



NIH PUBLIC ACCESS

Author Manuscript

Food Nutr Bull. Author manuscript; available in PMC 2015 February 02.

Published in final edited form as:

Food Nutr Bull. 2014 June ; 35(2): 230–243.

Childhood dual burden of under- and over-nutrition in low- and middle-income countries: a critical review

Emma Tzioumis and Linda S. Adair

University of North Carolina at Chapel Hill, Chapel Hill, North Carolina.

Abstract

Background—In low- and middle income countries, the distribution of childhood nutritional diseases is shifting from a predominance of undernutrition to a dual burden of under- and overnutrition. This novel and complex problem challenges governments and health organizations to tackle opposite ends of the malnutrition spectrum. The dual burden may manifest within a community, household, or individual, but these different levels have not been addressed collectively.

Objective—To critically review literature on the prevalence, trends, and predictors of the dual burden, with a focus on children from birth to 18 years.

Methods—We reviewed literature since January 1, 1990, published in English, using the PubMed search terms: nutrition transition, double burden, dual burden, nutrition status, obesity, overweight, underweight, stunting, body composition, and micronutrient deficiencies. Findings were classified and described according to dual burden level (community, household, individual).

Results—Global trends indicate decreases in diseases of undernutrition, while overnutrition is increasing. On the community level, economic status may influence the dual burden's extent, with obesity increasingly affecting the already undernourished poor. In a household, shared determinants of poor nutritional status among members can result in disparate nutritional status across generations. Within an individual, obesity may co-occur with stunting or anemia, due to shared underlying determinants or physiologic links.

Conclusions—The dual burden of malnutrition poses a threat to children's health in low- and middle-income countries. We must remain committed to reducing undernutrition while simultaneously preventing overnutrition, through integrated child health programs that incorporate prevention of infection, diet quality, and physical activity.

Keywords

Dual burden; undernutrition; overnutrition; nutrition transition; children; low- and middle-income countries

Please address all inquiries to the corresponding author, Emma Tzioumis, at: Carolina Population Center, 123 W. Franklin St University Square Chapel Hill, North Carolina, 27516. tzioumis@unc.edu.

None of the authors have conflicts of interest to declare.

Introduction

Undernutrition has traditionally been the focus of nutrition agendas in low and middle income countries (LMIC). However, rapid economic development and urbanization have given rise to a nutrition transition, where energy-dense diets replace traditional diets and sedentary lifestyles prevail [1]. This has led to an increase in obesity and diet-related chronic diseases. Despite economic development, substantial economic disparities remain and nutritional insufficiencies continue to affect many children in LMICs. The co-existence of under- and overnutrition, a phenomenon known as the “dual burden”, poses a novel public health challenge. It is imperative to find ways to eliminate undernutrition and its associated morbidity and mortality, without contributing to obesity and increasing the risk of nutrition-related chronic diseases as populations age.

There is substantial literature on the dual burden [2]. Although initially documented in adults, it has been also observed in children [3]. Most studies focus on high prevalence of underweight or stunting and overweight in the same population, or on households with both obese and stunted or underweight individuals [4,5]. Individuals may experience a dual burden of malnutrition, with overweight and obesity coupled with micronutrient deficiencies, or stunting coupled with high levels of central adiposity. In this critical review¹ and synthesis based on literature since January 1990, we focus on the dual burden of malnutrition among children and adolescents at three different, but interrelated, levels: the community, the household, and the individual. Prior literature has addressed the dual burden within each of these levels but has not synthesized findings across all levels. In addition to describing patterns and trends in the occurrence of the dual burden, we explore common and unique factors at each level. Finally, we highlight methodological issues for consideration when assessing the literature. We frame the review in the theoretical context of the nutrition transition.

Defining indicators used in dual burden research

The dual burden is often assessed by reporting the prevalence of a measure of undernutrition and the prevalence of a measure of overnutrition within households, specific communities, regions, or at the country level. Under- and overnutrition are non-specific terms used here to describe the opposite ends of the spectrum of nutritional status. Anthropometric indicators predominate, with undernutrition commonly defined as stunting or wasting (length or height-for-age, and weight-for length or BMI Z score < -2 based on the WHO Child Growth Standards [6]) among children, and as BMI <18.5 in adults [7]. Overnutrition is usually represented by BMI above established cut-points; overweight and obesity defined as BMI >25 and 30 kg/m² in adults, and the childhood equivalents of these cut-points established using the International Obesity Task Force (IOTF) or WHO reference [8]. Despite the limitations of BMI for representing child obesity [9], these anthropometric indicators are

¹We critically reviewed literature using the following search terms in PubMed: double burden, dual burden, nutrition transition, nutrition status, obesity, overweight, underweight, stunting, body composition, and micronutrient deficiencies. We identified 3188 articles. After restricting the search to non-intervention, human studies published in English since January 1, 1990, and unrelated to bariatric/laparoscopic surgery or gastrectomy, 747 studies remained. We reviewed the abstracts of these remaining studies according to the following criteria: 1) presented results from primary or secondary data analysis; 2) included measures of both undernutrition and overnutrition; 3) inclusion of children under 18y. Eighty-four studies remained and were considered for critical review.

commonly measured. Other indicators of undernutrition include biomarkers reflecting micronutrient status.

It is important to note that these indicators may reflect different points in a person's nutritional history. For example, stunting is a longer term indicator of chronic undernutrition, and typically develops during the first two years of life and tends to persist into later childhood and adulthood. Wasting is generally regarded as a more acute indicator of poor nutrition, and can develop quickly, as can micronutrient deficiencies. Thus, when exploring factors that contribute to the dual burden, it is important to consider the latency and sensitive windows for development of the different types of malnutrition. For example, the co-existence of stunting and obesity may reflect inadequate nutrition, poor diet quality and morbidity in the first two years, followed by excesses in energy intake in later childhood.

Global trends in stunting, underweight and obesity

The various forms of undernutrition (e.g.: stunting, wasting, micronutrient deficiencies) in children under five continue to pose a significant public health concern [10,11]. Linear growth retardation (stunting) remains highly prevalent worldwide. In 2010, an estimated 171 million children under five were stunted, with almost all occurring in developing countries [12]. Although the global prevalence of stunting has decreased recently (from 39.7% in 1990 to 26.7% in 2010), this trend has not been consistent in all regions of the world; stunting in Africa remains relatively unchanged around 40%. Projections for 2020 suggest the situation in Africa will not improve substantially, whereas rates in Asia and Latin America will continue to decrease. Similarly, micronutrient deficiencies in children under five continue to pose a global public health challenge; 47% are anemic (293 million children) [13] and 33% are vitamin A deficient (190 million children) [14].

Simultaneously, childhood rates of obesity have increased dramatically [11]. In 2010, 43 million children under five were overweight or obese, with an additional 93 million children at risk of overweight [15]. Global prevalence of overweight or obesity increased in all regions, from 4.2% in 1990 to 6.7% in 2010. Although the prevalence is higher in developed countries, developing countries have greater absolute numbers of affected children and higher relative increases. These increasing trends are projected to continue, with 60 million children under five expected to be overweight or obese in 2020.

These under- and overnutrition trends produce a dual burden at the country level. In 2000, de Onis and Blössner documented the emergence of the global overweight and obesity epidemic [16]. Global prevalence of overweight (weight-for-height (WHZ) > 2SD from the National Center for Health Statistics/WHO international reference median value [7]) was 3.3% (17.5 million children under five). They compared overweight prevalence with wasting (WHZ < 2 SD) prevalence in 94 countries, capturing both ends of the weight-for-height distribution. Global prevalence of wasting was higher than overweight (9.4% vs. 3.3%, respectively), but there was great variability on the country level. Although the prevalence of wasting was generally higher than overweight (45 countries had wasting prevalence >5% vs. 21 with overweight prevalence >5%), 42 of the countries had a higher

prevalence of overweight than stunting. These countries were typically wealthier, indicating a shift in disease burden as countries progress through the stages of the nutrition transition. Findings published more recently with comparable, but updated, data indicate similar trends, are shown in figure 1.

The nutrition transition is characterized by a shift in disease burden from undernutrition to overnutrition-related chronic disease. As the transition advances, traditional diets high in complex carbohydrates and fiber are replaced with diets high in fats and sweeteners. Physical activity levels decline and sedentary time increases [17]. Increased consumption of energy-dense foods and the lack of physical activity leads to obesity and the development of numerous chronic diseases. Obesity and related chronic diseases were once considered diseases of affluent Western societies; however, as a country's gross national product (GNP) increases, the burden of obesity shifts to lower socioeconomic groups [18]. People of lower socioeconomic status (SES) may be more at the mercy of an obesogenic environment; they do not have the economic means to avoid the micronutrient-poor, yet often ubiquitous, energy-dense food choices [19]. With urbanization, the consumption of oils, sweeteners, processed foods, and foods prepared away from the home increases. Improved transportation and food distribution systems, changes in household food preparation, and food production and processing technology contribute to this new dietary pattern [20]. Together, these changes contribute to the nutrition transition [21]. Owing to these important global trends, many in the lower socioeconomic groups of LMIC continue to struggle with undernutrition, even as obesity and overnutrition increase [21].

The community/population level

The childhood dual burden at the community/population level is usually represented by prevalence rates of stunting and/or wasting and overweight or obesity. Frequently, these statistics represent substantial economic disparities between subgroups, historically capturing the persistence of stunting and underweight in rural and poor urban segments of the population in low and middle income communities, and the development of overweight among economically better off segments. Recently, however, the burden of obesity has begun to shift to the poor [22], and the dual burden can be observed within low income communities. Country- or community-level estimates of the dual burden include instances in individuals and households, and separate distributions of under- and overnutrition in different subgroups.

Secular trends reflect global trends

The dual burden has been widely reported in Latin America, Africa, Middle East, and Asia [23–34]. Studies have assessed secular trends in nutritional status of children in LMIC as markers of overall population health; trends in each country reflect its progression through the nutrition transition. For example, comparisons of anthropometric trends in South African school children (ages 8–11 year) from 1994 to 2004 indicate that mild and moderate stunting have decreased, whereas overweight and obesity have increased [35]. Although stunting is decreasing, race/ethnic disparities remain; among boys, 6.8% of black children were moderately stunted, compared to 2.2% of mixed ancestry children and 0.5% of white children. Similar patterns were observed in girls. Coexistence of stunting and overweight/

obesity was lower in 2004 than in 1994, but this may be driven by the decrease in stunting. Similar anthropometric changes were assessed in one-year old Brazilian children between 1982 and 2004 [36]. Over this period, prevalence of overweight increased while stunting only decreased slightly. Additionally, prevalence of overweight increased among low SES children but decreased among high SES children, consistent with the progression of changes due to the nutrition transition.

In Chile, childhood stunting and underweight are no longer thought to affect the entire country but rather isolated sub-groups. Kain et al. examined trends in BMI and height from 1987 to 2002 in six-year old Chilean children [37]. Over this 15-year period, proportions of tall children steadily increased ($HAZ > 2SD$) and stunting steadily decreased. Similarly, prevalence of overweight/obesity increased and underweight decreased. Both tallness and stunting were associated with obesity; the tallness/obesity association was greater at all time-points, but the association of obesity with stunting increased more over the study period. This is reflective of the nutrition transition's rapid progression in Chile.

The role of socioeconomic status

Improvements in SES in low income countries initially increase rates of overweight and decrease rates of underweight. However, these improvements leave considerable inequality; underweight continues to be commonplace among the poor whereas overweight initially develops in the wealthy. Over time, energy-dense/nutrient-poor diets become the norm in low-income groups and as a result they struggle with the dual burden of malnutrition.

Differences in the extent of the burden vary by SES [38–48], as well as urbanicity [49–53], and gender [39,44,50,51,54]. In a study of school-aged children in urban and peri-urban areas of Burkina Faso, private schools had higher proportions of overweight or obese students, whereas stunting prevalence was higher in public schools [41]. Burkina Faso may still be in the early stages of the nutrition transition; those better off may be more susceptible to overweight/obesity, whereas the economically disadvantaged are still prone to undernutrition. Conversely, in low-income pre-school children in Brazil, a higher prevalence of overweight and obesity and a lower prevalence of stunting were detected, reflective of a more advanced stage of the nutrition transition [47].

Various economic factors may be related to the distribution of under- and overnutrition. The nutrition transition has progressed quickly in Latin America and the Caribbean, increasing overweight and obesity in the region; stunting prevalence in children 0–5 years was estimated at 13.7% (95% CI: 9.1–18.4) and overweight at 4.3% (95% CI: 3.5–5.2) [55]. In each of the sub-regions (the Caribbean, Central America, and South America), an inverse relationship was observed between stunting and overweight; however, when the prevalence of stunting was low (<20%), the prevalence of overweight varied more. Economic development (per capita GDP) was moderately inversely correlated with stunting prevalence ($r=-0.46$, $p < 0.001$) and weakly correlated with overweight ($r=0.26$, $p < 0.001$), but varied among the sub-regions. Regional differences in GDP may help explain the variability of the stunting/overweight relationship. An analysis of Brazilian children of the same age reported similar relationships; per capita income was inversely associated with stunting and

positively associated with obesity [56]. Again, these secular trends generally reflect global trends.

To provide further evidence of the relationship between the dual burden and economic situation in Latin America, we present Demographic and Health Survey data from selected Latin American countries in figure 2 [57]. The prevalence of stunting, wasting, and overweight in children under 5y is shown stratified by household wealth index; lowest versus highest quintile. Overall, stunting is decreasing over time, wasting trends are not pronounced, and overweight is increasing over time. Stunting prevalence is highest in the poorer countries (Haiti, Honduras) and overweight prevalence is highest in countries with more developed economies (Peru, Dominican Republic). Differences are observed between wealth strata within a country, where stunting remains high in the poorest children and overweight was highest in the wealthiest children. Of great concern, the prevalence of overweight is increasing amongst the poorest children, as well (Colombia, Dominican Republic, Honduras).

A prospective study in rural South African villages in the midst of the transition assessed determinants of stunting and obesity [58]. After a three-year follow-up (birth to three years), 48% of children were stunted, 18% were overweight, 24% were obese, and 19% were stunted and overweight or obese. Risk factors for stunting included larger household size and a mother who is a student, whereas a working mother was a risk factor for overweight. Larger household size, having a working or student mother, greater weight at one year, and shorter length at one year were risk factors for concurrent stunting and overweight. Large household size was also associated with concurrent stunting and overweight in Mexican children; however, it was associated with lower maternal education and lower SES [59].

Household

Household-based surveys find undernourished and overnourished persons living in a shared household environment with common macro-level social, environmental, and economic factors. Differences in nutritional status among them likely reflect individual differences in age and secular trend related exposure history, as well as current individual behaviors (e.g.: physical activity and work patterns), physiology and nutrient needs (per kg nutrient needs are substantially higher in growing children compared to adults). Differences may also reflect differential *susceptibility* or response to environmental factors according to age or developmental stage. Finally, there may be household-level differences in allocation of resources.

The dual burden household is defined as a household in which at least one member is underweight and at least one member is overweight. Distinct household typologies exist, including stunted child/overweight mother (SCOWT) [60] and overweight child/underweight adult households [61]. Mother-child dyads are often the unit of analysis when assessing weight discordance; it is assumed they share more resources, are in closer contact than other household members, and should be less likely to differ in weight status. One explanation for SCOWT households is that as family income increases, adults adopt increasingly sedentary lifestyles and are able to afford more energy-dense but nutrient-poor

foods for their household. This contributes to increased overweight in adults, and failure to meet linear growth potential in children.

Specific methodological concerns arise when characterizing SCOWT households. The prevalence of underweight in children under five has decreased since 1990 [62], whereas adult obesity has doubled since 1980 and continues to increase [63]. These trends tend to inflate the prevalence estimates of SCOWT pairs [64]. Using Demographic and Health Surveys (DHS) data from 54 countries, Dieffenbach and Stein concluded that SCOWT pairs are not statistically independent of either stunting or maternal overweight prevalence; SCOWT prevalence is largely dependent on maternal overweight prevalence [65]. Since the dual burden household is largely driven by the prevalence of its components, care must be taken when interpreting its determinants; a factor interpreted as predicting discordance within the household may only predict maternal obesity. Even so, these households should not be abandoned by public health interventions.

Some have tried to tease out the role of an energy-dense but nutrient-poor diet in the SCOWT pairs, examining the relationship between food variety scores and dual burden households. In a study of Malaysian mother-child pairs, 51% of mothers were overweight and 58% of children were underweight [66]. The most common household type was normal mother/underweight child (NW/UW, 31.8%), followed by overweight mother/underweight child (OW/UW, 25.8%). Energy intake was estimated from diet recalls and a food variety score was created for both mother and child. When the OW/UW household was compared to all other households, a higher maternal food variety score was associated with increased risk of OW/UW and obese women increased across tertiles of variety scores. However, a higher food variety score in children was associated with a decreased risk of OW/UW. These associations remained significant even when OW/UW households were compared only to NW/NW households. Diet variety may have different implications for mothers and children, and could be affected by higher physical activity levels in children and smaller quantity of food consumed by children.

When evaluating the role diet might play, it is important to consider the source and quality of the data. Mothers are often reporting both their diet and the diet of their children, potentially resulting in inaccurate reporting of either or both diets, whether intentionally or unintentionally. In a case study from Malaysia assessing the presence of dual burden households, UW/OW (underweight child/overweight mother) pairs were compared to NW/NW pairs [67]. Here, 52% of women were overweight, 27.1% of children were stunted, and 54 of the 140 households were UW/OW. Mothers reported diet and physical activity levels for themselves and their children. Overweight mothers appeared to be in negative energy balance, perhaps due to either diet underreporting or physical activity over-reporting. The reported energy and nutrient intakes for overweight mothers did not differ significantly from those of normal weight mothers, an inconsistent finding. Underweight children reported consuming less energy than normal weight children, but energy expenditure was the same between the two groups. Misreporting is present at least with the mother's data, but conclusions regarding its extent are cursory without further investigation.

The prevalence of dual burden households may be related to a country's economic development and nutrition transition stage. The middle of the socioeconomic range is often associated with the highest prevalence of dual burden households; countries in the chronic disease phase. This was initially shown in a study where data from national surveys of seven countries was used to classify households as normal weight, underweight, overweight, or dual burden [4]. In rank order of GDP (and phase of the nutrition transition), childhood underweight prevalence (BMI <18.5 kg/m²) and overweight prevalence (BMI ≥25 kg/m²) were as follows: 43.7% and 1.2% (Vietnam), 21.2% and 9.5% (China), 15.8% and 28.2% (Kyrgyz Republic), 32.2% and 5.1% (Indonesia), 11.4% and 13.0% (Russia), 12.3% and 9.4% (Brazil), and 7.5% and 23.3% (US). The Kyrgyz Republic, a country in the middle of the GDP range, had the highest prevalence of dual burden households (15.5%), whereas Vietnam and the United States, countries on the extremes of the GDP range, had the lowest prevalence. Dual burden households tended to be high income, and with the exception of Vietnam, dual burden households constituted 22–60% of households with an underweight person. However, many contextual nuances were described. For example, dual burden households were only different from overweight households in Brazil and Russia, where they were more likely urban and low-income. Therefore, the underweight individuals in dual burden households of the other five countries may be unintended recipients of household level obesity interventions. The public health community should be aware that people of dissimilar nutritional status may reside in the same household when designing targeted campaigns.

Other predictors

Fetal and childhood origins of adult obesity may play a role; a constrained fetal environment and childhood stunting have been linked to adult obesity and chronic disease. A group in Mexico used central adiposity (as measured by waist circumference, WC, and waist-to-hip ratio, WHR) to determine if in certain groups, undernutrition and obesity actually share common causes [68]. In this sample of 5974 mother-child pairs, prevalence of maternal overweight/obesity (classified by WHR) was 57.4%, childhood stunting prevalence was 17.3%, and dual burden household prevalence was 6.1%. In results adjusted for maternal height and BMI, and child's age, a WHR ≥0.85 increased the likelihood of having a stunted child by 33%. The likelihood of SCOWT was higher in less developed regions. The authors interpret their results as evidence of a biological link between childhood stunting and adult obesity.

Oddo et al. attempted to better understand these intergenerational predictors in a rural setting [69]. In 247,126 Indonesian and 168,317 Bangladeshi households, maternal short stature increased the risk of SCOWT, perhaps due to the intergenerational relationship of maternal height and offspring stunting (short mothers are more likely to have stunted children, who are more likely to become overweight adults). Childhood characteristics (female, older age, later birth), not breastfeeding at time of interview, and higher household SES were associated with an increased likelihood of SCOWT. SCOWT is not limited to urban settings where the effects of the nutrition transition on physical activity and diet may be more established.

Individual

The dual burden within an individual child often manifests as stunting or micronutrient deficiencies co-occurring with overweight or obesity. Trowbridge was one of the first to examine low height-for-age accompanied by high weight-for-age, in a sample of Peruvian children [70], attributing this phenomenon to a combination of body size, body composition, and body proportion factors. Martorell coined the phrase “short and plump” in reference to the stunted and overweight phenotype observed in Mexican-American children [71].

Cross-sectional surveys documenting prevalence of the different conditions do not provide insights in their sequence of development. One possibility is that common underlying factors contribute simultaneously to stunting and adiposity. This is a particularly troubling manifestation of the dual burden; if there is enough energy for a child to gain excess weight, then why do children fail to reach their linear growth potential and become stunted? The answer to this may reflect diet composition and quality. Golden described two types of nutrient deficiencies: type I deficiency (e.g.: iron, vitamin A) results in a depletion of body nutrient stores but maintenance of linear growth; type II nutrient deficiency (e.g.: zinc, protein) results in decreased linear growth [72] and is associated with poverty [73]. Zinc, for example, is found in red meat, poultry, and some seafood, and has catalytic, structural, and regulatory physiological functions, and plays a role in almost all major metabolic pathways [74]. Despite its critical role in metabolism, humans do not have zinc tissue reserves. Therefore, when dietary intake is inadequate, a child’s linear growth decreases to conserve body stores and maintain homeostasis. Although diets typical of the nutrition transition are energy-dense, and therefore provide adequate or excess calories, they lack sufficient micronutrients necessary for linear growth.

Another possibility is that poor early nutrition (i.e.: stunting, underweight) may alter physiology in ways that increase the likelihood of becoming overweight or obese later. Poor early nutrition fosters a “thrifty phenotype” with increased efficiency of fat storage. One example is the “thin fat” Indian baby phenotype; poor nutrition in utero results in small abdominal viscera and low muscle mass but high levels of adiposity [75,76]. These deficits in muscle mass relative to fat persist into adulthood, resulting an increased risk of nutrition-related chronic disease at lower BMI for Asians than for other race/ethnicities [77,78].

Childhood stunting is also associated with increased risk for adult diseases (e.g.: obesity, cardiovascular disease, and diabetes) [79–81] but less is known about how childhood stunting may affect concurrent chronic disease status. Clemente et al. assessed how mild stunting (HAZ <−1 to −2) relates to hypertension in children ages 9–19 in Brazil [82]. Although percent fat mass did not differ between mildly stunted and normal children, mildly stunted children had higher blood pressure than non-stunted children. Increased systolic blood pressure (SBP) and abdominal fat mass were correlated in mildly stunted children, but not in children of normal stature. Thus, even in mildly undernourished children, phenotypes of overnutrition are occur and may increase risk of chronic disease.

Several studies of children living in the slums of Brazil provide insight into how early stunting relates to future body composition changes. Martins et al. recruited 50 stunted and

non-stunted children (boys and girls) ages 11–15, and measured the relative quantities of lean body mass and fat body mass over a period of three years [83]. Boys and girls displayed similar changes; stunted children had a greater accumulation of fat mass and a lower lean mass gain when compared to their non-stunted counterparts over the three years of follow-up.

Hoffman et al. expanded on this work with an additional year of follow-up to analyze body fat distribution in the same children, focusing on changes in central adiposity [84]. After accounting for puberty and fat mass, stunted children had greater percent of truncal fat mass and greater four-year increase in truncal fat mass than their non-stunted counterparts, indicating they are more likely to deposit fat centrally when entering puberty, placing them at greater risk of chronic disease. These results may be explained by impaired fat oxidation in the stunted children versus non-stunted children, or alterations in cortisol metabolism, which may occur in utero or early childhood. Adolescence could be an important period to determine those at risk of obesity and its subsequent chronic diseases.

Inflammation is considered a precursor for chronic diseases, so detecting low-grade inflammation early in life may be important in the prevention of chronic diseases, in addition to improving early nutrition. The inflammatory status of stunted versus non-stunted and lean versus “over-fat” participants was compared in children ages 13–18 in South Africa [85]. “Over-fat” was defined as >30% body fat in girls and >25% body fat in boys. However, many of the children classified as “over-fat” were not overweight according to international BMI cut-points. In girls, tumor necrosis factor α (TNF- α , a pro-inflammatory cytokine) was higher in those stunted compared to those not stunted, and in a factor analysis it was associated with WHR. The etiology is not clearly understood yet, but it is possible that factors related to stunting and not fat mass may be driving the association between TNF- α and linear growth; in addition to its pro-inflammatory properties, TNF- α also inhibits bone growth. Stunted boys were more likely to be “over-fat” than their non-stunted counterparts, and in the factor analysis it was associated with C-reactive protein (CRP) and waist circumference. Small numbers of over-fat children impaired the ability to draw statistical conclusions regarding many of the inflammatory markers. Nutrition programs targeted at stunted children must consider how the interventions may effect body composition, fat accumulation, and inflammatory response that could increase the risk of chronic disease.

The theory of environmental-stress origin of stunting provides an additional perspective on the influence of early-life inflammation on growth. Environmental enteropathy, a subclinical disorder of bowel inflammation, occurs when children living in unsanitary conditions ingest high levels of fecal bacteria. This leads to impaired absorption of macro- and micronutrients and is associated with increases in systemic inflammation. Environmental enteropathy is linked to undernutrition and has been reported to account for 43% of poor linear growth [86–90]. A growing body of evidence suggests an independent association of frequent childhood diarrheal disease with increased risk of abdominal obesity, diabetes, and lipid levels [91]. Further research is needed to elucidate the mechanistic pathways of the “triple burden”, the intersection of gut inflammation, undernutrition, and overnutrition [92].

Obesity and micronutrient deficiencies

A common manifestation of obesity and co-occurring micronutrient deficiency is obesity coupled with anemia. Similar patterns may be expected with other micronutrients, such as zinc [93] and vitamin A [94], and these patterns may relate to calorie-rich but nutrient-poor diets [95]. As is the case with stunting and adiposity, there may be common underlying factors such as diets that are high in energy but deficient in micronutrients, or there may be metabolic changes associated with obesity that influence micronutrient metabolism. In the case of obesity and anemia, it has been suggested that obese individuals have increased iron requirements [96]. However, more recent research indicates obesity-related inflammation may impair iron absorption or iron utilization, and specifically highlights the role of hepcidin, a regulator of iron homeostasis, whose production is stimulated by inflammatory markers [97,98]. This suggests that total dietary iron intake may not be as important as the amount of bioavailable iron, and even so may be mediated by hepcidin. Much of the epidemiological research on obesity and iron has been conducted in high income settings, and shows that overweight children and adolescents are at increased risk of iron deficiency [99,100] and hepcidin is increased in overweight children independent of iron status [101].

In LMIC, evidence of a similar relationship between obesity and iron deficiency is emerging. Adiposity predicted iron deficiency in Indian and Moroccan children [102]. In Iranian adolescents, overweight girls had the highest prevalence of iron-deficiency anemia [103]. In a study of Mexican women and children [104], obesity was a strong predictor of iron deficiency, even though dietary iron intakes did not differ by weight status. Inflammatory markers were higher in the obese participants, and when inflammatory status was considered, obesity did not independently predict iron status. This may be due to increased levels of hepcidin which could have caused the reductions in circulating iron. Nutrition programs that target anemia and iron deficiency may not have the intended effect since obesity induced inflammation may mediate the absorption of iron.

Conclusion

Rapid economic development and urbanization have brought about a nutrition transition; diets become energy-dense but nutrient-poor, physical activity decreases and sedentary lifestyles predominate. The disease burden shifts from primarily stunting, to a high prevalence of both under- and overnutrition, and finally to mainly overweight/obesity. The dual burden at the population level reflects trends in the prevalence of each condition. Continuing or widening of economic disparities results in the persistence of pockets of undernutrition in regions or neighborhoods characterized by poverty and poor access to resources. Increasingly, obesity is affecting low income populations in urban centers, contributing to the challenges faced by national health care systems. At the household level, shared underlying determinants of poor nutritional status, as well as individual biological and behavioral differences among household members can result in disparate nutritional status across generations. Among individuals, a history of stunting may be coupled with current energy and micronutrient imbalances owing to shared underlying determinants or physiologic links.

Children are not immune from the “dual burden” of concurrent malnutrition disease states. Stunted children are more likely to accumulate central adipose tissue and be in a pre-chronic disease state than their normal height counterparts, whereas obese children are at increased risk of anemia. Although statistical independence of the dual burden household has been questioned recently, this phenomenon is increasingly common in developing countries and is likely influenced by perturbed dietary intake patterns, SES, and urbanicity.

Childhood is a crucial period of physical and cognitive development. The consequences of ignoring the changing nutrition needs and burden of disease are dire. Undernutrition is a drain on human capital and a society’s potential for growth; stunted children generally grow up to be adults of small stature with low lean body mass, limiting productivity and wages in occupations involving manual labor [105]. Undernutrition contributes to cognitive impairment, resulting in delayed school entry, poor school performance, and decreased graduation rates [105,106]. Overnutrition also affects the child’s potential contributions to society; childhood obesity is associated with increased risk of adult chronic diseases.

In order address the dual burden and improve the health of children, we need improved anthropometric monitoring; feeding programs designed with both undernutrition and overnutrition in mind; increased focus on water, sanitation, and hygiene (WASH); constant appraisal and revision of local and national public health policies; and a multifaceted and multi-institutional approach. Routine monitoring of overweight must occur with the same frequency and urgency as height. This will provide a more complete portrayal of childhood nutritional status, which is currently lacking in many areas that continue to focus solely on undernutrition. With accurate surveillance, governments will better understand the magnitude of the childhood nutritional problem, and be better informed when allocating resources to programs. When designing interventions and feeding programs, the quality of foods and supplements as well as the energy content must be considered, so as to not unintentionally add to the burden of overweight in the same children the program is striving to help. We can learn from existing feeding programs, such as Chile’s universal milk program, and shift away from feeding energy-dense foods high in fat and sugars to improve linear growth [107]. Promoting appropriate complementary feeding practices, increasing diet diversity, and providing multiple micronutrient supplements with iron may be methods to improve linear growth without contributing to overweight [108]. Incorporation of a WASH component may improve nutrient absorption, thereby improving the outcome of the feeding program or intervention and increasing cost-efficiency and resource utilization [109].

Countries and health organizations must continuously review and assess the current policies and programs to make sure they are consistent with the latest evidence-based recommendations, and consider actions that simultaneously address both under and overnutrition. As countries begin to enact new laws and policies, the global community should take note. For instance, Mexico’s effort to reduce consumption of energy-dense foods and sugar-sweetened beverages via a tax just went into effect. If this tax proves effective at improving diet quality, Mexico could be an example for other countries. Further evidence-based research on interventions that reduce overweight while improving stunting and micronutrient deficiencies is also needed. Many of these recommendations can be

unified under the multi-institutional and multi-faceted approach proposed by the WHO in their Global Strategy on Diet, Physical Activity and Health [110].

As the nutrition-related disease distribution shifts, so must the objectives of programs and policies. In an era of shrinking budgets, resources are evermore scarce. Policy makers and governments must determine how to allocate precious resources to address these disparate diseases: invest in reducing childhood stunting and nutritional deficiencies, or tackle the ever-growing burden of obesity and the future onset of its co-morbidities. Public health programs and policies must be able to address both ends of the malnutrition spectrum, and intervene appropriately. Awareness of the multilevel possibilities of the dual burden will help focus interventions and public health campaigns.

Acknowledgements

This project was supported by an educational grant from Wyeth Nutrition. We are grateful to the Carolina Population Center (R24 HD050924) for general support. LA and ET conceived the idea for the review; ET wrote the first draft of the manuscript; both authors contributed to, read, and approved the final manuscript.

References

1. Popkin BM. The nutrition transition in low-income countries: an emerging crisis. *Nutr. Rev.* 1994 Sep;52:285–298. [PubMed: 7984344]
2. Gillespie, SR.; Haddad, LJ. *The Double Burden Of Malnutrition In Asia : Causes, Consequences, And Solutions.* Sage Publications India Pvt Limited; 2003.
3. Popkin BM, Richards MK, Montiero CA. Stunting is Associated with Overweight in Children of Four Nations That Are Undergoing the Nutrition Transition. *J. Nutr.* 1996 Dec;126:3009–3016. [PubMed: 9001368]
4. Doak CM, Adair LS, Bentley M, Monteiro C, Popkin BM. The dual burden household and the nutrition transition paradox. *Int J Obes (Lond).* 2005 Jan;29:129–136. [PubMed: 15505634]
5. Doak CM, Adair LS, Monteiro C, Popkin BM. Overweight and underweight coexist within households in Brazil, China and Russia. *J. Nutr.* 2000 Dec;130:2965–2971. [PubMed: 11110855]
6. WHO Child Growth Standards based on length/height, weight and age. *Acta Paediatr Suppl.* 2006 Apr;450:76–85. [PubMed: 16817681]
7. World Health Organization. Report of a WHO Expert Committee. World Health Organization; 1995. Physical status: the use and interpretation of anthropometry; p. 854 Report No.
8. Cole TJ, Bellizzi MC, Flegal KM, Dietz WH. Establishing a standard definition for child overweight and obesity worldwide: international survey. *BMJ.* 2000 May;320:1240–1243. [PubMed: 10797032]
9. Flegal KM, Ogden CL. Childhood obesity: are we all speaking the same language? *Adv Nutr.* 2011 Mar;2:159S–166S. [PubMed: 22332047]
10. Black RE, Allen LH, Bhutta ZA, Caulfield LE, de Onis M, Ezzati M, Mathers C, Rivera J. Maternal and child undernutrition: global and regional exposures and health consequences. *Lancet.* 2008 Jan;371:243–260. [PubMed: 18207566]
11. Black RE, Victora CG, Walker SP, Bhutta ZA, Christian P, de Onis M, Ezzati M, Grantham-McGregor S, Katz J, Martorell R, Uauy R. the Maternal and Child Nutrition Study Group. Maternal and child undernutrition and overweight in low-income and middle-income countries. *Lancet.* 2013 Jun.
12. De Onis M, Blössner M, Borghi E. Prevalence and trends of stunting among pre-school children, 1990–2020. *Public Health Nutr.* 2012 Jan;15:142–148. [PubMed: 21752311]
13. McLean E, Cogswell M, Egli I, Wojdyla D, de Benoist B. Worldwide prevalence of anaemia, WHO Vitamin and Mineral Nutrition Information System, 1993–2005. *Public Health Nutr.* 2009 Apr;12:444–454. [PubMed: 18498676]

14. WHO. WHO Global Database on Vitamin A Deficiency. Geneva: World Health Organization; 2009. Global prevalence of vitamin A deficiency in populations at risk 1995–2005.
15. Onis, M de; Blössner, M.; Borghi, E. Global prevalence and trends of overweight and obesity among preschool children. *Am J Clin Nutr.* 2010 Nov.92:1257–1264. [PubMed: 20861173]
16. Onis, M de; Blössner, M. Prevalence and trends of overweight among preschool children in developing countries. *Am J Clin Nutr.* 2000 Oct.72:1032–1039. [PubMed: 11010948]
17. Ng SW, Popkin BM. Time use and physical activity: a shift away from movement across the globe. *Obes Rev.* 2012 Aug.13:659–680. [PubMed: 22694051]
18. Rivera JA, Barquera S, González-Cossío T, Olaiz G, Sepúlveda J. Nutrition transition in Mexico and in other Latin American countries. *Nutr. Rev.* 2004 Jul.62:S149–S157. [PubMed: 15387482]
19. Monteiro CA, Conde WL, Lu B, Popkin BM. Obesity and inequities in health in the developing world. *Int. J. Obes. Relat. Metab. Disord.* 2004 Sep.28:1181–1186. [PubMed: 15211362]
20. McGuire J, Popkin B. Beating the zero sum game: women and nutrition in the Third World. *Food and Nutrition Bulletin.* 1989; 11:38–63.
21. Popkin BM, Adair LS, Ng SW. Global nutrition transition and the pandemic of obesity in developing countries. *Nutr. Rev.* 2012 Jan.70:3–21. [PubMed: 22221213]
22. Jones-Smith JC, Gordon-Larsen P, Siddiqi A, Popkin BM. Is the burden of overweight shifting to the poor across the globe? Time trends among women in 39 low- and middle-income countries (1991–2008). *Int J Obes (Lond).* 2012 Aug.36:1114–1120. [PubMed: 21912397]
23. Jinabhai CC, Taylor M, Sullivan KR. Changing patterns of under- and over-nutrition in South African children-future risks of non-communicable diseases. *Ann Trop Paediatr.* 2005 Mar.25:3–15. [PubMed: 15814043]
24. Cattaneo A, Timmer A, Bomestar T, Bua J, Kumar S, Tamburlini G. Child nutrition in countries of the Commonwealth of Independent States: time to redirect strategies? *Public Health Nutr.* 2008 Dec.11:1209–1219. [PubMed: 18647428]
25. Motlagh ME, Kelishadi R, Amirkhani MA, Ziaoddini H, Dashti M, Aminae T, Ardalan G, Mirmoghtadaee P, Keshavarz S, Poursafa P. Double burden of nutritional disorders in young Iranian children: findings of a nationwide screening survey. *Public Health Nutr.* 2011 Apr.14:605–610. [PubMed: 20843401]
26. Ferreira, HdaS; Luciano, SCM. Prevalence of extreme anthropometric measurements in children from Alagoas, Northeastern Brazil. *Rev Saude Publica.* 2010 Apr.44:377–380. [PubMed: 20339640]
27. Gardner K, Bird J, Canning PM, Frizzell LM, Smith LM. Prevalence of overweight, obesity and underweight among 5-year-old children in Saint Lucia by three methods of classification and a comparison with historical rates. *Child Care Health Dev.* 2011 Jan.37:143–149. [PubMed: 20854448]
28. Jafar TH, Qadri Z, Islam M, Hatcher J, Bhutta ZA, Chaturvedi N. Rise in childhood obesity with persistently high rates of undernutrition among urban school-aged Indo-Asian children. *Arch. Dis. Child.* 2008 May.93:373–378. [PubMed: 17942586]
29. Wang X, Höjer B, Guo S, Luo S, Zhou W, Wang Y. Stunting and “overweight” in the WHO Child Growth Standards - malnutrition among children in a poor area of China. *Public Health Nutr.* 2009 Nov.12:1991–1998. [PubMed: 19656437]
30. Custodio E, Descalzo MA, Roche J, Molina L, Sánchez I, Lwanga M, Torres AM, Fernández-Zincke E, Bernis C, Villamor E, Baylin A. The economic and nutrition transition in Equatorial Guinea coincided with a double burden of over- and under nutrition. *Econ Hum Biol.* 2010 Mar. 8:80–87. [PubMed: 19959405]
31. Kimani-Murage EW, Kahn K, Pettifor JM, Tollman SM, Dunger DB, Gómez-Olivé XF, Norris SA. The prevalence of stunting, overweight and obesity, and metabolic disease risk in rural South African children. *BMC Public Health.* 2010; 10:158. [PubMed: 20338024]
32. Gross R, Lechtig A, López de Romaña D. Baseline evaluation of nutritional status and government feeding programs in Chiclayo, Peru. *Food Nutr Bull.* 2006; 27:S115–S121. [PubMed: 17455397]
33. Peña Reyes ME, Cárdenas Barahona EE, Lamadrid PS, Del Olmo Calzada M, Malina RM. Growth status of indigenous school children 6–14 years in the Tarahumara Sierra, Northern Mexico, in 1990 and 2007. *Ann. Hum. Biol.* 2009 Dec.36:756–769. [PubMed: 19852675]

34. Jildeh C, Papandreou C, Abu Mourad T, Hatzis C, Kafatos A, Qasrawi R, Philalithis A, Abdeen Z. Assessing the nutritional status of Palestinian adolescents from East Jerusalem: a school-based study 2002–03. *J. Trop. Pediatr.* 2011 Feb;57:51–58. [PubMed: 20675716]
35. Armstrong MEG, Lambert MI, Lambert EV. Secular trends in the prevalence of stunting, overweight and obesity among South African children (1994–2004). *Eur J Clin Nutr.* 2011 Jul; 65:835–840. [PubMed: 21505505]
36. Barros AJD, Victora CG, Santos IS, Matijasevich A, Araújo CL, Barros FC. Infant malnutrition and obesity in three population-based birth cohort studies in Southern Brazil: trends and differences. *Cad Saude Publica.* 2008; 24(Suppl 3):S417–S426. [PubMed: 18797717]
37. Kain J, Uauy R, Lera L, Taibo M, Albala C. Trends in Height and BMI of 6-Year-Old Children during the Nutrition Transition in Chile. *Obesity Research.* 2005; 13:2178–2186. [PubMed: 16421353]
38. Dasgupta P, Saha R, Nubé M. Changes in body size, shape and nutritional status of middle-class Bengali boys of Kolkata, India, 1982–2002. *Econ Hum Biol.* 2008 Mar;6:75–94. [PubMed: 17618843]
39. Ozgüven I, Ersoy B, Ozgüven AA, Erbay PD. Evaluation of nutritional status in Turkish adolescents as related to gender and socioeconomic status. *J Clin Res Pediatr Endocrinol.* 2010; 2:111–116. [PubMed: 21274324]
40. Bustos P, Muñoz S, Vargas C, Amigo H. Evolution of the nutritional situation of indigenous and non-indigenous Chilean schoolchildren. *Ann. Hum. Biol.* 2009 Jun;36:298–307. [PubMed: 19296262]
41. Daboné C, Delisle HF, Receveur O. Poor nutritional status of schoolchildren in urban and peri-urban areas of Ouagadougou (Burkina Faso). *Nutr J.* 2011; 10:34. [PubMed: 21504619]
42. Piperata BA, Spence JE, Da-Gloria P, Hubbe M. The nutrition transition in amazonia: rapid economic change and its impact on growth and development in Ribeirinhos. *Am. J. Phys. Anthropol.* 2011 Sep;146:1–13. [PubMed: 21541919]
43. Shi Z, Lien N, Nirmal Kumar B, Dalen I, Holmboe-Ottesen G. The sociodemographic correlates of nutritional status of school adolescents in Jiangsu Province, China. *Journal of Adolescent Health.* 2005 Oct;37:313–322. [PubMed: 16182142]
44. Reddy SP, Resnicow K, James S, Kambaran N, Omardien R, Mbewu AD. Underweight, overweight and obesity among South African adolescents: results of the 2002 National Youth Risk Behaviour Survey. *Public Health Nutr.* 2009 Feb;12:203–207. [PubMed: 18547451]
45. Romaguera D, Samman N, Farfán N, Lobo M, Pons A, Tur JA. Nutritional status of the Andean population of Puna and Quebrada of Humahuaca, Jujuy, Argentina. *Public Health Nutr.* 2008 Jun; 11:606–615. [PubMed: 17894917]
46. Jayatissa R, Ranbanda RM. Prevalence of challenging nutritional problems among adolescents in Sri Lanka. *Food Nutr Bull.* 2006 Jun;27:153–160. [PubMed: 16786981]
47. Shoeps DO, de Abreu LC, Valenti VE, Nascimento VG, de Oliveira AG, Gallo PR, Wajnsztein R, Leone C. Nutritional status of pre-school children from low income families. *Nutr J.* 2011; 10:43. [PubMed: 21549003]
48. Wickramasinghe V, Lamabadusuriya S, Atapattu N, Sathyadas G, Kuruparanantha S, Karunarathne P. Nutritional status of schoolchildren in an urban area of Sri Lanka. *Ceylon Medical Journal* [Internet]. 2010 May;49 Available from: <http://sljol.info/index.php/CMJ/article/view/1920>.
49. Jeemon P, Prabhakaran D, Mohan V, Thankappan KR, Joshi PP, Ahmed F, Chaturvedi V, Reddy KS. Double burden of underweight and overweight among children (10–19 years of age) of employees working in Indian industrial units. *Natl Med J India.* 2009 Aug;22:172–176. [PubMed: 20131480]
50. Mansourian M, Marateb HR, Kelishadi R, Motlagh ME, Aminaee T, Taslimi M, Majdzadeh R, Heshmat R, Ardalan G, Poursafa P. First growth curves based on the World Health Organization reference in a Nationally-Representative Sample of Pediatric Population in the Middle East and North Africa (MENA): the CASPIAN-III study. *BMC Pediatr.* 2012; 12:149. [PubMed: 22985219]

51. Tang HK, Dibley MJ, Sibbritt D, Tran HM. Gender and socio-economic differences in BMI of secondary high school students in Ho Chi Minh city. *Asia Pac J Clin Nutr.* 2007; 16:74–83. [PubMed: 17215183]
52. Dahinten SL, Castro LE, Zavatti JR, Forte LM, Oyhenart EE. Growth of school children in different urban environments in Argentina. *Ann. Hum. Biol.* 2011 Mar.38:219–227. [PubMed: 20849249]
53. Van Lierop A, Nam NV, Doak C, Hung LQ, Binh TQ, Hoekstra J, de Vries PJ. Regional clustering of anthropometric dimensions of primary school children in rural and suburban Vietnam. *Asia Pac J Clin Nutr.* 2008; 17:603–607. [PubMed: 19114397]
54. Jinabhai CC, Reddy P, Taylor M, Monyeke D, Kamabaran N, Omardien R, Sullivan KR. Sex differences in under and over nutrition among school-going Black teenagers in South Africa: an uneven nutrition trajectory. *Trop. Med. Int. Health.* 2007 Aug.12:944–952. [PubMed: 17697089]
55. Duran P, Caballero B, de Onis M. The association between stunting and overweight in Latin American and Caribbean preschool children. *Food Nutr Bull.* 2006 Dec.27:300–305. [PubMed: 17209471]
56. Saldiva SRDM, Escuder MML, Venâncio SI, Benicio MHD. Prevalence of obesity in preschool children from five towns in São Paulo State, Brazil. *Cad Saude Publica.* 2004 Dec.20:1627–1632. [PubMed: 15608865]
57. MEASUREDHS. STATcompiler [Internet]. Available from: <http://statcompiler.com/>.
58. Mamabolo RL, Alberts M, Steyn NP, Delemarre-van de Waal HA, Levitt NS. Prevalence and determinants of stunting and overweight in 3-year-old black South African children residing in the Central Region of Limpopo Province, South Africa. *Public Health Nutr.* 2005 Aug.8:501–508. [PubMed: 16153331]
59. Fernald LC, Neufeld LM. Overweight with concurrent stunting in very young children from rural Mexico: prevalence and associated factors. *Eur J Clin Nutr.* 2007 May.61:623–632. [PubMed: 17136036]
60. Garrett JL, Ruel MT. Stunted child-overweight mother pairs: prevalence and association with economic development and urbanization. *Food Nutr Bull.* 2005 Jun.26:209–221. [PubMed: 16060222]
61. Doak C, Adair L, Bentley M, Fengying Z, Popkin B. The underweight/overweight household: an exploration of household sociodemographic and dietary factors in China. *Public Health Nutr.* 2002 Feb.5:215–221. [PubMed: 12027287]
62. WHO. WHO; Underweight in children [Internet]. Available from: http://www.who.int/gho/mdg/poverty_hunger/underweight_text/en/index.html. [cited 2013 Mar 23]
63. WHO. WHO; Obesity [Internet]. Available from: http://www.who.int/gho/ncd/risk_factors/obesity_text/en/index.html. [cited 2013 Mar 23]
64. Jehn M, Brewis A. Paradoxical malnutrition in mother-child pairs: untangling the phenomenon of over- and under-nutrition in underdeveloped economies. *Econ Hum Biol.* 2009 Mar.7:28–35. [PubMed: 19246260]
65. Dieffenbach S, Stein AD. Stunted Child/Overweight Mother Pairs Represent a Statistical Artifact, Not a Distinct Entity. *J. Nutr.* 2012 Apr.142:771–773. [PubMed: 22378330]
66. Saibul N, Shariff ZM, Lin KG, Kandiah M, Ghani NA, Rahman HA. Food variety score is associated with dual burden of malnutrition in Orang Asli (Malaysian indigenous peoples) households: implications for health promotion. *Asia Pac J Clin Nutr.* 2009; 18:412–422. [PubMed: 19786390]
67. Khor GL, Sharif ZM. Dual forms of malnutrition in the same households in Malaysia—a case study among Malay rural households. *Asia Pac J Clin Nutr.* 2003; 12:427–437. [PubMed: 14672867]
68. Barquera S, Peterson KE, Must A, Rogers BL, Flores M, Houser R, Monterrubio E, Rivera-Dommarco JA. Coexistence of maternal central adiposity and child stunting in Mexico. *Int J Obes (Lond).* 2007 Apr.31:601–607. [PubMed: 17224933]
69. Oddo VM, Rah JH, Semba RD, Sun K, Akhter N, Sari M, de Pee S, Moench-Pfanner R, Bloem M, Kraemer K. Predictors of maternal and child double burden of malnutrition in rural Indonesia and Bangladesh. *Am. J. Clin. Nutr.* 2012 Apr.95:951–958. [PubMed: 22357721]

70. Trowbridge FL, Marks JS, Lopez de Romana G, Madrid S, Boutton TW, Klein PD. Body composition of Peruvian children with short stature and high weight-for-height. II. Implications for the interpretation for weight-for-height as an indicator of nutritional status. *Am. J. Clin. Nutr.* 1987 Sep;46:411–418. [PubMed: 3630960]
71. Martorell R, Mendoza FS, Castillo RO, Pawson IG, Budge CC. Short and plump physique of Mexican-American children. *Am. J. Phys. Anthropol.* 1987 Aug;73:475–487. [PubMed: 3661685]
72. Golden MH. Specific deficiencies versus growth failure: Type I and type II nutrients. *Journal of Nutritional and Environmental Medicine.* 1996; 6:301–308.
73. Golden MH. The nature of nutritional deficiency in relation to growth failure and poverty. *Acta Paediatr Scand Suppl.* 1991; 374:95–110. [PubMed: 1957635]
74. Brown KH, Rivera JA, Bhutta Z, Gibson RS, King JC, Lönnerdal B, Ruel MT, Sandtröm B, Wasantwisut E, Hotz C. International Zinc Nutrition Consultative Group (IZiNCG) technical document #1. Assessment of the risk of zinc deficiency in populations and options for its control. *Food Nutr Bull.* 2004 Mar;25:S99–S203. [PubMed: 18046856]
75. Yajnik CS, Fall CHD, Coyaji KJ, Hirve SS, Rao S, Barker DJP, Joglekar C, Kellingray S. Neonatal anthropometry: the thin-fat Indian baby. The Pune Maternal Nutrition Study. *Int. J. Obes. Relat. Metab. Disord.* 2003 Feb;27:173–180. [PubMed: 12586996]
76. Muthayya S, Dwarkanath P, Thomas T, Vaz M, Mhaskar A, Mhaskar R, Thomas A, Bhat S, Kurpad A. Anthropometry and body composition of south Indian babies at birth. *Public Health Nutr.* 2006 Oct;9:896–903. [PubMed: 17010256]
77. WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet.* 2004 Jan;363:157–163. [PubMed: 14726171]
78. Deurenberg P, Deurenberg-Yap M, Guricci S. Asians are different from Caucasians and from each other in their body mass index/body fat per cent relationship. *Obes Rev.* 2002 Aug;3:141–146. [PubMed: 12164465]
79. Uauy R, Kain J, Mericq V, Rojas J, Corvalán C. Nutrition, child growth, and chronic disease prevention. *Annals of Medicine.* 2008 Jan;40:11–20. [PubMed: 18246473]
80. Popkin BM. The shift in stages of the nutrition transition in the developing world differs from past experiences! *Public Health Nutr.* 2002 Feb;5:205–214. [PubMed: 12027286]
81. Fernandez-Twinn DS, Ozanne SE. Mechanisms by which poor early growth programs type-2 diabetes, obesity and the metabolic syndrome. *Physiol. Behav.* 2006 Jun;88:234–243. [PubMed: 16782139]
82. Clemente APG, Santos CD, Silva AAB, Martins VJ, Marchesano AC, Fernandes MB, Albuquerque MP, Sawaya AL. Mild stunting is associated with higher blood pressure in overweight adolescents. *Arquivos Brasileiros de Cardiologia.* 2012 Jan;98:06–12.
83. Martins PA, Hoffman DJ, Fernandes MTB, Nascimento CR, Roberts SB, Sesso R, Sawaya AL. Stunted children gain less lean body mass and more fat mass than their non-stunted counterparts: a prospective study. *Br. J. Nutr.* 2004 Nov;92:819–825. [PubMed: 15533271]
84. Hoffman DJ, Martins PA, Roberts SB, Sawaya AL. Body fat distribution in stunted compared with normal-height children from the shantytowns of São Paulo, Brazil. *Nutrition.* 2007 Sep;23:640–646. [PubMed: 17679045]
85. Kruger HS, Pretorius R, Schutte AE. Stunting, adiposity, and low-grade inflammation in African adolescents from a township high school. *Nutrition.* 2010 Jan;26:90–99. [PubMed: 20005466]
86. DeBoer MD, Lima AAM, Oría RB, Scharf RJ, Moore SR, Luna MA, Guerrant RL. Early childhood growth failure and the developmental origins of adult disease: do enteric infections and malnutrition increase risk for the metabolic syndrome? *Nutr. Rev.* 2012 Nov;70:642–653. [PubMed: 23110643]
87. Lin A, Arnold BF, Afreen S, Goto R, Huda TMN, Haque R, Raqib R, Unicomb L, Ahmed T, Colford JM, Luby SP. Household Environmental Conditions Are Associated with Enteropathy and Impaired Growth in Rural Bangladesh. *Am J Trop Med Hyg.* 2013 Jul;89:130–137. [PubMed: 23629931]

88. Campbell DI, Elia M, Lunn PG. Growth faltering in rural Gambian infants is associated with impaired small intestinal barrier function, leading to endotoxemia and systemic inflammation. *J. Nutr.* 2003 May.133:1332–1338. [PubMed: 12730419]
89. Lunn PG, Northrop-Clewes CA, Downes RM. Intestinal permeability, mucosal injury, and growth faltering in Gambian infants. *Lancet.* 1991 Oct.338:907–910. [PubMed: 1681266]
90. Lunn PG. Growth retardation and stunting of children in developing countries. *Br. J. Nutr.* 2002 Aug.88:109–110. [PubMed: 12144713]
91. Margolis R. Childhood Morbidity and Health in Early Adulthood: Life course linkages in a high morbidity context. *Adv Life Course Res.* 2010 Dec.15:132–146. [PubMed: 21516232]
92. Guerrant RL, DeBoer MD, Moore SR, Scharf RJ, Lima AAM. The impoverished gut--a triple burden of diarrhoea, stunting and chronic disease. *Nat Rev Gastroenterol Hepatol.* 2013 Apr. 10:220–229. [PubMed: 23229327]
93. Weisstaub G, Hertrampf E, López de Romaña D, Salazar G, Bugueño C, Castillo-Duran C. Plasma zinc concentration, body composition and physical activity in obese preschool children. *Biol Trace Elem Res.* 2007 Aug.118:167–174. [PubMed: 17873359]
94. De Souza Valente da Silva L, Valeria da Veiga G, Ramalho RA. Association of serum concentrations of retinol and carotenoids with overweight in children and adolescents. *Nutrition.* 2007 May.23:392–397. [PubMed: 17433621]
95. Pinhas-Hamiel O, Newfield RS, Koren I, Agmon A, Lilos P, Phillip M. Greater prevalence of iron deficiency in overweight and obese children and adolescents. *Int J Obes Relat Metab Disord.* 2003; 27:416–418. [PubMed: 12629572]
96. Yanoff LB, Menzie CM, Denkinger B, Sebring NG, McHugh T, Remaley AT, Yanovski JA. Inflammation and iron deficiency in the hypoferrremia of obesity. *Int J Obes (Lond).* 2007 Sep. 31:1412–1419. [PubMed: 17438557]
97. McClung JP, Karl JP. Iron deficiency and obesity: the contribution of inflammation and diminished iron absorption. *Nutr. Rev.* 2009 Feb.67:100–104. [PubMed: 19178651]
98. Bekri S, Gual P, Anty R, Luciani N, Dahman M, Ramesh B, Iannelli A, Staccini-Myx A, Casanova D, Ben Amor I, Saint-Paul M-C, Huet P-M, Sadoul J-L, Gugenheim J, Srai SKS, Tran A, Le Marchand-Brustel Y. Increased adipose tissue expression of hepcidin in severe obesity is independent from diabetes and NASH. *Gastroenterology.* 2006 Sep.131:788–796. [PubMed: 16952548]
99. Nead KG, Halterman JS, Kaczorowski JM, Auinger P, Weitzman M. Overweight children and adolescents: a risk group for iron deficiency. *Pediatrics.* 2004 Jul.114:104–108. [PubMed: 15231915]
100. Brotanek JM, Gosz J, Weitzman M, Flores G. Iron deficiency in early childhood in the United States: risk factors and racial/ethnic disparities. *Pediatrics.* 2007 Sep.120:568–575. [PubMed: 17766530]
101. Aeberli I, Hurrell RF, Zimmermann MB. Overweight children have higher circulating hepcidin concentrations and lower iron status but have dietary iron intakes and bioavailability comparable with normal weight children. *Int J Obes (Lond).* 2009 Oct.33:1111–1117. [PubMed: 19636315]
102. Zimmermann MB, Zeder C, Muthayya S, Winichagoon P, Chaouki N, Aeberli I, Hurrell RF. Adiposity in women and children from transition countries predicts decreased iron absorption, iron deficiency and a reduced response to iron fortification. *Int J Obes (Lond).* 2008 Jul.32:1098–1104. [PubMed: 18427564]
103. Eftekhari M, Mozaffari-Khosravi H, Shidfar F. The relationship between BMI and iron status in iron-deficient adolescent Iranian girls. *Public Health Nutr.* 2009 Dec.12:2377–2381. [PubMed: 19278566]
104. Cepeda-Lopez AC, Osendarp SJ, Melse-Boonstra A, Aeberli I, Gonzalez-Salazar F, Feskens E, Villalpando S, Zimmermann MB. Sharply higher rates of iron deficiency in obese Mexican women and children are predicted by obesity-related inflammation rather than by differences in dietary iron intake. *Am J Clin Nutr.* 2011 May.93:975–983. [PubMed: 21411619]
105. Victora CG, Adair L, Fall C, Hallal PC, Martorell R, Richter L, Sachdev HS. Maternal and child undernutrition: consequences for adult health and human capital. *Lancet.* 2008 Jan.371:340–357. [PubMed: 18206223]

106. Benton D. The influence of dietary status on the cognitive performance of children. *Mol Nutr Food Res*. 2010 Apr.54:457–470. [PubMed: 20077417]
107. Garmendia ML, Corvalan C, Uauy R. Addressing malnutrition while avoiding obesity: minding the balance. *Eur J Clin Nutr*. 2013 May.67:513–517. [PubMed: 23361159]
108. Bhutta ZA, Das JK, Rizvi A, Gaffey MF, Walker N, Horton S, Webb P, Lartey A, Black RE. Lancet Nutrition Interventions Review Group. Maternal and Child Nutrition Study Group. Evidence-based interventions for improvement of maternal and child nutrition: what can be done and at what cost? *Lancet*. 2013 Aug.382:452–477. [PubMed: 23746776]
109. Humphrey JH. Child undernutrition, tropical enteropathy, toilets, and handwashing. *Lancet*. 2009 Sep.374:1032–1035. [PubMed: 19766883]
110. WHO. Geneva: World Health Organization; 2004. Global strategy on diet, physical activity and health.

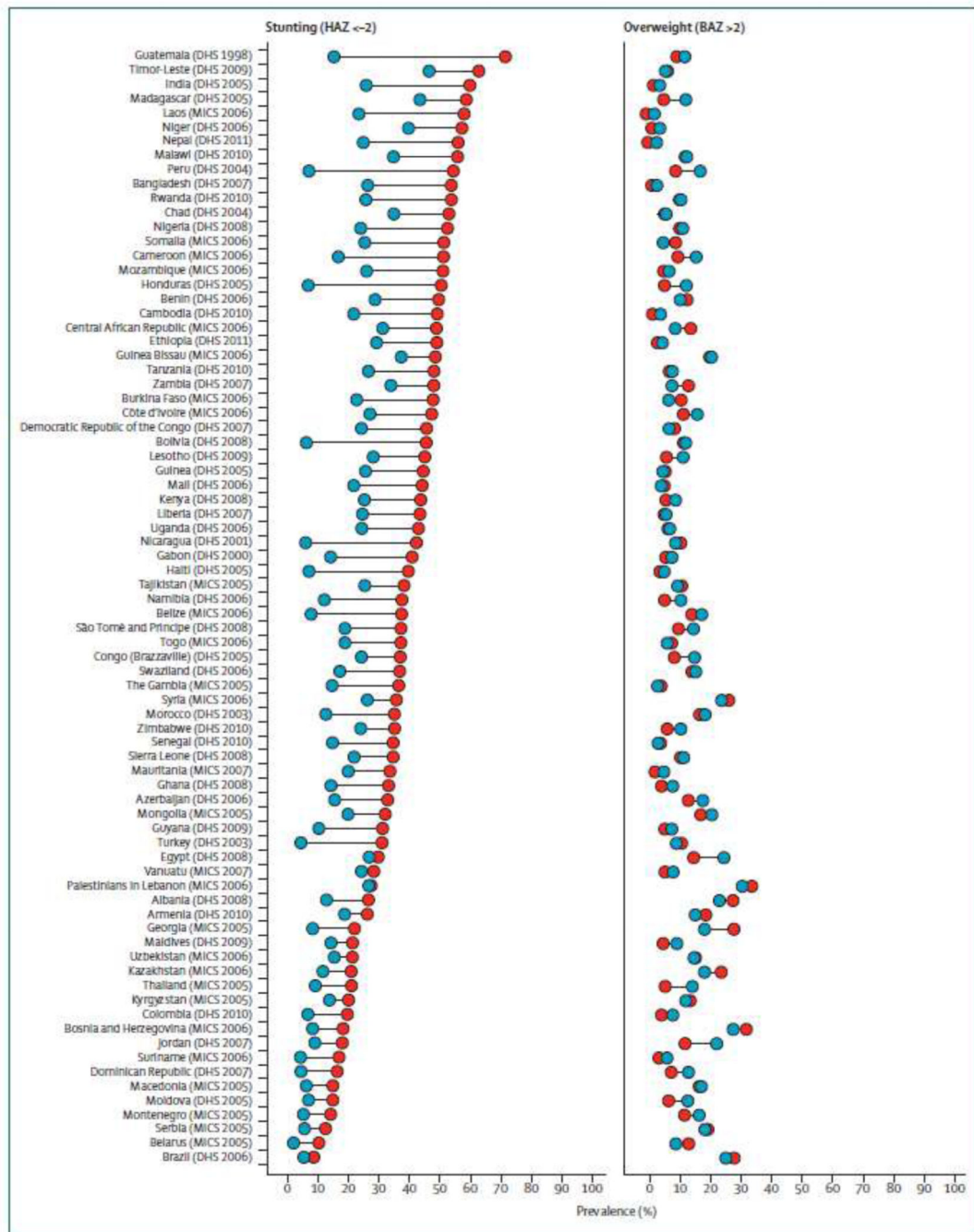


Figure 1. Prevalence of stunting (HAZ <-2) and overweight (BAZ >2) for lowest wealth quintile (red circles) and highest wealth quintile (blue circles) in children under 5y [11]. Reproduced with permission.

Country	Year	Stunted (%)			Wasted (%)			Overweight (%)		
		Total	Household wealth index		Total	Household wealth index		Total	Household wealth index	
			Lowest	Highest		Lowest	Highest		Lowest	Highest
Bolivia	2003	32.3	48.7	8.5	1.7	2.0	1.1	8.9	9.3	11.9
Bolivia	2008	27.1	45.9	6.5	1.4	2.2	1.6	8.5	7.3	11.3
Colombia	2005	15.9	25.2	5.2	1.5	1.7	1.3	4.2	2.7	7.6
Colombia	2010	13.2	19.4	6.8	0.9	1.2	0.4	4.8	3.5	7.3
Dominican Republic	2002	11.4	19.1	4.6	2.0	3.0	1.0	8.5	4.8	13.1
Dominican Republic	2007	9.8	15.8	4.7	2.2	2.0	1.8	8.1	6.5	12.1
Guyana	2009	18.2	29.6	9.8	5.3	4.8	5.1	6.2	4.2	6.1
Haiti	2000	28.7	37.9	11.1	5.4	6.8	5.8	3.0	3.7	2.9
Haiti	2005-06	29.4	41.0	8.0	10.2	10.1	6.4	3.8	3.2	3.8
Haiti	2012	21.9	31.0	6.6	5.1	5.6	3.2	3.6	3.5	3.1
Honduras	2005-06	30.0	50.4	6.7	1.3	2.1	0.8	5.7	3.2	11.5
Honduras	2011-12	22.6	42.1	8.0	1.4	1.8	1.3	5.1	3.5	9.1
Nicaragua	2001	24.9	42.2	5.5	2.2	3.5	1.7	6.7	7.9	8.9
Peru	2004-06	29.3	54.3	4.8	1.0	1.6	0.3	9.1	5.9	18.5
Peru	2009	23.8	45.3	4.2	0.6	0.7	0.5	10.2	5.8	17.8
Peru	2012	18.1	38.8	3.1	0.6	0.7	0.6	7.1	4.1	17.1

Figure 2. Percent of children under 5 classified as stunted (HAZ <-2), wasted (WHZ <-2), and overweight (WHZ >2) for selected Latin American countries (Data source: Demographic and Health Surveys, STATcompiler)