Recommended dietary allowances for growth, development and performance

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Sufficient scientific evidence has accumulated in our understanding of the impact of the quality of the diet during growth and development to suggest changes in the RDAs. We now recognize that: the quality of the prenatal diet has dramatic impact on growth and development in utero, on birth weight, and on infant mortality and on morbidity during childhood; the diet of the infant, especially during the first three years, has profound effects on the intellectual and physical (work capacity) performance potential during adolescence, and affords a decrease in the probability of morbidity and mortality; the quality of the diet during growth and development throughout adolescence has a life-long potential in the thwarting of chronic degenerative diseases. The implication is a decrease in health care costs and an increase in productivity. Certain limiting nutrient RDAs will need to be updated accordingly.

Introduction:
The outstanding characteristic of childhood and adolescence is growth. Of concomitant concern is mental development and thus behavior and performance. In addition, there is the need for optimal immune competence and thus resistance to infectious diseases. There is also concern that the early stages of chronic disease pathogenesis (such as atherosclerosis, hypertension, osteoporosis) are avoided. Since nutrients, non-nutrient aspects (such as fiber, quantity and quality of fat), environmental factors and lifestyle practices are all variables, the establishment of values as recommended dietary allowances or intakes is an exceedingly complex challenge requiring the acceptance of reasonable approximations that reflect current knowledge, but also an openness to accept and implement change as new knowledge emerges. However, it should be noted that the adoption of recommended dietary allowances for regulatory purposes, like labeling, tends to stifle change and such regulatory values should be independently evolved.

Growth and Development
The pattern of growth and development in all normal children is very similar (Figures 1 and 2)[1]. However, one can question if maximal adult stature is desirable[2]. There is considerable individual variation in growth and stature, especially in the age at which the adolescent spurt in growth occurs[3]. As a general rule the earlier the menarche, the greater the spurt in growth. The spurt occurs two years later in boys than in girls, but is greater and lasts longer in boys. The rate of growth decelerates quickly after this spurt[4].

Figure 1. Physical growth (weight) percentiles for boys and girls in USA

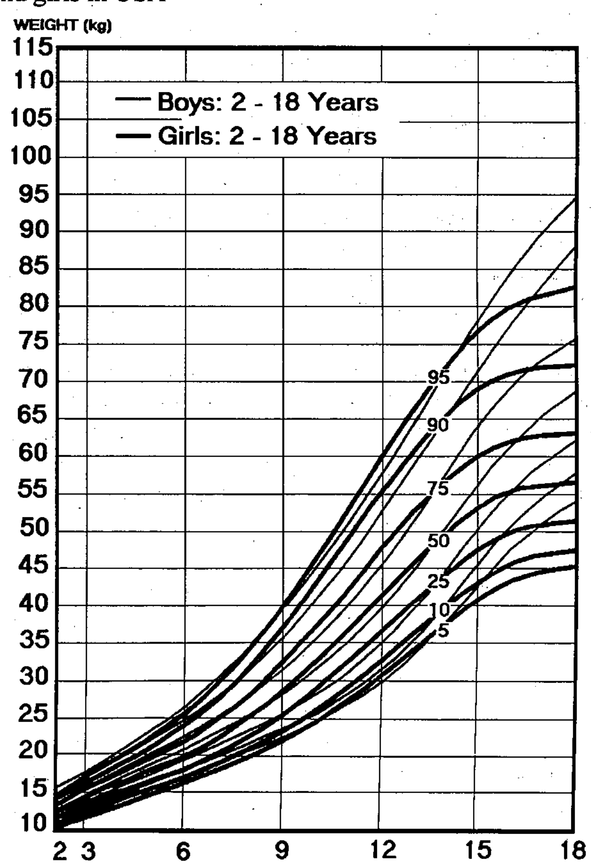

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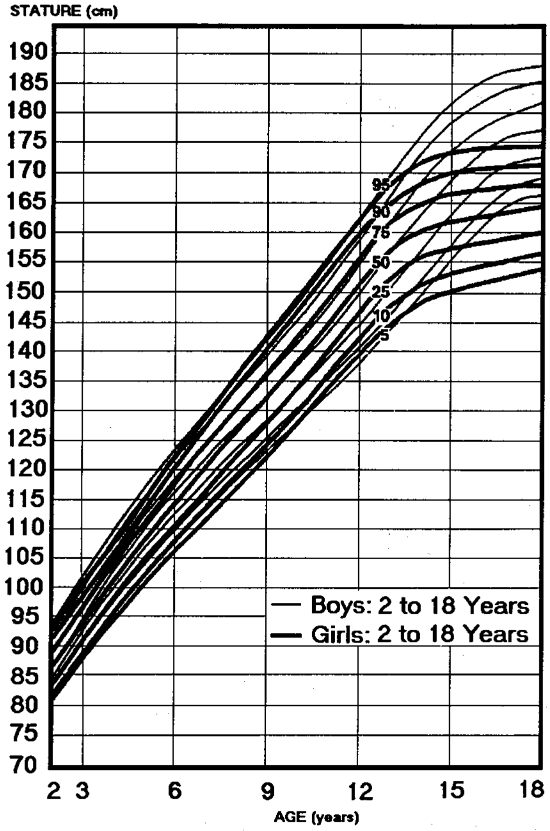
Figure 2. Physical growth (stature) percentiles for boys and girls in USA



Maturity as reflected in plateau in weight and stature occurs earlier in girls, otherwise status at earlier age is predictive of outcome. Stunting in early life can have permanent untoward outcome. Causes of stunting can be malnutrition or genetic hormonal deficiency.

Rapid development phases also occur at this time in major organ systems with the immune system exceeding that seen at any other stage in life. All systems continue development to maturity at approximately 18 years of age for women and 21 years of age for men. The question can be asked if the Recommended Dietary Allowances (RDAs) of necessary nutrients should be sufficient, or optimized, to provide for growth, spurts of growth and proper organ system development; or whether, in addition to optimal growth and development, nutrients to thwart the pathogenesis of chronic diseases should also be a consideration early in life. If the latter is to be included, the RDAs would need to be redefined, reexamined and the recommendations readjusted.

Effects of malnutrition on growth and performance

Maternal pre-pregnancy body size, which reflects maternal pre-pregnancy nutritional status, is a strong predictor of infant birth weight, infant growth and maternal postpartum nutritional status[5]. There is recent evidence[6] that there is a linear relationship between maternal diet and subsequent primitive neuroectodermal brain tumors in young children. For example, poorer diet leads to a higher incidence of tumors and vice versa. Length for age is a more accurate index of nutritional status in preschoolers than weight for age. Weight for height does not identify mild to moderately malnourished children.

Chronic mild to moderate malnutrition leads to growth faltering (stunting) very early in life. By three to four months of age, children begin to suffer permanent losses in their potential for normal growth and development[7,8]. Stunted children are more prone to illness than bigger children[9], and diarrhea and respiratory episodes are more likely to progress in severity in stunted children.

High quality diets, sufficient in energy and macronutrients are linked to positive outcomes. The high frequency consumption versus the low or nil consumption of protein and vitamins A, B_1, B_2, B_3, and iron in fortified tortilla flour over 2.5 years resulted in a 50% lower infant mortality, without a significant increase in birth weight, and 30% less morbidity in children under 5[11]. In school age children, current food intake is associated with physical and social activity level. Food intake between 7 and 9 years affects body weight, morbidity, and behavioral and cognitive development, a finding contrary to the earlier prevailing view that assumed no relationship[10].

Children's dietary intake influences their cognitive development. If iron deficiency anemia occurs during the first 2 years of life, it is associated with poor psychomotor performance and changes in behavior[12]. Infants with iron deficiency anemia show significantly lower mental and motor test scores, even after factors relating to birth, family background, and home environment are considered[12]. Preschool children who were anemic at 12 months show lower cognitive and motor scores than control children[13]. In school-age children, both past and current quality food intake are important for mental development and cognitive performance[14]. Studies have related vitamin and mineral supplementation to psychological functioning. Table 1 is a tabulation by Benton[15] of nine studies.

Table 1. Summary of studies on the effects of dietary supplementation on tests of verbal and nonverbal intelligence[15].

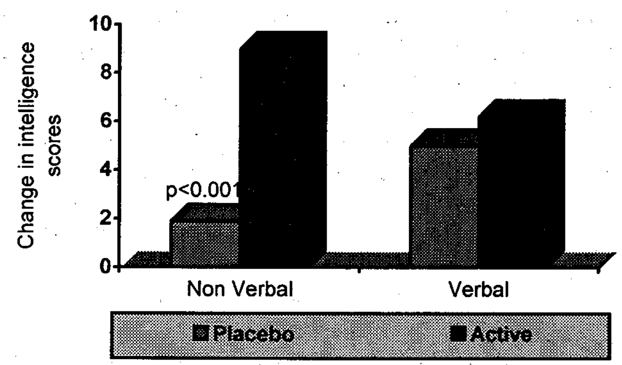
Investigators	Subjects	Age	n	Test	Response
1. Boggs et al. (1965)	Preschool (Head start)	<6	9	IQ	+
2. Schoenthaler (1987)	Young Criminals US Prisons	?	57	Mood	+
3. Benton and Roberts	British School-Children	12	60	Calvert	+
4. Schoenthaler et al. (1991)	Juvenile Offenders Oklahoma	15	40	Wechsler	+
5. Crombie et al. (1990)	British School-Children	11?	86	Calvert	N.S.
6. Benton and Buts (1990)	Belgian School-Children	13	167	Calvert	+
7. Heseker et al. (1987)	German University Students	17-29	197	Psycho-motor; Mood	+
8. Nelson et al. (1990)	British School-Children	7-12	227	IQ	N.S.
9. Benton and Cook (1991)	British School-Children	6-7	43	IQ	+

Seven of nine studies reported positive benefits. For complete citations see the Benton publication.

An example of such results from the research of Benton and Roberts[16] is given in figure 3. Sixty 12-year-old British school children were studied in a random, double blind over eight months. Non verbal intelligence IQ scores on the Calvert test were significantly higher in the students receiving the broad vitamin/ mineral supplement.

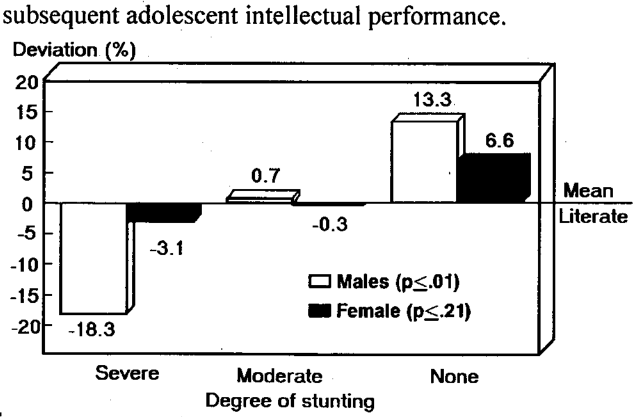
Mattorell[17] and Mattorell et al.[18] reported (Figure 4) the lagged effect of early childhood malnutrition in 18 year old males and females and was able to correlate a deviation from the norm with the degree of stunting in the males with a trend for the females. The symptoms of psychological disorders in young adult men have been related to mild to moderate vitamin deficiency[19]. In a controlled study, large doses of folate over three months to subjects with depression and lower than normal red blood cell folate, resulted in significant recovery[20]. The results of clinical trials on preschool and school-age children with iron deficiency anemia reveal that 2 months or more of dose-approved iron interventions resulted in major improvements in IQ and/ or cognitive attention span. Since there were no changes in children receiving the placebo, it appears that decrements in cognitive function are reversible[14,21]. Without exception, changes in mental development indices (such as Bayley scales) in moderate iron deficiency (Hgb < 100g/ L) have been reported in infants in both industrialized (USA, UK) and developing countries (Guatemala, Thailand, Indonesia)[21]. The mechanisms have not been determined but may possibly be attributable to brain nonheme iron levels and/ or enhanced oxygen transport.

Figure 3. Impact of vitamin/ mineral supplementation on the intelligence of 60 British schoolchildren.



Twelve-year-old schoolchildren received either a daily placebo or multi-vitamin/ mineral supplement. Data are changes in scores on verbal and nonverbal intelligence tests taken before and after 8 months of supplementation. Nonverbal intelligence was significantly improved.

Figure 4. Effect of early childhood malnutrition on subsequent adolescent intellectual performance.



Using the WHO definition, severe stunting was defined as values for length for age at 3 years 3 SDs or more below the reference median; with moderate stunting defined as values between 3 and 2 SDs below the reference median; and the absence of stunting defined as values within 2 SDs of the reference median. Deviations from the sex specific mean scores of 78.8% in males and 76.4% in females for literacy at 18 years of age was poorest for males with severe stunting at three years of age.

Work performance:

Age-adjusted physical performance means at maximum exertion revealed that oxygen consumption (VO_2Max) is 30% greater in males than in females, and increases with age in both sexes. Nutritional supplementation with an 180 ml atole (163 kcal+ 11.5g protein + vitamins and minerals) versus an 180 ml fresco (59 kcal from only sugar and no vitamins or minerals) during the first three years of life resulted in later in life (14-18.9 years of age) improvements in height and fat-free mass (especially in females) and in males the VO_2Max was significantly greater ($p < .05$) at 2.62 l/ min versus 2.30 l/ min, unrelated to the change in fat free mass[17,22,23]. In young adult males, a combined restriction of thiamin, riboflavin, pyridoxine, and vitamin C resulted in a decrease in physical performance (VO_2Max) within a few weeks[24].

There is a potential negative impact of excessive work/ exercise on growth and development[25]. Although skills may be built by repetitive exercise, the onset of puberty should occur before muscle building and endurance training activities. On the other hand, physical fitness profiles in developed countries are deteriorating and the incidence of obesity is increasing[26]. Recommendations for nutrients that promote health in the adolescent population also need to consider the change in body composition which is occurring.

Early disease prevention:

There is reason to believe that adverse prenatal nutrition experiences, such as maternal malnutrition predisposes to certain degenerative diseases in adulthood[27]. There are health care cost benefits of promoting prenatal and early life nutrition. Participation in the USDA Women, Infant and Children program versus non-participation health care savings have been measured. For every dollar of WIC program cost, a health care savings of US$1.92-$4.21 for newborns and mothers respectively has been reported[5,28].

Recommended dietary allowances:

The tenth edition of the RDA in the USA was issued in 1989. It was embroiled in controversy which will not be discussed here because it would serve no useful purpose. The Food and Nutrition Board of the Institute of Medicine, National Academy of Sciences in the United States is considering the reconceptualizing the RDAs[29,30]. The RDAs in the first 1943 edition were defined simply as "tentative goals which to aim at"..."to insure good nutrition and protection of all body tissues." In the second 1948 edition the RDAs were defined as the "levels of nutrient intakes which the Food and Nutrition Board recommends as normally desirable goals or objectives." From the 1974 edition to the present RDAs are described "as the levels of intake of essential nutrients that are adequate to meet the known nutrient needs of practically all healthy persons." Blumberg[31] asks "Can the onset of chronic diseases be delayed?" He states that research clearly suggests that for diseases considered threats to the public health (cancer, heart disease, osteoporosis), diet plays an important role. John Weisburger[32] has suggested that the RDAs should also give consideration to nutrient levels that would afford protection against environmental insults. It has been suggested that the RDAs provide antioxidant levels that afford protection from the free radical pathologies which play a role in wide variety of chronic degenerative diseases[33]. It appears that we are seeking[34] to recommend RDAs that

(1) promote optimal growth, development and cognitive as well as physical (work) performance; and

(2) delay or postpone the pathogenesis of chronic diseases stemming from

(a) endogenously generated excess free radicals (strenuous work/ exercise), and

(b) exogenously generated free radical sources (smog and occupation) which can not be otherwise controlled by lifestyle changes and reasonable environmental practices.

In other words, there is a need to select nutrient levels that benefit, by curtailing the deficiency of the nutrient, and recognizing the role the nutrient may have in thwarting immediate and longer term causes of morbidity, thus optimizing health.

The current RDAs and an outlook on change:

Table 2 presents the RDAs in the USA currently applicable to growth and development. Given the foregoing data on benefits which accrue to performance when selected nutrient levels are enhanced, I am suggesting that the following nutrient RDAs are candidates for upward revisions: all antioxidant nutrients: vitamin A (Including full recognition of carotenes, especially beta carotene or beta carotene equivalents); vitamin E; and vitamin C. Also folate (folic acid); calcium, magnesium and zinc. For the purposes of illustration, I have selected 2 vitamins for discussion: vitamin A and folate.

Vitamin A:

Prospective epidemiological data support the role of dietary vitamin A in reducing childhood mortality in developing countries[35]. Vitamin A provides an example of a nutrient that thwarts a specific morbidity (keratomalacia of the cornea and blindness) and is also believed to have an anti-infective benefit in measles. Stansfield et al.[36] reported that providing high-dose vitamin A decreased the incidence of xerophthalmia and blindness in Haitian children (6-83 months of age) but the prevalence of diarrhea increased. Underwood[37] discusses a meta-analysis of 12 studies, 6 of which improved the vitamin A status of deficient children and decreased mortality, but there was a variable effect on morbidity. This is not a new observation[38]. There is the questionable assumption that a decrease in morbidity signs should precede the observed decrease in mortality. What is more likely is that the phenomena represents a shift in the sequence of limiting nutrients. The incidence of protein energy malnutrition (PEM) is concomitantly high in such populations and the high dose irritates an already compromised gastrointestinal tract. Public health interventions need to be less tunnel-visioned and avoid the simple drug study design. In the case of vitamin A interventions, incorporating beta carotene would decrease the toxicity of the high dose, provide free radical scavenger protection, and possibly be as effective. Tunnel vision drug designs lead to unexpected negative results such as occurred in the Finnish study of beta carotene and vitamin E in long-term heavy smokers[39]. Not having a control group of non smokers, and not assuring adequate vitamin C intakes, when vitamin C is the front line in antioxidant protection, are critical oversights is such costly clinical trials. The benefits of carotenoids have been reviewed elsewhere[41]. My forecast is that the vitamin A RDA will be doubled but with at least 80% being provided by beta carotene equivalent to 5mg/ day in adults.

Folate:

It has long been suspected that folic acid is associated with neural tube formation. Neural tube defects include anencephaly and spina bifida. Folate approaching 400μg/ day appears necessary prior to conception for closure of the neural tube to occur at about the 26-28 day of fetal development[40]. Folate is also protective in cervical dysplasia in the presence of infection with the human papilloma virus[42,43]. Cervical cancer is the second most commonly occurring cancer in women worldwide and folate intakes are low throughout the life cycle. Oral contraceptive use and smoking can cause low blood levels[44]. Elevated plasma homocysteine occurs with low folate intakes and is a risk factor for occlusive vascular disease. Folate is involved in DNA and RNA synthesis and cell replication, and therefore DNA and RNA repairs necessitated by the mutagenic activity of environmental

Table 2. Recommended Dietaty Allowances in the USA (1989) for stages of growth and development

	Infants		Children			Males			Females	
Age (Years)	0.0-0.5	0.5-1.0	1-3	4-6	7-10	11-14	15-18	19-24	11-14	15-18
Weight (kg)	6	9	13	20	28	45	66	72	46	55
Height (cm)	60	71	90	112	132	157	176	177	157	163
Protein (g)	13	14	16	24	28	45	59	58	46	44
Vit A (RE)	375	375	400	500	700	1000	1000	1000	800	800
Vit D (μg)	7.5	10	10	10	10	10	10	10	10	10
Vit E (mg)	3	4	6	7	7	10	10	10	8	8
Vit K (mg)	5	10	15	20	30	45	65	70	45	55
Vit C (mg)	30	35	40	45	45	50	60	60	50	60
Thiamin (mg)	0.3	0.4	0.7	0.9	1.0	1.3	1.5	1.5	1.1	1.1
Riboflavin (mg)	0.4	0.5	0.8	1.1	1.2	1.5	1.8	1.7	1.3	1.3
Niacin (mg equiv)	5	6	9	12	13	17	20	19	15	15
Vit B6 (mg)	0.3	0.6	1.0	1.1	1.4	1.7	2.0	2.0	1.4	1.5
Folate (μg)	25	35	50	75	100	150	200	200	150	180
Vit B12 (μg)	0.3	0.5	0.7	1.0	1.4	2.0	2.0	2.0	2.0	2.0
Calcium (mg)	400	600	800	800	800	1200	1200	1200	1200	1200
Phosphorus (mg)	300	500	800	800	800	1200	1200	1200	1200	1200
Magnesium (mg)	40	60	80	120	170	270	400	350	280	300
Iron (mg)	6	10	10	10	10	12	12	10	15	15
Zinc (mg)	5	5	10	10	10	15	15	15	12	12
Iodine (μg)	40	50	70	90	120	150	150	150	150	150
Selenium (μg)	10	15	20	20	30	40	50	70	45	50

carcinogens. My forecast is that the RDA in the USA for folate will be returned to the 1980 adult value of 400μg, adjusted accordingly for age. Part of the food source for folate will be assured by requiring the existing wheat and corn flour "enrichment" regulation to include the restoration of folate loss in the milling process.

Intervention alternatives:
Given a recognized public health need, medical intervention is exceedingly costly because it is labor intensive. Introducing new agronomic practices and new foods requires generations to accomplish because the power of traditions is overriding. The fortification of existing food matrices has proven to be effective at the least cost. The fortification of wheat and corn flour, and rice was inaugurated in the USA in 1943 to curtail the recognized consequences of the prevalence of beriberi, ariboflavinosis, pellegra, and iron deficiency anemia (IDA). All but IDA became rare in a few years. Goiter and rickets were thwarted with the fortification of salt and milk respectively. Some developing countries have successfully fortified sugar with vitamin A (Guatemala and adjoining regions), and MSG with vitamin A (Indonesia). Ready to eat cereals are fortified with an array of nutrients to complement the accompanying milk. Meal replacer beverage and food bar products are nutrified with a profile of the RDA and are readily available in developed countries. The rationales and technologies of nutrient additions is available in one treatise[45].

Summary:
Sufficient scientific evidence has accumulated in our understanding of the impact of the quality of the diet during growth and development to suggest changes in the RDAs. We now recognize that:
(1) the quality of the prenatal diet has dramatic impact on growth and development in utero, on birth weight, and on infant mortality and on morbidity during childhood;
(2) the diet of the infant, especially during the first three years, has profound effects on the intellectual and physical (work capacity) performance potential during adolescence, and affords a decrease in the probability of morbidity and mortality;
(3) The quality of the diet during growth and development throughout adolescence has a life-long potential in the thwarting of chronic degenerative diseases. The implication is a decrease in health care costs and an increase in productivity. Certain limiting nutrient RDAs will need to be updated accordingly.

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Recommended dietary allowances for growth, development and performance

从生长、发育和行为考虑推荐的膳食供给量

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摘要：

制定营养素的推荐膳食供给量（ＲＤＡ）由于其涉及生长、发育、行为、免疫力和慢病预防而十分复杂。既要反映当今知识水平，又要为新知识的出现而有修改余地。作者认为最近营养科学的以下发展使人们认为需要修改某些营养素的ＲＤＡ：（１）妊娠期的膳食质量对胎儿在子宫内的生长和发育、出生体重、婴儿死亡及婴儿期患病，具有明显影响。（２）婴儿的膳食，特别是３岁以前的膳食，对青少年的智力和体力（工作能力）、行为潜能有深远的影响，而具有降低患病率和死亡率的可能性。（３）在青少年生长和发育整个期间的膳食质量对预防慢性病具有终生的影响。作者建议修改所有抗氧化营养素和维生素Ａ（胡罗卜素）、Ｅ、Ｃ以及叶酸、钙、镁和锌的ＲＤＡ。作者进一步建议维生素Ａ的ＲＤＡ应加倍，其中８０％由胡罗卜素提供；叶酸的ＲＤＡ应增加至成人每天４００微克。

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