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EARLY FEEDING PATTERNS, GROWTH AND THE AGING PROCESS

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Abstract. In recent years, significant increases in life expectancy have occurred. This increase has been most pronounced among females. As a result, there were 156 women for every 100 men over age 75 years in the U.S.A. in 1970. The sexual disparity in life expectancies has increased throughout this century. The reasons for this trend are unknown.

Males experience higher mortality rates to both infectious and degenerative diseases. The prognosis for a male patient is generally poorer no matter what stage of the disease at the time of diagnosis or the course of treatment.

It has been shown that the early stages of male growth and development are more susceptible to external perturbations. Thus, growth-maximizing diets have their greatest impact on male children. The connection between male responsiveness to environmental influences during early growth and vulnerability in the later years is significant to the understanding of the aging process.

Key words: Nutrition, growth, aging.

Introduction: Increasing lifespan: A worldwide phenomenon

Important changes in the demographic structure of the world's population are occurring. Increases in life expectancy, most pronounced in the industrialized nations, are being experienced in most parts of the world. Iceland, where a newborn female had a life expectancy of 79.2 years and a newborn male 73 years in 1976, have experienced the greatest increase (U.N. Statistical Yearbook, 1977). Citizens of seven other countries had life expectancies equal to or greater than those prevailing that year in the U.S.A., where females could expect to live 76.5. years and males 68.7 years. All of these countries were characterized by a high level of affluence.

However, increases in life expectancy have occurred in a number of the developing countries, while in some, 30 to 40 years is still the average. (For instance, Upper Volta males had a life expectancy of 32.1 years, females 31.1 years in 1975 and in Bangladesh both sexes had life expectancies at birth of 35.8 years in 1976.) Rapid increases in life expectancy have occurred in a number of Latin American countries since World War II. In Colombia, in 1976 male life expectancy was 59.2 and female 62.7 years and in Mexico, 62.76 and 66.57, respectively. Perhaps the most impressive change has taken place in Japan where the 1976 life expectancies were 72.15 years for males and 77.35 for females. These life expectancies are associated with one of the world's

lowest crude death rates, 6.3 per thousand. In comparison, crude death rates of 8.9 in the U.S.A., 7.2 in Canada and 10.5 in France are surprisingly high. Perhaps less surprising but nonetheless noteworthy are crude death rates of 28.1 in Bangladesh, 25.8 in Upper Volta and 22.7 in Nigeria.

From these figures, it would appear that industrialization with its associated benefits of improved nutrition, public health facilities and life-long medical care has been instrumental in creating an environment capable of maximizing the human life span. There are claims of greater life expectancy occurring in certain small, remote populations in South America and the Soviet Union, but in the absence of reliable records, these claims remain unsubstantiated (MAZESS and FORMAN 1979). It should also be emphasized that despite widespread increases in life expectancy at birth there has been relatively little change in longevity, even in the most affluent populations. Thus, a 65—70 year-old male in the U.S.A. has a life expectancy no longer than would have prevailed in the year 1900 (HAYFLICK 1974).

The lack of significant increase in longevity reflects the fact that increased life expectancies are the product of reductions in the number of "untimely" deaths. A major cause of untimely deaths in human populations has for some time been infectious disease. Reduction in mortalities attributable to tuberculosis, smallpox, scarlet fever and a variety of other diseases of early onset has meant that more people survive into and through middle age. We can only speculate on the likelihood that an individual surviving to age 50 by virtue of medical control of an infectious disease will live to an advanced age. We do not know, for instance, if innate characteristics that enhance survival of a young individual exposed to infectious disease, have similar properties later in life. The pattern is obscured in part because the chief causes of morbidity in the later years have been the so-called degenerative conditions. Much remains to be learned about the role of the immune system in the later years of life (MAKINODAN 1976, 1978, 1979) but it is entirely possible the segment of today's population reaching age 50 is of a different genetic composition than the one reaching age 50 in the year 1900. Thus, we are still unable to assess the true dimensions of human longevity.

While unable to determine the true potential of the human life span, we are nonetheless confronted with consistent evidence that the female potential is greater than that of the male. The disparity appears greatest in the countries where the life expectancies are greatest, although there is no clear correlation between the two variables. For instance, the most recent figures show life expectancy for females an average of 6.4 years greater than that of males in the nine countries where life expectancies are greatest. In at least 10 countries, male life expectancies are equal to or greater than female. These are all countries where life expectancies are still short (Liberia, Nigeria, Upper Volta, Bangladesh, Kampuchea, India, Indonesia, Jordon, Sabah Malaysia, Pakistan). Table 1 gives a breakdown of these figures and Table 2 provides similar comparisons for the most affluent populations. The greater risk of childbearing in the developing countries certainly influences these figures, as may be seen by inspection of the "birth rate" and "fertility" columns of Tables 1 and 2. But it is unlikely that the stress of childbearing is the sole reason for relative similarity of male and female life expectancies in most developing countries. Other factors are undoubtedly at work in the traditional societies where women are frequently discriminated against in many ways.

Table 1

Life expectancies at birth, birth rates, fertility, death rates and infant mortalities in nations where male life expectancy equals or exceeds female life expectancy at birth

Nation	Life expectancy		Births/1000	Fertility	Death/1000	Infant mortality/1000
	Male	Female				
Upper Volta	32.10	31.10	48.5	197.0	25.8	182.0
Bangladesh	35.80	35.80	49.5	231.7	28.0	?
Nigeria	37.20	36.70	49.3	217.8	22.7	?
India	41.89	40.55	34.5	136.7	14.4	122.0
Kampuchea	44.20	43.30	46.7	143.1	19.0	127.0
Liberia	45.80	44.00	49.8	161.2	20.9	159.2
Sabah Malaysia	48.79	45.43	35.0	179.4	14.4	31.6
Indonesia	47.50	47.50	42.9	175.7	16.9	125.0
Pakistan	53.72	48.80	36.0	174.8	12.0	124.0
Jordan	52.60	52.00	47.6	206.6	14.7	36.3

Source: *Statistical Yearbook: 1977*. New York, United Nations Department of Economic and Social Affairs. 1978

Table 2

Life expectancies at birth, birth rates, fertility, death rates and infant mortalities in industrial nations of high life expectancy

Nation	Life expectancy		Births/1000	Fertility	Death/1000	Infant mortality/1000
	Male	Female				
Canada	69.34	76.36	15.8	61.6	7.2	15.0
U.S.A.	68.70	76.50	14.7	58.5	8.9	15.1
Denmark	71.10	76.80	12.9	61.3	10.7	10.3
France	69.00	76.90	13.6	72.0	10.5	10.4
Netherlands	71.20	77.20	12.9	53.9	8.3	10.5
Japan	72.15	77.35	16.4	62.6	6.3	9.3
Sweden	72.07	77.65	11.9	56.4	11.0	8.7
Norway	71.50	77.83	13.3	64.1	9.9	11.1
Iceland	73.00	79.20	19.4	81.9	6.9	11.7

Source: *Statistical Yearbook; 1977*. New York, United Nations Department of Economic and Social Affairs. 1978

Relationships of growth performance, environment and longevity

Larger body size

Increases in life expectancy have been paralleled by increases in body size. The most pronounced changes have again occurred in the industrialized countries. The most recent statistics (EVELETH and TANNER 1976) indicate that in the U.S.A. the trend toward increased body size has peaked, with mean statures remaining unchanged for the past two decades. A similar pattern appears to be emerging in the other industrialized countries including Japan. Interestingly, the "secular trend" toward increased body size occurred over a much shorter period in Japan, being largely a post World War II phenomenon there. If it has indeed peaked, it would have taken only 35 years as compared to the approximately 100 years estimated for the U.S.A. and Western Europe.

Perhaps more significant than changes in stature are the observed increases in body weight associated with the secular trend. Table 3 shows some of the changes which have occurred between 1883 and 1971 in Sweden (BENGT—OLOV et al. 1974). While there is undoubtedly an increase in adipose fat included

Table 3

The secular trend for body weight in Sweden 1883—1971

Age Center (Yr.)	1883 Mean (kg)		1938—39 Mean (kg)		1965—71 Mean (kg)	
	Male	Female	Male	Female	Male	Female
7.0	21.7	21.2	23.5	23.0		
8.0	24.5	23.3	25.9	25.1		
9.0	27.5	26.0	28.3	28.2		
10.0	29.8	28.2	31.5	31.2	32.3	32.0
11.0	31.3	30.7	34.7	34.8	35.3	35.9
12.0	33.4	33.1	38.3	39.3	39.1	40.9
13.0	36.1	37.8	42.7	44.3	43.6	46.0
14.0	40.0	42.2	48.2	49.2	49.5	49.7
15.0	44.6	46.9	54.2	53.5	55.6	52.5
16.0	49.6	50.3	60.0	56.0	60.4	54.4
17.0	55.0	53.1	63.9	57.6	65.4	
18.0	59.5	55.5	66.2	58.5		

Adapted from Bengt-Olov, L.; A. Bergsten-Brucefors and G. Lindgren (1974) "The Secular Trend in Physical Growth in Sweden." *Annals of Human Biology 1;* 245—256.

in these weight increases, much is attributable to lean body mass. The fact is that much of the increase in young adult body weight observed in the industrialized nations is in the form of skeletal muscle. Thus, substantial increases in the amount of metabolically active tissue have occurred. The increase in the net metabolic activity prevailing on a sustained basis is considerable, especially when calculated over the course of a lifetime. Comparison of a 70 kg male with a 60 kg male over a day's activities (Table 4) shows that the heavier male would require nearly 500 kcal. (2092 kilojoules) more per day. These figures are, of course, only approximations and are based on an average for young adults. Changing body composition with increasing age will reduce total energy demand, with the rate of decrement in the basal rate being nearly linear after age 20 (FORBES 1976).

Table 4

Comparison of caloric requirements of a 70 kg U.S. male with those of a 60 kg Colombian male at similar activity levels

	U.S.A.	Colombia
Mean body weight (kg)	70	60
Caloric costs (kcal)		
Resting (8 hours)	570	480
Very light activity (6 hours)	630	540
Light labor (8 hours)	1624	1392
Moderate labor (2 hours)	602	516
Total	3426	2918

Sex differences in body composition

Adult female body composition differs from male sufficiently so that basal metabolic rates are significantly different (Table 5). Body size and body composition are implicated in the lower caloric requirements of females. Sexual dimorphism in human populations reflects these differences in varying ways. For instance, male statures average as much as 110% of female in some populations and as little as 104% in others (STINI 1972a). It is noteworthy that

Table 5

Relationship of adipose tissue to body weight in adult humans (from MASORO 1975)

Age (years)	Ratio adipose tissue mass: body mass	
	Male	Female
25	0.14	0.26
40	0.22	0.32
55	0.25	0.38

greater differences between the sexes occur among the more affluent populations. Even within populations, the more affluent segments exhibit greater sexual dimorphism for stature, as seen in Jamaica (ASHCROFT, HINEAGE and LOVELL 1966) and Ethiopia (DELLAPORTAS 1969). Some caution must be exercised in interpreting these findings, however, since measurements of young-adults frequently involve females who have all completed growth while many of the males are still growing. Even under optimal circumstances, females are about 10% ahead of males in growth and maturation. When conditions are stressful, the disparity is generally greater. For a variety of reasons, female growth is more canalized than male. The significance of this becomes quite apparent when well nourished and undernourished populations are compared with reference to specific sexually dimorphic characteristics.

For instance, radiographic determinations of skeletal maturity reveal more delay in the appearances of ossification centers and the closure of epiphyses of the hand and wrist of undernourished male children and adolescents than in the case for age-matched females in the same populations (STINI 1969). Lean tissue as estimated from measurement of arm circumference and skinfolds exhibits a similar pattern of male lability in growth when compared to the relative stability of the female (STINI 1972b). Table 6 shows values for arm muscle circumference in adults in a poorly nourished South American population compared to average values for males in the U.S.A. Note the reduction in sexual dimorphism in the Colombian population, females averging 81% of male values for muscle circumference. In U.S. populations female values are only about 66% of female. Muscle circumference of Colombian males is about 66% of that in U.S. males while Colombian females have about 81% the muscle circumference of U.S. females (STINI 1975).

Evolutionary hypotheses seeking an explanation for the apparent stability of female growth compared to that of males in our species have been presented elsewhere (STINI 1971, 1975, 1977a, 1977b, 1978a, 1978b). Here the emphasis

will be primarily on the relationship between early growth patterns and longevity.

Table 6

Mean upper arm muscle area of adults of both sexes: U.S.A. and Colombian values (from STINI 1975)

	Colombia		U.S.A.		Colombia % of U.S.A.	
	Arm muscle area (mm^2)	Body wt. kg	Arm muscle area (mm^2)	Body wt. kg	Muscle area	Body weight
Male	4267	60	6464	70	66	85
Female	3450	51	4272	58	81	88
Female % of male	81	85	66	83		

Human longevity: Actual and potential

The true dimensions of human longevity remain unknown. Nonetheless there has been considerable progress toward a point where extrinsic factors such as disease, trauma and starvation no longer act as the major causes of mortality. From this point on, the intrinsic factors serving to limit the life span will increasingly act as the determinants of longevity. Where cancer and cardiovascular disease are now the predominant causes of mortality, the intrinsic limits of the human life span may already be discernible. It is possible that the evolutionary process has endowed the species with a genetically determined limit to the length of life (KROOTH 1974, CUTLER 1975, 1979, DENCKLA 1975, KANUNGO 1975, MANNING 1976, SACHER 1978). If so, it could be that the life span in contemporary affluent populations is at, or close to, the species limit, and that any future increases in human longevity could be achieved only through manipulation of intrinsic factors.

But it is also possible, that even in the most affluent, longest-lived populations presently living, the environment is not optimal. It is, for instance, possible that at the same time the control of infectious disease has allowed more individuals to live long enough to be susceptible to degenerative diseases, there is also a trend toward greater susceptibility to degenerative diseases. The information needed to identify such a trend is not easily obtained. This is because individuals susceptible to early mortality to infectious disease would not necessarily be the same individuals susceptible to degenerative disease. Too many factors remain unknown to permit useful speculation about the relative effectiveness of the immune response at various stages of the life cycle in past populations. The search for clues to the true dimensions of human longevity must rely primarly on data from living populations. Even in these populations, it is impossible to estimate how many individuals dying of malaria, cholera or influenza might have lived to great age if they had been spared.

Despite the need for caution in interpreting the information we have, it is possible that factors reducing life expectancy in older individuals can be identified and, in some case, ameliorated. Improved understanding of the etiologies of cancer and of cardiovascular disease has permitted treatment and some prologation of life. Average life expectancy in the U.S.A. might be increased by as much as 10—15 years if cardiovascular disease and cancer were eliminated (SIEGEL 1975).

What can be learned from animal experiments?

Over the past 60 years, various animal experiments have shown that certain kinds of dietary restrictions can have the effect of increasing life expectancy. Beginning with the work of OSBORNE et al. (1917), followed by that of MCKAY, CROWELL and MAYNARD (1935), and continuing through the 40's and 50's with the work of TANNENBAUM (1945, 1959), evidence of beneficial effects of a growth-restricting diet has accumulated. Other investigators have continued this work, producing a considerable body of evidence supporting the argument that dietary restriction and reduced growth are associated with lower incidence of tumors and greater life expectancies in a number of species (KRAYBILL 1963, ROSS and BRAS 1971, PAYNE 1972, WALFORD, LIU and GERBASE-DELIMA 1973, JOSE and GOOD 1973, JOSE, STUTMAN and GOOD 1973, DRORI and FOLMAN 1976, CASTER 1976, HAZZARD 1976, ROSS 1976). Recent reviews have summarized aspects of the findings of these studies from several perspectives (LEAF 1973, NATIONAL DAIRY COUNCIL 1975, HAYFLICK 1975, WATKINS 1976, WINICK 1976, STOLTZNER 1977, BARROWS and KOKKONEN 1978, HOUCK 1979) reflecting continued interest in the topic.

GOODRICK (1973, 1977a, 1977b, 1978) has worked with normal and mutant strains of mice under dietary regimes involving restriction of protein and energy intakes. Of particular interest here is his conclusion (1978) that: "The duration and rate of body weight increment . . . appear to be more important predictors of longevity than mean body weight or food intake". Phrased another way, the longer the animal grows before attainment of maximum size, the longer it will live. Stated in yet another way, growth rate and longevity are negatively correlated. Table 7 (taken from GOODRICK 1977a) shows some of the results underlying this conclusion. In addition to the conclusion of GOODRICK, it might also be pointed out that the loss of body weight occurring between the attainment of peak weight and weight during the last month of life was most pronounced in the fastest growing, shortest-lived subjects.

Slowing of the rate of growth early in life will, if sufficiently prolonged, prevent the attainment of the full genetic potential for body size. The reduction in body size under most circumstances is allometric. Thus, it may be quite pronounced without impairment of functional capacity (PAŘÍZKOVÁ and

Table 7

Mean and standard error of the mean for measures of longevity, peak body weight, body weight at last month of life span and month of peak body weight (growth duration) for mutant and control mice (from GOODRICK 1977a)

Strain	L(Months)	BW-P (g)	BW-LM (g)	GD (Months)
Beige (bg)	23.4±1.08	30.4±0.18	27.6±0.47	17.4±1.09
Albino (c^3)	24.4±1.52	32.5±0.31	32.0±0.40	22.3±1.67
Control (C57Bl/6J)	27.9±0.93	35.3±0.42	30.6±0.34	21.3±1.60
Yellow (A)	22.2±0.84	53.7±1.61	35.5±5.01	16.3±0.47
Obese (ob)	13.0±1.89	55.2±5.76	27.3±6.56	7.6±0.62

Abbreviations: L = Life Span
BW-P = Peak Body Weight
BW-LM = Body Weight at Last Month of Life Span
GD = Growth Duration

MERHAUTOVA 1970). When it occurs, the effect is a reduction of both protein and energy requirements, especially the latter (NICHOLS et al. 1972). Moreover, the organism generally adjusts its appetite to the level of its needs (WIDDOWSON and MCCANCE 1975). The result may be lifelong reduction in nutrient requirements. If a lower level of total energy consumption summed over a lifetime influences tissue changes associated with aging, one of the beneficial effects of restricted diets on longevity may be so derived. Other factors which might be implicated are the reduction of the rate of tumor growth under conditions of low protein intake (SHILS 1975) and, possibly in other cases, tumor inhibition associated with adrenal hyperfunction (ALCANTARA and SPECKMANN 1976). A variety of changes in immune function can arise from dietary modifications (GOOD 1977, TYAN 1979) so that the permutations and combinations possible in the interaction of diet, growth and disease are many. But it does appear quite possible for an organism to attain a body size considerably smaller than its genetic potential, remain viable and achieve a greater life span than its larger conspecifics under laboratory conditions. Whether this is the case in wild populations has not been shown. But, in most of the species used as experimental animals, individuals from wild populations are, on the average, smaller than domesticated strains. Without more information concerning ongevity in the wild state, little more can be said.

Aging and body composition

In humans, as in laboratory animals, the process of aging is one in which cell number decreases and metabolic rate, therefore, declines (FORBES 1974, 1976, MALINA 1969, TZANKOFF and NORRIS 1978). This does not mean that the rate of energy conversion in the individual cells has slowed. Rather, the reduction in the number of actively metabolising cells leads to a net reduction in the total number of reactions requiring energy. The maintenance requirements of most tissues, dependent upon a supply of amino acids, do not decrease as rapidly as do total energy requirements (MUNRO and YOUNG 1978, MASORO et al. 1979). Thus, an across-the-board reduction in food intake to a level appropriate to the individual's reduced energy requirements may lead to inadequate protein intake (YOUNG and GERSOVITZ 1978). Only recently have some of the phenomena associated with the decline of human body size with age begun to be understood. Muscle makes a smaller contribution to total body protein metabolism in older people than in younger ones (UANY et al. 1978, YOUNG 1968, YOUNG et al. 1963, YOUNG and GERSOVITZ 1978, YOUNG and MUNRO 1978). Usually body composition is altered favoring the acquisition of fat (ROBERTS, ANDREWS and CAIRD 1975, MASORO 1977). Thus, even when weight stays the same, metabolic rate, strength and speed of reaction all decline (CLEMENT 1974, LAMPHEARE and MONTOYE 1976, MALINA 1975, MASORO 1975). Both males and females are subject to these changes (NOVAK 1972) but females begin the aging process with a higher proportion of fat and a lower basal metabolic rate than males and their fate of decline is thus not as pronounced (BURMEISTER 1965, FLYNN et al. 1970, NOVAK 1973, NOPPA et al. 1979).

The role of exercise in age-associated changes in body composition is of considerable significance, as has been shown by studies of hospitalized individuals (MACLENNAN, MARTIN and MASON 1975) and by longitudinal studies of individuals following their normal routine of activity (ROBINSON et al. 1975). The

beneficial effects of exercise programs, even when instituted in the later years of life, indicate that some of the changes seen in the underexercised and overfed populations of industrialized nations are not entirely irreversible (SUOMINEN, HEIKKINEN and PARKATTI 1977, PAŘÍZKOVÁ 1977, PAŘÍZKOVÁ and EISELT 1971, PAŘÍZKOVÁ et al. 1971). Exercise, improved aerobic capacity and the general improvement in condition they induce give evidence that one important facet of aging is a trend toward chronic hypoxemia (CHEBTAREV, KORKUSHKO and IVANOV 1974). Certainly, performance of exceptional athletes 60 years old (POLLACK, MILLER and WILMORE 1974a) and even up to age 75 (POLLACK, MILLER and WILMORE 1974b) give evidence that the decline in physical performance need not resemble the pattern predominant in the industrialized nations (CANTWELL and WATT 1974).

Early growth, aging and death

It was pointed out earlier that life expectancy at birth has increased the most in areas where the secular increase in body size has been most pronounced (VAN WIERINGEN 1978). Moreover, these are the same areas in which menarcheal age has also been reduced to what now appears to be its minimum (TANNER 1975). Also, the populations involved are those in which sexual dimorphism for body size is most pronounced and where the difference in male and female life expectancy is greatest.

A number of factors are implicated in the pattern of growth prevalent in contemporary industrialized societies. Improved medical care and diets abundant in both protein and energy sources are essential elements. It is uncertain, however, whether the increase in body size, dimorphism and life expectancy can all be attributed to the same dietary constituents. The uncertainty is heightened by a lack of populations where Western medical care has been adopted while non-Western dietary habits have been retained. Lacking evidence to the contrary, it could be argued that dietary practices that maximize growth may also limit life expectancy (KAGAWA 1978, DOCK and FUKUSHIMA 1979).

Male growth is more affected than female by environmental factors.There is a greater increase of male body size than female arising from nutritional abundance. This raises the question of whether enhancement and acceleration of male growth are implicated in the widening disparity between male and female life expectancies. Obesity has generally (if not always accurately) been conceded to have a role in predisposing the individual to heart disease and cancer. But where fat is increasing, it is often the case that lean body mass is increasing simultaneously (FORBES 1964). Early nutritional intake appears to have long-term effects on body composition (SVEGER et al. 1975, CHARNEY et al., 1976, MELBIN and VUILLE 1976). It may endow the organism with a metabolic demand that is not only costly to satisfy but also potentially detrimental to the attainment of maximum longevity.

It has long been believed that skeletal muscle, like nervous tissue, attained the adult complement of cells at or before birth (OLIVIER 1869, HAYERN 1875, BOMPAR 1887, MACCALLUM 1898, DURANTE 1902, BRAMWELL and MUIR 1907, BABLET and NORMET 1937). However, data from other studies indicate a capacity for the addition of skeletal muscle cells early in postnatal life (CHEEK 1968, CHEEK, BRASEL and GRAYSTONE 1968, MONTGOMERY 1962a), and even

as late as the fifth decade of life (ADAMS and DERUECK 1973). Work with autopsy material from severely malnourished individuals (MONTGOMERY 1962b) gives evidence of loss of fibers already present at birth rather than a reduction in the number of new fibers appearing in the postpartum period. The result is a smaller complement of muscle fibers, each of which appears to be of normal size. Experimental work with pigs switched to low protein diets immediately after weaning (STINI 1972c) has yielded evidence that in that species, the mechanism is most likely one of fiber loss. Other work with rats (SIDRANSKY and VERNEY 1970) supports that conclusion.

Although it is not known whether mild undernutrition induces muscle fiber loss, there is substantial evidence that nutritional abundance stimulates lean body mass growth through hypertrophy, hyperplasia, or possibly both. Work with experimental animals has shown that forced excess of nitrogen in the diet of young animals will lead to retention of up to 90% of the excess (ADOLPH 1972). WINICK, BRASEL and ROSSO (1972), attributed this retension to non-hyperplastic factors. LOWRY (1973) has found similar retention levels in human infants.

CHAVEZ and his colleagues, working in a poor rural population in Mexico, recorded greater efficiency of protein intake in male children during breast-feeding (CHAVEZ et al. 1973). In their study, male infants consumed essentially the same amount of breast milk during their first year of life but gained more weight (5.13 kg in males on the average and 4.28 kg in females). CHAVEZ calculated a weight increase of 4.9 grams per gram of protein ingested by male infants and 3.7 grams per gram for females. From these data, CHAVEZ concluded that males are more efficient synthesizers of carcass protein in circumstances of low protein intake. Similar sex differences have been recorded in baboons (a species of pronounced sexual dimorphism for body size), where male efficiency of weight gain measured 4.7 grams per gram of protein and female 3.7 grams per gram. In adolescence, the human male grows much more vigorously than the female (TANNER 1975, ZORAB 1969) although the female may possess slightly more muscle in the lower limb at ages 10 or 11 years (MARESH 1970). Certainly, male increase in strength is disproportionately large after adolescence (CARRON and BAILEY 1974).

Conclusions

It would be premature to claim a direct cause-and-effect relationship between the enhancement of male growth, increased sexual dimorphism for body size and the increased disparity between the life expectancies of males and females in industrialized societies. Nor would it be possible to assert simply that body size increases are detrimental to human longevity. But, the fact remains that males have higher mortality rates throughout most of the life cycle (with the exception of ages over 75 years) (MÁDAI 1978, OMACHI 1978). Male mortalities are higher for most kinds of cancer and the prognosis for the male patient is poorer than for the female patient upon the diagnosis of cancer, whatever the stage of the disease or type of treatment (CORREA and HAENSZEL 1975).

The slowdown of increases in body size in the industrialized countries gives evidence that maximum body size is being attained. Along with increases in body size, there has been earlier attainment of adult size and sexual maturity

in both sexes. In GOODRICK's animal experiments, reductions in the time taken to attain adult body size are correlated with reductions in life span. While no correlations of this nature have yet been identified in human populations, it is not possible to rule out the possibility that acceleration of growth, maximization of body size and early attainment of sexual maturity is not an optimal outcome (STINI 1979). The human growth curve is unique in that it contains an adolescent growth spurt following 10 to 15 years of slow but steady growth. Human longevity is relatively great compared to that of other mammals and, in general, late maturity and greater longevity are correlated in mammalian species. It does appear that humans live longer than would be predicted on the basis of total caloric activity per kilogram of body weight calculated over a lifetime (CUTLER 1979). Whether the nutritional habits that prevail in modern industrialized nations have the potential of altering that relationship is an open question. The "natural" diet of humans is not known for certain (GAULEN and KONNER 1977). But it is highly unlikely that it resembled the high protein, high fat, high energy, high density one prevalent in much of the world today. It may be that dietary factors responsible for the acceleration of growth are also implicated in the failure of the affluent members of the human species to attain their full longevity.

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