetics* **17**, 281–286.

Satyanarayana K, Nadamuni Naidu A, Swaminathan MC & Narasinga Rao BS (1981) Effect of nutritional deprivation in early childhood on later growth – community study without intervention. *American Journal of Clinical Nutrition* **34**, 1636–1637.

Satyanarayana K, Prasanna Kishna T & Narasinga Rao BS (1986) Effect of early childhood undernutrition and child labor on growth and adult nutritional status of rural Indian boys around Hyderabad. *Human Nutrition: Clinical Nutrition* **40**, 131–140.

Satyanarayana K, Radaiah G, Murali Mohan R, Thimmayamma BVS, Pralhad Rao N & Narasinga Rao BS (1989) The adolescent growth spurt of height among rural Indian boys in relation to childhood nutritional background: an 18 year longitudinal study. *Annals of Human Biology* **16**, 289–300.

Schroeder DG, Martorell R & Flores R (1999) Infant and child growth and fatness and fat distribution in Guatemalan adults. *American Journal of Epidemiology* **149**, 177–185.

Schroeder DG, Martorell R, Rivera JA, Ruel MT & Habicht J-P (1995) Age differences in the impact of nutritional supplementation on growth. *Journal of Nutrition* **125**, 1051S–1059S.

Seckler D (1982) 'Small but healthy': a basic hypothesis in the theory, measurement, and policy of malnutrition. In *Newer Concepts in Nutrition and their Implications for Policy*, pp. 127–137 [PV Sukhatme, editor]. Pune, India: Maharashstra Association for the Cultivation of Science Research Institute.

Shetty PS (1984) Adaptive changes in basal metabolic rate and lean body mass in chronic undernutrition. *Human Nutrition: Clinical Nutrition* **38**C, 443–451.

Shetty PS (1993) Chronic undernutrition and metabolic adaptation. *Proceedings of the Nutrition Society* **52**, 267–284.

Shetty PS (1999) Adaptation to low energy intakes: the responses and limits to low intakes in infants, children and adults. *European Journal of Clinical Nutrition* **53**, Suppl. 1, S14–S33.

Simondon KB, Simondon F, Simon I, Diallo A, Benefice E, Traaissac P & Maire B (1998) Preschool stunting, age at menarche and adolescent height: a longitudinal study in rural Senegal. *European Journal of Clinical Nutrition* **52**, 412–418.

Soares MJ & Shetty PS (1991) Basal metabolic rates and metabolic efficiency in chronic undernutrition. *European Journal of Clinical Nutrition* **45**, 363–373.

Spurr GB (1984) Physical activity, nutritional status, and physical work capacity in relation to agricultural productivity. In *Energy Intake and Activity*, pp. 207–261 [E Pollitt and P Amante, editors]. New York: AR Liss.

Spurr GB (1988) Marginal malnutrition in childhood: implications for adult work capacity and productivity. In *Capacity for Work in the Tropics*, pp. 107–140 [KJ Collins and DF Roberts, editors]. Cambridge: Cambridge University Press.

Spurr GB (1990) The impact of chronic undernutrition on physical work capacity. In *Diet and Disease in Traditional and Developing Societies*, pp. 24–61 [GA Harrison and JC Waterlow, editors]. Cambridge: Cambridge University Press.

Spurr GE & Reina JC (1987) Marginal malnutrition in school-aged Colombian girls: dietary intervention and daily energy expenditure. *Human Nutrition: Clinical Nutrition* **41**C, 93–104.

Spurr GB, Reina JC, Dahners HW & Barac-Nieto M (1983) Marginal malnutrition in school-aged Colombian boys: functional consequences in maximal exercise. *American Journal of Clinical Nutrition* **37**, 834–847.

Tanner JM (1986) Growth as a mirror of the conditions of society: secular trends and class distinction. In *Human Growth: A Multidisciplinary Review*, pp. 3–34 [A Demirjian, editor]. London: Taylor and Francis.

Tanner JM (1989) *Foetus into Man*. Ware, Herts.: Castlemead Publications.

Torun B (1990) Short and long term effects of low or restricted energy intakes on the activity of infants and children. In *Activity, Energy Expenditure and Energy Requirements of Infants and Children*, pp. 335–358 [B Schurch and NS Scrimshaw, editors]. Lausanne, Switzerland: IDECG.

Ulijaszek SJ (1996) Long-term consequences of early environmental influences on human growth: a developmental perspective. In *Long Term Consequences of Early Environment: Growth, Development and the Lifespan Developmental Perspective*, pp. 25–43 [CJK Henry and SJ Ulijaszek, editors]. Cambridge: Cambridge University Press.

van Lenthe FJ, Kemper HCG & van Mechelen W (1996*a*) Rapid maturation in adolescence results in greater obesity in adulthood: The Amsterdam Growth and Health Study. *American Journal of Clinical Nutrition* **64**, 18–24.

van Lenthe FJ, Kemper HCG, van Mechelen W, Post GB, Twisk JWR, Welten DC & Snel J (1996*b*) Biological maturation and the distribution of subcutaneous fat from adolescence into adulthood. *International Journal of Obesity* **20**, 121–129.

Vaz M, Thangham TA, Prabhu A & Shetty PS (1996) Maximal voluntary contraction as a functional indicator of adult chronic undernutrition. *British Journal of Nutrition* **76**, 9–15.

Waaler HT (1985) Height, weight and mortality: the Norwegian experience. *Acta Medica Scandinavica* **215**, Suppl. 679.

Waterlow JC (1990) Mechanisms of adaptation to low energy intakes. In *Diet and Disease in Traditional and Developing*

Societies, pp. 5–23 [GA Harrison and JC Waterlow, editors]. Cambridge: Cambridge University Press.

Wilkinson RG (1996) *Unhealthy Societies: The Afflictions of Inequality*. London: Routledge.

Winick M, Meyer KK & Harris HC (1975) Malnutrition and environmental enrichment by early adoption. *Science* **190**, 1173–1175.

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