Adolescent Undernutrition: Global Burden, Physiology, and Nutritional Risks

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Abstract

Background: Adolescents, comprised of 10–19 year olds, form the largest generation of young people in our history. There are an estimated 1.8 billion adolescents in the world, with 90% residing in low- and middle-income countries. The burden of disease among adolescents has its origins in infectious and injury-related causes, but nutritional deficiencies, suboptimal linear growth, and undernutrition are major public health problems, even as overweight may be on the rise in many contexts. **Summary and Key Messages:** Girls are most vulnerable to the influences of cultural and gender norms, which often discriminate against them. Dietary patterns and physical activity, in addition to schooling and countervailing social norms for early marriage, influence

health and nutritional well-being of adolescents. Nutrient requirements – including those for energy, protein, iron, calcium, and others – increase in adolescence to support adequate growth and development. In settings where dietary intakes are suboptimal, anemia and micronutrient deficiencies are high. Endocrine factors are essential for promoting normal adolescent growth and are sensitive to undernutrition. Growth velocity increases during puberty when peak height velocity occurs and catch-up is possible; in girls, about 15–25% of adult height is attained. A premature pregnancy can halt linear growth and increase the risk of adverse birth outcomes. Research is needed to fill the huge data gaps related to nutrition and growth during adolescence, in addition to testing interventions during this second window of opportunity to enhance growth and development, improve human capital, and to end the intergenerational cycle of growth failure.

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Definitions and Demographics

Adolescence is a physiological and social process through which a child matures into an adult. The word is borrowed from the Latin *adolēscētia*, and its first known use was in the 15th century [1]. The UN defines young people as those aged 10–24, early adolescents as those aged 10–14 years, and late adolescents as those aged 15–19 years. The world is currently home to 1.8 billion young people; this is the largest generation in human history. Their number and population share continue to grow in many places, and nearly 90% of young people live in low- and middle-income countries (LMICs). In 15 countries in Sub-Saharan Africa (plus Afghanistan and Timor-Leste), more than half of the population are under the age of 18. Each year, nearly 120 million youth become old enough to work [2]. If this growing workforce is coupled with lower fertility and improved health, nutrition, and education, many countries may benefit from the explosive economic growth associated with the demographic dividend [3]. Young people merit special attention not only because they have special health and nutrition needs but because they are our future teachers, problem-solvers, and global leaders. The degree to which this generation responds to the challenges of tomorrow and promotes economic growth relies on optimal health and development throughout adolescence.

Globally, there were nearly 1 million adolescent deaths in 2015. Approximately 60% (588,000) of these were among those aged 15–19. The leading cause of death among 10–14 year olds was HIV/AIDS due to its prevalence in Sub-Saharan Africa and the Caribbean [4] (Fig. 1). Intestinal and lower respiratory infections, diarrheal diseases, road injuries, and drowning are also leading causes of death in early adolescents. Among 15–19 year olds, road injuries, HIV/AIDS, interpersonal violence, and self-harm are leading causes of death. But malaria and infections including diarrhea and TB are also common. Maternal mortality is a serious concern for female adolescents. The maternal mortality ratio (MMR) for 10–19 year olds is 278 per 100,000, as compared to the global MMR for 25–29 year olds of 132 per 100,000. And the rate of decline in MMR for adolescents has been slower than for other age groups [4]. Given the leading causes of mortality, existing global adolescent health programs focus primarily on HIV and sexual and reproductive health services [5]. However, there are clear needs to address mental health, substance use, violence, and injury. Nutrition is essential, too; it is the leading risk factor contributing to many of the predominant causes of adolescent death.

Ranking for ages 10–14

	Female	Male
HIV/AIDS	1	1
Intestinal infectious disease	2	4
Lower respiratory infections	3	5
Road injuries	4	2
Diarrheal disease	5	6
Congenital anomalies	6	9
Meningitis	7	7
Drowning	8	3
Malaria	9	8
Leukemia	10	10

Ranking for ages 15–19

	Female	Male
Road injuries	1	1
Self harm	2	3
HIV/AIDS	3	6
Malaria	4	9
Lower respiratory infections	5	7
Diarrheal disease	6	8
Intestinal infectious disease	7	10
Tuberculosis	8	11
Maternal hemorrhage	9	
Maternal hypertensive disorders	10	
Interpersonal violence	14	2
Collective violence, legal intervention	20	4
Drowning	11	5

Fig. 1. Global ranking of the top 10 causes of death in 2015, stratified by age and sex (data source [4]).

Global Burden of Poor Nutrition in Adolescents

Micronutrient Deficiencies

Micronutrient deficiencies are a leading, underlying risk factor contributing to the global burden of disease [6]. Iron deficiency and iron deficiency anemia account for the majority of disability-adjusted life years (DALYs) associated with micronutrient deficiencies (>2,500 DALYs per 100,000 adolescents) [7]. The prevalence of iron deficiency and iron deficiency anemia is higher among adolescent females than males; the prevalence is higher among lower social development index (SDI) countries (Fig. 2a). Iodine deficiency contributes substantially to the burden of micronutrient deficiencies, and it is also more common among female adolescents. The prevalence among young adolescent girls in low SDI countries is 3.4% (95% CI 3.0–4.0), and it is 4.6% (95% CI 3.9–5.3) among older (15–19 year-old) female adolescents (Fig. 2b). Vitamin A defi-

Fig. 2. Estimated global prevalence of micronutrient deficiencies, stratified by age, sex, and social development index (SDI). The panels show the prevalence of (a) iron deficiency anemia, (b) iodine deficiency, and (c) vitamin A deficiency (data source Institute for Health Metrics and Evaluation 2017).

ciency (VAD) contributes to relatively few DALYs in this age group [7], but this merely reflects the low burden of night blindness associated with VAD as this is the only symptom that contributes to DALYs. The prevalence of VAD using biochemical indicators is estimated to be 20% (95% CI 17–24) among 10–14 year-old girls and 18% (95% CI 16–22) among 15–19 year-old girls in lowSDI countries. VAD is higher among adolescent males in middle, low-middle, and low-SDI countries (Fig. 2c). Global estimates suggest that the burden of vitamins B, C, D, and

calcium, zinc, and selenium, combined are relatively low, although this is based on somewhat few population-based micronutrient surveys [7].

Underweight

Physical growth is a key indicator of child health, and this holds true for adolescence. The global prevalence of underweight (thinness) among children and adolescents – defined as less than 2 SDs from median for body mass index (BMI) by age and sex – is 8.4% for girls and 12.4% in

Fig. 3. Prevalence of underweight (<1 to -2 SD BMI), moderate and severe underweight (<-2 SD BMI), overweight (>1 to 2 SD BMI), and obesity (>2 SD BMI) – stratified by sex and geographic region (data source [8]).

boys. Prevalence has not declined much in the past 3 decades [8]. The prevalence of moderate and severe underweight is highest in South Asia; one in 5 girls aged 5–19 years and nearly one-third of their male peers are underweight [8]. According to the Global School-Based Student Health Survey, about 4% of girls aged 13–15 years are underweight, although more than 10% of surveyed girls were underweight in Mauritius, Sudan, Bangladesh, Maldives, Cambodia, and Vietnam [7]. In 2016, the mean BMI estimates for youths aged 10–19 in South Asia, Southeast Asia, East Africa, West Africa, and Central Africa were <20 for

both male and female adolescents (Fig. 3). The lowest BMIs were seen in Ethiopia, Niger, Senegal, India, Bangladesh, Myanmar, and Cambodia [8]. While the lowest mean BMIs for children (aged 5–9 years) are found in East Africa, the lowest mean BMIs in adolescence are found in South Asia [8].

Overweight

Emerging evidence suggests that overnutrition is a growing population health concern among adolescents in LMICs. Globally, the prevalence of obesity (BMI >2 SD

BMI-for-age z score) has risen from <1% in 1975 to more than 5% in girls and nearly 8% in boys age 5–19 in 2016 [8]. Obesity has increased in all regions of the world, with the largest proportional increase in southern Africa—about 400% per decade from very low levels of 1975 [8]. More than one in 4 adolescents are overweight or obese in most of countries in the Eastern Mediterranean, Western Pacific, and the Americas regions [7]. The burden of high-fasting plasma glucose and diabetes mellitus (types I and II) increases with age throughout the young adult period, and the burden of these diseases is highest among adolescents in Middle East and North Africa and the Latin America and Caribbean regions [7]. Overweight, obesity, and poor metabolic profile in adolescence are associated with chronic disease and mortality later in life [9, 10].

Although the prevalence of overweight and obesity has increased globally, the prevalence of underweight has remained somewhat stagnant in recent decades. Thus, the global distribution of BMI has widened. Further, it is important to note that the global burden of moderate or severe underweight remains higher than that of overweight and obesity [8]. This is especially stark in South Asia and Sub-Saharan Africa where the prevalence of underweight is much higher than that of overweight and obesity (Fig. 3).

Stunting

Stunting (height-for-age below 2 SD of the World Health Organization (WHO)/CDC reference standards) in adolescence reflects poor nutrition, infection, and environmental stress accumulated from the fetal period through young adulthood. Limited data regarding adolescent stunting has been published to date, as BMI estimates have been favored as a reporting metric for children and adolescents. However, BMI growth references for adolescents are not based on perspective cohort studies to identify BMIs associated with optimal health outcomes. Median bodyweight is still low in many LMIC countries, and high BMI may be driven, in part, by stunting and suboptimal linear growth [11]. Further, stunting is especially important to consider among adolescent girls because adolescent pregnancy is very common worldwide, and young girls may stop growing. Pre-pregnancy stunting is a risk factor for poor pregnancy outcomes including small-for-gestational age (SGA) and preterm birth [12]. The limited published estimates of stunting in girls aged 15–19 range from 52% in Guatemala and 44% in Bangladesh to 8% in Kenya and 6% in Brazil [6]. We pooled data from the most recent demographic and health surveys in 58

countries and looked at a height of more than 240,000 recent mothers aged 15–19. Figure 4a shows that the distribution of height-for-age z-scores (HAZ) for these adolescent girls is significantly shifted to the left; more than a quarter of these girls are 2 or more SDs below the mean height-for-age as compared to the WHO/CDC reference population.

Framework for Viewing Adolescent Nutrition

A comprehensive framework for adolescent health considers both the life course and social determinants of health (Fig. 5). The social, economic, and political context looms over the entire framework; it includes the policies – or lack thereof – and health systems that promote healthy adolescent growth and development. As noted in the WHO framework for nutritional status in adolescents, economic factors and food systems matter too. These affect community and household access to nutritious food, food supply deficits, and changes in access to processed and unhealthy food markets. Cultural and gender norms are essential structural determinants of health and nutrition. Women and girls may be expected to eat last or eat the least. Gender norms may further dictate acceptable types of work and free time activities, and these in turn affect physical activity, energy expenditure, and income. Gender norms may become increasingly important during late adolescence, as noted by the divergent causes of death between males and females during this period. Adolescent pregnancy not only affects growth for many girls around the world, it is also a determinant of poor fetal growth. A life course approach to adolescent health is necessary because poor nutrition during gestation and childhood is an additional critical determinant of adolescent health. Finally, as discussed below, significant physiological processes affect adolescent health.

Nutritional Requirements and Diet

Nutritional Requirements and Interventions for Promoting Adolescent Growth

Adequate energy is important for supporting appropriate growth during adolescence, and notably 4% of total energy requirement supports growth in adolescence as compared to 3% at 1 year of age. Caloric requirements are high and determined by physical activity, growth, and lean tissue accretion; the requirements are higher for boys than girls. US dietary reference intakes for calcium sug-

Fig. 4. Stunting in older adolescents. **a** Shows the distribution of height-for-age z-scores (HAZ) among 15–19 years old girls. **b** Shows the proportion of these girls measuring <–3 HAZ, –3 to <–2 HAZ, and ≥–2 HAZ (data source: demographic and health surveys (DHS) – the most recent survey data from 58 countries, $n = 243,806$).

gest that requirements are the highest in adolescence compared to other life stages; Adequate Intake (AI) is 1,300 mg at ages 9–18 years (for both boys and girls) compared with 1,000 mg for children 4–8 years and adults [13]. Similarly, the AI for iron increases at ages 14–18, especially for females (15 mg for females as compared to 11 mg for males); this is related largely to the onset of menarche and increased iron needs of monthly menstrual blood loss. AIs for other minerals, such as phospho-

rus and magnesium, critical to bone mineralization are highest during adolescence (14–18 years). The RDA for protein are those set for adults in girls (46 g/day) and almost-adult levels for boys (52 g/day). The need for essential amino acids is critical during adolescence to support the pubertal growth spurt. Inadequacy of protein in many LMIC settings may be the result of both lower intake and low protein quality, likely exacerbated by infection and inflammation.

Fig. 5. A comprehensive framework for adolescent health.

Diet

Special attention is needed to adolescent dietary intake, which is an important contributor to adolescent nutrition although data are limited. Longitudinal data in a survey from India show that dietary diversity is lower in girls than boys at most ages, and the female disadvantage is largest in adolescence [14]. A review of 63 studies on macronutrient intake in adolescent girls found that global protein intake was adequate (mean $\pm$ SD = 58 ± 17 g/day), except among older adolescents living in Africa (mean $\pm$ SD = 39 ± 3 g/day) [15]. Despite AI, protein inadequacy still occurs due to poor quality of protein and suboptimal protein utilization in areas of high infection burden. In contrast, global carbohydrate intake was adequate or high among adolescent girls [15]. Globally, total energy, protein, and fat intakes are higher among younger adolescent girls than older girls. Protein and fat intake were also higher in urban areas. Fruit and vegetable intake was relatively low with just over one third of girls reporting eating vegetables daily, and less than half reporting daily fruit consumption [15]. A review of 25 studies found that most adolescents have inadequate fruit and vegetable intake (<400 g of fruit and vegetables

daily) [15]. For example, one study estimated that 97% of girls in India had inadequate fruit intake [15]. One out of 5 adolescent girls reported eating fast and convenience foods; overall consumption was highest in Africa (52%) and India (84%), although daily consumption was highest in Latin America and the Caribbean [15]. Older adolescents more commonly drank sugar sweetened beverages as compared to younger girls (35 vs. 21%) [15]. One limitation of the existing data is that the majority of dietary studies among adolescents have been conducted in school settings and may not be representative of girls who are not enrolled in school. Another limitation is that relatively few dietary assessment tools have been developed and validated for adolescents in LMICs [16]. A recent study validating a food frequency questionnaire in urban Peru noted that older children and young adolescents (aged 8–14 years) must be involved in dietary assessments to achieve valid results [17]. This may be related to the counterintuitive fact that young adolescents eat more meals away from home than older adolescents [15]. Nonetheless, the existing body of evidence clearly shows that important dietary patterns vary by age, gender, urbanicity, and region.

Adolescent Growth and Development

Regulation of Pubertal Growth: The Hypothalamus-Pituitary-Gonadal Axis

Puberty is the process of physical changes by which adolescents reach sexual maturity, that is, adolescents become capable of reproduction. Puberty is marked by both gonadotrophic and somatotrophic processes; the former is marked by sexual maturation including onset of ovulation and spermatogenesis, whereas the latter represents accelerated linear growth and changes in lean, fat, and bone tissue. Somatic growth and maturation are influenced by the interplay of numerous factors that can be broadly classified as hormonal, environmental (with nutrition playing an important role), and genetic.

Significant advances have been made in the understanding of the endocrinology of pubertal growth and development [18–20] and are simply summarized here. The interaction between gonadal and adrenal steroid hormones along with growth hormone (GH) is essential for a normal adolescent growth spurt and maturation to occur. Puberty is marked by the secretion of gonadotropin releasing hormone (GnRH) that stimulates the release of leuteinizing hormone/follicle stimulating hormone (LH/FSH) from the pituitary gland; LH/FSH in turn stimulates the production of sex steroids in ovaries and testes [19]. These endocrine factors are sensitive to undernutrition, which may cause delays in signals to initiate puberty by impacting the amplitude and pulsatility of GnRH. Additional endocrine factors are also in play. Leptin, produced by adipocytes and a regulator of energy balance/appetite, plays a permissive role for puberty. For example, lower leptin concentrations are associated with later onset of menses. On the other hand, ghrelin produced by the digestive tract during energy insufficiency, may inhibit GnRH release. Estrogen in girls and testosterone in boys reach adulthood circulating levels in adolescence. These sex hormones play a significant role in sexual maturation and growth stimulation. Testosterone stimulates spermatogenesis, development of secondary sexual characteristics, body composition changes, muscle development, and brain development of gender dimorphic areas. Estrogen stimulates the maturation of female reproductive tract and onset of menarche, mammary gland development, body compositional changes, GH secretion, bone mineralization, and closure of bone plate. The GH/insulin-like growth factor 1 (IGF-1) axis is stimulated by enhanced sex hormone production in puberty and is highly sensitive to nutrition. GH produced by the pituitary gland

stimulates the production of IGF-1 in liver. IGF-1 may also have paracrine and autocrine effects locally in bone and is associated with both-accelerated accumulation of lean body mass and bone mineralization via osteocalcin production.

Linear and Skeletal Growth and Catch-Up

Growth velocity, that is, the rate of linear growth, is the highest during the fetal period, continues to be high in the first 2–3 years of life, but declines thereafter. However, growth velocity increases again during puberty when peak height velocity occurs. In girls, it is estimated to be 9 cm/year beginning at age 12. In girls, about 15–25% of adult height is attained during this time, and in healthy environments, girls will gain about 25 cm in height during adolescence on average [21]. Similar changes occur with weight; 50% of adult weight is gained in adolescence. Linear growth in adolescence is marked by the lengthening of long bones at the growth plate followed by epiphyseal closure when growth is completed. The mechanism involves cells called chondrocytes at the growth plate depositing new matrix. The bone also grows in size and the matrix must then be mineralized. Nearly 40% of peak bone mass is attained during puberty. Calcium comprises a third of bone mineral, and calcium deposition in bones is occurring at almost 300 mg/day at its peak [22, 23]. Bone mineralization depends on calcium intake, but also other minerals such as phosphorus and magnesium, exercise, and genetics. Estrogen is associated with growth plate fusion in both sexes, and significant differences in bone mineral content and density by sex have been reported [24]. Body compositional changes also occur during adolescence with fat free mass and fat mass increasing. There are major sex differences in the accrual of fat compared to lean body mass. Boys have about twice as much lean mass than girls; percent body fat is much higher in females [25].

Catch-Up Growth

Given the patterns of growth described above, adolescence may be a second window of opportunity for intervening. Linear growth faltering is commonly seen in LMICs in the first 2 years of life [26]. Catch-up growth is possible in children older than 2 years, although stunting is often well established by this age in many LMICs. Using longitudinal data from 5 different countries, HAZs were shown to improve during adolescents in each of the 3 tertiles of height categories [27]. The only exception was the cohort from India in which catch-up did not occur. In a study that followed children until adulthood in the Gambia, boys and girls eventually achieved height

comparable to a UK cohort, illustrating significant catch up growth by 20–23 years of age [27]. This study gives credence to the idea that adolescence may be a period during which there is an opportunity for catch-up growth. However, the factors allowing for catch-up growth are not clear, given the contrasting evidence from India. Differences in catch up growth during adolescents between African and Asian populations need further investigation given higher rates of maternal stunting in South and Southeast Asia compared to many parts of Africa. Early life factors may be important to consider in addition to size at birth and nutritional status during pregnancy of the mother.

Adoption studies provide another piece of evidence that catch-up growth is possible. One example, although there are several, is a study among Indian girls adopted into Sweden [28]. Height was examined when they were adopted, at 2 years after arrival, and at menarche; there was an overall increase in HAZ, albeit adult height was lower among the adopted Indian girls relative to the National Center for Health Statistics standards. The adopted Indian girls experienced an early onset of menarche and a shortened growth period post menarche. The biology of this phenomenon needs to be better understood. In a more recent study, adopted girls in Denmark entered puberty earlier (by about 1.3 years) than the Danish born girls, and they did not achieve the same height but weighed the same as their local counterparts [29].

Skeletal growth slows with age in mammals largely due to a phenomenon of programmed growth plate senescence in childhood [30]. A decline in growth plate senescence occurs over time and with cumulative replication of chondrocytes. Catch-up growth may occur following “growth inhibitory conditions” [30] as demonstrated in animal models. In rabbits exposed to glucocorticoids [31] and in rats induced to hypothyroidism [32] or tryptophan deficiency [33] a phenomenon of catch-up growth in part driven by delayed growth plate senescence has been shown. These animal studies demonstrate that any adverse exposures that slow growth plate chondrocyte proliferation and stop growth result in slowing of senescence, but once the condition is removed, the growth plates that are less senescent show an accelerated growth rate than expected for age resulting in catch-up [31]. In undernourished populations, growth is suppressed in part to conserve nutrients for other uses; endocrine control of growth is highly responsive to nutritional intake. In such situations, growth that is postponed may likely not be lost irreversibly. Adolescence is perhaps the time to facilitate and promote such catch-up growth.

Assessment of Growth and Development

Historically, the assessment of somatic or linear growth in adolescence has been a challenge, largely related to the inability to distinguish normal variations in maturation from those associated with health risks. This has resulted in failure to adequately address poor growth in adolescence, which is an important determinant of future health, work capacity, and cognitive function that all contribute to human capital. WHO growth charts constructed by combining the original US growth charts with Multicentre Growth Reference Study child data through age 6 years allow assessment of growth across the adolescent age range. Thus, height for age and BMI for age for girls and boys from 5 to 19 years of age can be used to define stunting and wasting rates. For modeling growth velocity, Tanner [21], Preece-Baines [34], and the ICP models [35] are commonly applied.

Refinement of cognitive abilities and voluntary control of behaviors is constantly being enhanced during the period of adolescence. Brain function that controls response inhibition and working memory that support cognitive control changes with age. Findings from studies using functional magnetic imaging indicate age-related differences in the use of areas of prefrontal cortex in children and adolescents compared to adults when performing tasks requiring cognitive control; use of dorsolateral prefrontal cortex is higher in adolescents suggesting the need for more effort and attention [36]. Executive functioning in adolescents is still developing and not reached adult levels. For example, correct inhibitory responses in a functional test increased in a linear fashion across age categories of 8–12, 13–17, and 18–27 years [37], suggestive of continued development during this period.

Interventions

Few studies have systematically examined nutritional interventions in settings where dietary inadequacy and micronutrient deficiencies exist. The most studied is calcium, as calcium absorption and bone mineralization have been shown to increase during early pubertal development among girls with consequences for long-term bone health [22, 38]. Supplementation with calcium, however, has been found to be of limited benefit even in settings where calcium intake was low. For example, in a Gambian cohort of children 10.3 years (Tanner stage 1) at enrolment, calcium supplementation (1,000 mg/day) increased bone mineral content of digital radius, but had no impact on bone size or linear growth [39, 40]. Exercise and weight-bearing activity in early puberty in US girls improved bone mineralization [41], although excessive

physical activity (such as among gymnasts) is also associated with delayed growth and pubertal maturation [18]. Promising interventions for promoting linear growth beyond the critical first 1,000-day window were recently described in a systematic review [42]. Several effective nutritional interventions were identified across a wide range of ages, duration, and baseline status; few studies were from LMICs. The overall findings were that interventions with zinc, vitamin A, multiple micronutrients, and protein had a significant impact on improving height; the effect size increases in linear growth ranged from 0.05 HAZ for vitamin A to 0.68 HAZ for protein. Perhaps surprisingly, interventions including iron, calcium, iodine, and food supplements showed no significant benefit, although sample sizes for the pooled studies for these were low (approximately 500–1,100). The small effect size with vitamin A may be of little clinical relevance and was shown to be significant perhaps due to the large sample size ($n = 23,000$) across studies. The sample size for the protein studies was low ($n = 939$), and yet the effect size was the highest. The multiple micronutrient intervention had modest significant benefits and may be a better way to combine provision of individual nutrients. In addition, randomized trials of iron supplementation have been shown to significantly improve hemoglobin concentrations among adolescents (standardized mean difference 1.83, 95% CI 0.59–3.08) based on pooled analysis of 7 studies [43]. Despite the lack of impact on linear growth, this intervention may be worth considering, given the benefit of iron supplementation on cognition in school age children [44]. The systematic review did not examine the impact of calcium or vitamin D intervention on bone mineral density and attainment of peak bone mass or other outcomes of bone health, which, as described above may be improved.

Adolescent Pregnancy

Worldwide 16 million girls aged 15–19 years give birth every year [45]. The highest proportion of adolescent births, approximately 20% of all births, occurs in Sub-Saharan Africa and Latin American countries. In absolute terms, Bangladesh, India, and Nigeria together account for 30% of the world's adolescent births [45]. The highest prevalence of early child bearing is in Guinea, Mali, and Niger, where about 10% of women gave birth before the age of 15. Nearly all (90%) adolescent births occur within marriage. More than one third of girls in LMICs marry before they are 18; data from UNICEF show that 720 mil-

lion girls were married or were in a union before the age of 18 [46]. Approximately 14% of girls marry before they reach age 15. Adolescent pregnancies are more likely in poor, uneducated and rural communities. Sociocultural norms surrounding marriage and social pressure to become pregnant after marriage are important contributors to adolescent marriage and pregnancy. Family planning programs have an important role in delaying the age of first pregnancy.

Consequences of Adolescent Pregnancy on Birth Outcomes

Adolescent pregnancy has numerous adverse consequences including increased risk of maternal mortality, obstructed delivery, and risk of cephalo-pelvic disproportion and C-section delivery [6]. A meta-analysis using data from 14 LMIC birth cohorts shows that nulliparous, adolescent mothers (<18 years old) have 1.5–3 times the adjusted odds for adverse birth outcomes including SGA, preterm birth, as well as neonatal and infant mortality. Compared to older women [47], in many settings, young pregnancy age is associated with socioeconomic and cultural factors. Recently, a detailed analysis from the longitudinal data of the COHORTS group – a 5-country birth cohort collaboration shed important light on the consequences of young maternal age and adverse birth and child outcomes. In an adjusted analysis (for maternal height, socioeconomic status, and parity), younger maternal age was associated with increased risk of low birth weight, preterm birth, stunting at 2 year of age, and higher adult glucose concentrations [48]. Additional analyses revealed that younger mothers (15–19 years) had less schooling and were more likely to be primiparous than older mothers, but there was no association with wealth index, urban location, race, or height [49]. Short maternal stature, which is quite common among adolescent mothers (Fig. 4b), is associated with an increased risk of SGA and preterm births in LMICs [12]. Younger age, which is accompanied by primiparity accounted for the increased risk of adverse outcomes [49], although previous studies have also found second pregnancies during adolescence to be associated with an increased risk of preterm delivery and stillbirth [50] in high-income countries. Maternal stunting has been associated with adverse obstetric outcomes, fetal growth failure, and poor birth size that likely perpetuates the intergenerational cycle of growth failure [6]. These findings support encouraging delay in the first pregnancy especially in many setting where age of marriage is early.

Consequences of Adolescent Pregnancy for the Girl Herself

There is evidence that becoming pregnant prematurely may have adverse consequences for the nutritional health of the young mother. Adolescent girls often enter pregnancy with inadequate nutritional stores, and pregnancy leads to competition for dietary energy and nutrients between the fetus and the mother. Based on adolescent animal models, nutrient partitioning is complex and depends on numerous hormonal and physiologic adaptations to promote fetal growth, as well as maternal nutritional status during pregnancy [51]. Daily required intakes for adolescent pregnancy account for the higher needs for nutrients [13]. In a study in rural Bangladesh, when adolescent pregnancy was halted, linear growth continued [52]. This study enrolled 2 groups of girls who were of the same chronologic age and had the same age of onset of menarche. The study examined annual changes in anthropometric measurements by pregnancy status. Pregnant adolescents did not gain height during the follow-up period; never-pregnant girls increased stature by 0.35 cm. Similarly, pregnant adolescents had lower weight, BMI, mid-upper arm circumference, upper-arm-muscle area, and percent body fat by 6 months postpartum compared to non-pregnant adolescents who gained in each of these ponderal dimensions over a 1-year period. Annual losses in triceps skinfold and upper-arm fat area were greater, and gain in subscapular skinfolds was lower among pregnant than never-pregnant girls. Differences in annual changes between pregnant and nonpregnant adolescents were statistically significant. This study, however, was unable to follow-up the girls into adulthood to see if, in fact, they had different heights as adults. In a study in Brazil, pregnant adolescents had a similar reduction in height, but intriguingly they had higher BMIs (due to more weight gain) relative to their counterparts [53]. More recently, a South African study found no difference in growth between previously pregnant and non-pregnant adolescents [54]. We need to better understand what will happen in different environments where low BMI or high BMI are common. Delayed first pregnancy beyond the teenage years and increased birth spacing following an early pregnancy may provide adolescent girls an opportunity for nutritional recovery and linear growth. Thus, in some LMIC settings where over a third to half of women give birth prior to their 20th birthday, a premature pregnancy may contribute to lower attained stature among adult women. Even less is known about the lactation success and breast milk quality of adolescent mothers and the long-term impact of this on child health outcomes. One

small study of 22 lactating mothers, half of whom were adolescents, found milk production was lower by 37–54% among adolescent mothers, in part related to ethnic and racial differences rather than behavioral differences, but biological differences could also account for the poorer lactation performance [55].

Programmatic Gaps and Research Agenda

The need for comprehensive research in adolescent nutrition is huge. We have identified 4 prioritized areas of necessary research. First, we must fill the major data gap that exists for this age group, especially among younger adolescents. Much of the existing data, as described above, are derived from demographic and health surveys among women of reproductive age who have had a previous pregnancy or small-scale school-based surveys. Longitudinal, dietary, anthropometry, and micronutrient status data gaps need to be filled with systematic work, and global indicators regarding health and nutrition in adolescents should be developed. Additionally, stunting and suboptimal linear growth in adolescence, leading to short attained adult height, are underrecognized problems. Tracking pubertal growth and understanding factors associated with the age of onset of puberty, peak height velocity, and duration of linear growth and bone mineralization in representative populations in LMICs would inform potential interventions. Simultaneously, data on the sociocultural and economic drivers of nutritional status and food choices are needed, especially those driving low vs. high BMI is needed. A second area of research is rigorous evaluations of nutritional interventions to determine the right combination and dosage of macro and micronutrients, age and duration for intervening to enhance optimal growth and development outcomes in adolescence and later in life outcomes. It is also important to promote healthy growth and simultaneously have minimal risk of overweight, adiposity, and metabolic risks [11]. Interventions to delay pregnancy beyond adolescence should be developed and tested for their ability to improve adolescent nutritional status including attained height, pelvic size, and body composition. The third important evidence gap related to adolescent health is in implementation and programmatic research. Evidence related to the effectiveness of various delivery platforms to efficiently roll out appropriate policies and programs is needed. Integrated approaches (e.g., across education, family and sexual and reproductive health sectors) may be cost effective and more beneficial. The fourth area for

research is related to the structural determinants of adolescent health. Key issues are how to improve gender equality and enhance individual agency in influencing decision making around age of marriage and first pregnancy. Education and women's economic empowerment are also likely to improve nutritional and health outcomes in adolescence.

Investing in adolescent nutrition has the potential to improve economic productivity, reproductive health, and chronic disease outcomes of populations. Further, these investments are key to address the cycle of intergenerational growth failure and poverty in many LMIC contexts. Political will and funding to address these research

and implementation knowledge gaps are urgently needed to ensure we optimally invest in health and development of the largest generation in human history.

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