ANNALS OF THE NEW YORK ACADEMY OF SCIENCES
Issue: *Women's and Adolescent Nutrition*
REVIEW

Nutrition in adolescents: physiology, metabolism, and nutritional needs

Jai K. Das,[1] Rehana A. Salam,[1] Kent L. Thornburg,[2] Andrew M. Prentice,[3] Susan Campisi,[4] Zohra S. Lassi,[5] Berthold Koletzko,[6] and Zulfiqar A. Bhutta[4,7]

[1]Division of Women and Child Health, Aga Khan University, Karachi, Pakistan; and South Australian Health and Medical Research Institute, and University of Adelaide, Adelaide, Australia. [2]Moore Institute for Nutrition and Wellness, Oregon Health and Science University, Portland, Oregon. [3]MRC Unit, The Gambia, and MRC International Nutrition Group, London School of Hygiene & Tropical Medicine, London, UK. [4]Centre for Global Child Health, The Hospital for Sick Children, Toronto, Ontario, Canada. [5]Robinson Research Institute, University of Adelaide, Adelaide, South Australia, Australia. [6]Ludwig Maximilians University of Munich, Munich, Germany. [7]Center of Excellence in Women and Child Health, Aga Khan University, Karachi, Pakistan

Address for correspondence: Zulfiqar A. Bhutta, Centre for Global Child Health, The Hospital for Sick Children, 686 Bay Street, Toronto, ON M6S 1S6, Canada. zulfiqar.bhutta@sickkids.ca

Adolescence is the period of development that begins at puberty and ends in early adulthood. Most commonly, adolescence is divided into three developmental periods: early adolescence (10–14 years of age), late adolescence (15–19 years of age), and young adulthood (20–24 years of age). Adolescence is marked by physical and sexual maturation, social and economic independence, development of identity, acquisition of skills needed to carry out adult relationships and roles, and the capacity for abstract reasoning. Adolescence is characterized by a rapid pace of growth that is second only to that of infancy. Nutrition and the adolescent transition are closely intertwined, since eating patterns and behaviors are influenced by many factors, including peer influences, parental modeling, food availability, food preferences, cost, convenience, personal and cultural beliefs, mass media, and body image. Here, we describe the physiology, metabolism, and nutritional requirements for adolescents and pregnant adolescents, as well as nutrition-related behavior and current trends in adolescent nutrition. We conclude with thoughts on the implications for nutrition interventions and priority areas that would require further investigation.

Keywords: adolescent nutrition; physiology; nutritional requirements; adolescents

Background

Adolescence is the period of development that begins at puberty and ends at adulthood. The World Health Organization (WHO) defines *adolescence* as age between 10 and 19 years and *youth* as between 15 and 24 years, while *young people* encompass the entire age group of 10- to 24-year-olds.[1] The recent *Lancet* commission on adolescent health and well-being further divided this time in the life cycle into three 5-year age categories: early adolescence (10–14 years), late adolescence (15–19 years), and young adulthood (20–24 years).[2] Physiologically, early adolescence is dominated by puberty and sexual development; late adolescence (15–19 years) is also characterized by pubertal maturation but less obviously than early adolescence; and young adulthood (typically 20–24 years) corresponds to the adoption of adult roles and responsibilities.[1] In some poorer populations, the initiation of puberty may be delayed and its duration extended, whereas in other poorer populations (e.g., in the United States), the initiation of puberty may be advanced, which appears to be related to body size and body fat mass.[3]

Epidemiologically, the age group from about 10 to 24 years is a quarter of the global population and, in any population, it is the healthiest of any age group and the group best able to raise the economic productivity of the resident country. In 2012, there were 1.8 billion adolescents in the world,

doi: 10.1111/nyas.13330

90% of whom lived in low- and middle-income countries (LMICs).[4] Owing to the success of child survival initiatives over the last few decades, there has been a dramatic rise in the population of adolescents, especially in LMICs, making this the largest generation of young people in history.[5]

Adolescence is a period of rapid physiological, sexual, neurological, and behavioral changes, and it lays the foundation for adopting adult roles and responsibilities, including the transition to employment and financial independence, as well as the formation of life partnerships.[6] Since it is a period of rapid growth, adequate nutrition is crucial for achieving full growth potential, and failure to achieve optimal nutrition may lead to delayed and stunted linear growth and impaired organ remodeling.[7] Iron-deficiency anemia is the leading cause of years lived with disability among children and adolescents, affecting an estimated 619 (95% confidence interval (CI), 618–621) million in 2013.[8] While undernutrition, including stunting and wasting, is on the decline in children <5 years of age, there are growing concerns about increasing rates of overweight and obesity among children and adolescents. Childhood overweight is associated with multiple immediate and long-term risks, including raised cholesterol, triglycerides, and glucose; type 2 diabetes; high blood pressure; and an elevated risk of developing adult obesity and its associated consequences.[9,10] Many LMICs now bear a double burden of malnutrition,[11,12] with the increasing burden of overweight and obesity along with existing high burden of undernutriton.[13,14]

Recently, there has been a growing interest in adolescent nutrition in developing countries as a means to improve the health of women and future generations of children on the basis of the reasoning that interventions targeted at adolescents allow time for the interventions to have the maximum impact on optimizing health in the years ahead, including the health of women during future pregnancies and hence also the related health of the next generation.

Current trends in adolescent nutrition

Body size during adolescence can be used as a proxy for nutritional status, with overnutrition manifesting as overweight and obesity, while undernutrition can manifest as stunting and/or wasting or as nutrient deficiencies without change in body size (so-called *hidden hunger*). Recent findings from the global burden of diseases and injuries among children and adolescents suggest that protein-energy malnutrition is among the top 10 causes of death among children and adolescents, accounting for 225,906 deaths in 2013.[15] Globally, around 34 deaths per 100,000 children and adolescents are attributed to malnutrition, and this number significantly varies between developing (38.5 per 100,000) and developed countries (0.2 per 100,000).[15] Global nutrition trends in adolescents have been discussed in detail in a companion paper,[16] but, briefly, overweight and obesity affect one in every three adolescents worldwide.[17] In 2011, an estimated 43 million (7%) children younger than 5 years were overweight globally, marking a 54% increase from an estimated 28 million in 1990,[2] and most of these overweight children (32 million) lived in LMICs. The prevalence of underweight among adolescent females aged 13–17 years across the five regions of the world and about 60 countries is generally less than 5%; however, in some LMICs in Africa and Asia, almost 10% or more of younger adolescent girls (13–15 years) are too thin for their age and height.[17] Though data on the burden of stunting among adolescent females are limited, it has been estimated that in some countries as many as half of all adolescents are stunted, reflecting persistent and cumulative effects of growth retardation from an early age.[18] The extent and severity of wasting among adolescents are less clear compared with the effects in children under 5 years of age.[19] Little focus has been given to micronutrient deficiencies among adolescents, despite their increased nutritional vulnerability.[20] Deficiencies in multiple micronutrients are of particular importance to adolescent health because of their direct effects, such as iron-deficiency anemia and iodine-deficiency disorders. The largest contributors to micronutrient deficiency burden among female youth globally are iron deficiency and iron deficiency–related anemia, which are responsible for about 700–1200 disability-adjusted life years per 100,000 girls in the 10–14 years age group, 300–900 in 15- to 19-year-olds, and 300–1100 among 20- to 24-year-old females.[8]

Physiology

Maturation of the body owing to hormonal changes during adolescence leads to dramatic changes in body composition.[21] These differences appear most obviously as the maturation of the sexual organs, but they also manifest in different proportions of

lean and fat body mass. While puberty usually takes place between the ages defined during adolescence, it can begin as early as 8 years of age and can extend beyond 19 years of age. Pubertal sex hormones and growth hormones generally increase together and are responsible for the enhanced skeletal growth and sexual maturation. During normal puberty, height and body weight increase (50% of adult body weight is gained during adolescence), bone mass and muscle mass increase, blood volume expands, and the heart, brain, lungs, liver, and kidney all increase in size.[22] The high rate of growth during puberty is second to that in infancy, but is greater in duration, and therefore total nutritional requirements during puberty may be greater than during any other period in life. Current evidence suggests that adolescents grow in distinct spurts that occur only rarely (about 5% of the time), as has been established for infants and young children.[23] In infants, growth spurts follow periods of sleep;[24] thus, one can speculate that adolescent sleeping patterns may accommodate growth and may require adequate energy and nutrient consumption. Adolescent sleep patterns are characterized by a natural propensity to stay up later in the evening and sleep late in the morning, and it has been argued that the chronotype of an adolescent reverses and individuals wake up naturally at an earlier time, marking the end of the adolescent period; this reversal occurs at a younger age in girls than in boys.[25] Since sleep and growth are related, it is important that robust nutritional support and opportunities for adequate sleep are considered in the health of adolescent girls and boys, who may be in the workforce and forced to sleep out of synchrony with their natural clocks.

The timing and duration of body composition changes are linked directly to sexual maturation; thus, nutritional requirements depend more on sexual maturity than on chronological age. Tanner stages[26] are most commonly used to assess sexual maturation, but other puberty stage classifications also exist. In girls, there are five Tanner stages for breast development (stages B1–B5) and pubic hair development (stages PH1–PH5). The long prepubertal Tanner stage B1 is followed by the initial breast development stage B2 at the onset of puberty, which is usually observed at 10.2 years of age but can range from 8 to 12 years of age. Transition to the next breast stage takes about 12–18 months.[27] Pubertal hair stage 3 (PH3) occurs around 11.6 years and ranges from 9.3 to 13.9 years.[28] Menarche occurs mostly at stage B4 but can extend into B5 about 2–3 years after the onset of breast development.[29] Age of menarche varies between populations, ranging from 11.0 to 14.1 years, with the mean of 12.6 years, and this has decreased significantly over the last century.[28] Height velocity peaks at B3 between 11 and 12 years of age.[30] Peak weight gain lags behind peak height velocity by 6 months and in most cases reaches 8.3 kg/year. Although height velocity decreases after menarche, final or adult height is usually achieved 1 year after menarche and also includes increased bone mineralization and fat accumulation. However, in low-income settings, the pubertal growth spurt is frequently extended, thus allowing a substantial degree of catch-up in height and weight compared with the international growth reference standards.[3]

There are five Tanner stages for genital development (stages G1–G5) and pubic hair (stages PH1–PH5) for boys. Stage G1 reflects the prepubertal stage in boys, and puberty begins with an increase in testicular volume (stage G2) at around 11.5 years but can begin as early as 9.5 years and extend to 13.3 years. The increase in testicular volume precedes the development of pubic hair. Peak height velocity occurs around PH4 and G4 at around 14 years of age.[27] Boys' height velocity begins 1–2 years after girls, and, as a result, boys' growth in height occurs for a longer period than girls, and final adult height is usually achieved by 17 years of age.[28,30] In boys, peak weight velocity occurs at about the same time as peak height velocity and averages 9 kg/year.[31]

Metabolism

Metabolism is directly related to total energy requirements and indirectly to growth and consists of energy cost of growth (ECG), basal metabolic rate (BMR), and activity energy expenditure (AEE). Figure 1 depicts the relationship between ECG, BMR, and AEE. Basal metabolism is the energy required for cellular and tissue maintenance—it increases rapidly to 2 years of age and levels off throughout adolescence[32]—while ECG is a small component compared with BMR and AEE.[33] Excessive total energy intake may lead to overweight and obesity; however, if a decreased total energy intake falls below BMR, then ECG and AEE will be compromised and can lead to growth stunting,

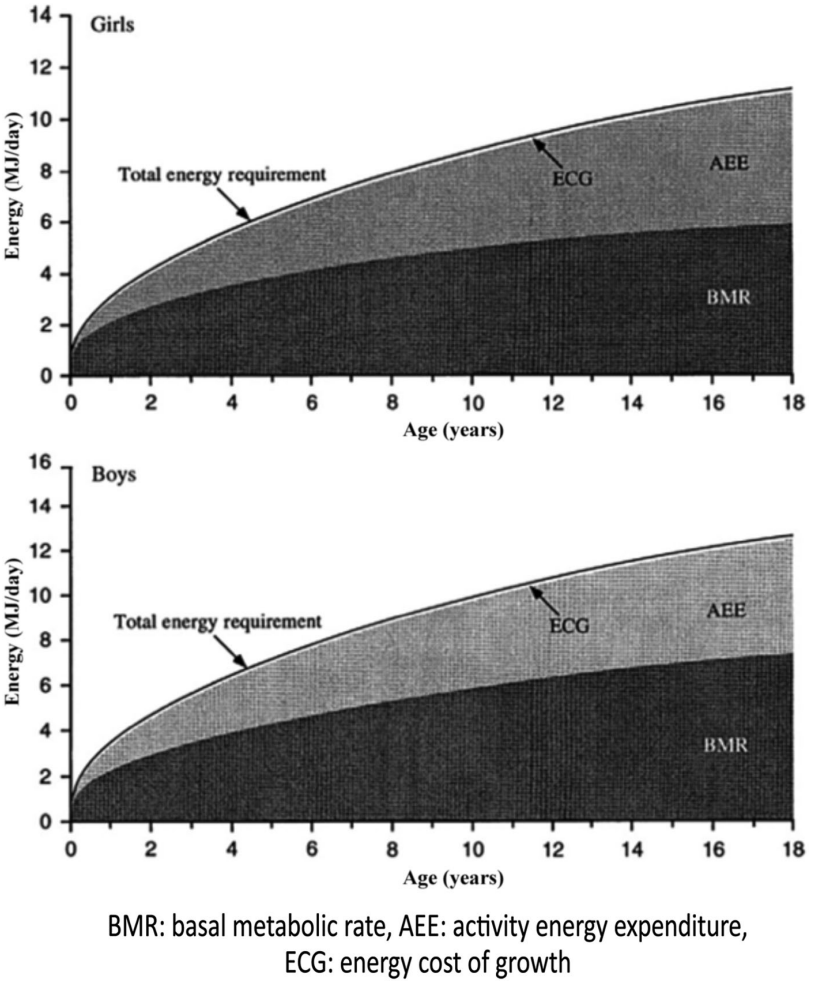

Figure 1. Energy requirements of girls and boys from birth to 18 years of age.[33]

pubertal delay, menstrual abnormalities in girls, and interference with bone mass accumulation. The WHO has endorsed the Schofield equations for estimating BMR that take into account sex, age, and body weight.[34,35] For males 10–18 years of age, BMR (megajoules (MJ)/day) = (0.074 × body weight (kg)) + 2.754 and BMR (kcal/day) = (17.69 × body weight (kg)) + 658. For females 10–18 years of age, BMR (MJ/day) = (0.056 × body weight (kg)) + 2.898 and BMR (kcal/day) = (13.38 × body weight (kg)) + 693.

Nutritional requirements during adolescence

The growth spurt in adolescence requires rapid tissue expansion with special nutrient requirements, including amino acids for growth of striated muscle, as well as calcium and vitamin D to accommodate bone growth. Energy and nutrition requirements must match the needs of the adolescents as they typically engage in physical work or recreational exercise (boys on average more than girls), which benefits striated muscle mass enlargement. Appetite increases during adolescence, and sedentary individuals are more likely to accumulate fat if they have access to high-energy food. Thus, low activity levels among adolescents are a key factor that underlies increases in adolescent obesity across the globe. The caloric requirement of adolescent males is higher than that of adolescent females, owing to greater increases in height, weight, and lean body mass. Dietary recommendations suggest

Table 1. Examples of population reference nutrient intakes: dietary reference intakes (DRIs) and adequate intakes (AIs) for adolescents in the United States[50]

DRIs and AIs: recommended intakes for adolescents: vitamins and minerals						
	Females			Males		
	9–13 years	14–18 years	19–30 years	9–13 years	14–18 years	19–30 years
Energy (kcal/day)	2071	2368	2403	2279	3152	3067
Carbohydrates (g/day)	130	130	130	130	130	130
Total fiber (g/day)	26	28	25	31	38	38
n-6 Polyunsaturated fat (g/day)	10	11	12	12	16	17
n-3 Polyunsaturated fat (g/day)	1.0	1.1	1.1	1.2	1.6	1.6
Protein (g/day)	34	46	46	34	52	56
Vitamins						
Vitamin A (μg/day)	**600**	**700**	**700**	**600**	**900**	**900**
Vitamin C (mg/day)	**45**	**65**	**75**	**45**	**75**	**90**
Vitamin D (μg/day)	**5**	**5**	**5**	**5**	**5**	**5**
Vitamin E (mg/day)	**11**	**15**	**15**	**11**	**15**	**15**
Vitamin K (μg/day)	**60**	**75**	**90**	**60**	**75**	**120**
Thiamin (mg/day)	**0.9**	**1.0**	**1.1**	**0.9**	**1.2**	**1.2**
Riboflavin (mg/day)	**0.9**	**1.0**	**1.1**	**0.9**	**1.3**	**1.3**
Niacin (mg/day)	**12**	**14**	**14**	**12**	**16**	**16**
Vitamin B_6 (μg/day)	**1.0**	**1.2**	**1.3**	**1.0**	**1.3**	**1.3**
Folate(μg/day)	**300**	**400**	**400**	**300**	**400**	**400**
Vitamin B_{12} (μg/day)	**1.8**	**2.4**	**2.4**	**1.8**	**2.4**	**2.4**
Pantothenic acid (mg/day)	**4**	**5**	**5**	**4**	**5**	**5**
Biotin (μg/day)	**20**	**25**	**30**	**20**	**25**	**30**
Choline (mg/day)	**375**	**400**	**425**	**375**	**550**	**550**
Elements						
Calcium (mg/day)	1300	1300	1000	1300	1300	1000
Chromium (μg/day)	21	24	25	25	35	35
Copper (μg/day)	700	890	900	700	890	900
Fluoride (mg/day)	2	3	3	2	3	4
Iodine (μg/day)	120	150	150	120	150	150
Iron (mg/day)	8	15	18	8	11	8
Magnesium (mg/day)	240	360	310	240	410	400
Manganese(mg/day)	1.6	1.6	1.8	1.9	2.2	2.3
Molybdenum (μg/day)	34	43	45	34	43	45
Phosphorus (mg/day)	1250	1250	700	1250	1250	700
Selenium (μg/day)	40	55	55	40	55	55
Zinc (mg/day)	8	9	8	8	11	11

Note: This table presents RDAs in bold type and AIs in ordinary type. RDAs and AIs may both be used as goals for individual intake. RDAs are set to meet the needs of almost all (97–98%) individuals in a group. The AI is believed to cover needs of all adolescents in the group, but lack of data or uncertainty in the data prevents the ability to specify with confidence the percentage of individuals covered by this intake. The data are derived from reports from the Institute of Medicine, Food and Nutrition Board, Standing Committee on the Scientific Evaluation of Dietary Reference Intakes,© by the National Academy of Sciences, courtesy of the National Academies Press, Washington, DC. (http://www.nap.edu/)

that 50% or more of total daily calories should come from carbohydrates, with no more than 10–25% of calories derived from sugars, such as sucrose and high fructose corn syrup.[36] Protein requirements per unit of height are highest for females in the 11- to 14-year age range and for males in the 15- to 18-year age range, corresponding to the usual timing of peak height velocity. Population reference intakes, for example, the dietary reference intakes (DRIs) of the United States, do not list specific requirements

for total fat intake, but do make recommendations for the intake of linoleic (*n*-6) and α-linolenic (*n*-3) polyunsaturated fatty acids (Table 1).

The U.S. DRI for calcium for 9- to 18-year-olds is 1300 mg/day, and the recommended dietary allowance (RDA) for iron is 8 mg/day for 9- to 13-year-olds, 11 mg/day for males aged 14–18, and 15 mg/day for females aged 14–18 years, as the onset of menstruation imposes additional iron needs for girls. The U.S. RDA for zinc for males and females aged 9–13 is 8 mg/day, while for males and females aged 14–18 years the RDAs are 11 and 9 mg/day, respectively. To ensure adequate body stores of vitamin A, boys and girls aged 9–13 years should consume 600 μg/day, females aged 14–18 years should consume 700 μg/day, and males aged 14–18 years should consume 900 μg/day. For the United States, the RDA for vitamin E for 9- to 13-year-olds is 11 mg/day and is 15 mg/day for 14- to 18-year-olds. The U.S. RDA for vitamin C is 45 mg/day for 9- to 13-year-olds, 75 mg/day for males aged 14–18 years, and 65 mg/day for females aged 14–18 years. The U.S. RDA for folate is 300 μg/day for 9- to 13-year-olds and 400 μg/day for 14- to 18-year-olds. Findings from a recent systematic review[37] evaluating nutrition interventions for adolescents suggest that micronutrient supplementation among adolescents can significantly decrease anaemia prevalence (relative risk (RR): 0.69; 95% CI: 0.62–0.76).[37]

Nutritional requirements for adolescent pregnancy

Sixteen million babies are born annually to adolescent girls 15–19 years of age, which accounts for over 10% of the total births each year. Adolescent fertility is three times higher in LMICs owing to various contextual factors, including traditional marriage practices, poverty, lack of education and employment, restricted access to care, weak health systems, abuse, unplanned or unwanted pregnancies, and the absence of autonomy or support in their social arrangements. One area of human biology in need of more research is the competition for nutrients between mother and fetus in pregnancies where mothers are still growing. Several studies have shown that adolescent women are able to grow during pregnancy if their nutrition is adequate,[38,39] while others have shown that pregnancy may limit maternal growth.[40] Studies in adolescent sheep show that excess calories given during pregnancy will be allocated to the mother's growth over the growth of the fetus;[41] the degree to which this is true in humans has not been adequately studied. Existing data suggest that, when energy is constrained, the physiology of younger adolescents invests in growth, while that of older adolescent females privileges reproductively valuable adipose tissue.[42]

When an adolescent becomes pregnant, there is increased competition for nutrients with the fetus, and pregnant adolescents are at higher risk for becoming stunted[43,44] and at elevated risk of adverse neonatal outcomes, including low birth weight (LBW), preterm delivery, anemia, and postpartum outcomes, like excessive weight retention, owing to a combination of physiological, socioeconomic, and behavioral factors.[45,46] Adolescent girls are two to five times more likely to die from pregnancy-related causes than women aged 20–29 years.[47] Girls younger than 19 years of age have a 50% increased risk of stillbirths and neonatal deaths, as well as an increased risk for preterm birth, LBW, and asphyxia. These health risks further increase for girls who become pregnant earlier than 15 years of age and are somewhat reduced for older adolescents aged 18–19 years. Nutrition in pregnant teens is crucial, since their bodies are not physically ready for pregnancy and they tend to give low priority to nutrition despite having enhanced needs for nutrients owing to their pregnant state. Prepregnancy low body mass index (<18.5) significantly increases perinatal risks, including stillbirths, preterm births, and small for gestational age and LBW babies.[2] For a lactating mother, her micronutrient status determines the health and development of her breast-fed infant, especially during the first 6 months of life.[47,48] Nutrient needs during pregnancy and lactation are higher relative to other physiological stages in the life cycle and, likewise, the requirements for most micronutrients are also higher. Findings from a recent systematic review suggest that interventions to improve the nutritional status of pregnant women result in a statistically significant improvement in mean birth weight (SMD: 0.25; 95% CI: 0.08–0.41), reduced LBW (birth weight < 2500 g; RR: 0.70; 95% CI: 0.57–0.84), and preterm birth (before 37 weeks; RR: 0.73; 95% CI: 0.57–0.95). The intervention strategies included the provision of micronutrient supplementation, such as calcium

Table 2. Examples of population reference nutrient intakes: reference dietary allowances for pregnant adolescents for the United States[50]

Components	Unit	RDA		
		Prepregnancy	Pregnancy	Lactation
Macronutrients				
Protein (g)	g	60	71	71
Fat (g)	g	ND	ND	ND
Omega-6	g	12	13	13
Omega-3	g	1.1	1.4	1.3
Carbohydrates (g)	g	130	175	210
Micronutrients				
Vitamin A (retinol)	μg	700	770	1300
Vitamin B_1 (thiamin)	mg	1.1	1.4	1.4
Vitamin B_2 (riboflavin)	mg	1.1	1.4	1.6
Vitamin B_3 (niacin)	mg	14	18	17
Vitamin B_5 (pantothenic acid)	mg	5	6	7
Vitamin B_6 (pyridoxine)	mg	1.3	1.9	2.0
Vitamin B_7 (biotin)	μg	30	30	35
Vitamin B_9 (folate)	μg	400	600	500
Vitamin B_{12} (cobalamine)	μg	2.4	2.6	2.8
Vitamin C (ascorbate)	mg	75	85	120
Vitamin D (cholecalciferol)	IU	600	600	600
Vitamin E (tocopherol acetate)	mg	15	15	19
Vitamin K (phytomenadione)	μg	90	90	90
Calcium	mg	1300	1300	1300
Copper	μg	900	1000	1300
Iodine	μg	150	220	290
Iron	mg	18	27	10
Magnesium	mg	360	400	360
Selenium	μg	55	60	70
Zinc	μg	9	12	13

and zinc, in addition to the routine iron–folic acid supplementation to adolescent mothers, or engaging them in nutritional education sessions to enable them to improve nutritional intake. Long-term nutritional counseling was frequently employed whereby pregnant adolescents would have access to a nutritionist whom they would consult as part of antenatal care. Table 2 describes the RDAs for prepregnant, pregnant, and lactating women.

It has become increasingly clear that several conditions in pregnancy predispose both mother and offspring for later disease, including hypertension, cardiovascular disease, and type 2 diabetes.[49] In the general population, it appears that teenagers are less likely to acquire preeclampsia than older pregnant women. However, prepregnancy obesity and excessive weight gain during pregnancy dramatically increase the risk for preeclampsia among adolescents.[50,51] The fact that the prevalence of obesity among adolescent girls is increasing worldwide imposes a need to develop a heightened awareness of the health implications for current and future generations. Thus, prevention of overweight and management of obesity through lifestyle and nutritional programs is urgent and should be a top-priority topic for international bodies.

In a systematic review, Bellamy estimated that the relative risk for a mother who suffered preeclampsia at any age to acquire hypertension within 14 years was 3.7 (95% CI: 2.70–5.05), to acquire ischemic heart disease and stroke within 10 years was 2.16 (95% CI: 1.86–2.52) and 1.81 (95% CI: 1.45–2.27), respectively, and to acquire thromboembolic venous disease within 5 years was 1.79 (95% CI: 1.37–2.33). Thus, one can predict that the global trend for excess weight gain among adolescent girls will lead to elevated rates of diabetes and heart disease among affected women and their

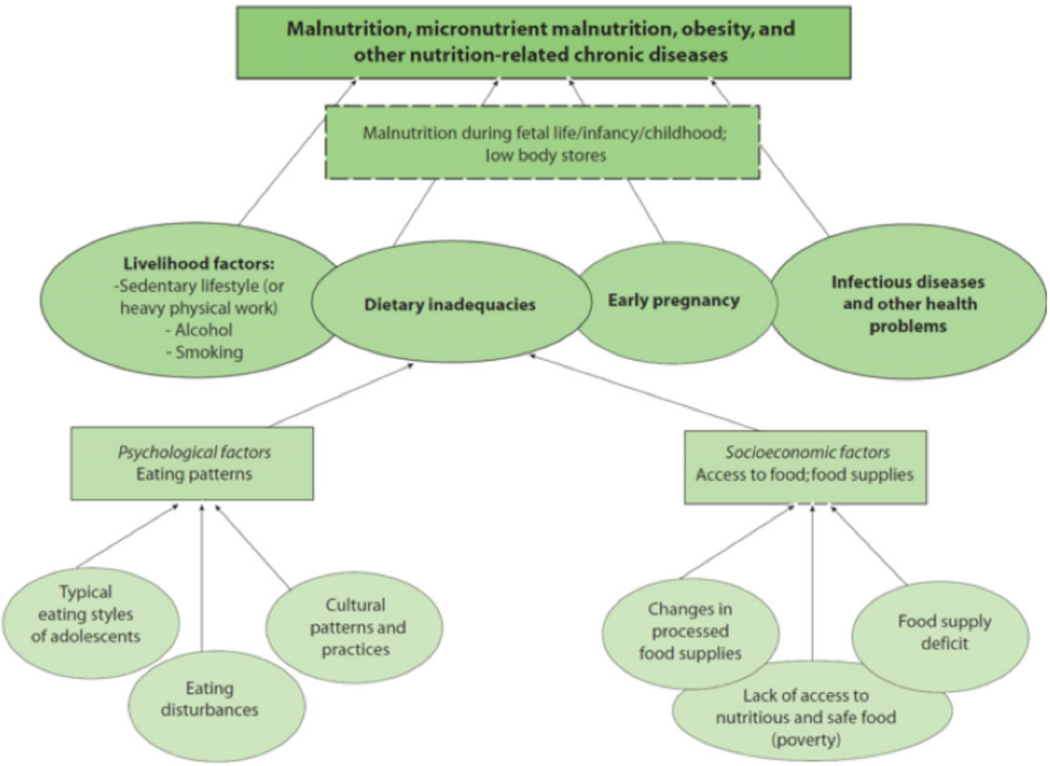

Figure 2. Adolescent transition and nutrition: a complex interaction. Adapted from Ref. 31.

offspring. Adolescents who were exposed to maternal preeclampsia as fetuses showed structural and functional changes in their hearts, including greater relative wall thickness and reduced left ventricular end-diastolic volume compared with controls.[52] Because at-risk groups of adolescent girls more often have LBW babies and babies born prematurely or suffering neonatal death,[53,54] survivors will have elevated risks for chronic conditions, including hypertension over the life span,[55] early-onset renal disease,[56] type 2 diabetes,[57] mental disorders,[58] and many other chronic conditions. [59]

Adolescent behavior and nutrition

Eating patterns and behaviors are influenced by many factors during adolescence, including peer influences, parental modeling, food availability, food preferences, costs, convenience, personal and cultural beliefs, mass media, and body image.[60] These could be broadly classified as *personal factors*, including attitudes, beliefs, food preferences, self-efficacy, and biological changes; *environmental factors*, including family, friends, peer networks, school, fast food outlets, and social and cultural norms; and *macrosystem factors*, including food availability, food production, distribution systems, mass media, and advertising (Fig. 2).[60,61] Teens as a group tend to snack and graze, miss meals, eat away from home, consume fast foods, and diet (especially females) more frequently than younger children.[60,62] Nutrition surveys show that many adolescents have inadequate intakes of vitamins and minerals, which is more pronounced in females than in males.[63] Recently, there has been an increase in the trend toward excess consumption of total fat and saturated fat, cholesterol, sodium, and sugar. There appears to be an increasing prevalence of obesity among adolescents worldwide, explained by widespread nutrition transitions to lipid-rich diets and a decrease in physical activity, especially among urban adolescents.[62,63] Other unhealthy behaviors, such as smoking, drinking, and illicit drug use, often begin during adolescence and are closely related to physiological and nutritional aspects.[64,65] Anorexia nervosa (AN) and bulimia nervosa (BN) are two specified eating disorders: about 0.3%

of adolescents aged 13–18 years have AN, 0.9% have BN, and 1.6% have a binge-eating disorder.[66] AN is relatively more common among young women.[67]

Recent studies into adolescent eating behaviors suggest that personal factors identified during adolescence were found to be predictive of both persistent dieting and disordered eating from adolescence into young adulthood, as well as initiation of these behaviors during young adulthood.[63,68] Body image is important in adolescence, and disturbances are in relation to obesity, dietary disorders, and psychological discontent. Many theories have been proposed to explain body image disturbances and their link with eating disorders, but most researchers appear to agree that the strongest influence in Western societies is the sociocultural factor, the theory best supported by available data.[69] A recent systematic review evaluating variations in population-level physical activity in European children and adolescents suggests that the reported levels of physical activity and prevalence of compliance to physical activity recommendations in youth vary widely across European countries owing to variation in physical activity as well as variation in assessment methods and reported outcome variables.[70] Over the last decade, many studies have been conducted to evaluate the impact of lifestyle changes, health behavior modifications, and nutrition interventions among children, adolescents, and youth in various population groups.[71–76] These include the European Youth Heart Survey (EYHS), the Healthy Lifestyle in Europe by Nutrition in Adolescence (HELENA) study, the Identification and Prevention of Dietary and Lifestyle Induced Health Effects in Children and Infants (IDEFICS) study, the Health Behavior in School Ages Children (HBSC) study, and the Dortmund Nutritional and Anthropometric Longitudinally Designed Study (DONALD). The focus of all these large cohorts has been to study the risk factors associated with noncommunicable diseases in later life and identify the effective integrated lifestyle, nutrition, and behavioral interventions to address these risk factors. More recently, there has been an epidemiological transition in LMICs, since these countries now bear a double burden of undernutrition and overnutrition.[77,78] However, there is a lack of rigorous nutrition program evaluations and large study cohorts from these countries,[79] and most of the large-scale initiatives exist in Brazil, Mexico, and Chile, where there have been efforts to systematically address obesity.[79]

Beyond the behavioral context, there are other social contexts that directly or indirectly affect adolescent nutrition, growth, and development. These include issues like child labor, food security, poverty, conflicts, and humanitarian emergencies.[80] The recent *Lancet* Series on Adolescent Health suggested that structural factors, such as national wealth, income inequality, and access to education, are the strongest determinants of adolescent health.[81] Furthermore, families, peers, institutions (like schools), and the broader social environment are also strong predictors of adolescent health.[81] However, these issues are not discussed in detail here since these are out of the scope of the review.

Future research areas

Adolescents are vulnerable and deserve special attention in nutrition because adult health can be affected by nutrition during adolescence. Adolescence is a time for catch-up growth; the extent to which pubertal growth can contribute to stunting recovery at this time is under much investigation, but studies of children who migrated or were adopted may offer more insight into later linear growth increases.[3,82,83] It has become increasingly important to target this vulnerable group, as today's adolescents are more exposed to nutritional risks, harmful alcohol consumption, sexually transmitted diseases, and other risks than in the past and face other new challenges, such as social media.[84–86] Additionally, wide variations exist between and within regions in adolescent health profiles in relation to the prevalence of risk factors for noncommunicable diseases in adulthood, substance abuse, overweight, and sedentary lifestyles.[86] There is a need to better define and measure adolescent health indicators and extend the data coverage for this specific subgroup.[86] Future work is needed to better understand the adolescent nutrition needs in varying contexts, its possible prediction by factors in early life, such as imprinting of eating behavior, taste preferences, and food choices, and its relation to physiology of sleep, physical activity, puberty, growth spurt, and adult health (Box 1), without undermining the fact that adolescents should be the focus of future policy and implementation, as investing in this generation can yield dividends for generations to come.

Box 1: High-priority areas of investigation among adolescents

- Delineation of nutrition requirements for adolescents (early (12–15) and late (15–19)) across various contexts, including LMICs
- Identification of modifiable predictors of adolescent nutritional practice, such as early imprinting of eating behavior and taste and food preferences
- The physiology of pregnancy among adolescent girls who have different dietary histories and body composition
- Impact of adolescent nutritional practice on subsequent pregnancies, pregnancy outcomes, and offspring health
- Relationship of nutrition status and interventions to growth spurt and final height in adolescence
- Relationship of nutrition status to puberty onset and development
- Relationship of sleep and activity patterns with nutritional status in adolescence
- Role of intervening in adolescents for healthy reversal of stunting
- Role of nutrition in augmentation of brain development in previously malnourished adolescents
- Determining the likelihood of obesity and diabetes and wasting among adolescent girls with different nutritional histories

Acknowledgments

All authors contributed to writing this manuscript. The views presented in this paper are the personal views of the authors, who contributed their time for this project gratis.

Competing interests

The authors declare no competing interests.

References

1. Patton, G.C., S.M. Sawyer, J.S. Santelli, *et al.* 2016. Our future: a Lancet commission on adolescent health and well-being. *Lancet* **387:** 2423–2478.
2. Black, R.E., C.G. Victora, S.P. Walker, *et al.* 2013. Maternal and child undernutrition and overweight in low-income and middle-income countries. *Lancet* **382:** 427–451.
3. Avenell, A., W.J. Gillespie, L.D. Gillespie & D. O'Connell. 2005. Vitamin D and vitamin D analogues for preventing fractures associated with involutional and post-menopausal osteoporosis. *Cochrane Database Syst. Rev.* **3:** CD000227. doi: 10.1002/14651858.CD000227.pub3.
4. Cappa, C., T. Wardlaw, C. Langevin-Falcon & J. Diers. 2012. Progress for children: a report card on adolescents. *Lancet* **379:** 2323–2325.
5. United Nations, Department of Economic and Social Affairs, Population Division. 2015. World population prospects: the 2015 revision, key findings and advance tables.Working Paper No. ESA/P/WP.241.
6. Hellerstedt, W.L. 2005. Adolescent growth and development: Trends in Adolescent Sexual Behavior, Contraceptive Use, Pregnancy, and Pregnancy Resolution. In *Guidelines for Adolescent Nutrition Services.* M. Story & J. Stang, Eds.: Chapter 1. Minneapolis, MN: Center for Leadership, Education and Training in Maternal and Child Nutrition, Division of Epidemiology and Community Health, School of Public Health, University of Minnesota. http://www.epi.umn.edu/let/pubs/adol_book.shtm.
7. Story, M. 1992. Nutritional requirements during adolescence. In *Textbook of Adolescent Medicine.* E.R. McAnarney, R.E. Kreipe, D.E. Orr & G.D. Comerci, Eds.: 75–84. Saunders.
8. Patton, G.C., C. Coffey, S.M. Sawyer, *et al.* 2009. Global patterns of mortality in young people: a systematic analysis of population health data. *Lancet* **374:** 881–892.
9. Koplan, J.P., C.T. Liverman & V.I. Kraak. 2005. Preventing childhood obesity: health in the balance: executive summary. *J. Am. Diet. Assoc.* **105:** 131–138.
10. Lloyd, L.J., S.C. Langley-Evans & S. McMullen. 2012. Childhood obesity and risk of the adult metabolic syndrome: a systematic review. *Int. J. Obes.* **36:** 1–11.
11. Lobstein, T., L. Baur & R. Uauy. 2004. Obesity in children and young people: a crisis in public health. *Obes. Rev.* **5:** 4–85.
12. Popkin, B.M. & P. Gordon-Larsen. 2004. The nutrition transition: worldwide obesity dynamics and their determinants. *Int. J. Obes.* **28:** S2–S9.
13. Nichols, M.S., A.M. de Silva-Sanigorski, J.E. Cleary, *et al.* 2011. Decreasing trends in overweight and obesity among an Australian population of preschool children. *Int. J. Obes.* **35:** 916–924.
14. Rokholm, B., J.L. Baker & T.I. Sørensen. 2010. The levelling off of the obesity epidemic since the year 1999—a review of evidence and perspectives. *Obes. Rev.* **11:** 835–846.
15. Collaboration GBoDP. 2016. Global and national burden of diseases and injuries among children and adolescents between 1990 and 2013. *JAMA Pediatr.* **170:** 267–287.
16. Thomas, R.E., M. Russell & D. Lorenzetti. 2010. Interventions to increase influenza vaccination rates of those 60 years

and older in the community. *Cochrane Database Syst. Rev.* **9:** CD005188. doi: 10.1002/14651858.CD005188.pub2.
17. World Health Organization. 2014. World health statistics 2014. http://apps.who.int/iris/bitstream/10665/112738/1/9789240692671_eng.pdf. Accessed October 15, 2016.
18. Khara, T. & E. Mates. 2015. *Adolescent nutrition: policy and programming in SUN+ countries.* Save the Children, London. Accessed March 10, 2017. https://www.savethechildren.org.uk/sites/default/files/images/Adolescent_Nutrition.pdf.
19. UNICEF. 2013. Improving child nutrition: the achievable imperative for global progress. New York. UNICEF.
20. PAHO. 2011. Underweight, short stature, and overweight in adolescents and young women in Latin America and the Caribbean. Accessed October 10, 2016. http://www.paho.org/hq/index.php?option=com_docman&task=cat_view&gid=3426&limit=10&limitstart=0&order=hits&dir=ASC&Itemid=3482&lang=fr.
21. Eckert, K.L., V.A. Loffredo & K. O'Connor. 2009. Adolescent physiology. In *Behavioral Approaches to Chronic Disease in Adolescence.* W.T. O'Donohue & L.W. Tolle, Eds.: 29–45. Springer.
22. Corkins, M.R., S.R. Daniels, S.D. de Ferranti, *et al.* 2016. Nutrition in children and adolescents. *Med. Clin. North Am.* **100:** 1217–1235.
23. Lampl, M., J.D. Veldhuis & M.L. Johnson. 1992. Saltation and stasis: a model of human growth. *Science* **258:** 801–803.
24. Lampl, M. & M.L. Johnson. 2011. Infant growth in length follows prolonged sleep and increased naps. *Sleep* **34:** 641–650.
25. Roenneberg, T., T. Kuehnle, P.P. Pramstaller, *et al.* 2004. A marker for the end of adolescence. *Curr. Biol.* **14:** R1038–R1039.
26. Tanner, J.M. 1962. *Growth at Adolescence.* Oxford: Blackwell.
27. Robeva, R. & P. Kumanov. 2016. Physical changes during pubertal transition. In *Puberty: Physiology and Abnormalities.* P. Kumanov & A. Agarwal, Eds.: 39–64. Cham: Springer International Publishing.
28. Bordini, B. & R.L. Rosenfield. 2011. Normal pubertal development: part II: clinical aspects of puberty. *Pediatr. Rev.* **32:** 281–292.
29. Parent, A.S., G. Teilmann, A. Juul, *et al.* 2003. The timing of normal puberty and the age limits of sexual precocity: variations around the world, secular trends, and changes after migration. *Endocr. Rev.* **24:** 668–693.
30. Eckert, K.L., V.A. Loffredo & K. O'Connor. 2009. Adolescent physiology. In *Behavioral Approaches to Chronic Disease in Adolescence: A Guide to Integrative Care.* W.T. O'Donohue, Ed.: 29–45. New York, NY: Springer.
31. Fellmeth, G.L., C. Heffernan, J. Nurse, *et al.* 2013. Educational and skills-based interventions for preventing relationship and dating violence in adolescents and young adults. *Cochrane Database Syst. Rev.* **6:** CD004534.
32. Holliday, M. 1971. Metabolic rate and organ size during growth from infancy to maturity and during late gestation and early infancy. *Pediatrics* **47:** 169–179.
33. Norgan, N.G., B. Bogin & N. Cameron. 2012. Nutrition and growth. In *Human Growth and Development.* 2nd ed. N. Cameron & B. Bogin, Eds.: 123–152. Boston: Academic Press.
34. Schofield, W. 1984. Predicting basal metabolic rate, new standards and review of previous work. *Hum. Nutr. Clin. Nutr.* **39:** 5–41.
35. Schofield, C. 1985. Annotated bibliography of source material for basal metabolic rate data. *Hum. Nutr. Clin. Nutr.* **39** (Suppl. 1): 42–91.
36. Hellerstedt, W.L. 2005. Adolescent growth and development: Trends in Adolescent Sexual Behavior, Contraceptive Use, Pregnancy, and Pregnancy Resolution. In *Guidelines for Adolescent Nutrition Services.* M. Story & J. Stang, Eds.: 1–8. Minneapolis, MN: Center for Leadership, Education and Training in Maternal and Child Nutrition, Division of Epidemiology and Community Health, School of Public Health, University of Minnesota. http://www.epi.umn.edu/let/pubs/adol_book.shtm.
37. Salam, R.A., M. Hooda, J.K. Das, *et al.* 2016. Interventions to improve adolescent nutrition: a systematic review and meta-analysis. *J. Adolesc. Health* **59:** S29–S39.
38. Scholl, T.O. & M.L. Hediger. 1993. A review of the epidemiology of nutrition and adolescent pregnancy: maternal growth during pregnancy and its effect on the fetus. *J. Am. Coll. Nutr.* **12:** 101–107.
39. Scholl, T.O., M.L. Hediger & I.G. Ances. 1990. Maternal growth during pregnancy and decreased infant birth weight. *Am. J. Clin. Nutr.* **51:** 790–793.
40. Rah, J.H., P. Christian, A.A. Shamim, *et al.* 2008. Pregnancy and lactation hinder growth and nutritional status of adolescent girls in rural Bangladesh. *J. Nutr.* **138:** 1505–1511.
41. Wallace, J.M., R.P. Aitken, J.S. Milne & W.W. Hay Jr. 2004. Nutritionally mediated placental growth restriction in the growing adolescent: consequences for the fetus. *Biol. Reprod.* **71:** 1055–1062.
42. Reiches, M.W., S.E. Moore, A.M. Prentice, *et al.* 2013. The adolescent transition under energetic stress: body composition tradeoffs among adolescent women in The Gambia. *Evol. Med. Public Health* **2013:** 75–85.
43. Gigante, D.P., K.M. Rasmussen & C.G. Victora. 2005. Pregnancy increases BMI in adolescents of a population-based birth cohort. *J. Nutr.* **135:** 74–80.
44. Rah, J.H., P. Christian, A.A. Shamim, *et al.* 2008. Pregnancy and lactation hinder growth and nutritional status of adolescent girls in rural Bangladesh. *J. Nutr.* **138:** 1505–1511.
45. Hediger, M.L., T.O. Scholl & J.I. Schall. 1997. Implications of the Camden Study of adolescent pregnancy: interactions among maternal growth, nutritional status, and body composition. *Ann. N.Y. Acad. Sci.* **817:** 281–291.
46. Strobino, D.M., M.E. Ensminger, Y.J. Kim & J. Nanda. 1995. Mechanisms for maternal age differences in birth weight. *Am. J. Epidemiol.* **142:** 504–514.
47. Fesenfeld, M., R. Hutubessy & M. Jit. 2013. Cost-effectiveness of human papillomavirus vaccination in low and middle income countries: a systematic review. *Vaccine* **31:** 3786–3804.
48. Ward, D., A. Drahota, D. Gal, *et al.* 2008. Care home versus hospital and own home environments for rehabilitation of older people. *Cochrane Database Syst. Rev.* **4:** CD003164. doi: 10.1002/14651858.CD003164.pub2.
49. Rich-Edwards, J.W., A. Fraser, D.A. Lawlor & J.M. Catov. 2014. Pregnancy characteristics and women's future

cardiovascular health: an underused opportunity to improve women's health? *Epidemiol. Rev.* **36:** 57–70.
50. Bellamy, L., J.P. Casas, A.D. Hingorani & D.J. Williams. 2007. Pre-eclampsia and risk of cardiovascular disease and cancer in later life: systematic review and meta-analysis. *BMJ* **335:** 974.
51. Baker, A.M. & S. Haeri. 2012. Estimating risk factors for development of preeclampsia in teen mothers. *Arch. Gynecol. Obstet.* **286:** 1093–1096.
52. Timpka, S., C. Macdonald-Wallis, A.D. Hughes, *et al.* 2016. Hypertensive disorders of pregnancy and offspring cardiac structure and function in adolescence. *J. Am. Heart Assoc.* **5**: pii: e003906.
53. de Vienne, C.M., C. Creveuil & M. Dreyfus. 2009. Does young maternal age increase the risk of adverse obstetric, fetal and neonatal outcomes: a cohort study. *Eur. J. Obstet. Gynecol. Reprod. Biol.* **147:** 151–156.
54. Mombo-Ngoma, G., J.R. Mackanga, R. Gonzalez, *et al.* 2016. Young adolescent girls are at high risk for adverse pregnancy outcomes in sub-Saharan Africa: an observational multicountry study. *BMJ Open* **6:** e011783.
55. Barker, D.J. & C. Osmond. 1988. Low birth weight and hypertension. *BMJ* **297:** 134–135.
56. Lackland, D.T., H.E. Bendall, C. Osmond, *et al.* 2000. Low birth weights contribute to high rates of early-onset chronic renal failure in the Southeastern United States. *Arch. Intern. Med.* **160:** 1472–1476.
57. Barker, D.J., C.N. Hales, C.H. Fall, *et al.* 1993. Type 2 (non-insulin-dependent) diabetes mellitus, hypertension and hyperlipidaemia (syndrome X): relation to reduced fetal growth. *Diabetologia* **36:** 62–67.
58. Mina, T.H., M. Lahti, A.J. Drake, *et al.* 2016. Prenatal exposure to very severe maternal obesity is associated with adverse neuropsychiatric outcomes in children. *Psychol. Med.* **47:** 353–362.
59. Negrato, C.A. & M.B. Gomes. 2013. Low birth weight: causes and consequences. *Diabetol. Metab. Syndr.* **5:** 49.
60. Story, M. & J. Stang. 2005. Understanding adolescent eating behaviors. In *Guidelines for Adolescent Nutrition Services.* M. Story & J. Stang, Eds.: 9–19. Minneapolis, MN: Center for Leadership, Education and Training in Maternal and Child Nutrition, Division of Epidemiology and Community Health, School of Public Health, University of Minnesota. http://www.epi.umn.edu/let/pubs/adol_book.shtm.
61. Moreno, L.A., G. Rodriguez, J. Fleta, *et al.* 2010. Trends of dietary habits in adolescents. *Crit. Rev. Food Sci. Nutr.* **50:** 106–112.
62. Story, M., D. Neumark-Sztainer & S. French. 2002. Individual and environmental influences on adolescent eating behaviors. *J. Am. Diet. Assoc.* **102:** S40–S51.
63. Schneider, D. 2000. International trends in adolescent nutrition. *Soc. Sci. Med.* **51:** 955–967.
64. Henkel, D. 2011. Unemployment and substance use: a review of the literature (1990–2010). *Curr. Drug Abuse Rev.* **4:** 4–27.
65. World Health Organization. 2014. Health for the world's adolescents: a second chance in the second decade. http://apps.who.int/adolescent/second-decade/. Accessed November 24, 2016.
66. Swanson, S.A., S.J. Crow, D. Le Grange, *et al.* 2011. Prevalence and correlates of eating disorders in adolescents: results from the national comorbidity survey replication adolescent supplement. *Arch. Gen. Psychiatry* **68:** 714–723.
67. Smink, F.R., D. Van Hoeken & H.W. Hoek. 2012. Epidemiology of eating disorders: incidence, prevalence and mortality rates. *Curr. Psychiatry Rep.* **14:** 406–414.
68. Christie, D. & R. Viner. 2005. Adolescent development. *BMJ* **330:** 301–304.
69. Heinberg, L.J. & J.K. Thompson. 1996. Body image. In *Adolescent Nutrition—Assessment and Management.* V.I. Rickert, Ed.: 136–156. New York: Chapman & Hall Inc.
70. Van Hecke, L., A. Loyen, M. Verloigne, *et al.* 2016. Variation in population levels of physical activity in European children and adolescents according to cross-European studies: a systematic literature review within DEDIPAC. *Int. J. Behav. Nutr. Phys. Act.* **13:** 70.
71. Ruiz, J.R., N.S. Rizzo, A. Hurtig-Wennlöf, *et al.* 2006. Relations of total physical activity and intensity to fitness and fatness in children: the European Youth Heart Study. *Am. J. Clin. Nutr.* **84:** 299–303.
72. Kroke, A., F. Manz, M. Kersting, *et al.* 2004. The DONALD study. *Eur. J. Nutr.* **43:** 45–54.
73. World Health Organization. 2004. Health behaviour in school-aged children (HBSC) study: international report from the 2001/2002 survey. World Health Organization.
74. Pigeot, I., S. Henauw & T. Baranowski. 2015. The IDEFICS (Identification and prevention of Dietary-and lifestyle-induced health EFfects In Children and infantS) trial outcomes and process evaluations. *Obes. Rev.* **16:** 2–3.
75. Poortvliet, E., A. Yngve, U. Ekelund, *et al.* 2003. The European Youth Heart Survey (EYHS): an international study that addresses the multi-dimensional issues of CVD risk factors. *Forum Nutr.* **56:** 254–256.
76. Moreno, L.A., M. Gonzalez-Gross, M. Kersting, *et al.* 2008. Assessing, understanding and modifying nutritional status, eating habits and physical activity in European adolescents: the HELENA (HEalthy Lifestyle in Europe by Nutrition in Adolescence) study. *Public Health Nutr.* **11:** 288–299.
77. Feigin, V.L., G.A. Roth, M. Naghavi, *et al.* 2016. Global burden of stroke and risk factors in 188 countries, during 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet Neurol.* **15:** 913–924.
78. Wang, H., M. Naghavi, C. Allen, *et al.* 2016. Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980–2015: a systematic analysis for the Global Burden of Disease Study 2015. *Lancet* **388:** 1459–1544.
79. Popkin, B.M., L.S. Adair & S.W. Ng. 2012. Global nutrition transition and the pandemic of obesity in developing countries. *Nutr. Rev.* **70:** 3–21.
80. Ruel, M.T., H. Alderman & Maternal and Child Nutrition Study Group. 2013. Nutrition-sensitive interventions and programmes: how can they help to accelerate progress in improving maternal and child nutrition? *Lancet* **382:** 536–551.
81. Viner, R.M., E.M. Ozer, S. Denny, *et al.* 2012. Adolescence and the social determinants of health. *Lancet* **379:** 1641–1652.

82. Teivaanmäki, T., Y.B. Cheung, E. Kortekangas, *et al.* 2015. Transition between stunted and nonstunted status: both occur from birth to 15 years of age in Malawi children. *Acta Paediatr.* **104:** 1278–1285.
83. Lundeen, E.A., J.R. Behrman, B.T. Crookston, *et al.* 2014. Growth faltering and recovery in children aged 1–8 years in four low-and middle-income countries: young lives. *Public Health Nutr.* **17:** 2131–2137.
84. Sawyer, S.M., R.A. Afifi, L.H. Bearinger, *et al.* 2012. Adolescence: a foundation for future health. *Lancet* **379:** 1630–1640.
85. Catalano, R.F., A.A. Fagan, L.E. Gavin, *et al.* 2012. Worldwide application of prevention science in adolescent health. *Lancet* **379:** 1653–1664.
86. Patton, G.C., C. Coffey, C. Cappa, *et al.* 2012. Health of the world's adolescents: a synthesis of internationally comparable data. *Lancet* **379:** 1665–1675.