Chapter 2

NUTRITIONAL CHALLENGES FOR ECONOMIC DEVELOPMENT

From an economic development perspective, malnutrition among women of reproductive age and among young children is of particular concern. This is because prevalence rates tend to be highest among these groups, their nutritional well-being is most crucial for future generations' prosperity, and targeted interventions have been found to be most cost-effective during the period from pregnancy to when a child is 24 months of age (Bryce et al. 2008; Engle et al. 2007; Horton et al. 2010).

Malnutrition among pregnant women can have serious long-term effects because of the biological intergenerational transmission of malnutrition in utero (see below). And malnourished mothers tend to be less able to provide sufficient and nutritious breast milk and adequate care to their children, which, in turn, increases the children's risk of being malnourished. Malnutrition during early childhood reduces cognitive and physical capacity and productivity in children's later life, so children born from malnourished mothers tend to be economically disadvantaged from the beginning compared to their well-nourished peers.

Due to the importance of maternal and child nutrition for economic and social development, our analysis focuses mainly on the nutritional status of preschool children and of women of reproductive age—especially the children's mothers. Nutritional status is determined by anthropometric measures. Anthropometric measurements, particularly for children under five years of age, have been shown to be reliable nutrition indicators and, more broadly, critical development indicators for several reasons.

First, focusing on young children, who are typically the weakest household members, captures aspects of intrahousehold distribution of resources (including food and care time) that are ignored when using household-level indicators such as poverty (as measured by household income) or food and nutrient consumption derived from standard household consumption surveys. Second, anthropometrics are measurements of the human body and hence indicators of nutritional (and health) outcomes (unlike food and nutrient consumption/intake). Third, they aggregately capture adequacy of food intake in terms of macro- and micronutrients, health status, and the interaction of nutrient absorption and diseases in the human body. Fourth, young children's nutritional status tends to be more responsive to changing living conditions and shocks than that of adults due to children's high physiological nutrient requirements for growth, special dietary needs, and high vulnerability to diseases common in underdeveloped settings. Fifth, high prevalence rates of malnourished children are often associated with poor delivery of basic public services (especially in the health sector), poor quality of drinking water and sanitation infrastructure, low educational levels, gender inequality, and high population growth rates.

Using selected anthropometric indicators, the following two sections examine the double burden of malnutrition and the growth-nutrition disconnect—Egypt's two main nutritional challenges—in detail.

The Double Burden of Malnutrition

Under- and overnutrition can coexist at the population, the family, and even the individual levels. Common forms of the double burden of malnutrition at the population level are high prevalence rates of stunting among children and overweight among adults; at the family level, stunted children with overweight mothers; and at the individual level, stunted but overweight children. Other common forms of the double burden of malnutrition include the coexistence of micronutrient deficiencies and overweight. Because this study is concerned with chronic malnutrition and the link to (future) economic development, it focuses on the coexistence of stunting among children under five years of age and overweight among them and among their mothers.

From a development policy analysis point of view, a situation where the double burden of malnutrition is prevalent at the population level but under- and overnutrition do not coexist in the same family or the same individual among a considerable share of the population may be of less concern compared to a situation where the double burden of malnutrition is also prevalent at the family and the individual levels. The former situation does not constitute a novel problem and may rather be an outcome of other, well-studied development challenges such as large inequalities in the distribution of wealth and access to public services. For example, one may imagine a situation in a country where undernutrition affects almost exclusively the poor and overnutrition almost exclusively the rich (Corsi, Kyu, and Subramanian 2011; Subramanian, Perkins, and Khan 2009). Because the problems of undernutrition and overnutrition are separate here, some policy implications from analyses looking at under- and overnutrition independently of each other should be transferable to this situation (given comparable settings).

However, such transferability of findings is inappropriate and can even lead to counterproductive policy recommendations where the double burden of malnutrition is also prevalent at the family and the individual levels, as in the case of Egypt (as we will show below). In such cases, households may face circumstances that can simultaneously contribute to both under- and overnutrition, and policies that influence these circumstances require careful consideration of their potential effects on both under- and overnutrition. Moreover, these circumstances must occur at large in order to result in prevalence rates of under- and overnutrition coexisting in families and individuals that are sufficiently high to be a public health concern.

Physiological Pathways to the Double Burden of Malnutrition

In the search for factors that cause and contribute to the double burden of malnutrition, it is important to note that there are genetic conditions that favor its manifestation and severity. Due to lack of suitable cohort data, we cannot estimate nutritional effects because of genetic factors and assess their importance relative to those of socioeconomic factors captured in our analyses (presented later in this book). Genetic conditions' contribution to the prevalence of malnutrition among the current generation, however, is likely to be of minor relevance for our study, which focuses on factors that can be influenced by policy changes in the present. Yet factors that cause and contribute to malnutrition in the current generation also increase the risk of malnutrition among future generations. The following literature review therefore describes such physiological pathways and draws particular attention to those that facilitate the phenomena of overweight mothers having stunted children and of stunted children being overweight at the same time.

The health literature provides robust evidence that under- and overnutrition is partly predetermined in utero. It also provides comprehensive evidence for several interlinked pathways by which fetal nutrition affects patterns of physical growth and body composition and increases the risk of nutrition-related noncommunicable diseases (NCDs) in the individuals' later lives. Most of these pathways build on or are consistent with Barker's well-known "mismatch hypothesis" (Barker 1988)—also known as "thrifty phenotype hypothesis" (Hales and Barker 1992, 2001). The hypothesis offers an explanation for the intergenerational transmission of malnutrition in terms of both over- and undernutrition and related NCDs. According to the hypothesis, poor nutrition during fetal life causes irreversible metabolic changes that are possibly designed to ensure survival in similar conditions as those faced in utero but increase the risk of nutritional diseases during adulthood when facing different conditions (Barker 1988).

To be specific, undernutrition during pregnancy in terms of macro- and micronutrient deficiencies is associated with higher risks of fetal growth retardation, premature birth, and low birth weight (Han et al. 2011; Haider and Bhutta 2012; Scholl and Hediger 1994; Yi, Han, and Ohrr 2013). Consistent with Barker's hypothesis, growth retardation can be seen as a mechanism to compensate for insufficient nutrient supply without compromising the development of vital organs. Low birth weight increases the risk of cardiovascular diseases, hypertension, and type 2 diabetes in adulthood—NCDs that are typically associated with overweight and obesity (Barker 2004).

Although overweight and obese women are less likely to give birth to low-weight babies (1,500–2,499 grams) than normal-weight women, they are more likely to give birth prematurely and to bear very low birth weight babies (< 1,500 grams), aggravating the risk of stillbirth (McDonald 2010). In addition, women with excess weight gain and associated symptoms of cardiovascular diseases during pregnancy are at an elevated risk of having children with these birth abnormalities (Eriksson et al. 1999; Gluckman and Hanson 2008). Being overweight during pregnancy can affect placental growth, leading to impaired nutrient supply to the fetus and thus fetal growth retardation as well (Wu et al. 2004).

It is worth noting here that low birth weight is an evident indicator of suboptimal intrauterine growth, but its absence does not necessarily imply optimal development for the fetus. Fetal undernutrition can affect cognitive development by causing structural damage to the brain and by impairing infant motor development and exploratory behavior (Victora et al. 2008). A cohort study from the United States suggests that differences in mean IQs are directly associated with birth weight, while each 0.5 IQ-point difference in male siblings at seven years of age corresponds to a 100-gram difference in birth weight, even within the normal birth weight range (Matte et al. 2001). Moreover, a systematic review study indicates that maternal obesity adversely affects breastfeeding success (Turcksin et al. 2014): compared with their normal-weight counterparts, obese women are less likely to intend to breastfeed. Maternal obesity is also associated with a delayed onset of milk secretion, a less adequate milk supply, and a decreased initiation and shortened duration of breastfeeding.

Undernutrition in utero and during infancy may also lead to permanent metabolic changes that increase the risk of overweight and obesity and related NCDs in adulthood (Barker 2004; Uauy et al. 2008). Children born from undernourished mothers may have developed a survival mechanism that allows them to better draw dietary energy from food and to more readily store it in their body as fat. However, when these individuals face a nutritional environment in childhood or adulthood that encourages obesity, they tend to gain weight faster than their healthy peers because of the prenatal metabolic programming (Hales and Barker 2001). In addition, people who were small at birth are more susceptible to hypertension and type 2 diabetes than people who were big. The development patterns of the two disorders are identical and coincide with that of coronary heart disease (Barker 2004). The risk of NCDs declines with increasing birth weight and rises with rapid weight gain in early childhood (Barker 2004; Koletzko et al. 2012).

Overnutrition during pregnancy may also cause permanent metabolic changes in the unborn that increase the risk of overweight and related NCDs later in life (Dabelea 2007; Dabelea et al. 2000; Freinkel 1980). The "fuel-mediated teratogenesis hypothesis" (Freinkel 1980) proposes that excess glucose supply in utero can give rise to irreversible dysfunctions of the body's control systems, including glucose intolerance, hunger regulation, and fat accumulation (Plagemann 2008). While maternal glucose is freely transferred to the fetus, maternal insulin does not cross the placenta, causing the fetus's pancreas to absorb high glucose levels by producing high volumes of insulin (Freinkel 1980). Although evidence from human studies is still limited, existing data point to a higher risk of diabetes in persons exposed to diabetes in utero, independent of adiposity and genetic predisposition of diabetes (Dabelea and Crume 2011). Yet the mechanism through which diabetes exposure in utero affects cardiovascular diseases is still not fully understood. Impaired insulin secretion has been proposed as a possible mechanism, considering that studies in newborns of diabetic mothers have revealed an enhanced insulin secretion in response to a glycemic stimulus. It is still unclear, however, if this is a transitory phenomenon or a symptom of impaired glucose tolerance later in life, when insulin resistance becomes important (Dabelea and Crume 2011). Nonetheless, there is compelling evidence that children from overweight women or women with excessive weight gain during pregnancy are at an increased risk of overweight and related NCDs, independent of genetic characteristics.

Finally, in utero growth retardation and low birth weight are associated with higher risks of postnatal child stunting and retarded head circumference growth, while children with reduced head circumference are more likely to be delayed in psychomotor development (Bove et al. 2012). Although stunting mainly manifests itself postnatally and usually peaks around 24 months (Victora et al. 2010), children born prematurely or small for gestational age are more likely to remain physically retarded in their growth compared to children born with normal stature and weight (Hediger et al. 1999; Karlberg and Albertsson-Wikland 1995; Leger et al. 1997; Strauss and Dietz 1998). Under suboptimal nutritional and health conditions, growth faltering cannot be caught up easily and may even accumulate over time, given the rapid development that children undergo, particularly during their first two years of life. Stunted children are more likely to become overweight in their later lives (Bove et al. 2012; El Taguri et al. 2009; Popkin et al. 1996; Schroeder, Martorell, and Flores 1999). Early evidence on the potential physiological mechanism involved in this process suggests that stunted children have a lower fat oxidation than their well-nourished peers and are therefore at higher risks of adiposity (Sawaya and Roberts 2003; Hoffman et al. 2000a, 2000b).

In conclusion, "chronic diseases are not the inevitable lot of humankind; they are the result of the changing pattern of human development," as Barker (2012; p. 185) puts it. Many children are malnourished and have an increased risk of NCDs in their later lives because their mothers were malnourished during pregnancy and lactation. In addition to the genetic loading, children with malnourished mothers have an increased risk of malnutrition because they tend to share the same or a similarly inadequate diet. Household diets are determined by household incomes, food prices, education, and many other factors—some of which can be influenced by economic and social policies.

Egypt's Double Burden of Malnutrition in the Global Comparison

The double burden of malnutrition is most pronounced in two developing regions, namely the MENA and LAC regions (Garrett and Ruel 2005). While undernutrition is still common in both regions, overnutrition has been rising rapidly in the wake of high economic growth and structural transformation in recent decades. In fact, along with North America, the MENA and LAC regions exhibit the highest prevalence of overweight and obesity, with rates among women (20 years of age and older) of more than 60 percent for overweight and 30 percent for obesity (Finucane et al. 2011; Stevens et al. 2012).

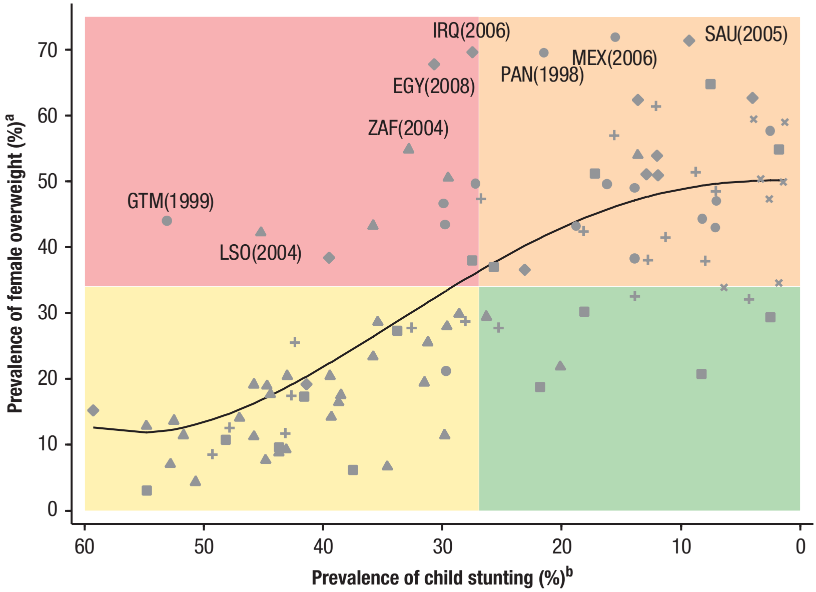
The prevalence of female overweight and obesity and the prevalence of child stunting are inversely correlated across countries. Considering that the countries are at different stages of economic development, the inverse relationship may also mark the typical course of decreasing child stunting and increasing female overweight and obesity that an average country follows throughout its development process. Our estimated functions suggest that the relationship has a flattened S-shaped curve (Figure 2.1 and Figure 2.2).

Yet several countries deviate from the general relationship. For example, relative to the prevalence of child stunting, the prevalence of female overweight and obesity is extremely high in Egypt, Guatemala, Iraq, Lesotho, Mexico, Panama, South Africa, and Saudi Arabia. Among these countries, the prevalence of child stunting exceeds 30 percent in Egypt, Guatemala, Lesotho, and South Africa. The gap between the actual prevalence rate of female overweight and obesity and the expected one given the actual child stunting rate is widest in Egypt, particularly for female obesity—the more severe form of overnutrition. Hence, according to this measure, the double burden of malnutrition (at the population level) in Egypt is most pronounced in the global comparison.

Egypt's prevalence of female overweight and obesity alone is among the highest in the world. Egypt ranks among the top 10 countries with the highest rate of obese women, ahead of the United States, Mexico, and Iraq and behind only Kuwait, Libya, Qatar, and some small Caribbean island states (Ng et al. 2014). Data from the Egypt DHSs in 2000, 2003, 2005, and 2008 suggest that a stable proportion of around 80 percent of all (nonpregnant) women 20–49 years of age were overweight between 2000 and 2008 (MOH, El-Zanaty and Associates, and Macro International 2008; MOHP, NPC, and ORC Macro 2000; MOHP et al. 2003; MOHP et al. 2005). The prevalence rate of female obesity in 2008 was about 40 percent, roughly the same as in 2000.[1] Considering a longer study period of 33 years, Ng et al. (2014) find the largest average increase in the female obesity rate to be in Egypt, followed by Saudi Arabia, Oman, Honduras, and Bahrain. Hence, the public health problem of chronic overnutrition and related NCDs in Egypt has increased considerably in severity in recent decades (Herman et al. 1995). In fact, Egypt has one of the highest death rates from cardiovascular diseases and diabetes in the developing world (Alwan et al. 2010). Abegunde et al. (2007) estimate that Egypt lost a cumulative US$1.26 billion to chronic diseases between 2005 and 2015; most of the loss is due to cardiovascular diseases and diabetes.

1 See Tables A.1 and A.2 in the Appendix.

FIGURE 2.1 Relationship between the prevalence of female overweight and child stunting



Source: Authors' estimation based on data from World Bank (2014).

Note: The sample includes the latest observations of the prevalence of female overweight and child stunting (measured within a period of less than five years) for 101 countries.

The regions are North America and Western Europe high-income countries (+); Arab world (♦); Africa south of the Sahara (▲); Latin America and Caribbean (●); East Asia and Pacific (■); and West, Central, and South Asia and Eastern Europe (✕).

Countries with a high prevalence of female overweight relative to their prevalence of child stunting include Egypt (EGY), Guatemala (GTM), Iraq (IRQ), Lesotho (LSO), Mexico (MEX), Panama (PAN), Saudi Arabia (SAU), and South Africa (ZAF).

The estimated relationship between the prevalence of female overweight and the prevalence of child stunting is obtained in two steps. In the first step, a nonparametric regression is applied to the data, and the estimated function is graphed to gain evidence on the form of a parametric function that yields the best data fit. A locally weighted regression is run, using Stata's "lowess" (locally weighted scatter plot smoothing) command. (The chosen bandwidth of the lowess curve is 0.8, which is Stata's standard bandwidth. The results of the nonparametric regression are not reported.) Based on the found shape of the curve, a fractional polynomial regression of degree 2 and a robust estimator of variance are applied to the data in the second step to determine the specific functional form and plot the predicted line of the relationship.

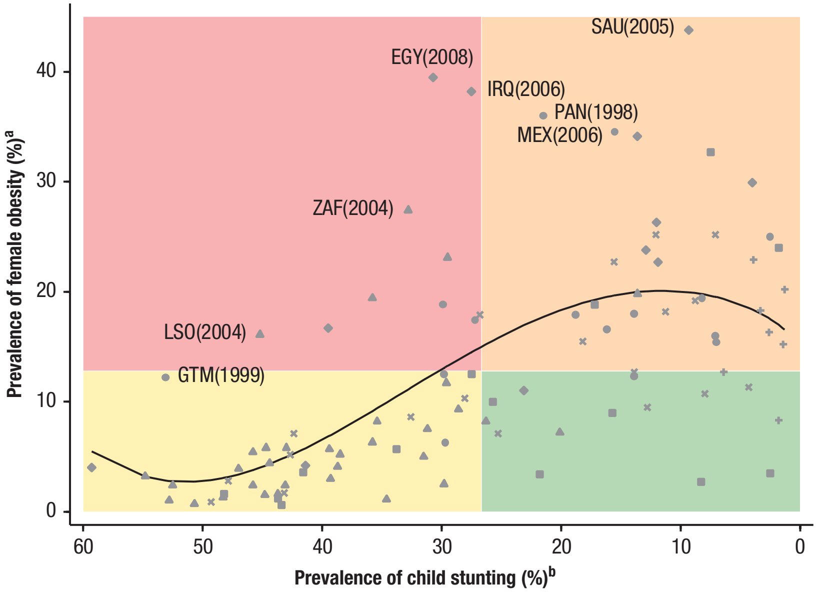
The estimated function is: $y = -1.370 * (((x/10)^3) - 19.556) + 0.668 * (((x/10)^3) * \ln(x/10) - 19.381) + 36.360$, where y is a country's female overweight rate, and x is its child stunting rate. Statistical significance and explanatory power of the model are high (F = 56.65, R-sq. = 0.536).

The sample means divide the sample into four quadrants, which group the countries according to the severity of the double burden of malnutrition. The color of the quadrant indicates the severity of the double burden of malnutrition, with green indicating low severity and red indicating high severity.

[a] The prevalence of female overweight is defined as the proportion of women 15 years of age and older with a body mass index (BMI) of 25 or higher.

[b] The prevalence rate of child stunting is defined as the proportion of children under five years of age with height-for-age z-scores (HAZs) below –2.

FIGURE 2.2 Relationship between the prevalence of female obesity and child stunting



Source: Authors' estimation based on data from World Bank (2014).

Note: The sample includes the latest observations of the prevalence of female obesity and child stunting (measured within a period of less than five years) for 100 countries.

The regions are North America and Western Europe high-income countries (+); Arab world (♦); Africa south of the Sahara (▲); Latin America and Caribbean (•); East Asia and Pacific (■); and West, Central, and South Asia and Eastern Europe (×).

Countries with a high prevalence of female obesity relative to their prevalence of child stunting include Egypt (EGY), Guatemala (GTM), Iraq (IRQ), Lesotho (LSO), Mexico (MEX), Panama (PAN), Saudi Arabia (SAU), and South Africa (ZAF).

The estimated relationship between the prevalence of female overweight and the prevalence of child stunting is obtained in two steps. In the first step, a nonparametric regression is applied to the data, and the estimated function is graphed to gain evidence on the form of a parametric function that yields the best data fit. A locally weighted regression is run, using Stata's "lowess" (locally weighted scatter plot smoothing) command. (The chosen bandwidth of the lowess curve is 0.8, which is Stata's standard bandwidth. The results of the nonparametric regression are not reported.) Based on the found shape of the curve, a fractional polynomial regression of degree 3 and a robust estimator of variance are applied to the data in the second step to determine the specific functional form and plot the predicted line of the relationship.

The estimated function is: $y = 7.119 * (((x/10)^{0.5}) - 1.633) - 1.095 * (((x/10)^3) - 18.957) + 0.546 * (((x/10)^3) * \ln(x/10) - 18.582) + 14.999$, where y is a country's prevalence rate of female obesity (in percent) and x is its prevalence rate of child stunting (in percent). Statistical significance and explanatory power of the model are moderately high ($F = 20.80$, R-sq. = 0.375).

The sample means divide the sample into four quadrants, which group the countries according to the severity of the double burden of malnutrition. The color of the quadrant indicates the severity of the double burden of malnutrition, with green indicating low severity and red indicating high severity.

[a] The prevalence of female obesity is defined as the proportion of women 15 years of age and older with a body mass index (BMI) of 30 or higher.

[b] The prevalence rate of child stunting is defined as the proportion of children under five years of age with height-for-age z-scores (HAZs) below –2.

Patterns of the Double Burden of Malnutrition within Egypt

Data from the HIECS in 2011 (CAPMAS and WFP 2011) suggest that almost every third young child in Egypt is stunted. Hence, their physical development is retarded as a result of inadequate nutrition and poor health conditions, which will remain a life-long health impairment in most cases—with all its implications for individual well-being and economic development. In addition to chronic child undernutrition, child overnutrition is widespread. The 2011 HIECS data suggest that overweight among children ages 6–59 months is almost as prevalent as stunting in this age group, while a large proportion suffer from stunting and overweight at the same time (Table 2.1).

TABLE 2.1 Prevalence patterns of the double burden of malnutrition in Egypt

	DBM at population level				DBM at family level	DBM at individual level
	Child stunting	Child overweight	Female overweight	Female obesity	Stunted child with overweight mother	Stunting and overweight in children
Total (%)	**31.2**	**29.2**	**72.6**	**33.9**	**22.3**	**14.0**
By region and residential area[a] (%)						
Metropolitan	30.8	29.5	74.0	33.9	26.9	15.6
Lower Egypt	27.1	30.7	78.3	40.9	20.6	12.7
Urban	24.7	27.1	78.5	39.7	20.4	11.9
Rural	27.8	31.7	78.2	41.4	20.7	12.9
Upper Egypt	34.7	27.0	65.6	25.8	21.5	14.3
Urban	39.2	28.1	71.5	29.4	25.6	16.2
Rural	33.4	26.7	62.8	24.1	20.2	13.7
By income quintile and residential area (%)						
Quintile 1 (poorest)	34.0	27.9	66.2	27.6	20.0	14.6
Quintile 2	32.3	28.9	71.2	32.4	22.2	15.0
Quintile 3	29.2	28.7	75.1	36.6	22.8	11.9
Quintile 4	27.2	26.6	75.3	37.5	20.3	12.5
Quintile 5 (richest)	33.1	33.7	75.0	35.5	26.1	15.9
Urban	31.9	28.8	74.3	34.2	25.3	15.1
Quintile 1 (poorest)	33.0	26.4	72.6	31.4	21.8	13.2
Quintile 2	32.3	29.9	77.0	36.7	25.5	16.6
Quintile 3	31.9	27.4	74.2	35.8	26.4	16.3
Quintile 4	33.0	30.4	76.1	36.2	29.9	14.1
Quintile 5 (richest)	29.4	30.1	71.6	30.7	22.9	15.4

	DBM at population level				DBM at family level	DBM at individual level
	Child stunting	Child overweight	Female overweight	Female obesity	Stunted child with overweight mother	Stunting and overweight in children
Rural	30.8	29.3	71.3	33.8	20.6	13.4
Quintile 1 (poorest)	32.3	27.1	62.3	25.5	17.6	13.9
Quintile 2	33.8	28.2	69.6	31.0	22.4	14.5
Quintile 3	30.7	31.6	71.0	33.4	22.4	12.1
Quintile 4	26.8	25.9	74.9	37.3	18.8	11.7
Quintile 5 (richest)	30.3	34.0	78.8	41.8	21.9	14.7

Source: Authors' calculation based on data from CAPMAS and WFP (2011).

Note: DBM = double burden of malnutrition.

The child stunting sample includes children ages 6–59 months with biologically plausible height-for-age z-scores (HAZs) (–6 ≤ HAZ ≤ 6) and has 3,852 observations. The child overweight sample includes children ages 6–59 months with biologically plausible body-mass-index-for-age z-scores (BMIZs) (–5 ≤ BMIZ ≤ 5) and has 3,631 observations. The female overweight/obesity sample includes nonpregnant women 20–49 years of age with biologically plausible body mass indexes (BMIs) (5.2 ≤ BMI ≤ 52.1) and has 9,778 observations. All others are subsamples of these samples with observations in both original samples. The child stunting and maternal overweight sample has 3,661 observations, and the child stunting and overweight sample has 3,577 observations.

The prevalence rate of child stunting is defined as the proportion of children with HAZs below –2, and the prevalence of child overweight is defined as the proportion of children with BMIZs of 2 or above. The prevalence of female overweight is defined as the proportion of women with BMIs of 25 or above, and the prevalence of female obesity is defined as the proportion of women with BMIs of 30 or above.

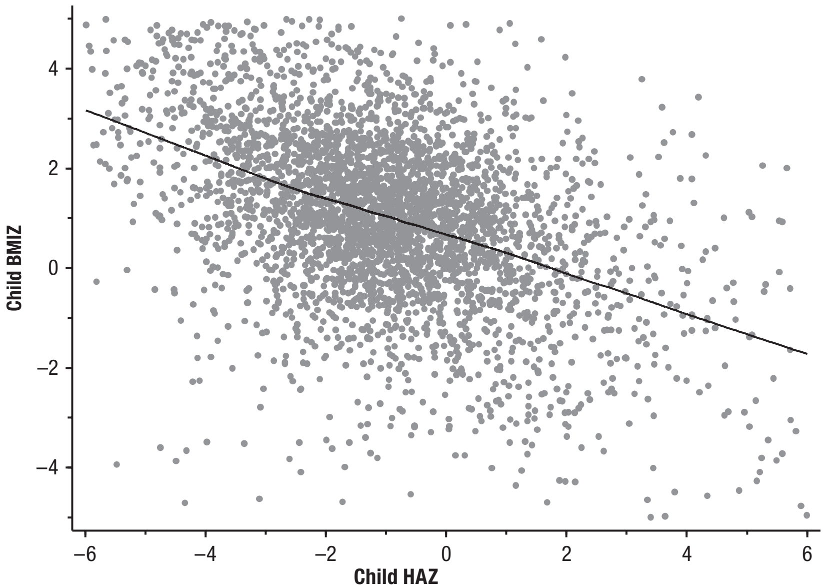
The household disaggregation by wealth/income quintile in the Demographic and Health Surveys samples (MOH, El-Zanaty and Associates, and Macro International 2008; Egypt, MOHP, NPC, and ORC Macro 2000; MOHP et al. 2003; MOHP et al. 2005) is based on the distribution of asset wealth (measured by a composite index), and the household disaggregation in the Household Income, Expenditure, and Consumption Survey sample (CAPMAS and WFP 2011) is based on the distribution of household income per capita.

[a] Prevalence rates for Frontier Governorates are not reported because of insufficient observations.

While 31.2 percent of the children are stunted, 29.2 percent are overweight.[2] Of those children who are stunted, about 45 percent are overweight. In total, 14.0 percent of all children ages 6–59 months are stunted and overweight. Children's height-for-age z-scores (HAZs; indicating stunting) are distinctly negatively and linearly correlated with their body-mass-index-for-age z-scores (BMIZs; indicating overweight and obesity) (Figure 2.3); the correlation coefficient is –0.431. Hence, in Egypt shorter children tend to be fatter than their taller peers. Note that child wasting (indicating acute child undernutrition)

2 Children are classified as stunted if their height-for-age z-scores (HAZs) are below –2 and as overweight if their body-mass-index-for-age z-scores (BMIZs) are 2 or above.

FIGURE 2.3 Relationship between children's BMIZs and HAZs in Egypt



Source: Authors' estimation based on data from CAPMAS and WFP (2011).

Note: The sample includes children ages 6–59 months with biologically plausible body-mass-index-for-age z-scores (BMIZs) (−5 ≤ BMIZ ≤ 5) and height-for-age z-scores (HAZs) (−6 ≤ HAZ ≤ 6) and has 3,577 observations.

The relationship between children's BMIZs and HAZs is estimated nonparametrically, using a locally weighted regression estimation inbuilt in Stata. The chosen bandwidth is 0.8, which is Stata's default bandwidth of the "lowess" (locally weighted scatter plot smoothing) command.

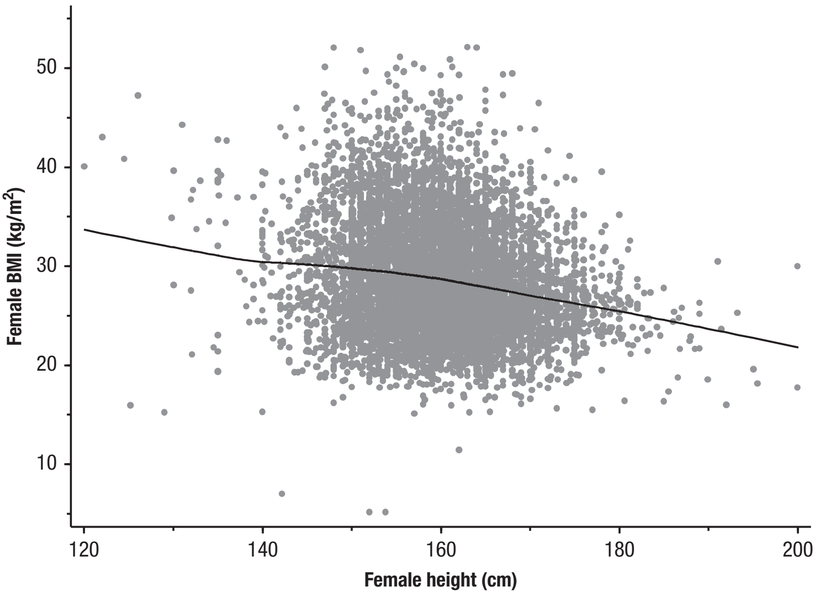
and child underweight (indicating overall child undernutrition) have been low in Egypt, with national prevalence rates of below 8 percent since 2000.[3]

Moreover, the 2011 HIECS data (CAPMAS and WFP 2011) suggest that about 71 percent of all stunted Egyptian children have an overweight mother.[4] Out of all Egyptian child-mother pairs, 22.3 percent are child-mother pairs with a stunted child and an overweight mother (Table 2.1). According to the HIECS data, 72.6 percent of (nonpregnant) Egyptian women 20–49 years

3 Children are classified as wasted if their weight-for-height z-score (WHZ) is below −2 and as underweight if their weight-for-age z-score (WAZ) is below −2. For disaggregated prevalence rates of child nutrition indicators, see Tables A.3–A.6 in the Appendix.

4 The HIECS (CAPMAS and WFP 2011) does not explicitly indicate the child's mother. However, we are able to identify the likely mother—or female caretaker—using information from the household member characteristics module. See Box 4.2 in the subsection "Survey Data and Estimation Variables" below. In the following, we omit "caretaker" for ease of readability.

FIGURE 2.4 Relationship between women's BMIs and body heights in Egypt



Source: Authors' estimation based on data from CAPMAS and WFP (2011).

Note: cm = centimeters; kg/m² = kilograms per square meter.

The sample includes nonpregnant women 20–49 years of age with biologically plausible body mass indexes (BMIs) (5.2 ≤ BMI ≤ 52.1) and has 9,778 observations.

The relationship between women's BMIs and heights is estimated nonparametrically using a locally weighted regression estimation inbuilt in Stata. The chosen bandwidth is 0.8, which is Stata's default bandwidth of the "lowess" (locally weighted scatter plot smoothing) command.

of age are overweight, and 33.9 percent are obese.[5] For these women, we also find a linear, negative relationship between their BMIs and absolute body heights (Figure 2.4), although the correlation, with a coefficient of −0.193, is less strong than that for children. Hence, shorter Egyptian women tend to be fatter than their taller peers. Note that stunting in women—which manifests itself mainly during early childhood—is not very prevalent in Egypt today. The 2011 HIECS data suggest that only 1.6 percent of all (nonpregnant) women 20–49 years of age are shorter than 145 centimeters (cm). And female underweight is almost nonexistent in Egypt. According to the HIECS

5 The 2011 HIECS data (CAPMAS and WFP 2011) indicate somewhat lower prevalence of female overweight and obesity than the 2008 DHS data (MOP, El-Zanaty and Associates, and Macro International 2008; MOHP, NPC, and ORC Macro 2000; MOHP et al. 2003; MOHP et al. 2005) did. These differences may partly be due to different survey years and sample populations.

data, 0.8 percent of all (nonpregnant) women 20–49 years of age are underweight.[6] DHS data from the survey rounds in 2000, 2003, 2005, and 2008 indicate that underweight affected less than 1 percent of all (nonpregnant) women throughout the 2000s (MOH, El-Zanaty and Associates, and Macro International 2008; MOHP, NPC, and ORC Macro 2000; MOHP et al. 2003; MOHP et al. 2005).

These first results have several important implications: The double burden of malnutrition in Egypt is not only highly prevalent at the population level but also at the family and the individual levels. The findings that under- and overnutrition exist in child-mother pairs and in the same children at considerable prevalence rates, and that individual body height and weight are negatively correlated among young children and women of reproductive age, suggest that lack of sufficient staple foods can be ruled out as a cause of widespread undernutrition. In other words, chronic hunger should not be an issue of public concern. This notion is also supported by low prevalence rates of wasting and underweight among children and of underweight among women.

Instead, these correlations point to a nutritional situation where calorie-rich foods are readily and cheaply available, which encourages overconsumption of these foods; where a large share of the population cannot afford or does not prefer a diversified diet dense in micronutrient-rich foods (such as meat, fish, dairy products, pulses, vegetables, and fruits); and where individual health conditions compromise optimal nutritional outcomes. The results also imply that the same circumstances in households' environments, the same behavior of their members, and the same individual characteristics may indeed contribute to both under- and overnutrition at the same time.

Furthermore, the HIECS data (CAPMAS and WFP 2011) show no distinct patterns in the prevalence of undernutrition, overnutrition, and the double burden of malnutrition between urban and rural areas and between regions (Table 2.1). This might be surprising given that there are clear economic development gaps between urban and rural areas, the Metropolitan areas and the rest of Egypt, and Lower and Upper Egypt. The regional patterns of some forms of malnutrition appear to even be inconsistent with those of economic development. For example, child stunting is somewhat more prevalent in the Metropolitan areas than in urban Lower Egypt—although less prevalent than in urban Upper Egypt. In Upper Egypt, the prevalence of child stunting is higher in urban areas than in rural areas. Female overweight and obesity are more prevalent in both urban and rural areas of Lower Egypt

6 Adults are classified as underweight if their BMIs are below 18.5.

than in the Metropolitan areas, whereas they are least prevalent in Upper Egypt. The double burden of malnutrition at the family and the individual levels is most prevalent in the Metropolitan areas and urban Upper Egypt.[7] The absence of distinct spatial prevalence patterns of under- and overnutrition and unexpectedly high prevalence rates in some parts of Egypt may be explained by the coverage of the food subsidy system, the local health environment, and differential impacts of the crises in the 2000s (Kavle et al. 2015a) to some extent.

Breaking down the malnutrition prevalence rates by income quintiles reveals no clear income-dependent patterns for any considered form of malnutrition—with the exception of female overnutrition in rural areas (Table 2.1).[8] Thus, unlike most other developing countries, Egypt shows no typical tendency of high rates of undernutrition among poor rural households and of high rates of overnutrition among rich urban households. In contrast, undernutrition and overnutrition are highly prevalent among both the poor and the rich at relatively similar rates. These results—obtained from the 2011 HIECS data (CAPMAS and WFP 2011)—are largely consistent with the patterns found in the DHSs conducted in the 2000s (MOH, El-Zanaty and Associates, and Macro International 2008; MOHP, NPC, and ORC Macro 2000; MOHP et al. 2003; MOHP et al. 2005).[9] In addition, the HIECS data also do not reveal, either in rural or in urban areas, obvious income-dependent patterns of the double burden of malnutrition at the family and the individual levels. Overall, these findings provide some initial evidence that household income (and monetary poverty) alone is unlikely to be the main driver of malnutrition in Egypt.

The next section explores how the prevalence of chronic child undernutrition—one component of the double burden of malnutrition—changed over time in Egypt relative to national economic growth and compared to other developing countries, as well as between different regions in Egypt and among different income/wealth groups of the Egyptian population.

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7 See Tables A.7 and A.8 in the Appendix.

8 This also holds true when using household expenditure instead of household income for defining quintiles. In this study we use reported household incomes from the income section of the HIECS (CAPMAS and WFP 2011) because household expenditures are influenced by the amount of subsidies the households receive. We checked the robustness of all results using household expenditures instead of household incomes and did not find significant deviations. Note that the coefficient of the correlation of household income and expenditures is 0.906 across the entire sample (and 0.909 for urban households and 0.844 for rural households).

9 See Tables A.1–A.8 of the Appendix.

The Growth-Nutrition Disconnect

Economic growth leads to reduced undernutrition—at least in the long term—through two main routes. First, economic growth increases household incomes. Higher incomes allow households to purchase more and more nutritious food and to spend more on health, education, and housing that may result in improved nutrition indicators. Hence, the degree to which growth translates into reduced prevalence rates of undernutrition mainly depends on the proportion of the undernourished population that experiences income increases and the rates at which that population's income grows—in other words, it depends on the inclusiveness of economic growth.

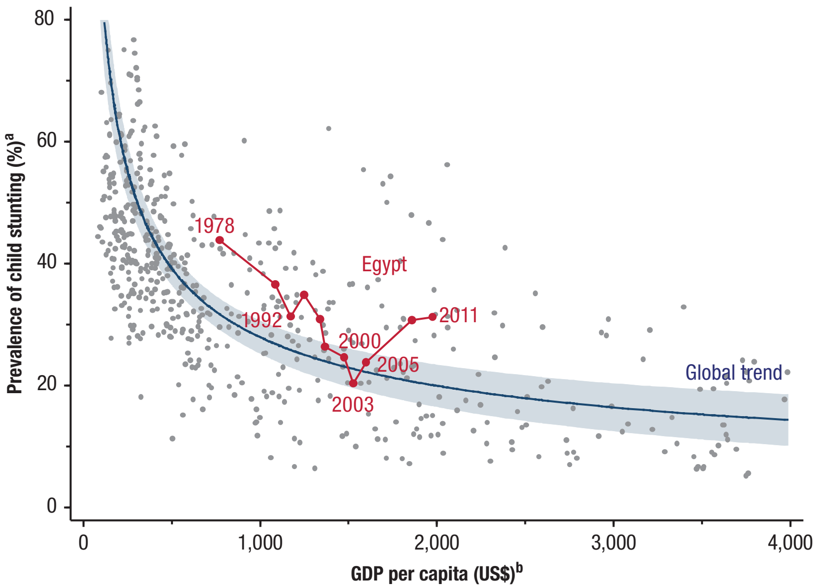
Second, economic growth tends to increase the public budget through higher tax revenues, state-owned enterprises, and other revenue sources. These additional revenues provide fiscal space for income redistribution policies (affecting individual nutrition through the first route), investments in nutrition-sensitive infrastructure and public services (such as water and sanitation and basic healthcare), and health and nutrition-specific interventions (such as micronutrient supplementation and fortification and nutrition information and education programs). In addition, policies often play a critical role in influencing consumers' choices: they can either create an environment that encourages people to use increasing income for improving nutrition or that discourages them from nutrition-beneficial spending. An example of a policy that can incentivize unhealthy diets is food subsidies—as hypothesized in this book.

Thus, national policy can influence the degree to which economic growth translates into reduced prevalence rates of undernutrition through the amount of additional public budget generated from economic growth, the allocation and efficiency of the additional resources, the quality (and effectiveness) of the public services provided, and the design of policies enabling or interfering with nutritional improvement (Bryce et al 2008; Gillespie et al. 2013; Haddad et al. 2003; Smith and Haddad 2002).

Egypt's Chronic Child Undernutrition and Economic Growth in a Global Comparison

Relative to the national income level, chronic child undernutrition has been considerably more prevalent in Egypt than in most other developing countries—with the exception of a period in the late 1990s and early 2000s when it reached average prevalence rates (Figure 2.5). On a global level, countries' child stunting rate has declined, at decreasing margins, with increasing GDP

FIGURE 2.5 Relationship between the prevalence of chronic child undernutrition and national income



Source: Authors' estimation based on data from World Bank (2014), complemented with data from other sources. Child stunting rates for Egypt in 2011 are derived from CAPMAS and WFP (2011) data. Missing year observations of gross domestic product (GDP) per capita levels are replaced by computed GDP per capita levels, using GDP growth rates derived from IMF (2014) and UNSTAT (2014) and population data from UN-DESA (2014).

Note: The sample includes child stunting and GDP data for 141 countries with child stunting rates of 5 percent and above. The sample has a total of 628 observations, spanning the period from 1966 to 2012. The figure presents an excerpt, including countries with GDP per capita below US$4,000 (composing 89 percent of all observations).

The estimation of the "global trend" graph is bivariate and cross-country (based on the full sample) and includes two steps. In the first step, a nonparametric regression is applied, and the results are plotted to gain evidence on the general functional form of the relationship between the prevalence of child stunting and GDP per capita levels. A locally weighted regression is run, using Stata's "lowess" (locally weighted scatter plot smoothing) command. (The chosen bandwidth of the lowess curve is 0.8, which is Stata's standard bandwidth. The results of the nonparametric regression are not reported.) Given the shape of the curve, a fractional polynomial regression of degree 1 and a robust estimator of variance are applied to the data in the second step to determine the specific functional form and plot the predicted line of the relationship. The regression is weighted by population size.

The estimated function is: $y = 8.604 * (((x/10{,}000)^{\wedge}(-0.5)) - 2.385) + 21.220$, where y is a country's prevalence rate of child stunting (percentage) and x is its GDP per capita level in the same year. Statistical significance and explanatory power of the model are high (F = 124.7, R-sq. = 0.625). We tested alternative specifications of the functional form that had GDP per capita levels lagged by one to five years but found no clear evidence for general time lags. The shaded area around the global trend line marks the 95 percent confidence interval.

[a] The prevalence rate of child stunting is defined as the proportion of children under five years of age with height-for-age z-scores (HAZs) below –2.

[b] GDP per capita is measured at constant 2000 prices.

TABLE 2.2 Country comparison of child stunting reduction and economic growth

Country	Around 2010			Around 2000			Annual average change in the prevalence of child stunting (percentage points)	Annual average GDP per capita growth (%)	Arc child stunting-growth elasticity[c]
	Prevalence of child stunting (%)[a]	GDP per capita (US$)[b]	Year	Prevalence of child stunting (%)[a]	GDP per capita (US$)[b]	Year			
Egypt	**31.2**	**1,977**	**2011**	**24.6**	**1,476**	**2000**	**0.6**	**2.7**	**0.22**
Countries with similar stunting rates[d]									
Angola	29.2	563	2007	61.7	272	1996	–3.0	6.8	–0.43
Cameroon	32.6	666	2011	36.7	570	1998	–0.3	1.2	–0.26
Haiti	29.7	385	2006	28.3	424	2000	0.2	–1.6	–0.15
Honduras	29.9	1,354	2006	34.5	1,150	2001	–0.9	3.3	–0.28
Togo	29.8	266	2010	29.8	283	1998	0.0	–0.5	0.00
Zimbabwe	32.3	323	2010	33.7	556	1999	–0.1	–4.8	0.03
Countries with similar GDP per capita levels[e]									
Algeria	15.9	2,115	2005	23.6	1,794	2000	–1.5	3.3	–0.46
Azerbaijan	26.8	1,574	2006	24.1	655	2000	0.5	15.7	0.03
China	9.4	2,426	2010	17.8	949	2000	–0.8	9.8	–0.09
Guatemala	48.0	1,856	2009	50.0	1,717	2000	–0.2	0.9	–0.26
Morocco	14.9	1,941	2011	29.0	1,215	1997	–1.0	3.4	–0.30
Syria	27.5	1,509	2009	24.3	1,209	2000	0.4	2.5	0.14
Other Arab countries[f]									
Iraq	27.5	710	2006	28.3	1,063	2000	–0.1	–6.5	0.02
Jordan	8.3	2,577	2009	12.0	1,871	2002	–0.5	4.7	–0.11
Oman	9.8	11,192	2009	12.9	8,343	1999	–0.3	3.0	–0.10

Somalia	42.1	288	2006	29.2	277	2000	2.2	0.6	3.48
Sudan	38.3	582	2006	47.6	445	2000	–1.6	4.6	–0.34
Tunisia	9.0	2,776	2006	16.8	2,245	2000	–1.3	3.6	–0.36
West Bank and Gaza	11.8	984	2007	16.1	979	2002	–0.9	0.1	–7.54
Yemen	59.6	574	2006	59.3	508	1997	0.0	1.4	0.02
Other large developing countries[g]									
Bangladesh	41.4	588	2011	50.8	364	2000	–0.9	4.5	–0.19
Brazil	7.1	4,298	2007	13.5	3,628	1996	–0.6	1.6	–0.37
Ethiopia	44.2	230	2011	57.4	124	2000	–1.2	5.8	–0.21
India	47.9	622	2006	51.0	441	1999	–0.4	5.0	–0.09
Indonesia	39.2	1,145	2010	42.4	773	2000	–0.3	4.0	–0.08
Nigeria	36.0	566	2011	39.7	361	1999	–0.3	3.8	–0.08
Pakistan	43.0	672	2011	41.5	511	2001	0.2	2.8	0.05
Philippines	33.6	1,413	2011	38.3	1,017	1998	–0.4	2.6	–0.14
Vietnam	23.3	723	2010	43.4	402	2000	–2.0	6.1	–0.33

Source: Authors' calculation based on data from World Bank (2014), complemented with data from other sources. Child stunting rates for Egypt in 2011 are derived from CAPMAS and WFP (2011) data; child stunting rates for Yemen in 2006 are derived from CSO (2006) data. Missing year observations of gross domestic product (GDP) per capita levels are replaced by computed GDP per capita levels, using GDP growth rates derived from IMF (2014) and UNSTAT (2014) and population data from UN-DESA (2014).

Note: The sample includes countries with populations above 5 million people, a child health survey in the period 2005–2013, a child health survey in the period 1996–2004, a difference of at least five years between the two surveys, and GDP data reported for the years of the child health surveys.

[a] The prevalence rate of child stunting is defined as the proportion of children under five years of age with height-for-age z-scores (HAZs) below –2; [b] GDP per capita is measured in US dollars at constant 2000 prices; [c] Arc elasticities are presented in gray if countries experienced negative GDP per capita growth.

The sample includes 91 countries. The countries were selected according to the following criteria: [d] Countries with child stunting rates in the range of 2 percentage points around Egypt's rate; [e] Countries with GDP per capita levels in the range of US$500 around Egypt's GDP; [f] Arab countries that do not fall into the first two categories, where Arab countries include all 21 Arab League member states and Syria (which was suspended from the League in 2011); [g] Countries with populations over 80 million people that do not fall into the first two categories (Egypt's population in 2011 was 82.5 million).

per capita levels. Egypt followed this global trend throughout the 1980s and 1990s and even experienced over-proportionate reduction rates in the second half of the 1990s. However, Egypt's progress reversed in the early 2000s, and the prevalence of child stunting steadily increased throughout the 2000s. In fact, despite high economic growth in the 1990s and 2000s, child stunting in Egypt in 2011 (31.2 percent) was as prevalent as it was in 1992 (31.3 percent).[10]

Between 1992 and 2000, the prevalence of child stunting in Egypt declined by an average annual rate of 0.83 percentage points, so each 1 percent GDP per capita growth was associated with an average annual decrease in the child stunting rate of 0.29 percentage points. Globally, each 1 percent GDP per capita growth was associated with an average annual decrease in the child stunting rate of only 0.12 percentage points (at a GDP per capita level equivalent to Egypt's average GDP per capita during this period). However, between 2000 and 2011, the prevalence of child stunting in Egypt increased by an average annual rate of 0.60 percentage points, and each 1 percent GDP per capita growth in Egypt was associated with an average annual increase of 0.22 percentage points in the prevalence of child stunting (Table 2.2). In contrast, the global trend suggests an average annual decrease of 0.10 percentage points (at a GDP level equivalent to Egypt's period-average GDP per capita).

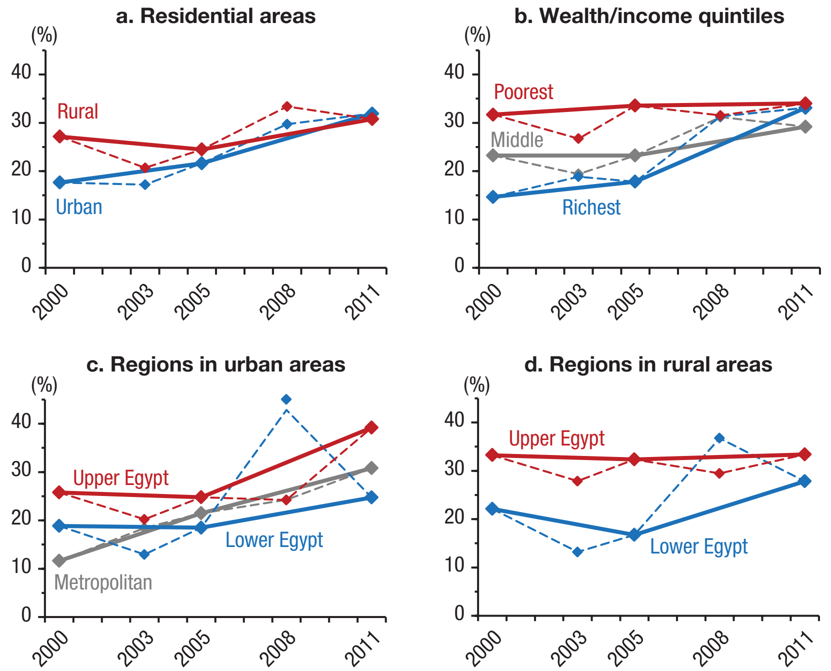
International comparisons confirm that Egypt's nutrition-growth disconnect is particularly pronounced (Table 2.2). Countries with prevalence rates of child stunting similar to Egypt's have considerably lower GDP per capita levels. Conversely, countries with similar GDP per capita levels have substantially lower prevalence rates of child stunting, with the exception of Guatemala—another country facing a pronounced double burden of malnutrition (Figure 2.1 and Figure 2.2). Further, only a few other countries experienced increasing chronic child undernutrition in the face of economic growth in the 2000s (Table 2.2). Most of these countries were affected by civil war or were in a postconflict transition period during the observation period—unlike Egypt. They include countries like Armenia, Azerbaijan, Macedonia, Pakistan, Sierra Leone, Somalia, Timor-Leste, and Yemen.

Patterns of Chronic Child Undernutrition within Egypt

In the 2000s, Egypt's prevalence rate of child stunting rose faster in urban areas than in rural areas (Figure 2.6a). Child stunting in 2011 was even slightly more prevalent in urban areas, whereas in most other developing

10 Egypt's decade-average GDP per capita growth rate was 2.3 percent in the 1990s and 3.0 percent in the 2000s.

FIGURE 2.6 Prevalence of child stunting in Egypt, by different subnational disaggregations, 2000–2011



Source: Authors' estimation based on Demographic and Health Surveys (DHS) data (MOH, El-Zanaty and Associates, and Macro International 2008; MOHP, NPC, and ORC Macro 2000; MOHP et al. 2003; MOHP et al. 2005) and data from CAPMAS and WFP (2011).

Note: The samples include children ages 6–59 months with biologically plausible height-for-age z-scores (HAZs) ($-6 \leq$ HAZ ≤ 6). The prevalence rate of child stunting is defined as the proportion of children with HAZs below -2. The samples exclude the Frontier Governorate region because of insufficient observations.

The DHSs from 2000, 2005, and 2008 (MOH, El-Zanaty and Associates, and Macro International 2008; MOHP, NPC, and ORC Macro 2000; MOHP et al. 2003; MOHP et al. 2005) are standard DHSs; the respective samples contain 7,737, 10,352, and 8,196 observations. The 2003 DHS is an interim DHS, which oversampled Menia Governorate (in Upper Egypt) and the slum areas of Greater Cairo (in the Metropolitan area). The 2003 DHS sample contains 5,200 observations. The 2011 Household Income, Expenditure, and Consumption Survey (HIECS) sample (CAPMAS and WFP [2011]) contains 3,815 observations. The 2008 DHS data yield implausible subnational prevalence rates.

The solid lines connect "trusted" prevalence rates, derived from standard DHS and HIECS data. The dashed lines connect prevalence rates derived from all DHS and HIECS data.

countries, it has been considerably more prevalent in rural areas (Fotso 2007; Smith, Ruel, and Ndiaye 2005). Egypt's rural-urban gap closed over the past decade because children's nutrition deteriorated faster in urban areas than in rural areas and not because progress in reducing chronic child undernutrition in rural areas caught up with the advances made in urban areas. Rural-urban migration and rapid population growth in urban slums with a poor health environment may explain increasing child stunting in urban areas only to some extent. Compared to 2000, the increase in the prevalence rate of child stunting in 2011 was highest in the Metropolitan areas, where the slums are located (Figure 2.6c). However, child stunting also became considerably more prevalent in urban areas outside the big cities, and particularly in Upper Egypt. The rapid increase in urban Upper Egypt contributes to a widening north-south gap in child nutrition for urban areas.

The situation is quite different for rural areas. Throughout the 2000s, the prevalence rate of child stunting was fairly stable in rural Upper Egypt, at around 33 percent. It rapidly increased in rural Lower Egypt, at least between 2005 and 2011 (Figure 2.6d). Unlike for urban areas, this development has led to a narrowing of the north-south gap in child nutrition for rural areas. The 2011 child stunting rate in Upper Egypt is actually considerably lower in rural areas than in urban areas, which is the reverse of the situation in 2000 and the situation in Lower Egypt (Table 2.1 and Figure 2.6c and Figure 2.6d).

The difference in the prevalence rate of child stunting between the rich and the poor also narrowed throughout the 2000s, so the prevalence rate among the richest and poorest wealth/income quintiles was quite close in 2011 (Figure 2.6b). This narrowing is due to a rapidly increasing prevalence among the rich rather than a decreasing prevalence among the poor. In fact, the prevalence rate among the poorest wealth/income quintile increased only marginally. These results preliminarily suggest that child stunting is poorly correlated with household wealth/income across space and time.

The next chapter identifies four potential drivers that may explain part of these exceptional patterns.