THE LANCET

December, 2019 www.thelancet.com

The Double Burden of Malnutrition

"Malnutrition is a global challenge that all countries need to address. Despite some progress, the world is not on track to meet globally agreed goals and targets for nutrition."

A Series by *The Lancet*

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

The Lancet—London
125 London Wall,
London EC2Y 5AS,
UK
T +44 (0) 20 7424 4950
editorial@lancet.com

The Lancet—New York
230 Park Avenue, Suite 800,
New York, NY 10169,
USA
T +1 212 633 3810
F +1 212 633 3853

The Lancet—Beijing
Unit 1–6, 7F, Tower W1,
Oriental Plaza, Beijing
100738, China
T + 86 10 85208872
F + 86 10 85189297

THE LANCET

The Double Burden of Malnutrition · December, 2019

Comment

Series

Previously published online

See www.thelancet.com for supplementary material

Version verified by CrossMark

Bartosz Hadyniak/Getty Images

Information for Authors
See www.thelancet.com for guidelines for authors.

A new nutrition manifesto for a new nutrition reality

Published Online
December 15, 2019
https://doi.org/10.1016/S0140-6736(19)32690-X

See Online/Series
https://doi.org/10.1016/S0140-6736(19)32497-3,
https://doi.org/10.1016/S0140-6736(19)32472-9,
https://doi.org/10.1016/S0140-6736(19)32506-1,
and https://doi.org/10.1016/S0140-6736(19)32473-0

Malnutrition is a global challenge that all countries need to address. Despite some progress, the world is not on track to meet globally agreed goals and targets for nutrition.[1] While more than 149 million children have stunted growth, childhood overweight and obesity are increasing almost everywhere,[2] and suboptimal diets are responsible for one in five (22%) adult deaths globally.[3] *The Lancet*'s Series on the Double Burden of Malnutrition[4–7] highlights this new nutrition reality: we can no longer characterise countries as low-income and undernourished or high-income and only concerned with obesity.

Good nutrition is fundamental to human health and for achieving sustainable development. The Sustainable Development Goals (SDGs) enshrine the dual targets of ending malnutrition in all its forms and ensuring access to nutritious food for all people by 2030 as major global priorities.[8] These goals reinforce human rights to adequate food, nutrition, and health,[9,10] and build on the growing momentum for ending hunger and malnutrition.[11,12] Going forward, the UN Decade of Action on Nutrition (2016–2025) seeks to accelerate action to achieve these goals.

Malnutrition has an important common denominator: food systems that fail to provide all people with healthy, safe, affordable, and sustainable diets. The economic, social, and environmental costs of inaction will hinder the growth and development of individuals and societies for decades to come.[4–7,13,14] As this new Series shows, the complex interconnected biological and social pathways of all forms of malnutrition are difficult to disrupt through siloed interventions and require societal shifts that can be scaled up and sustained over decades.[4–7]

Prevention of malnutrition, especially in the first 1000 days, has lifelong health and economic benefits.[15] For food systems to deliver healthy, safe, affordable, and sustainable diets for all, we must address the underlying drivers that incentivise endless market and consumption growth over human and planetary health.[16,17] Meaningful change will require action across food systems—from production and processing, through trade and distribution, pricing, marketing, and labelling, to consumption and waste—driven from the bottom up by communities, cities, regions, and nations. All relevant policies and investments must be radically re-examined.

Silos must be broken down. Magic bullets do not exist. The message of a 2013 *Lancet* Comment that only collective action will end undernutrition remains true today.[18] But given the political economy of food, the commodification of food systems, and growing patterns of inequality worldwide, a broader response is now required. New stakeholders need to join existing ones to power a food system revolution if ending all forms of malnutrition is the goal (table).[14] Among these stakeholders are advocates for planetary health; faith-based leaders; innovators and investors who are financing fair and green companies; and city mayors

Stakeholder groups	Role and responsibility
Governments	Prioritise solving the problem; regulate to set standards and their enforcement; implement policies that are equitable, inclusive, and financed; collect and use data to inform action; and mobilise public investments
UN	Convene and connect actors; demonstrate cost-effective solutions; monitor implementation of commitments and achievement of targets
Civil society	Advocate, organise, mobilise people; monitor commitments; and create a generation of activists
Academia	Generate a diverse evidence base; build capacity and conduct research to solve problems, create sustainable solutions, and promote interdisciplinary systems thinking and research
Media	Inform public opinion, tell stories, create debate; facilitate demand for public accountability; focus on structural drivers not individuals and avoid stigma
Philanthropy and multi/bilaterals	Foster innovation; embrace complexity; fund systems-based problem solving; and convene stakeholders
Private sector	Commit to responsible business by production and distribution of affordable nutritious foods; prioritise population health and wellness agenda over profits; consent to appropriate conduct by removal of undue influence on relevant policy and research; and abide by national and international marketing and other codes and regulations
Regional economic platforms	Reshape trade and investment policies in line with public health policies and protect policy space for nutrition

***Table:* Roles and responsibilities of stakeholder groups who must create the systemic changes needed to end malnutrition**

with responsibility for enabling access to nutritious and affordable food for the world's increasingly urbanised populations. There is a role for food producers. Farmers are increasingly affected by extreme weather events and small and medium enterprises are central to efforts to achieve environmental sustainability and more inclusive growth. Consumer associations also have a role in demanding transparency and accountability. Needless to say, the meaningful engagement of children, adolescents, and young people is vital.

Although new strategic partnerships are essential, we must recognise the damage and mistrust that result from incompatible partnerships with stakeholders whose behaviour runs counter to human or planetary health.[16] The food industry has an important role in implementing and delivering change. However, companies cannot be allowed to influence and interfere in public policy making or bias the science that underpins this process.[17] While constructive dialogue is necessary, a default seat at the table for private-sector representatives should not be assumed and policy development processes need to be firewalled from vested interests.

This multistakeholder effort to end malnutrition must prioritise the engagement, inclusion, and empowerment of rights-holders, such as women, smallholder farmers, young people, and marginalised groups. Any policy action or governance arrangement must begin with the question: whom does our food system ultimately serve, and for what purpose?

More than 60 countries have joined the Scaling Up Nutrition (SUN) Movement, committing to accountable actions and measured results. The SDGs, the UN Decade of Action on Nutrition (2016–2025), and the 2020 Nutrition for Growth Summit in Japan offer further opportunities for stakeholders to expand their commitments. Sustained accountability is crucial to ensure these commitments are translated into actions that deliver good nutrition for all, everywhere. Empty promises fail populations and risk delaying progress.

The new nutrition reality calls for a broadened community of nutrition stakeholders who occupy common ground, speak a shared language, work in mutually reinforcing and interconnected ways, and act on a global scale. Such work can draw inspiration from the contributions of grassroots actors as well as non-state actors, and can be guided by existing goals, targets, frameworks, and action plans.[9,12,19,20]

In this nutrition decade, a new global nutrition movement is emerging that needs to take the lead in demanding food systems change locally, regionally, and globally. It is within our collective power; we owe it to our children and future generations.

**Francesco Branca, Alessandro Demaio, Emorn Udomkesmalee, Phillip Baker, Victor M Aguayo, Simon Barquera, Katie Dain, Lindsay Keir, Anna Lartey, Gladys Mugambi, Stineke Oenema, Ellen Piwoz, Ruth Richardson, Sudhvir Singh, Lucy Sullivan, Gerda Verburg, Patrizia Fracassi, Lina Mahy, Lynnette M Neufeld*

Department of Nutrition for Health and Development, World Health Organization, Geneva 1211, Switzerland (FB, LM); VicHealth, Victoria, Australia and Melbourne School of Population and Global Health, University of Melbourne, Melbourne, VIC, Australia (AD); Institute of Nutrition, Mahidol University, Salaya, Nakhon Pathom, Thailand (EU); Institute for Physical Activity and Nutrition, School of Exercise and Nutrition Sciences, Deakin University, Geelong, VIC, Australia (PB); Nutrition Programme Division, United Nations Children's Fund (UNICEF), New York, NY, USA (VMA); Instituto Nacional de Salud Pública, Cuernavaca, Morelos, Mexico (SB); NCD Alliance, Geneva, Switzerland (KD); Wellcome Trust, London, UK (LK); Nutrition and Food Systems Division, Food and Agriculture Organization of the United Nations (FAO), Rome, Italy (AL, PF); Ministry of Health, Nairobi, Kenya (GM); United Nations System Standing Committee on Nutrition (UNSCN) Coordinator c/o FAO, Rome, Italy (SO); Bill & Melinda Gates Foundation, Seattle, WA USA (EP); Global Alliance for the Future of Food, Toronto, ON, Canada (RR); EAT Foundation, Oslo, Norway (SS); Feed the Truth, Washington, DC, USA (LS); Scaling Up Nutrition (SUN) Movement Secretariat, Geneva, Switzerland (GV); and Global Alliance for Improved Nutrition, Geneva, Switzerland (LMN)

brancaf@who.int

We declare no competing interests. FB contributed to paper three of this Series, by Hawkes and colleagues. AD contributed to paper two of this Series, by Wells and colleagues. We gratefully acknowledge Genevieve Moseley and Niisoja Torto for their support in preparing this Comment.

1 UN General Assembly. Implementation of the United Nations Decade of Action on Nutrition (2016–2025). A/RES/72/306. New York: United Nations, 2018.

2 United Nations Children's Fund, WHO, International Bank for Reconstruction and Development/The World Bank. Levels and trends in child malnutrition: key findings of the 2019 Edition of the Joint Child Malnutrition Estimates. Geneva: World Health Organization, 2019.

3 GBD 2017 Diet Collaborators. Health effects of dietary risks in 195 countries, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. *Lancet* 2019; **393:** 1958–72.

4 Popkin BM, Corvalan C, Grummer-Strawn LM. Dynamics of the double burden of malnutrition and the changing nutrition reality. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32497-3.

5 Wells JC, Sawaya AL, Wibaek R, et al. The double burden of malnutrition: aetiological pathways and consequences for health. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32472-9.

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

6 Hawkes C, Ruel MT, Salm L, Sinclair B, Branca F. Double-duty actions: seizing programme and policy opportunities to address malnutrition in all its forms. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32506-1.

7 Nugent R, Levin C, Hale J, Hutchinson B. Economic effects of the double burden of malnutrition. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32473-0.

8 UN General Assembly. Transforming our world: the 2030 Agenda for Sustainable Development. A/RES/70/12015. New York: United Nations, 2015.

9 UN General Assembly. Universal Declaration of Human Rights. A/RES/217(III)1948. New York: United Nations, 1948.

10 Convention on the Rights of the Child. A/RES/44/251989. New York: United Nations, 1989.

11 Horton R, Lo S. Nutrition: a quintessential sustainable development goal. *Lancet* 2013; **382:** 371–72.

12 Food and Agriculture Organization of the United Nations, WHO. Report of the Joint FAO/WHO Secretariat on the Conference. Rome: Food and Agriculture Organization of the United Nations, 2015.

13 Swinburn BA, Kraak VI, Allender S, et al. The global syndemic of obesity, undernutrition, and climate change: *The Lancet* Commission report. *Lancet* 2019; **393:** 791–846.

14 Willett W, Rockström J, Loken B, et al. Food in the Anthropocene: the EAT–*Lancet* Commission on healthy diets from sustainable food systems. *Lancet* 2019; **393:** 447–92.

15 Shekar M, Kakietek J, D'Alimonte M, et al. Investing in nutrition: the foundation for development—an investment framework to reach the global nutrition targets. Washington, DC: World Bank, Results for Development, Bill & Melinda Gates Foundation, CIFF, 1,000 Days, 2016.

16 Kraak VI, Swinburn B, Lawrence M, Harrison P. The accountability of public-private partnerships with food, beverage and quick-serve restaurant companies to address global hunger and the double burden of malnutrition. *United Nations System Standing Committee on Nutrition: News* 2011; **39:** 11–24.

17 Nestle M. Unsavory truth: how food companies skew the science of what we eat. New York: Basic Books, 2018.

18 Taylor A, Dangour AD, Reddy KS. Only collective action will end undernutrition. *Lancet* 2013; **382:** 490–91.

19 WHO. Comprehensive implementation plan on maternal, infant and young child nutrition. Geneva: World Health Organization, 2014.

20 WHO. Tackling NCDs: "best buys" and other recommended interventions for the prevention and control of noncommunicable diseases. Geneva: World Health Organization, 2017.

Double Burden of Malnutrition 1

Dynamics of the double burden of malnutrition and the changing nutrition reality

Barry M Popkin, Camila Corvalan, Laurence M Grummer-Strawn

The double burden of malnutrition (DBM), defined as the simultaneous manifestation of both undernutrition and overweight and obesity, affects most low-income and middle-income countries (LMICs). This Series paper describes the dynamics of the DBM in LMICs and how it differs by socioeconomic level. This Series paper shows that the DBM has increased in the poorest LMICs, mainly due to overweight and obesity increases. Indonesia is the largest country with a severe DBM, but many other Asian and sub-Saharan African countries also face this problem. We also discuss that overweight increases are mainly due to very rapid changes in the food system, particularly the availability of cheap ultra-processed food and beverages in LMICs, and major reductions in physical activity at work, transportation, home, and even leisure due to introductions of activity-saving technologies. Understanding that the lowest income LMICs face severe levels of the DBM and that the major direct cause is rapid increases in overweight allows identifying selected crucial drivers and possible options for addressing the DBM at all levels.

Published **Online**
December 15, 2019
https://doi.org/10.1016/S0140-6736(19)32497-3

This is the first in a **Series** of four papers about the double burden of malnutrition

Department of Nutrition and Carolina Population Center, University of North Carolina at Chapel Hill, Chapel Hill, NC, USA (Prof B M Popkin)**; Institute of Nutrition and Food Technology (INTA), University of Chile, Santiago, Chile** (C Corvalan PhD)**; and Department of Nutrition for Health and Development, World Health Organization, Geneva, Switzerland** (L M Grummer-Strawn PhD)

Correspondence to:
Prof Barry M Popkin, Carolina Population Center, University of North Carolina, Chapel Hill, NC 27516, USA
popkin@unc.edu

Introduction

The global health community has been slow to acknowledge the challenge of the large proportion of low-income and middle-income countries (LMICs) facing the double burden of malnutrition (DBM), which is the coexistence of undernutrition (ie, micronutrient deficiencies, underweight, and childhood stunting and wasting) and overweight, obesity, and diet-related non-communicable diseases. 2·28 billion[1] or more children and adults worldwide are estimated to be overweight and more than 150 million children are stunted.[2,3]

Several studies describe the double burden of nutritional deficiencies (childhood stunting or wasting, and micronutrient deficiencies) and overweight and obesity affecting countries, households, and individuals. Included are the first studies that measured the DBM at the household level,[4–6] now this is represented by growing literature that has focused on understanding the dimensions of the problem, causes, consequences, and possible solutions.[7–13] The analysis has pinpointed several reasons for this health crisis, many related to the stage of the nutrition transition dominated by reduced physical activity and increased access to less healthy, highly processed foods and beverages.[14–21] However, how to translate this evidence into effective actions is unclear.

Building on the 2013 *Lancet* Series on maternal and child undernutrition[22] and complementing other major scientific initiatives such as the EAT-*Lancet* Commission on healthy diets from sustainable food systems[23] and the *Lancet* Commission on the global syndemic of obesity, undernutrition, and climate change,[24] this Series highlights the new nutrition reality: that there are multiple forms of malnutrition that overlap in different ways and in different places.[13] Additionally, addressing all forms of malnutrition will require new ways of designing, targeting, and implementing programmes and policies to accelerate progress in improving nutrition globally.

We also want to acknowledge that the effect of undernutrition over the past four to five decades will affect our health for many future years. Although stunting has declined greatly from the early 1990s, the stunting from the past three to four decades will subsequently have a great effect decades later on increases in visceral fat and greater risks of major non-communicable diseases as discussed by Wells and colleagues,[25] in the second paper in this Series,[26] and in the key cohort studies.[27–29]

This 2019 Series is timely, with the recent UN Decade of Action on Nutrition and the Sustainable Development Goals shifting focus from predominantly undernutrition, or single sides of malnutrition, to all forms of malnutrition.[30,31] Further, major UN and other international institutions and donors are revisiting their strategies to reconsider the scope of nutrition priorities, developing strategy documents, and formulating initiatives to focus on overweight and obesity as well as undernutrition.[32]

The papers in this Series take this concern for malnutrition in all forms a step further and focus on not only the epidemiology and larger societal changes in the food system and other major demographic and economic dimensions, but also the biological underpinnings of stunting and subsequent adiposity and the risk of non-communicable disease.[25]

The papers in this Series also take the issue of the DBM into the programme and policy area[33] by building on the work of double-duty interventions,[11] which focus on reducing both undernutrition and overweight and obesity. This Series also shows how ignoring obesity in programmes focused on preventing malnutrition at various ages has affected obesity and the DBM[33] and focuses on the economic effects of health programmes and policies.[34] This Series paper introduces the epidemiology of the DBM, presents changes in global estimates of the DBM and its components, and uses

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

Key messages

- In low-income and middle-income countries (LMICs), stunting and wasting, and thinness in women are declining while overweight is increasing in most age groups. According to the most recent surveys, a severe double burden of malnutrition (DBM) is defined as wasting in more than 15% and stunting in more than 30% of children aged 0–4 years, thinness in women (body-mass index <18·5 mg/kg^2) in more than 20% of females aged 15–49 years, and adult or child overweight, was found in 48 countries using the 20% overweight prevalence threshold, 35 countries using the 30% overweight prevalence threshold, and ten countries using the 40% overweight prevalence threshold of all LMICs.
- Severe levels of the DBM have shifted to the countries in the poorest income quartile. Although, in the 1990s, the DBM was typically seen in the highest income bracket countries among the LMICs, today the DBM predominates in poorest LMICs that have much lower gross domestic product per capita, particularly in south and east Asia and sub-Saharan Africa.
- Increases in overweight are the result of changes in the global food system that make less nutritious food cheaper and more accessible, as well as to the decrease in physical activity due to major technological shifts in the workplace, home, and transportation. In south Asian and sub-Saharan African countries, the risk of overweight and obesity is greater among the higher-wealth households and urban areas, and in many other LMICs the risk of the DBM is starting to concentrate among people with low incomes and in rural areas.
- LMICs face a new nutrition reality. The shifts in the global food system are accelerating increases in overweight. Concurrently these same shifts have changed the diet of children aged 0–4 years but the effect on stunting needs further research. LMICs need to implement interventions to improve diet quality to address undernutrition and overnutrition across the lifecycle.

See Online for appendix

repeated household surveys to explore aspects of the DBM. We then provide an overview of the nutrition and food system transitions that explain the large increase in the DBM, particularly among the lowest-income LMICs. The final section of this Series paper discusses the consequences of the problem and possible solutions.[7,8,12,35–37]

The DBM and its prevalence: country and household epidemiology

In this paper and the subsequent papers in this Series we use the word malnutrition to refer to both wasting, stunting, and thinness, and overweight and obesity. Although micronutrient malnutrition is recognised as a component of undernutrition, we have not been able to include this form of malnutrition in our DBM estimates, because of insufficient data.

Country-level DBM

The DBM at the country level is defined as having a high prevalence of both undernutrition and overweight and obesity in at least one population group. We examined which LMICs had a DBM (ie, a prevalence of wasting of >15%, stunting of >30%, and thinness in women of >20%, and an adult or child overweight prevalence of >20%, >30%, or >40%). The cutoffs for undernutrition are defined as follows: a weight-for-height Z score of less than –2 for wasting; a height-for-age Z score of less than –2 for children aged 0–4 years for stunting; and a body-mass index (BMI) of less than 18·5 kg/m² for thinness in adult women. For overweight, the cutoffs are a BMI Z score of greater than 2 in children younger than 18 years, and a BMI of greater than 25 kg/m² in more than 20%, 30%, or 40% of the adult (older than 18 years) population[35] (figure 1; appendix pp 6–11). We use a combination of overweight and obesity because extensive epidemiological research associates BMI of 25 kg/m² or higher (or possibly an even lower threshold) with the risks of non-communicable diseases across LMICs.[36–42]

Of the 123 LMICs with data from the 1990s, the number of countries facing the DBM was 45 (37%) based on a 20% overweight prevalence cutoff, 22 (18%) based on a 30% cutoff, and 15 (12%) based on a 40% cutoff (figure 1A; appendix pp 6–8). Of the 126 LMICs with data from the 2010s, the number of countries facing the DBM was 48 (38%) based on a 20% overweight prevalence cutoff, 35 (28%) based on a 30% cutoff, and ten (8%) based on a 40% cutoff (figure 1B; appendix pp 9–11).

The DBM is especially prevalent in sub-Saharan Africa, south Asia, and east Asia and the Pacific. In countries with data for both time periods, increases and decreases were roughly balanced, using the 30% and 40% cutoffs. Increases in the DBM were observed particularly in Asia, whereas improvements were observed in Latin America and the Caribbean, and the Middle East and north Africa.

Economic development and the DBM at the national level

Examining the changes in the DBM status by quartile of gross domestic products (GDP) per capita in 1990, we can see that increases in the number of countries with a DBM from the 1990s to the 2010s are within the lowest income quartile, whereas the number of countries with a DBM has declined in the top three income quartiles (figure 2). The largest increases in number of the DBM countries were observed using the 20% and 30% adult overweight prevalence cutoffs.

This highlights the driving role of overweight in shaping countries now facing a high DBM, with the greatest effect among the countries in the lowest quartile of GDP per capita. Figure 3 further highlights these changes in the DBM by income quartile. At the same time, the total number of countries with a severe DBM (ie, defined using the 40% overweight cutoff) declined, related mainly to substantial declines in wasting and stunting.

Household-level DBM

The DBM at the household level was defined as one or more individuals with wasting, stunting, or thinness and one or more individuals with overweight or obesity within the same household. The DBM can occur in one of four ways: a child is both stunted and overweight; the mother is overweight and one of her children younger than 5 years has wasting the mother is overweight and one of her children younger than 5 years is stunted; or the mother is thin and one of her children is

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

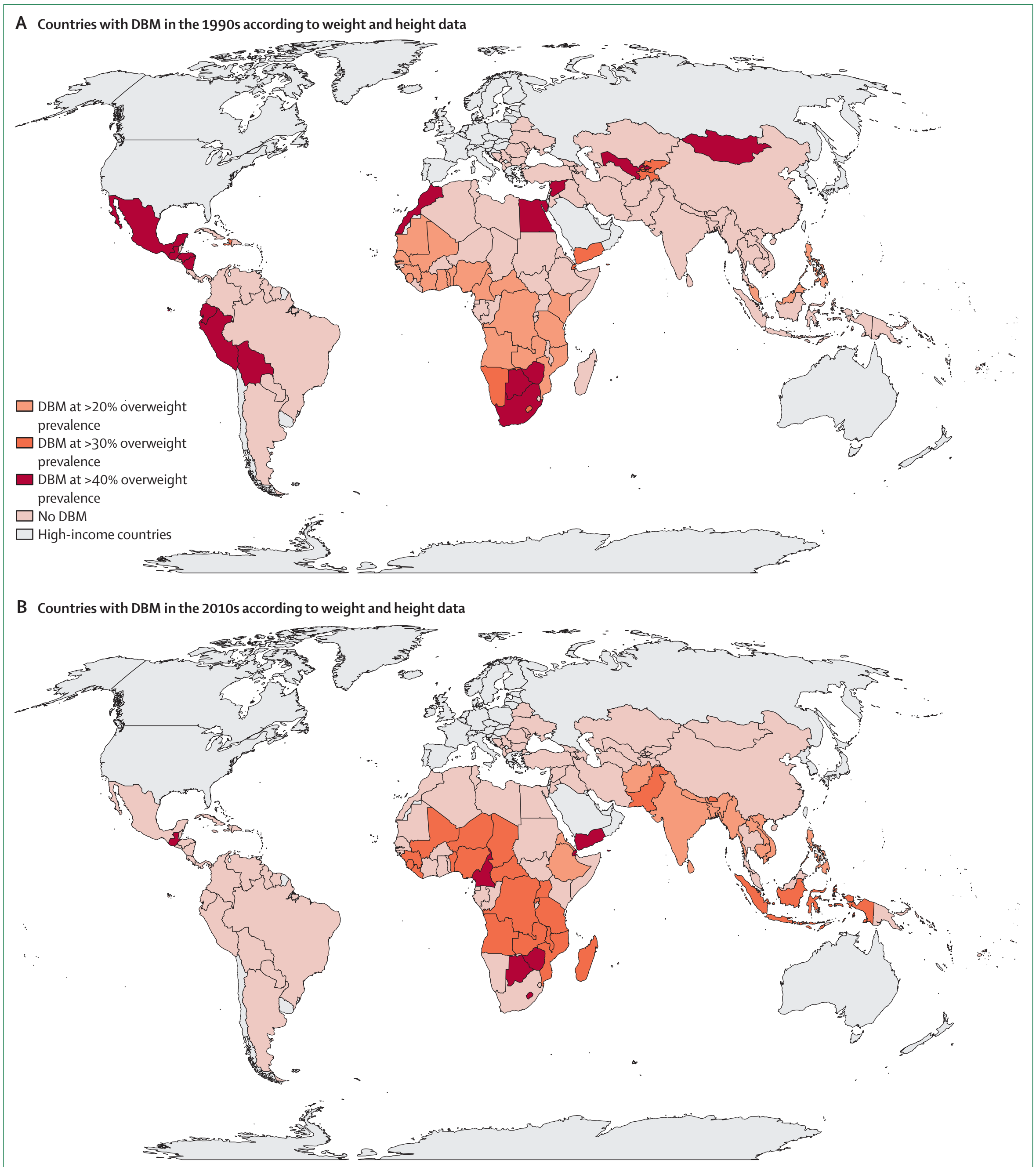

Figure 1: **The global DBM in low-income and middle-income countries based on weight and height data from the 1990s (A) and 2010s (B)**
DBM at the country level was defined as a high prevalence of both undernutrition (wasting and stunting in children aged 0–4 years, and thinness in adult women) and overweight and obesity (defined according to three different overweight prevalence thresholds: 20%, 30%, and 40%) in at least one population group. Data sources are UNICEF, WHO, World Bank, and NCD-RisC estimates, supplemented with selected Demographic and Health Surveys and other country direct measures. DBM=double burden of malnutrition.

For more on the **Demographic and Health Surveys** see https://dhsprogram.com/

overweight. Appendix pp 14–15 shows the prevalence of each of these scenarios and the total prevalence of household DBM.

Household-level DBM is driven primarily by the combination of women with overweight and children with stunting (highest prevalence of all four possible combinations in every country with the highest level of change where increases in the prevalence of the DBM are occurring). By contrast, the contribution of the fourth category of the DBM (mother with thinness and child with overweight) is extremely small, with less than 1% prevalence in most countries.

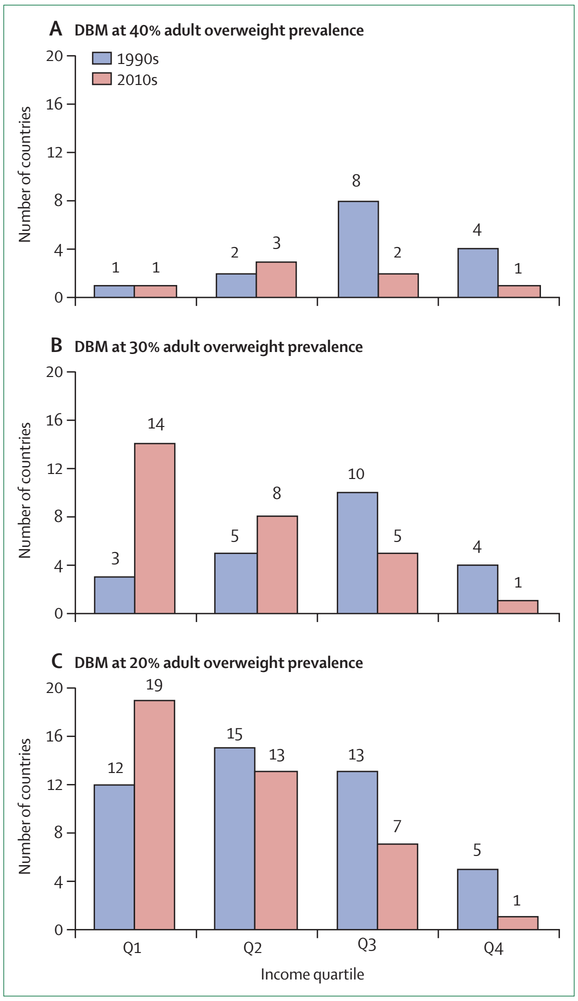

Figure 2: **Countries with a high DBM in the 1990s and 2010s, by overweight prevalence threshold and income quartile**
DBM at the country level was defined as a high prevalence of both undernutrition (wasting and stunting in children aged 0–4 years, and thinness in adult women) and overweight and obesity, defined according to three different overweight prevalence thresholds: 20% (A), 30% (B), and 40% (C), in at least one population group. Countries were only included here if they had DBM data available for both time periods (1990s and 2010s). Q1 is lowest wealth and Q4 is highest wealth according to gross domestic product per capita (purchasing power parity). Data sources are UNICEF, WHO, World Bank, and NCD-RisC estimates, supplemented with selected Demographic and Health Surveys and other country direct measures for the 1990s. DBM=double burden of malnutrition. Q=quartile.

The prevalence of total household-level DBM ranges from less than 3% to nearly 35%, with over a quarter of households experiencing a DBM in Azerbaijan, Guatemala, Egypt, Comoros, and São Tomé and Príncipe. The total household-level DBM is shown in appendix p 22 for 22 countries with at least two surveys of more than 15 years apart. Comparing the earliest and the latest surveys in these countries, five countries showed reductions in their DBM and 15 (including India, China, and Indonesia) showed increases (appendix p 22).

Economic development and the DBM at the household level
In appendix pp 12–15, the GDP per capita and household-level DBM relationship is similar in the two time periods examined (1990s and 2010s), with the highest levels of household DBM seen in the middle of the income range (figure 4). However, the entire curve has shifted slightly upward (about 2–3 additional percentage points) in the most recent time period at all income levels.

Individual-level DBM

The second paper in this Series[26] addresses the underlying developmental origins biology that can lead to a child having both stunting and overweight. The prevalence of individual-level DBM for children aged 0–4 years is shown in the appendix (pp 14–15, 22). The proportion of children who have both stunting and overweight ranges from less than 1% in Myanmar, Colombia, and Nepal to more than 15% in Albania. Among the 20 countries with earliest-to-latest-year data spanning 15 or more years, about half showed that the DBM was declining in children at the individual level and half showed that it was increasing (appendix p 22).

Changes in malnutrition over time: equity considerations

Nearly all countries saw declines in child wasting or stunting, with a third declining by more than one percentage point per year (appendix pp 16–17). Conversely, nearly all countries saw an increase in overweight among women, with over a third of countries increasing by more than one percentage point per year (appendix pp 18–19).

From a public health perspective, where this overweight is emerging and whether the burden is now, or in the future, and greater among people living in poverty is important to know. To provide some sensitivity to where increased overweight is occurring, we looked at the prevalence and annualised changes in prevalence in all countries with 2 years of anthropometric data and examined the differences in the changes in the prevalence of overweight and obesity between the poorest and the highest-wealth quintiles in the earliest and most recent survey periods (figure 5).

A positive annualised difference indicates that people in the poorest quintile face a greater prevalence of overweight and obesity than do those in higher wealth quintiles and suggests increasing disparities between the lowest and the highest wealth quintiles (see appendix p 19 for the data and appendix pp 1–5 for the methodology). We show a growing prevalence of overweight and obesity among lower-wealth households in most countries in Latin America and the Caribbean, eastern Europe and central Asia, and east Asia (led by China and Indonesia). By contrast, sub-Saharan Africa and south Asia have the largest increases in prevalence of overweight and obesity among higher-wealth households (appendix p 19). We cannot predict with existent data whether these regions will see a shift toward greater overweight among lower-wealth households in the future. A study shows that in all LMICs other than south Asia and sub-Saharan Africa, rural overweight and obesity is growing faster than in urban areas and highlights the need for shared solutions targeted to both urban and rural areas.[43]

The next section explores the global food systems linked with a new nutrition reality now affecting even the poorest LMICs.

Transitions that explain the current shifts in the DBM towards lower-income countries

We found that of the countries that have a new DBM at any overweight or obesity prevalence threshold, the largest proportion were in the lower quartiles GDP per capita purchasing power parity (figure 3). At the same time, the number of countries in the upper income quartiles with a DBM decreased. This result reflects increasing overweight among lower-income countries that have not reduced stunting, wasting, or thinness below the WHO-UNICEF cutoff levels. We focus the following discussion on the changing food system and new nutrition reality that these poorest LMICs are facing.

Economic change certainly has been crucial to the reductions in wasting, stunting, and thinness as well as declines in physical activity and major shifts in the food system that have resulted in an increase in consumption of ultra-processed foods.[16,20,21,44–49] In longitudinal studies in China, the increases in overweight were fuelled by a massive reduction in physical activity and most probably also in energy expenditure derived from the introduction of modern technology in market economic work, home production (eg, rice cookers, refrigerators, stoves), and transportation systems.[18–21,48,50]

However, very rapid changes in the diets and the food systems of most LMICs are where most of the recent change in energy imbalance that causes weight gain is focused. Offsetting the effects of any ultra-processed food is difficult—eg, by drinking a 355 mL bottle of sugar-sweetened beverage, the consumer would be required to undertake a 1·5 mile walk or run for at least 15 min.[39] Thus, we discuss later on mainly the shifts in the food system and diet changes over time. We conceptualise the food system as the entire process from production to the consumer.[51–53]

This system includes the activities, infrastructure, and people involved in feeding the global population. Over the past several decades the relative influence and power among the various actors who affect and direct food production has increased, as has the penetration of modern food retailing and marketing throughout most LMICs. In this Series paper we touch on these dramatic changes only briefly. A more in-depth discussion of these changes can be found elsewhere[16] and in more detail for Latin America and the Caribbean.[53] These food system changes are clearly important for weight gain and overweight status but the literature on how these changes

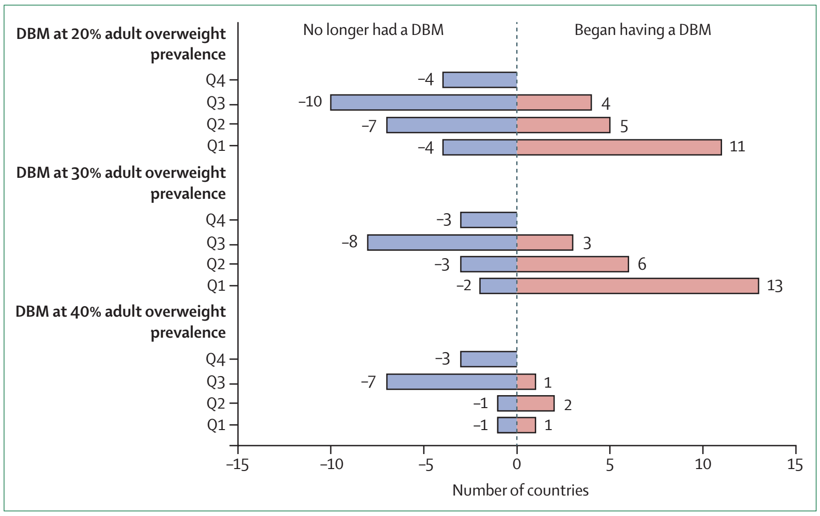

Figure 3: **Number of countries that changed DBM status from the 1990s to 2010s, by gross income quartile**
DBM at the country level was defined as a high prevalence of both undernutrition (wasting and stunting in children aged 0–4 years, and thinness in adult women) and overweight and obesity (defined according to three different overweight prevalence thresholds: 20%, 30%, and 40%), in at least one population group. Countries were only included here if they had DBM data available for both time periods (1990s and 2010s). Q1 is lowest wealth and Q4 is highest wealth according to gross domestic product per capita (purchasing power parity). Data sources are UNICEF, WHO, World Bank, and NCD-RisC estimates, supplemented with selected Demographic and Health Surveys and other country direct measures for the 1990s. DBM=double burden of malnutrition. Q=quartile.

affect undernutrition is unclear and this association is understudied.

The general concept of the nutrition transition is that in each region of the world (not only countries but subregions within countries), a transformation in the way people eat, drink, and move at work, at home, in transport, and in leisure has affected the distribution of body composition and created nutritional problems.[54,55]

The transition has produced remarkable shifts in physical activity and diets in LMICs and a rapid increase in overweight, obesity, and nutrition-related non-communicable diseases.[20,21,48,50] As we have noted we only have suggestive information on ways that this transition affects infant diets for those facing a high risk of stunting and wasting.[45,47,49,56] In the past decade, no studies have been done on the exact causes of country-level or even household-level DBM. But we will describe briefly a new nutrition reality that is rapidly becoming the major driver of overweight and obesity among lower-income countries and also has unclear but increasing effects on undernutrition.

The new nutrition reality

The new nutrition reality is particularly important to acknowledge, because diet is an important driver of the DBM.[57,58] Although we understand that changes in the past several decades in food marketing, access, and purchase of packaged processed foods have demarcated a new nutrition reality across the globe, this Series paper focuses on the effect on all LMICs while attempting to

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

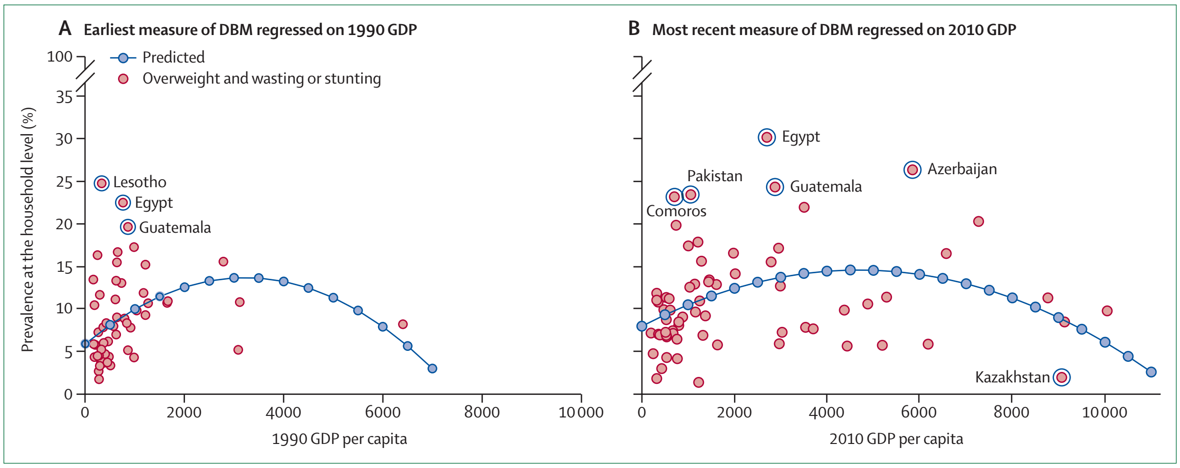

Figure 4: **The association between GDP per capita (purchasing power parity) and regressions relating GDP per capita to prevalence of household-level DBM**
DBM at the household level was defined as one or more individuals with wasting, stunting, or thinness and one or more individuals with overweight or obesity within the same household. Data sources are the Demographic and Health Surveys with the exceptions of China (China Health and Nutrition Survey), Indonesia (Indonesian Family Life Survey), Mexico (Mexico National Survey of Health and Nutrition), Brazil (Brazil National Health Survey), and Vietnam (Vietnam Living Standards Survey). DBM=double burden of malnutrition. GDP=gross domestic product.

understand the effects on countries in different regions and with varying income levels.[59–61] The growth in retail food[59,62,63] and the control of the entire food chain in many countries by agribusinesses, food retailers, food manufacturers, and food service companies have changed markedly.[53,64] This change has been accompanied by the increased consumption of ultra-processed food purchases in LMICs.[65,66] Ultra-processed, packaged foods rich in refined carbohydrates, fat, sugar, and salt are relatively inexpensive and often ready to eat.[67]

Evidence suggests these ultra-processed foods play a major role in increased obesity and non-communicable diseases. A randomised controlled trial done by a team at the US National Institutes of Health showed that adults with normal weight lost 0·9 kg in 2 weeks when fed a real-food diet and gained 0·9 kg when fed a diet composed of ultra-processed foods, following a cross-over design.[14]

Whether the hyperpalatability of ultra-processed food or the much higher energy density of these foods causes such a weight gain is unclear from this study. Additionally, two large European cohorts have shown a strong positive relation between ultra-processed foods and cardiovascular disease and all-cause mortality.[68–70] The role of ultra-processed foods on stunting remains less clear, although, we would expect a negative effect if they are replacing nutrient-rich, energy-dense foods.[14,69,70] Intake of ultra-processed foods during the first 1000 days of life is increasing and represents a newly emerging probable contributor of stunting,[44–47,49,56,71,72] therefore perpetuating the DBM.

The retail revolution

The retail revolution, which has led to fresh markets increasingly disappearing and large and small food retailers replacing them, has swept the globe, as a series of studies by Reardon and others shows.[59,60,62–64,73] In Latin America and the Caribbean, sales of packaged processed food increased from about 10% of all food expenditures (both in-cash and in-kind sales) in 1990 to 60% in 2000. The bulk of this increase appears to be in ultra-processed unhealthy foods and beverages and the growth continues in this region.[53,62,74] Similar increases in the penetration of modern food retailers emerged at different rates across Asia, Africa, and the Middle East.[63]

The Persian Gulf states[63] have not been studied as carefully, although increased penetration of modern food retailers in this region presumably occurred even earlier than in other regions, because the area urbanised and had rapid growth in incomes. The changes vary across sub-Saharan Africa, north Africa, east Asia, southeast Asia, and south Asia but are accelerating in most countries in these regions.[59,60,75] Urban areas were already dominated by a modern food retail sector in the mid-1990s, but most growth in the sub-Saharan African market has occurred in the 21st century.[61]

Controlling the food supply

The actors who control the food supply are changing. At the time of the green revolution and the growth of the global agricultural research sector between 1950 and 1969, countries, large agribusiness firms in the seed and fertiliser sector, and global foundations generated the change.[51,53,64,73,74,76] Although these players still have major roles in producing new technology, control of the food chain is transforming. Case studies from China, Bangladesh, and India were the first to remark on this transformation[64] and later research showed similar trends in Africa.[60]

These studies showed that the global and national public sectors were no longer the major influences of diets in LMICs. Rather, food retailers, food agribusinesses, global food companies, and the food service sector and their domestic local counterparts have contracts directly with farmers.

Non-essential foods and beverages

Sales of non-essential foods and beverages are growing rapidly. Sales volume data from Euromonitor International shows trends in increasing sales of non-essential or junk foods and sugar-sweetened beverages in Chile, South Africa, the Philippines, and Malaysia (appendix p 23).[77] Appendix pp 20–21 present the sales data from Euromonitor International used to model the GDP relationship with both sales volumes and annualised changes in sales of sugar-sweetened beverages (appendix p 24) and junk foods (appendix p 25) in LMICs using data regressions.[78]

These results show that sales of sugar-sweetened beverages were already high in lower-income countries by 2017. The rapid growth of junk foods and sugar-sweetened beverages in these countries exemplifies how aggressive this food sector is. India and China are two of the top five markets for sugary beverage manufacturers (appendix pp 26–27) and sugary beverages are expected to become these countries major markets in the next decade. The speed of change is particularly important in understanding how this nutrition reality is shifting.

Key drivers of the new nutrition reality

In LMICs, urbanisation, migration to cities, income growth, infrastructure improvements, and global trade policy liberalisation have spurred private investment in the food sector.[54,61,74,77] The roles of income growth[78] and other drivers associated with diet changes should not be downplayed. Equally important is how the increase in the number of women working outside the home[53,79] and the value of their time in work have shaped the demand for food that is ready to eat or ready to heat.[80] Monteiro calls this convenience in food preparation and consumption the ultra-processed food revolution.[65,66]

Modern marketing and access to mass media have added to changes in conceptions of the ideal set of foods. Although power is shifting to large-scale food retailers, manufacturers, and food-service companies,[53,64] the informal sector and smaller local companies remain understudied components of the food sector who are often important sources of food for low-income and rural populations.

Conclusion

This Series paper has shown that LMICs continue to have a high DBM; however, countries with a high DBM have lower incomes than the countries that had a high DBM in the early 1990s. The analysis of the dynamics of undernutrition and obesity suggest that the high DBM is being driven by the rapid increases in the prevalence of overweight and obesity occurring in these lower-income

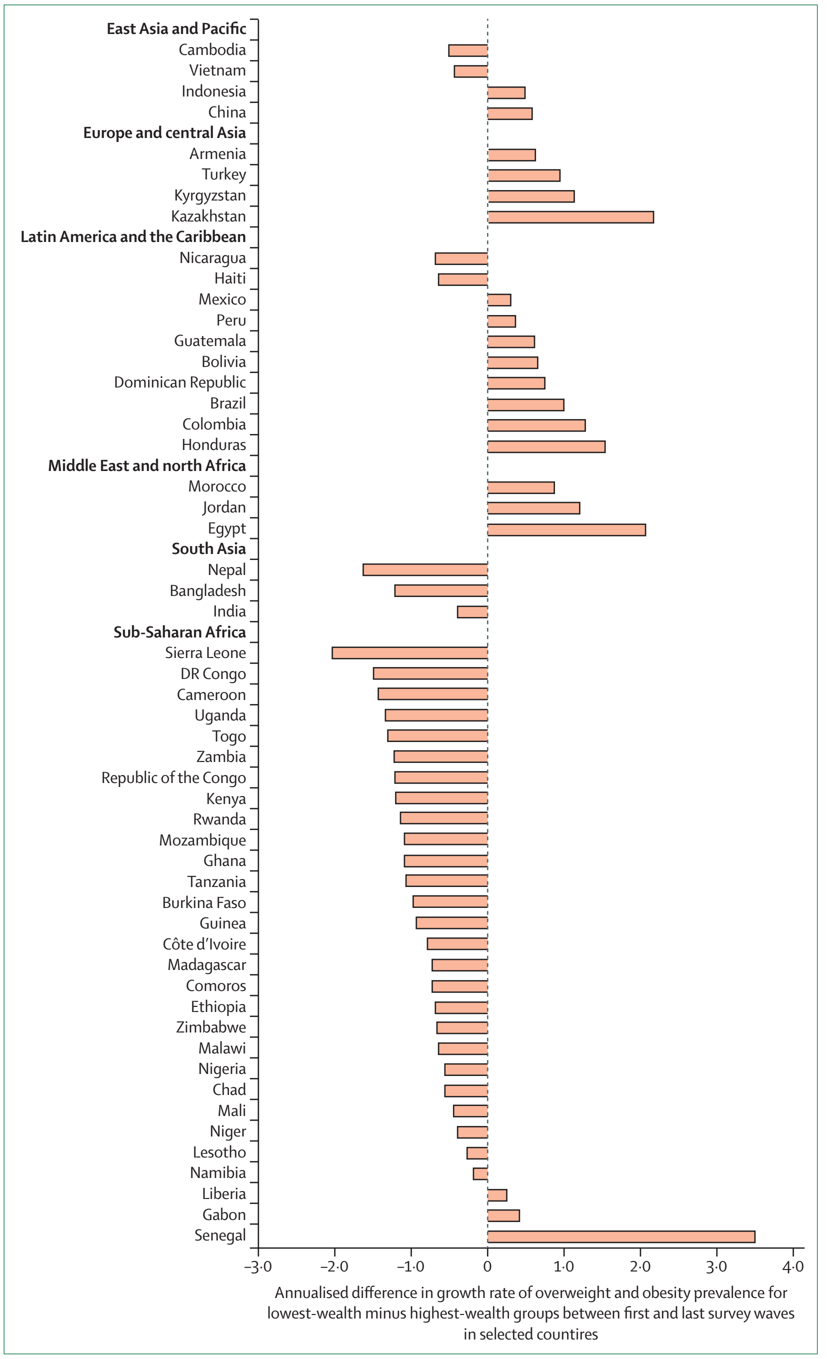

Figure 5: **The shifting burden of overweight and obesity from higher-wealth to lower-wealth populations in sample countries**

Positive difference indicates higher annualised growth in overweight and obesity prevalence for the lowest-wealth quartile. Countries presented here had earliest-to-latest-year data spanning 15 or more years, latest-year data after 2010, and a population greater than approximately 15 million (with the exception of Jordan and Kyrgyzstan, which both had smaller populations but were included for regional representation). The data presented are from years spanning 1988 to 2016, but exact years vary by country. The span of earliest-to-latest years collected ranges from 15 years to 24 years. All data are from the Demographic and Health Surveys with the exceptions of China (China Health and Nutrition Survey), Indonesia (Indonesian Family Life Survey), Mexico (Mexico National Survey of Health and Nutrition), Brazil (Brazil National Health Survey), and Vietnam (Vietnam Living Standards Survey).

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

countries that are also experiencing a slower decline in the prevalence of undernutrition. A greater number of new countries with a high DBM were in the lowest GDP per capita (purchasing power parity) quartile of LMICs than in the 1990s. We show that this new nutrition reality is driven by important and rapid changes that have taken place in the food system. Conversion of the global and domestic retail food, food service, and agribusiness sectors of the economy, along with other transformations,[52,53,60,64] have resulted in an important increased availability of ultra-processed foods in LMICs.

Ultra-processed food consumption has been linked to the risk of overweight and obesity, and non-communicable disease, and preliminary evidence shows that the consumption of these foods during the first 1000 day (pregnancy and infancy) early-life window could be also linked to stunting. Understanding and tackling the drivers of the food system shift, and enacting effective policies that address the challenges of the DBM, are urgently needed.

Contributors

BMP drafted the paper, tables, and figures. LMG-S and CC reviewed, suggested major revision of the structuring of the paper, and suggested additional analyses. All authors reviewed and edited the final revision.

Declaration of interests

We declare no competing interests.

Acknowledgments

Funding is from the National Institutes of Health (R01DK108148 and P2C HD050924). Funding for the preparation of the Series was provided by WHO, through a grant from the Bill & Melinda Gates Foundation. The funder had no role in the analysis and interpretation of the evidence or in writing the paper and the decision to submit for publication. We thank Karen Ritter and Emily Busey for great programming, research, and graphics support, Frances Burton for administrative support, and Francesco Branco and the other authors of the Double Burden of Malnutrition Series for suggestions.

References

1 World Health Organization. Obesity and overweight factsheet. 2016. http://www.who.int/mediacentre/factsheets/fs311/en/ (accessed May 1, 2017).
2 International Food Policy Research Institute. 2017 global food policy report. Washington, DC: International Food Policy Research Institute (IFPRI), 2017.
3 FAO, IFAD, UNICEF, WFP, WHO. The state of food security and nutrition in the world 2018: building climate resilience for food security and nutrition. 2018. https://www.who.int/nutrition/publications/foodsecurity/state-food-security-nutrition-2018/en/ (accessed Nov 7, 2019).
4 Doak CM, Adair LS, Bentley M, Monteiro C, Popkin BM. The dual burden household and the nutrition transition paradox. *Int J Obes* 2005; **29:** 129–36.
5 Doak CM, Adair LS, Monteiro C, Popkin BM. Overweight and underweight coexist within households in Brazil, China and Russia. *J Nutr* 2000; **130:** 2965–71.
6 Garrett JL, Ruel MT. Stunted child-overweight mother pairs: prevalence and association with economic development and urbanization. *Food Nutr Bull* 2005; **26:** 209–21.
7 Victora CG, Rivera JA. Optimal child growth and the double burden of malnutrition: research and programmatic implications. *Am J Clin Nutr* 2014; **100:** 1611S–12S.
8 Rivera JA, Pedraza LS, Martorell R, Gil A. Introduction to the double burden of undernutrition and excess weight in Latin America. *Am J Clin Nutr* 2014; **100:** 1613S–16S.
9 Kroker-Lobos MF, Pedroza-Tobías A, Pedraza LS, Rivera JA. The double burden of undernutrition and excess body weight in Mexico. *Am J Clin Nutr* 2014; **100:** 1652S–58S.
10 World Health Organization. Double-duty actions for nutrition: policy brief. 2017. https://www.who.int/nutrition/publications/double-duty-actions-nutrition-policybrief/en/ (accessed Nov 7, 2019).
11 Hawkes C, Demaio AR, Branca F. Double-duty actions for ending malnutrition within a decade. *Lancet Glob Health* 2017; **5:** e745–46.
12 Tzioumis E, Adair LS. Childhood dual burden of under- and overnutrition in low- and middle-income countries: a critical review. *Food Nutr Bull* 2014; **35:** 230–43.
13 Global Nutrition Report independent expert group. 2018 global nutrition report: shining a light to spur action on nutrition. 2018. https://globalnutritionreport.org/reports/global-nutrition-report-2018/ (accessed Nov 7, 2019).
14 Hall KD, Ayuketah A, Brychta R, et al. Ultra-processed diets cause excess calorie intake and weight gain: A one-month inpatient randomized controlled trial of ad libitum food intake. *Cell Matab* 2019; **30:** 1–10.
15 Popkin BM. Nutrition, agriculture and the global food system in low and middle income countries. *Food Policy* 2014; **47:** 91–96.
16 Popkin BM. Relationship between shifts in food system dynamics and acceleration of the global nutrition transition. *Nutr Rev* 2017; **75:** 73–82.
17 Popkin BM, Reardon T. Obesity and the food system transformation in Latin America. *Obes Rev* 2018; **19:** 1028–64.
18 Monda KL, Adair LS, Zhai F, Popkin BM. Longitudinal relationships between occupational and domestic physical activity patterns and body weight in China. *Eur J Clin Nutr* 2008; **62:** 1318–25.
19 Monda KL, Popkin BM. Cluster analysis methods help to clarify the activity-BMI relationship of Chinese youth. *Obes Res* 2005; **13:** 1042–51.
20 Ng SW, Norton EC, Guilkey DK, Popkin BM. Estimation of a dynamic model of weight. *Empir Econ* 2012; **42:** 413–43.
21 Ng SW, Popkin BM. Time use and physical activity: a shift away from movement across the globe. *Obes Rev* 2012; **13:** 659–80.
22 Horton R, Lo S. Nutrition: a quintessential sustainable development goal. *Lancet* 2013; **382:** 371–72.
23 Willett W, Rockström J, Loken B, et al. Food in the Anthropocene: the EAT-*Lancet* Commission on healthy diets from sustainable food systems. *Lancet* 2019; **393:** 447–92.
24 Swinburn BA, Kraak VI, Allender S, et al. The Global Syndemic of Obesity, Undernutrition, and Climate Change: *The Lancet* Commission report. *Lancet* 2019; **393:** 791–846.
25 Wells JCK, Wibaek R, Poullas M. The dual burden of malnutrition increases the risk of cesarean delivery: evidence from India. *Front Public Health* 2018; **6:** 292.
26 Wells JC, Sawaya AL, Wibeak R, et al. The double burden of malnutrition: aetiological pathways and consequences for health. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32472-9.
27 Adair LS, Fall CH, Osmond C, et al. Associations of linear growth and relative weight gain during early life with adult health and human capital in countries of low and middle income: findings from five birth cohort studies. *Lancet* 2013; **382:** 525–34.
28 Stein AD, Wang M, Martorell R, et al. Growth patterns in early childhood and final attained stature: data from five birth cohorts from low- and middle-income countries. *Am J Hum Biol* 2010; **22:** 353–59.
29 Victora CG, Adair L, Fall C, et al. Maternal and child undernutrition: consequences for adult health and human capital. *Lancet* 2008; **371:** 340–57.
30 WHO. United Nations decade of action. 2016. http://www.who.int/nutrition/decade-of-action/en/ (accessed March 1, 2019).
31 Nilsson M, Griggs D, Visbeck M. Policy: map the interactions between Sustainable Development Goals. *Nature* 2016; **534:** 320–22.
32 UN Children's Fund. Implementing taxes on sugar-sweetened beverages: an overview of current approaches and the potential benefits for children. 2019. https://scalingupnutrition.org/news/implementing-taxes-on-sugar-sweetened-beverages-an-overview-of-current-approaches/ (accessed March 19, 2019).
33 Hawkes C, Ruel MT, Salm L, Sinclair B, Branca F. Double-duty actions: seizing program and policy opportunities to address malnutrition in all its forms. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32506-1.

34 Nugent R, Levin C, Hale J, Hutchison B. Economic effects of the double burden of malnutrition. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32473-0.

35 de Onis M, Borghi E, Arimond M, et al. Prevalence thresholds for wasting, overweight and stunting in children under 5 years. *Public Health Nutr* 2019; **22:** 175–79.

36 Colin Bell A, Adair LS, Popkin BM. Ethnic differences in the association between body mass index and hypertension. *Am J Epidemiol* 2002; **155:** 346–53.

37 Albrecht SS, Mayer-Davis E, Popkin BM. Secular and race/ethnic trends in glycemic outcomes by BMI in US adults: the role of waist circumference. *Diabetes Metab Res Rev* 2017; **33:** e2889.

38 Expert Consultation WHO. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* 2004; **363:** 157–63.

39 Heyward VH, Gibson A. Advanced fitness assessment and exercise prescription 7th edition. Champaign Illinois: Human Kinetics Publishing, 2014.

40 Misra A. Ethnic-specific criteria for classification of body mass index: a perspective for Asian Indians and American Diabetes Association position statement. *Diabetes Technol Ther* 2015; **17:** 667–71.

41 Nair M, Prabhakaran D. Why do South Asians have high risk for CAD? *Glob Heart* 2012; **7:** 307–14.

42 Joshi P, Islam S, Pais P, et al. Risk factors for early myocardial infarction in South Asians compared with individuals in other countries. *JAMA* 2007; **297:** 286–94.

43 NCD Risk Factor Collaboration (NCD-RisC). Rising rural body-mass index is the main driver of the global obesity epidemic in adults. *Nature* 2019; **569:** 260–64.

44 Pries AM, Huffman SL, Mengkheang K, et al. Pervasive promotion of breastmilk substitutes in Phnom Penh, Cambodia, and high usage by mothers for infant and young child feeding. *Matern Child Nutr* 2016; **12** (suppl 2): 38–51.

45 Pries AM, Huffman SL, Mengkheang K, et al. High use of commercial food products among infants and young children and promotions for these products in Cambodia. *Matern Child Nutr* 2016; **12** (suppl 2): 52–63.

46 Vitta BS, Benjamin M, Pries AM, Champeny M, Zehner E, Huffman SL. Infant and young child feeding practices among children under 2 years of age and maternal exposure to infant and young child feeding messages and promotions in Dar es Salaam, Tanzania. *Matern Child Nutr* 2016; **12** (suppl 2): 77–90.

47 Huffman SL, Piwoz EG, Vosti SA, Dewey KG. Babies, soft drinks and snacks: a concern in low- and middle-income countries? *Matern Child Nutr* 2014; **10:** 562–74.

48 Bell AC, Ge K, Popkin BM. Weight gain and its predictors in Chinese adults. *Int J Obes Relat Metab Disord* 2001; **25:** 1079–86.

49 Pries AM, Filteau S, Ferguson EL. Snack food and beverage consumption and young child nutrition in low- and middle-income countries: a systematic review. *Matern Child Nutr* 2019; **15** (suppl 4): e12729.

50 Bell AC, Ge K, Popkin BM. The road to obesity or the path to prevention: motorized transportation and obesity in China. *Obes Res* 2002; **10:** 277–83.

51 Anand SS, Hawkes C, de Souza RJ, et al. Food consumption and its impact on cardiovascular disease: importance of solutions focused on the globalized food system: a report from the workshop convened by the World Heart Federation. *J Am Coll Cardiol* 2015; **66:** 1590–614.

52 Reardon T, Timmer CP. The economics of the food system revolution. *Annu Rev Resour Econ* 2012; **4:** 225–64.

53 Popkin BM, Reardon T. Obesity and the food system transformation in Latin America. *Obes Rev* 2018; **19:** 1028–64.

54 Popkin BM. The shift in stages of the nutrition transition in the developing world differs from past experiences! *Public Health Nutr* 2002; **5:** 205–14.

55 Popkin BM, Adair LS, Ng SW. Global nutrition transition and the pandemic of obesity in developing countries. *Nutr Rev* 2012; **70:** 3–21.

56 Pries AM, Huffman SL, Adhikary I, et al. High consumption of commercial food products among children less than 24 months of age and product promotion in Kathmandu Valley, Nepal. *Matern Child Nutr* 2016; **12** (suppl 2): 22–37.

57 Forouzanfar MH, Alexander L, Anderson HR, et al. Global, regional, and national comparative risk assessment of 79 behavioural, environmental and occupational, and metabolic risks or clusters of risks in 188 countries, 1990–2013: a systematic analysis for the Global Burden of Disease Study 2013. *Lancet* 2015; **386:** 2287–323.

58 Black RE, Allen LH, Bhutta ZA, et al. Maternal and child undernutrition: global and regional exposures and health consequences. *Lancet* 2008; **371:** 243–60.

59 Reardon T, Timmer CP, Minten B. Supermarket revolution in Asia and emerging development strategies to include small farmers. *Proc Natl Acad Sci USA* 2012; **109:** 12332–37.

60 Reardon T, Tschirley D, Minten B, et al. Transformation of African agrifood systems in the new era of rapid urbanization and the emergence of a middle class. In: Badiane O, Makombe T, eds. Beyond a middle income Africa: transforming African economies for sustained growth with rising employment and incomes. Washington: International Food Policy Research Institute, 2015: 62–74.

61 Tschirley D, Reardon T, Dolislager M, Snyder J. The rise of a middle class in East and Southern Africa: Implications for food system transformation. *J Int Dev* 2015; **27:** 628–46.

62 Reardon T, Berdegué JA. The rapid rise of supermarkets in Latin America: challenges and opportunities for development. *Dev Policy Rev* 2002; **20:** 371–88.

63 Reardon T, Timmer CP, Barrett CB, Berdegue JA. The rise of supermarkets in Africa, Asia, and Latin America. *Am J Agric Econ* 2003; **85:** 1140–46.

64 Reardon T, Chen KZ, Minten B, et al. The quiet revolution in Asia's rice value chains. *Ann N Y Acad Sci* 2014; **1331:** 106–18.

65 Monteiro CA, Levy RB, Claro RM, de Castro IR, Cannon G. Increasing consumption of ultra-processed foods and likely impact on human health: evidence from Brazil. *Public Health Nutr* 2011; **14:** 5–13.

66 Monteiro CA, Moubarac JC, Cannon G, Ng SW, Popkin B. Ultra-processed products are becoming dominant in the global food system. *Obes Rev* 2013; **14** (suppl 2): 21–28.

67 Poti JM, Mendez MA, Ng SW, Popkin BM. Is the degree of food processing and convenience linked with the nutritional quality of foods purchased by US households? *Am J Clin Nutr* 2015; **101:** 1251–62.

68 Lawrence MA, Baker PI. Ultra-processed food and adverse health outcomes. *BMJ* 2019; **365:** l2289.

69 Rico-Campà A, Martínez-González MA, Alvarez-Alvarez I, et al. Association between consumption of ultra-processed foods and all cause mortality: SUN prospective cohort study. *BMJ* 2019; **365:** l1949.

70 Srour B, Fezeu LK, Kesse-Guyot E, et al. Ultra-processed food intake and risk of cardiovascular disease: prospective cohort study (NutriNet-Santé). *BMJ* 2019; **365:** l1451.

71 Feeley AB, Ndeye Coly A, Sy Gueye NY, et al. Promotion and consumption of commercially produced foods among children: situation analysis in an urban setting in Senegal. *Matern Child Nutr* 2016; **12** (suppl 2): 64–76.

72 Pries AM, Rehman AM, Filteau S, Sharma N, Upadhyay A, Ferguson EL. Unhealthy snack food and beverage consumption is associated with lower dietary adequacy and length-for-age z-scores among 12–23-month-olds in Kathmandu Valley, Nepal. *J Nutr* 2019; published online July 16. DOI:10.1093/jn/nxz140.

73 Reardon T, Barrett CB, Berdegué JA, Swinnen JFM. Agrifood industry transformation and small farmers in developing countries. *World Dev* 2009; **37:** 1717–27.

74 Reardon TA, Berdegué JA, Farrington J. Supermarkets and farming in Latin America: pointing directions for elsewhere? Overseas Development Institute, 2002. https://www.odi.org/sites/odi.org.uk/files/odi-assets/publications-opinion-files/1949.pdf (accessed Nov 7, 2019).

75 Hu D, Reardon T, Rozelle S, Timmer P, Wang H. The emergence of supermarkets with Chinese characteristics: challenges and opportunities for China's agricultural development. *Dev Policy Rev* 2004; **22:** 557–86.

76 Neven D, Odera MM, Reardon T, Wang H. Kenyan supermarkets, emerging middle-class horticultural farmers, and employment impacts on the rural poor. *World Dev* 2009; **37:** 1802–11.

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

77 Euromonitor. Market sizes- historical- total volume- kilograms or litres per capita-packaged food, soft drinks, and hot drinks. [Statistics]. Euromonitor Passport International London, United Kingdom: Euromonitor; 2018.

78 Bennett M. The world's food. *Am J Agric Econ* 1954; **36:** 350–51.

79 Novta N, Wong J. Women at work in Latin America and the Caribbean. 2017. https://www.imf.org/en/Publications/WP/Issues/2017/02/14/Women-at-Work-in-Latin-America-and-the-Caribbean-44662 (accessed Nov 7, 2019).

80 Mincer J. Market prices, opportunity costs, and income effects. In: Christ CF, Friedman M, Goodman LA, et al, eds. Measurement in economics: studies in mathematical economics and econometrics in memory of Yehuda Grunfeld. Stanford: Stanford University Press, 1963: 67–82.

Double Burden of Malnutrition 2

The double burden of malnutrition: aetiological pathways and consequences for health

Jonathan C Wells, Ana Lydia Sawaya, Rasmus Wibaek, Martha Mwangome, Marios S Poullas, Chittaranjan S Yajnik, Alessandro Demaio**

Malnutrition has historically been researched and addressed within two distinct silos, focusing either on undernutrition, food insecurity, and micronutrient deficiencies, or on overweight, obesity, and dietary excess. However, through rapid global nutrition transition, an increasing proportion of individuals are exposed to different forms of malnutrition during the life course and have the double burden of malnutrition (DBM) directly. Long-lasting effects of malnutrition in early life can be attributed to interconnected biological pathways, involving imbalance of the gut microbiome, inflammation, metabolic dysregulation, and impaired insulin signalling. Life-course exposure to early undernutrition followed by later overweight increases the risk of non-communicable disease, by imposing a high metabolic load on a depleted capacity for homoeostasis, and in women increases the risk of childbirth complications. These life-course trajectories are shaped both by societal driving factors—ie, rapidly changing diets, norms of eating, and physical activity patterns—and by broader ecological factors such as pathogen burden and extrinsic mortality risk. Mitigation of the DBM will require major societal shifts regarding nutrition and public health, to implement comprehensive change that is sustained over decades, and scaled up into the entire global food system.

Published Online December 15, 2019 https://doi.org/10.1016/S0140-6736(19)32472-9

This is the second in a Series of four papers about the double burden of malnutrition

*Joint senior authors

Childhood Nutrition Research Centre, UCL Great Ormond Street Institute of Child Health, London, UK (Prof J C Wells PhD, M S Poullas MRes)**; Department of Physiology, Federal University of São Paulo, São Paulo, Brazil** (Prof A L Sawaya PhD)**; Department of Nutrition, Exercise and Sports** (R Wibaek PhD) **and School of Global Health, Department of Public Health** (A Demaio PhD)**, University of Copenhagen, Copenhagen, Denmark; Clinical Epidemiology, Steno Diabetes Center Copenhagen, Copenhagen, Denmark** (R Wibaek)**; Kenya Medical Research Institute Wellcome Trust Research Program, Kilifi, Kenya** (M Mwangome PhD)**; Diabetes Unit, KEM Hospital and Research Centre, Pune, India** (Prof C S Yajnik MD)**; EAT Foundation, Oslo, Norway** (A Demaio)**; Melbourne School of Population and Global Health, University of Melbourne, Melbourne, VIC, Australia** (A Demaio)**; and VicHealth, Melbourne, VIC, Australia** (A Demaio)

Correspondence to: Prof Jonathan C Wells, Childhood Nutrition Research Centre, Population, Policy and Practice Research and Teaching Programme, UCL Great Ormond Street Institute of Child Health, London WC1N 1EH, UK **jonathan.wells@ucl.ac.uk**

See Online for appendix

Introduction

Undernutrition and overweight have historically been considered separate challenges affecting distinct populations, and with contrasting risk factors. Undernutrition was linked with poverty, food insecurity, and infection, whereas obesity was linked with affluence, dietary richness, and sedentary behaviour. Increasingly, the two forms of malnutrition co-occur within communities, families, and even individuals, such as those who are both stunted and overweight.[1] The current manifestation of this global double burden of malnutrition (DBM) is summarised in the first paper of this Series.[2] Obesogenic environments are expanding while the causes of undernutrition persist,[2] and an increasing proportion of individuals who are overweight were undernourished earlier in life.[3] To understand the implications of the DBM for health at the individual level, the explanatory framework must shift from descriptive epidemiology to biology.

In their *Lancet* Commission on the global syndemic of obesity, undernutrition, and climate change, Swinburn and colleagues[4] reconceptualised the two extremes of malnutrition within a single ecological framework, relating them to common drivers that also underlie climate breakdown. Here, we develop this overarching perspective, focusing on the biological interconnections between undernutrition and overweight. First, we describe the cause of malnutrition across life courses and generations. Both undernutrition and overweight can propagate long-term effects, especially if they develop early in life, and each might increase risk of the other occurring. Moreover, increasing numbers of people are being exposed to both forms of malnutrition at different points in the life course, due to the rapid nature of global nutrition transition. Second, we show that individuals who experience the DBM through their life course have increased risk of diverse forms of ill health. Third, we examine why the DBM is affecting more people worldwide, and highlight populations with high susceptibility. We provide an evolutionary perspective that can help to understand these biological interactions and their health consequences in different settings. Our framework might help to identify effective strategies for double-duty actions that address both forms of malnutrition, as discussed in subsequent papers in this Series.[5,6]

Life-course manifestation of malnutrition

Malnutrition is a complex phenotype that manifests across the life course in different ways (appendix pp 2–5), yet its categorisation remains unsophisticated. Regarding undernutrition, simple anthropometry is used to categorise low birthweight, stunting (low height for age) or wasting (low weight for age) during infancy or childhood, and short stature or low body-mass index (BMI) in adulthood. Assessed thus, undernutrition is most prevalent among younger age groups. Undernutrition can also be assessed in terms of depleted stores or circulating concentrations of nutrients, reflecting dietary inadequacy. Micronutrient deficiencies remain prevalent in adults and are of particular concern among women of reproductive age.[7]

Excess weight can likewise emerge in late fetal life (macrosomia), but usually develops from early childhood through cumulative exposure to obesogenic factors acting on both individuals and societies.[4] Many studies link elevated adiposity, especially abdominal fat, with ill health. Despite being weakly associated with adiposity, the simple anthropometric BMI provides a useful

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

Key messages

- Malnutrition has historically been researched and addressed in two distinct silos, focusing either on chronic or acute undernutrition, energy inadequacy, and micronutrient deficiencies, or on overweight, obesity, and dietary excess. Through rapid changes in food environments and living conditions, global nutrition transition is generating a new double burden of malnutrition (DBM) in which undernutrition and overweight are impossible to separate; however, the recognition of this double burden should reveal opportunities to address both issues simultaneously.
- Malnutrition harms health throughout the life course, but the emergence of malnutrition early in life is particularly harmful. A variety of physiological mechanisms propagate effects of early-life malnutrition across the life course, and adolescent and adult malnutrition can transmit effects to the next generation.
- Different forms of malnutrition can interact through the life course and across generations. In some settings, early stunting might predispose an individual to a more central distribution of adiposity at later ages, and the extent to which maternal obesity adversely affects early growth and development of the offspring might be exacerbated if the mother was undernourished in early life.
- Life-course exposure to the DBM (early undernutrition followed by later overweight) increases the risk of non-communicable disease, by imposing a high metabolic load on a depleted capacity for homoeostasis. The health costs of adult obesity are therefore exacerbated among individuals who have previously had undernutrition. In women, life-course exposure to the DBM increases the risk of childbirth complications.
- Exclusive and appropriate breastfeeding protects infants against all forms of malnutrition, and protects mothers against diabetes and breast cancer, in part through healthy-weight benefits. However, maternal obesity, diabetes, and micronutrient deficiencies alter the biology of lactation, and should be addressed to maximise the success of breastfeeding.
- Exposure to the DBM can only be fully understood in the context of broader societal drivers acting across culture, behaviour, and technology. Various groups are at high risk of the double burden through elevated exposure to these drivers, often exacerbated by biological susceptibility.
- Developmental responses to malnutrition in early life are shaped by ecological factors, such as pathogen burden and extrinsic mortality risk. An evolutionary perspective, focusing on how our biological plasticity was shaped in ancestral environments to promote survival and reproduction, might help design interventions that promote linear growth and lean tissue accretion rather than excess adiposity.
- Intergenerational cycles of malnutrition have proven difficult to disrupt through public health interventions. Major societal shifts are required regarding nutrition and public health to implement comprehensive change that is sustained over decades, and scaled up into the entire global food system.

metabolic risk marker for populations.[8] The main limitation of BMI is its inconsistent association with non-communicable disease risk across populations.[9] As with undernutrition, indices of nutritional excess extend beyond the body to traits such as diet composition and physical inactivity, both of which can perturb metabolism.

The concept of malnutrition should also incorporate the gut microbiome, representing millions of genes from microorganisms. The microbiome generates a collective metabolic activity that affects and responds to the human host. Diverse forms of malnutrition are associated with dysbiosis, propagating adverse metabolic consequences (appendix pp 6–9), although findings for obesity are heterogeneous. The microbiome shows resilience within individuals, with implications for health maintenance and disease risk,[10] but can also respond to interventions (appendix pp 6–9).[11]

Malnutrition harms health throughout life, but its early emergence has particularly harmful consequences. Development is characterised by a succession of sensitive periods or so-called critical windows, when phenotype is particularly responsive to nutritional influences. Physiological mechanisms characterising these periods include the differential growth of organs and tissues, establishment of hormonal set points and epigenetic variability, telomere attrition, and microbiome maturation (panel 1). Crucially, these mechanisms respond to both inadequate and excessive levels of nutritional supply in early life, meaning that they contribute to intergenerational effects in both contexts. Such physiological sensitivity explains why early nutrition and growth have major implications for immediate survival, long-term health, and human capital.[21,22]

Many critical windows close early during development, reducing the sensitivity of specific traits to environmental influences. For example, some epigenetic effects are restricted to the periconceptional period,[23] others to early infancy.[18] Likewise, from late infancy linear growth becomes less sensitive to nutritional intake,[24] hence the environmental contribution to short adult stature is primarily attributable to early stunting. However, other traits subsequently become plastic, and adolescence represents a key period of sensitivity to nutritional factors, especially relating to reproductive biology.

At the individual level, the DBM can thus be assessed through diverse somatic, dietary, and behavioural traits, as well as the microbiome, all of which might be targeted by appropriate interventions. Through the mechanisms of plasticity we have highlighted, different forms of malnutrition interact through the life course and across generations.

Intergenerational emergence of the DBM

Although malnutrition manifests within the life course, the causes of this condition span generations. For example, early sensitive periods fall within pregnancy and lactation, making maternal phenotype the key nutritional factor shaping early development.[19,23,25] Through rapid nutrition transition, increasing numbers of people in low-income and middle-income countries (LMICs) are exposed to both nutritional deficiencies and fuel excess at different ages, a scenario termed double teratogenesis.[26]

Many LMIC populations have experienced chronic undernutrition, characterised by intergenerational cycles of disadvantage. Maternal undernutrition compromises fetal growth and increases the risk of childhood underweight, stunting, and micronutrient deficiency (figure 1; appendix pp 13–18). Stunting is a cumulative process, often apparent by birth but worsening until around the age of 2 years when growth becomes canalised.[27] Faltering of linear growth during infancy is exacerbated by

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

episodes of wasting,[28] which helps explain why stunting is associated with an elevated risk of mortality. Such intergenerational cycles have proven difficult to disrupt through interventions: maternal supplementation from mid-pregnancy to term with both macronutrients and micronutrients has modest effects on birthweight, but does not benefit growth in the longer term.[29–31]

Intergenerational effects are equally relevant to mothers with obesity or perturbed metabolism. Maternal obesity is associated with elevated fetal adiposity, especially when compounded by gestational diabetes (figure 1; appendix pp 13–18). More generally, a high amount of nutrition in early life (greater gestational weight gain, higher birthweight, faster postnatal weight gain) is associated with greater risk of obesity, abdominal adiposity, and insulin resistance in adulthood.

However, changes in food systems are breaking down the separation between these intergenerational cycles of nutritional deficiency and excess; we summarise evidence for interactions between these cycles (figure 1 and appendix pp 13–18). We highlight numerous ways in which exposure to undernutrition alters the consequences of subsequent exposure to obesity; for example rapid gain in BMI following early undernutrition might predispose an individual to central adiposity and non-communicable diseases. We show that the reverse pathway is also relevant; for example, the offspring of mothers who are obese might have poor growth and development in early life,[32] although heterogeneity within each of these examples is substantial.

Whether early undernutrition predisposes to later adiposity depends on postnatal patterns of growth and nutrition, including complementary feeding patterns. Growth faltering in early pregnancy might induce a catch up in fat mass for the fetus before birth,[33] and individuals who were small when born often undergo accelerated weight gain in infancy or childhood.[34] This catch up might induce elevated adiposity, and in high-income countries rapid infant weight gain is associated with later obesity.[35] However, studies in LMICs typically associate faster infant weight gain with greater adult height and lean mass,[36] and in these settings, rapid weight gain seems to promote adiposity after about age 2 years,[22] although the pattern of association might change in concert with nutrition transition.

Associations between stunting and later body composition are complex. In the short term, compensatory weight gain immediately following undernutrition might prioritise accretion of fat over lean body mass, through mechanisms of energy sparing.[37] In some studies in South America, early stunting was found to predict excess abdominal adiposity, mediated by changes in fuel metabolism (panel 2).[41] However, stunting was not associated with impaired fat oxidation in young children aged 2–6 years from Cameroon,[42] and in Peru, the height of children aged 3·0–8·5 years was positively associated with adiposity at low altitude, but inversely associated at high altitude.[43] In malnourished young children aged 6–23 months from Burkino Faso, 93·5% of weight gained during a food supplementation programme consisted of lean tissue.[44] These findings indicate complex developmental links between growth patterns and adiposity, whereby growth might either be accelerated across all traits, or characterised by trade-offs between traits. Regardless of whether early stunting elevates abdominal adiposity, a consistent finding is that early undernutrition permanently reduces lean mass and its functional correlates such as grip strength.[45–47]

Although maternal obesity is associated with higher birthweight, it is also associated with micronutrient deficiencies that might impair offspring development, and maternal hypertension is associated with increased

Panel 1: Physiological mechanisms through which exposure to undernutrition or nutritional excess during early life is associated with long-term phenotypic variability

- Early nutrition generates long-term effects on organ size, structure, and function. Mammalian growth in fetal life and early infancy comprises hyperplasia (cell proliferation), crucial for the development of organ structure, whereas from late infancy, growth comprises hypertrophy (increases in cell size).[12] Early growth variability has long-term effects—eg, infants with a low birthweight have altered cardiac structure and small liver, kidneys, and spleen,[13,14] whereas macrosomic infants might have organomegaly.
- Early nutrition affects hormonal axes regulating growth and appetite. Both undernutrition and excess nutrition in the perinatal period affect insulin metabolism and hypothalamic circuits regulating food intake.[15] Infants with low birthweight might be insulin-sensitive at birth, but are susceptible to insulin resistance in association with faster weight gain in childhood.
- Both interuterine growth retardation and maternal diabetes expose the fetus to oxidative stress, affecting cardiac and vascular structure, haemodynamics, and endothelial function.
- Gene expression in the offspring is shaped by maternal nutrition in pregnancy and by nutritional experience after birth. For example, periconceptional exposure to maternal famine has been associated with epigenetic changes in *IGF1* expression that persisted into early old-age,[16] while when the offspring was conceived in rural Africa was associated with diverse epigenetic effects in infancy.[17] Gestational diabetes is associated with epigenetic effects on genes associated with metabolic disease. Some epigenetic changes might have adverse long-term health effects.
- Early exposure or lack of exposure to different food tastes might shape food preferences and diet choices at later ages.
- Telomere length provides a marker of cellular ageing that is sensitive to early nutritional experience. For example, placental and neonatal telomere length are both associated with some components of maternal nutritional status, and predict postnatal body composition,[18] and exclusive breastfeeding might reduce telomere attrition.[19]
- The gut microbiome rapidly matures in early life, and early undernutrition can disrupt this process. For example, among twins discordant for kwashiorkor the affected sibling developed narrower gut microbiome diversity, and transplanting this biota to germ-free mice induced weight loss.[20]
- Collectively, these mechanisms contribute to a profound imprint of early malnutrition on later phenotype, affecting both the risk and the metabolic effects of subsequent overweight.

An expanded, fully referenced version of this panel is available in the appendix (pp 10–12).

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

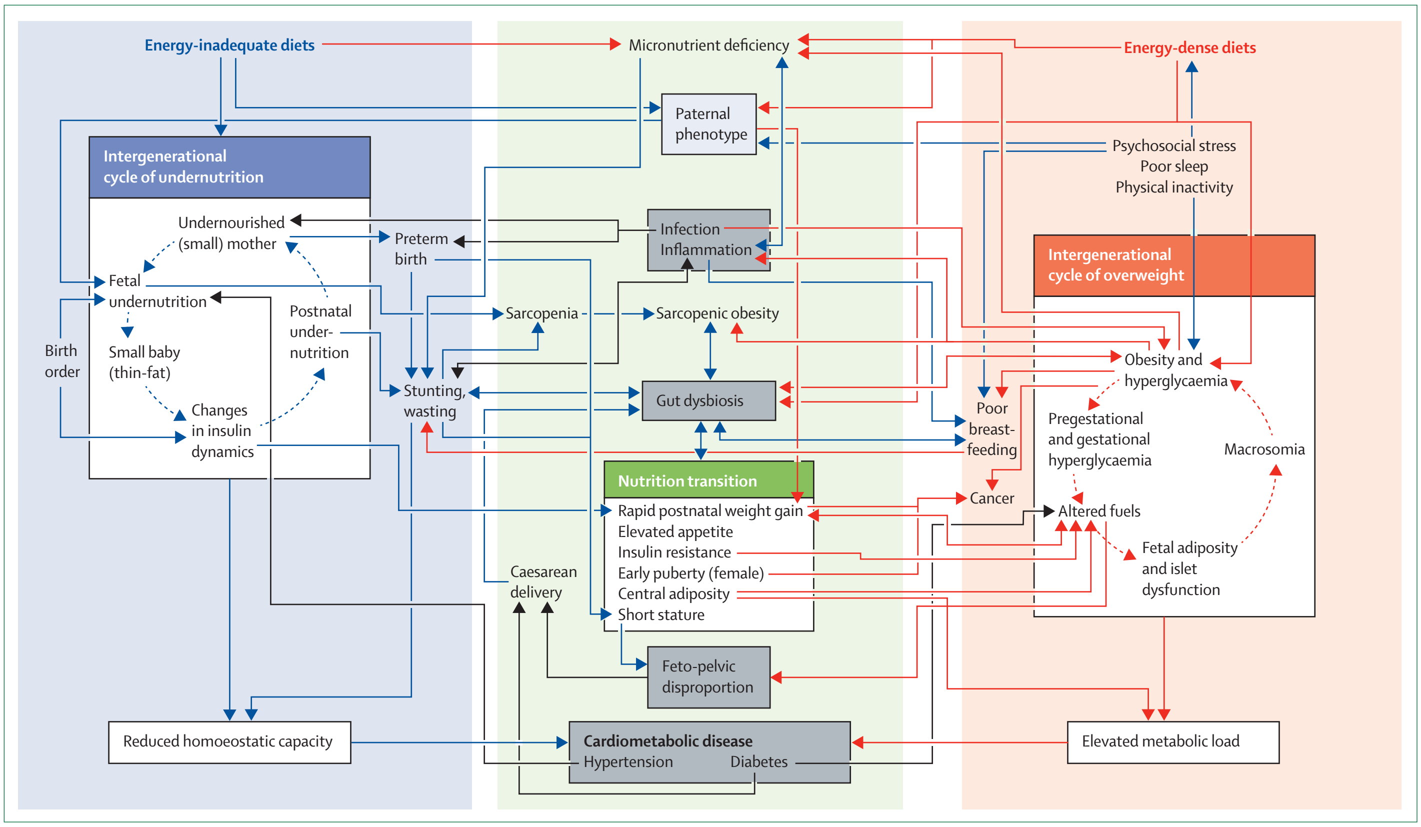

Figure 1: **Complex interconnections between intergenerational cycles of undernutrition and nutritional excess and the effect of nutrition transition**
The intergenerational cycle of undernutrition (blue) associated with energy-inadequate diets and micronutrient deficiencies constrains growth and reduces the metabolic capacity for homoeostasis. The intergenerational cycle of overnutrition (red) associated with energy-dense diets is characterised by excess metabolic fuel and elevated adiposity, each of which challenges homoeostasis. Both cycles of malnutrition contribute to a wide range of adverse health outcomes (grey boxes), and specific diseases also increase the risk of malnutrition (black arrows). Through nutrition transition, individuals shift between these cycles within the life course, both increasing the risk and exacerbating the magnitude of health consequences. This framework helps identify how nutrition transition generates biological connections between many different forms of ill health (eg, low birthweight, stunting, central obesity, diabetes, and caesarean delivery).

risk of offspring having a low birthweight, a scenario exacerbated by antihypertensive pharmaceutical agents.[48] Obesity is generally associated with worse micronutrient status,[49] mediated by chronic inflammation and nutrient-poor diets, and maternal obesity might also contribute to dysbiosis in the offspring.[50]

For more on the **Demographic and Health Survey data** see http://www.dhs.org/

Using Demographic and Health Survey data from 12 LMICs we analysed how markers of maternal malnutrition (short stature, overweight, and obesity) interact in association with the risk of stunting in the offspring. Although short maternal stature (defined as having a height in the bottom quartile of the population sample) increases the risk of stunting, maternal overweight or obesity typically reduces this risk relative to normal BMI, providing the mother does not have a short stature. However, this protective effect disappears if the mother is both overweight and short (figure 2A). The consequences of maternal obesity for the next generation therefore depend on the mother's own developmental experience. In a Swedish study, the intergenerational transmission of obesity was three times greater among mothers who were obese and born small for gestational age than in mothers of normal birthweight.[51]

Although evidence remains scarce, paternal metabolic phenotype can also affect offspring development. For example, paternal smoking and dietary intake during adolescence have been associated with offspring BMI,[52] mediated by imprinting of the sperm.[53] Paternal genes might be especially relevant in early life because they contribute to placental function. Bariatric surgery in men has been associated with remodelling of sperm DNA methylation, in particular of genes associated with appetite control.[53] However, beyond father–child correlations in height and BMI,[54] the understanding of paternal biological contributions to the DBM in LMICs is minimal.

The life-course and intergenerational physiological pathways that we have summarised underlie associations of the DBM with several forms of ill health, as we discuss next.

The DBM and the risk of non-communicable disease

Associations of adult obesity and unhealthy lifestyle with non-communicable diseases are well recognised,[55] but the evidence that exposure to undernutrition in early life exacerbates these relationships is compelling.

To elucidate this interconnection, we present a capacity-load conceptual model.[56,57]

Initially, associations of the risk of non-communicable disease with birthweight were attributed to long-term consequences of fetal undernutrition. The so-called thrifty phenotype hypothesis proposed that inadequate fetal nutrition reduced growth of some organs (eg, pancreas, liver, kidney) to protect the brain. Later, such individuals would be more likely to develop ill health as a result of obesity and energy-dense diets, elevating the risk of non-communicable diseases.[38] However, birthweight is inversely associated with the risk of non-communicable disease across most of its range,[58,59] although infants who are macrosomic have an increased risk of non-communicable disease.[60] This variability refutes the notion that fetal undernutrition is the primary developmental mechanism of non-communicable diseases. The capacity-load model addresses continuous associations of both developmental and adult traits with the risk of non-communicable disease, and can be applied to diverse traits through the life course and to various non-communicable disease outcomes (appendix pp 22–26). Metabolic capacity refers to traits that are strongly dependent on growth and metabolic exposures during early life, and that have life-long implications for the capacity for homoeostasis.[56] Relevant traits include pancreatic β-cell mass and function, nephron number, organ and tissue mass, airway and blood vessel diameter, and cardiac structure. All these traits scale with the magnitude of growth during the period of hyperplasic growth. Environmentally induced epigenetic variability and microbiome development can be considered within the same conceptual framework,[61,62] although the extent to which early variability in these traits persists long term remains uncertain.[63] Size at birth and early postnatal growth patterns act as useful, though imperfect, composite markers of metabolic capacity.

Metabolic load refers to traits that challenge homoeostasis, including excess adiposity, physical inactivity, lipogenic diet, smoking, infection, and psychosocial stress.[56,57] These traits broadly show dose–response associations with the risk of non-communicable disease, and are all associated with increased oxidative damage. Metabolic load can increase early in life in association with catch-up growth, which elevates not only adiposity but also molecular markers of non-communicable disease risk (epigenetic effects, telomere attrition).[64] Macrosomic infants already have elevated adiposity (high metabolic load), and potentially also low metabolic capacity, by birth.

According to this conceptual model, the risk of non-communicable disease decreases in association with metabolic capacity and increases in association with metabolic load. Substantial evidence supports the model,[58,59] but the majority is from studies in high-income countries. Evidence from Asian and sub-Saharan African populations is summarised in the appendix (p 27–31), focusing on anthropometric markers of low capacity and elevated load.

Panel 2: Developmental links between stunting, obesity, and cardiometabolic risk in Brazil

- Undernutrition in early life promotes survival by energy sparing, selectively preserving some tissues and organs over others.[37,38] This adaptation is achieved by endocrine changes affecting growth, energy expenditure, and body composition, which then interact with the composition and energy content of the diet.
- Among children from shanty towns in Brazil, stunting is associated with reduced lean mass but greater adiposity, especially central abdominal fat. These physical traits are associated with increased insulin sensitivity, reduced insulin production, higher cortisol, and a reduced capacity for fat oxidation.[39]
- By adulthood, the adverse effects of overweight on cardiometabolic traits are exacerbated among individuals who are also stunted. Among adults who are overweight, stunting is associated with lower tri-iodothyronine, higher insulin resistance, and higher glycated haemoglobin. In women who are overweight, stunting is also associated with dyslipidaemia and higher blood pressure.[40]
- Adequate treatment of undernutrition during childhood with recovery in height and weight might lead to normalisation of insulin activity, leptin, cortisol stress response, body composition, and bone mineral density.[39]

An expanded, fully referenced version of this panel is available in the appendix (pp 19–20).

One caveat is that the specific role of linear growth in this model varies by outcome. For cardiovascular disease, diabetes, and hypertension, linear growth promotes metabolic capacity, indicated by elevated risk of these non-communicable diseases among individuals with poor early growth or short adult stature (appendix p 32). This association is probably explained by height being a good proxy for organ growth and development through the life course. However, for many forms of cancer, linear growth might better be considered a marker of metabolic load than of metabolic capacity, because faster growth and taller height are associated with elevated cancer risk (appendix pp 33–34).[65,66] These associations indicate that efforts to reduce low birthweight and stunting in LMICs might increase future cancer incidences.

This conceptual model helps explain why the DBM is strongly associated with the risk of non-communicable disease. Low birthweight, childhood stunting, and wasting all deplete components of metabolic capacity, whereas overweight and unhealthy environmental exposures exacerbate metabolic load. Importantly, the extent to which early undernutrition leads to overt non-communicable diseases depends strongly on subsequent nutritional status. For example, survivors of severe malnutrition during early life in Malawi had long-term deficits in height, lean mass, and grip strength; however, the risk of

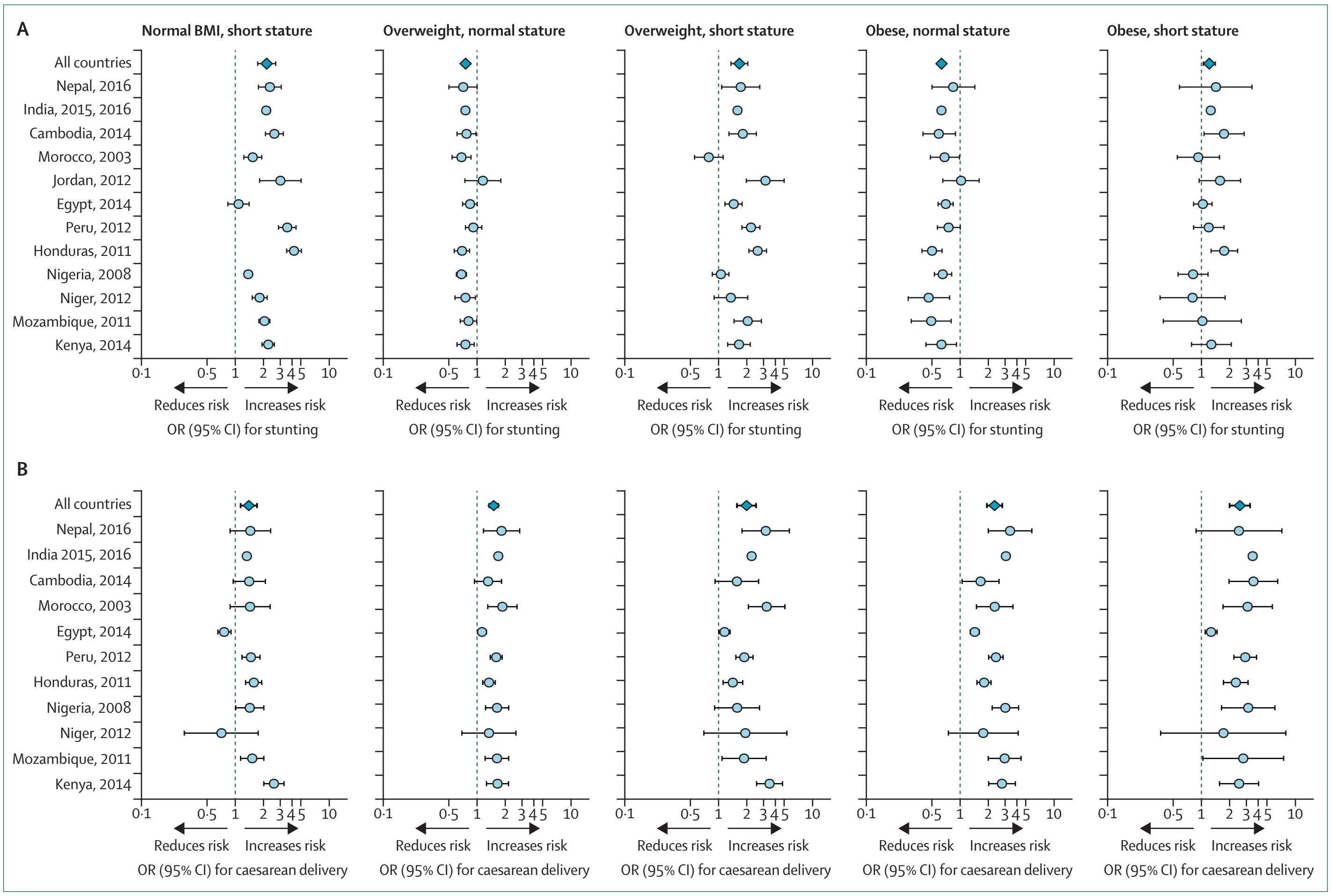

***Figure 2*: Risk of stunting and caesarean delivery according to maternal phenotype in selected LMIC populations relative to mothers of normal height and normal BMI**
(A) Associations between stunting and maternal nutritional phenotype in 12 populations. (B) Associations between caesarean section and maternal nutritional phenotype in 11 populations. Based on Demographic and Health Survey data. All models adjust for wealth, parity, and offspring sex. Full details are given in the appendix (p 21). BMI=body-mass index. LMIC=low-income and middle-income countries. OR=odds ratio.

non-communicable disease was negligibly affected, most probably because these children remained relatively thin and had low metabolic load.[47] The combination of poor early growth and subsequent elevated BMI associated with nutrition transition appears to be the important factor contributing to the risk of adults developing non-communicable diseases.[67]

Figure 3 illustrates how nutrition transition is driving the epidemic of non-communicable disease in Pune, India, combining data from three cohorts at different stages of economic development.[68–73] Following multigenerational exposure to energy scarcity and micronutrient deficiencies, the rural cohort (recruited 1993–96) has shown a secular increase in adult size compared with parents, but 190 (28·7%) of 663 young adults have developed prediabetes, and 15% of 100 young mothers have gestational diabetes. These trends are more extreme in the matched urban cohort of 357 offspring (recruited 1987–89), with 75 (21·0%) developing overweight or obese, 66 (18·5%) developing prediabetes, and 8 (2·2%) developing diabetes by the age of 21 years. Finally among an urban cohort born to mothers with diabetes in pregnancy (recruited 1990–2010), 48 (24%) of 200 children were overweight or obese, 30 (37%) of 81 tested had prediabetes, and 4 (5%) had diabetes at mean age of 15 years.

The increased toxicity of obesity among individuals who were initially undernourished has been shown in diverse populations (appendix pp 27–31).[58,75] Studies in Brazil have revealed some of the physiological mechanisms through which childhood stunting might predispose that individual to central fat deposition and the risk of non-communicable disease (panel 2). Notably, however, a combination of better diet quality and prevention of infections appears to be able to reverse these effects.[39]

Likewise, nutritional supplementation in early life might potentially promote metabolic capacity and thus reduce the risk of non-communicable disease. Supplementation during pregnancy reduces the risk of low

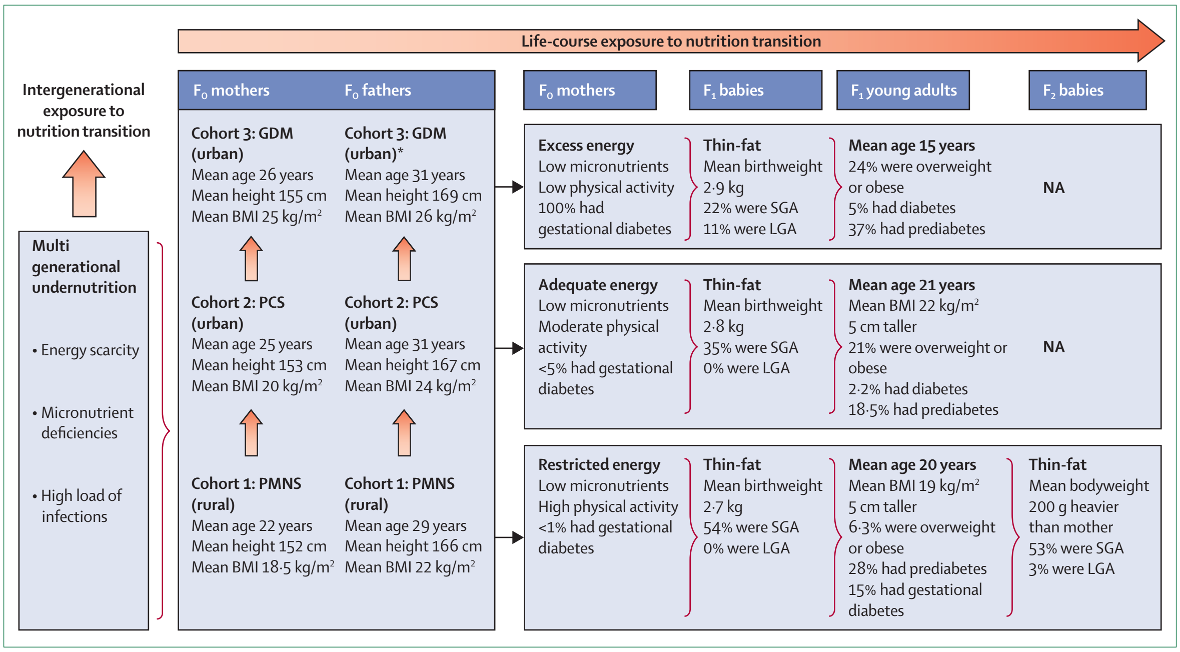

***Figure 3*: Rapid transition and evolution of the double burden of malnutrition in Pune, India, over the past 40 years, based on data from rural and urban cohorts**

Average age in index pregnancy, height, and BMI of mothers and fathers are shown for three cohorts set up in Pune, India in the 1980–90s.[68–73] Rural mothers (F_0 generation, cohort 1: PMNS, recruited 1993–96) show the legacy of multigenerational undernutrition (stunted and underweight, low energy intake and excess physical activity from subsistence farming, multiple micronutrient deficiencies, and low prevalence of gestational diabetes). Parents in the contemporary urban cohort (cohort 2: PCS, recruited 1987–89) were somewhat taller and had a higher BMI. Parents in the diabetic pregnancy cohort (cohort 3: GDM, recruited 1990–2010) were the tallest and heaviest, almost half being overweight or obese (BMI>25 kg/m^2), reflecting the effects of socioeconomic transition. F_1 babies born to cohort 1 mothers had low average weight and a characteristic thin-fat composition (ie, low lean mass but high fat mass compared with European babies). F_1 babies in cohort 2 had somewhat higher birthweight but 35% were still SGA by INTERGROWTH criteria.[74] Babies born to mothers in cohort 3 were the heaviest (11% LGA), but 22% were still SGA. F_1 young adults in cohort 1 were taller and heavier than their parents, although still thin (low BMI) but adipose (high body fat). F_2 babies were 200 g heavier at birth than their mothers' birthweight, highlighting an intergenerational effect of the DBM. In cohort 2, F_1 children were similarly taller and heavier than their parents, with 75 (21%) of 357 being overweight or obese. In cohort 3, these intergenerational effects were more marked, with 48 (24%) of 200 F_1 children being overweight or obese. BMI=body-mass index. DBM=double burden of malnutrition. GDM=gestational diabetes. LGA=large for gestational age. NA=not available. PCS=Pune Children's Study. PMNS=Pune Maternal Nutrition Study. SGA=small for gestational age. *Husbands of women with gestational diabetes.

birthweight[29,76] but propagates few beneficial effects into childhood,[31,77] whereas a community food supplementation programme provided to both pregnant women and their offspring in early childhood improved childhood growth and was associated with greater height and lean mass but not adiposity in early adulthood.[78] However, the longer-term consequences of this intervention for the risk of non-communicable disease were mixed,[79] possibly because the intervention spanned several different developmental stages and might have affected both metabolic capacity and load.

The optimal timing in early life for interventions to prevent the DBM therefore needs further research. We suggest that alongside the preconception period and pregnancy,[23] early infancy might be another crucial window of opportunity.[5] For example, the period of exclusive breastfeeding is simultaneously a developmental period when many mechanisms of plasticity respond to nutritional influences (appendix pp 35–36), an important period for the development of metabolic capacity, and a period when metabolic load can be suppressed.[57] However, the success of breastfeeding is itself threatened by maternal malnutrition (panel 3), indicating that interventions targeting mothers who are breastfeeding might simultaneously improve maternal health while also mitigating the DBM in the next generation.

Although malnutrition damages health in all populations, the manifestation and physiological consequences of the DBM vary. First, the prevalence and consequences of malnutrition often differ between the sexes. Low adult BMI and childhood stunting tend to be slightly more common in male individuals than female individuals in LMICs,[86,87] whereas adult women show higher prevalence than men for obesity and anaemia.[88,89] Moreover, the life-course development of the risk of non-communicable disease also differs by sex.[90]

Second, ethnicity contributes to variability in the health consequences of the DBM. Human morphology and physiology vary with geography, in ways that also change through the life course.[91] For example, south Asian populations have a high prevalence of low birthweight and stunting and relatively short adult stature, all indicative of reduced metabolic capacity, but also a high fat-to-lean ratio and abdominal adiposity, indicative of

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

elevated metabolic load for a given BMI value.[92,93] These traits are strongly implicated in the elevated susceptibility of non-communicable disease in south Asian populations, although whether these traits arise from genetic factors or mechanisms of intergenerational plasticity is unclear. However, these traits are also overlaid by economic and cultural factors, including diet preferences, migration patterns, and social inequality.

Malnutrition and inflammation

The full consequences of the DBM for ill health relate not only to physical phenotype (growth and nutritional status), but also to the local ecological setting. The life-course manifestation of malnutrition varies markedly across ecological settings. In high-income countries, the obesity epidemic developed in the context of low burdens of communicable disease and childhood undernutrition.

Panel 3: The pivotal role of breastfeeding in mitigating the double burden of malnutrition

- Breastfeeding has the potential to reduce the risk of both components of malnutrition in the offspring, and to promote maternal health. First, breastfeeding is strongly protective against diarrhoea and infections in the offspring, and therefore reduces the risk of mortality, stunting, and wasting in early life.[80] Second, breastfeeding constrains excess body-mass index in the offspring during early sensitive periods, and is suggested to protect against later obesity, as well as non-communicable diseases such as diabetes, although it is not associated with all non-communicable disease risk markers.[80] Third, breastfeeding might be considered to mitigate some of the maternal metabolic stresses induced by pregnancy. For example, prolonged breastfeeding is associated with increased maternal insulin sensitivity that persists for at least 2 years after weaning, and reduces long-term risk of diabetes in mothers and the risk of breast cancer.[81] Given these beneficial effects, breastfeeding is an ideal target for interventions, as explored in the third paper of this Series.[5]
- Successful breastfeeding is challenged not only by societal constraints on women's autonomy and employment, but also by both forms of maternal malnutrition. Among mothers who are severely undernourished, low breastmilk volume and micronutrient status might affect growth and micronutrient status of the offspring.[82] Poor maternal diet reduces the diversity of the maternal microbiome, which is then transmitted to the offspring and is associated with increased risk of severe malnutrition. Human milk oligosaccharides, unique to our species, play a key role in the establishment of a healthy gut microbiome. Animal studies have shown that promoting specific types of oligosaccharides increases lean tissue accretion in early life.[83]
- At the other extreme, maternal obesity is associated with lower likelihood of breastfeeding and reduced duration of any or exclusive breastfeeding. At the level of physiology, glucose intolerance during pregnancy might impede milk synthesis and contribute to delayed lactogenesis, and excessive gestational weight gain is associated with raised inflammatory markers in breastmilk.[84] Studies of women with diabetes show that a longer duration of breastfeeding than in women without diabetes is necessary to achieve the beneficial protective effects against childhood obesity (appendix pp 37–38).[85]
- Both social support and metabolic health of the mother are therefore crucial to maximising the success of breastfeeding and capturing the health benefits to both mother and offspring. Maternal dietary intake, non-communicable disease status, and the composition of the maternal microbiome are all potential targets for interventions, but further work is required to better understand the mechanisms and to develop effective solutions.

In LMICs, however, both extremes of malnutrition coexist with persistent burdens of infections. Both undernutrition and overweight are associated with inflammation,[94,95] effectively generating a triple challenge to metabolic health with major implications for the risk of non-communicable disease.

Poor nutrition in early life (fetal growth faltering, postnatal stunting, suboptimal breastfeeding) has been associated with elevated markers of inflammation in childhood and young adulthood, and obesity is also a chronic inflammatory condition.[94,96–98] Although research from LMICs is scarce, studies from high-income countries indicate that the inflammatory load of obesity might be exacerbated by undernutrition in early life.[99,100]

An unfavourable gut microbiome might contribute to these associations (appendix pp 6–9). Microbiota immaturity, increased enteropathogen burden, and gut barrier dysfunction are inter-related factors associated with inflammation in early life.[101] The microbiota of Indian children with stunting was depleted in probiotic species and enriched in inflammogenic taxa, relative to controls.[102] In adulthood, dysbiosis contributes to associations of obesity with insulin resistance and systemic inflammation.[103,104] Among adults who are obese with similar BMI, those with greater dysbiosis have a higher risk of non-communicable disease,[105] and dysbiosis also contributes to the inflammatory process associated with sarcopenia.[106] Thus, in LMICs the nutrition transition could exacerbate the inflammatory burden of malnutrition.

Manipulation of the microbiome—eg, by providing probiotics or faecal transplantation—might beneficially modify markers for the risk of non-communicable disease (appendix pp 6–9), but further research is needed to understand how this treatment could achieve long-lasting effects mitigating both forms of malnutrition.

The DBM and childbirth complications

Although much emphasis has been placed on the implications of the DBM for the risk of non-communicable disease, both short stature and overweight are also independent risk factors for obstructed labour, related to maternal mortality.[107] The DBM might therefore affect health outcomes of mothers and offspring related to childbirth. For women who are short, the primary underlying mechanism is likely to be reduced pelvic dimensions, whereas for women who are overweight a key mechanism is perturbed fuel metabolism, increasing birthweight.[107] Globally, many mothers who were stunted in early life become overweight before reproducing, and women who are overweight are also more likely to develop gestational diabetes if previously stunted.[107] The combined effect is predicted to increase the risk of obstructed labour.

We analysed Demographic and Health Surveys from 11 LMICs. Both maternal stunting and overweight each increase risk of caesarean delivery (a correlate of obstructed labour); however, the risk tends to be further amplified among women who are stunted and overweight or obese (figure 2B). Although increases in the number of caesareans have been linked with indices of wealth, the financial incentives of health-care providers, and defensive medicine to minimise litigation,[108,109] the emerging DBM might be an additional factor.[3]

Given the profound health consequences of the DBM in individuals, we need to understand what is driving the global epidemic. We show in the next section that biological susceptibility interacts with societal factors and provide an evolutionary perspective to help understand how we might combat the DBM most effectively.

Societal driving factors

The biological interlinkages between undernutrition and overweight that we have described can only be fully understood in the context of broader societal drivers, which mediate differential exposure to the causes of malnutrition. The global DBM is closely associated with rapid economic development and increased income per capita,[2] but also incorporates a constellation of societal trends acting across culture, behaviour, and technology.[4] Many of the individuals most exposed to these trends are not the wealthiest and are the least empowered to resist adverse societal and corporate influences.[5,57] The metabolic consequences of unhealthy diets that are both energy dense and micronutrient poor[110] are exacerbated by increases in sedentary behaviour,[111] and exposure to psychosocial stress has been associated with unhealthy food choices and eating patterns, and with perturbed appetite, weight gain, and central adiposity.[112,113]

Many factors reduce individual agency. For example, at a societal level, gender inequality exacerbates female exposure to the DBM.[114,115] From a political perspective, governments struggle to restrain commercial activities in the interests of population health, but might also contribute to the DBM through the promotion of international trade and national economic development, and the opening of domestic markets to multinational corporations. Policy makers are increasingly exploring strategies to reduce malnutrition that place less emphasis on economic growth, by addressing issues such as food sovereignty, gender equality, education, and healthy food systems.[114,116–118] Nevertheless, the emerging DBM is a stark indication of how a large proportion of the global population, especially in LMICs, is poorly protected from multiple factors driving malnutrition in all its forms.

Groups at high risk

Although the DBM affects many countries,[2,4] various groups are at high risk of both undernutrition and overweight and merit attention. This susceptibility relates to diverse factors spanning both biology and environmental stressors.

In high-income countries such as Canada, Australia, and the UK, for example, First Nation, Indigenous, and ethnic minority populations, respectively, typically show higher levels of low birthweight and childhood undernutrition than do the general population, but also increased risk of obesity and non-communicable diseases in later life (appendix pp 39–42). Similarly, African Americans show persisting deficits in birthweight relative to people of European ancestry, additionally, Hispanic and African Americans have greater prevalence of adult obesity than Americans of European descent.[119] Similar patterns increasingly apply to minority groups within LMICs, such as tribal populations in India.[120]

To track such population-specific susceptibility, ethnic differences in physique and metabolism should be addressed. In the UK, for example, adjusting for ethnic differences in the relationship between BMI and adiposity reveals that obesity is most prevalent and increasing fastest in children of south Asian ethnicity compared with other ethnic groups.[121] Moreover, body fat is more toxic in children of south Asian descent than in those of European descent, since it has a stronger association with insulin resistance.[122] These patterns help explain why some ethnic groups show high susceptibility to non-communicable diseases in early adulthood, even at relatively low BMI thresholds.

As noted in the first paper of this Series,[2] within LMICs, rural to urban migration exposes increasing numbers of people to drastic changes in diet, physical activity, and living conditions. Rural populations have a high prevalence of childhood stunting,[123] whereas migration to cities is typically associated with rapid increases in BMI and abdominal fat. This adiposity elevates the risk of non-communicable disease in comparison with the rural population, but typically to lower levels than in established urban populations.[124] These health consequences might increase through lengthier urban residence and at older ages; however, research on malnutrition in older people (aged >65 years) in LMICs remains very scarce.[125,126]

Adolescents are another particularly important group because of the imminence of reproduction. Surveys indicate high prevalences of underweight, overweight, and anaemia in adolescents aged 10–19 years, although they vary by country.[127,128] Adolescents are also among the first groups to adopt new diets and lifestyles, in part through their tendency to migrate in search of new economic opportunities. The combination of overweight and anaemia in adolescent women is difficult to address, because obesity-mediated inflammation might impede iron absorption and reduce the efficacy of supplementation programmes.[127] Finally, infants are susceptible to complementary foods that are fattening and also deficient in micronutrients.[129]

An evolutionary perspective

The profound health risks associated with life-course exposure to the DBM might seem puzzling. First, why does nutrition transition not resolve the effects of multigenerational undernutrition? Why do children with stunting often remain short in adulthood and become overweight, rather than growing tall and remaining lean? Second, why is the combination of early stunting and later overweight so detrimental to health? An evolutionary perspective might help explain why different forms of malnutrition interact and shape the risk of non-communicable disease. Evolutionary life-history theory assumes that every organism allocates energy between four competing functions: maintenance, growth, reproduction, and defence, resulting in trade-offs between these functions.[130] The optimal allocation strategy for maximising reproductive fitness is expected to vary in association with developmental trajectory and ecological conditions.[131]

A key factor influencing these allocation decisions is extrinsic mortality risk. In high-risk environments, selection favours discounting the future, diverting energy away from maintenance and growth towards defence (short-term survival) and reproduction. This insight helps understand the combination of high fertility and lower birthweights in chronically undernourished populations with high infectious burdens: fitness is maximised by producing more offspring, but investing less in each. Suboptimal fetal nutrition not only constrains the development of metabolic capacity, but also is associated with long-term central adiposity and inflammation, promoting immune function through the life course.[57,98] Economic development increases dietary energy availability and alters life history strategy; however, the nature of this change depends on both extrinsic mortality risk and diet composition. In high-pathogen and food-insecure environments, if energy supply increases during childhood it is too late to allocate this energy to maintenance because the physiological critical window has already closed. Instead, the surplus is primarily diverted to survival (pro-inflammatory state, energy stores) and reproduction (gaining weight during adolescence). This strategy helps to explain why individuals who were initially undernourished do not entirely resolve their growth deficit, and are prone to central adiposity and elevated inflammation.[99,100,132] Reduced fertility among women who are obese[133] suggests that these trade-offs evolved in ancestral environments characterised by energy scarcity, and in contemporary settings they might be exacerbated through exposure to processed foods high in energy but low in protein and micronutrients. Inflammation disrupts many components of homoeostasis relevant to the risk of non-communicable disease, such as appetite, sleep, insulin metabolism, arterial health, and oxidative balance.[134] The result is a high metabolic load superimposed on a depleted capacity, provoking non-communicable diseases at relatively low thresholds of age and overweight.

In food-secure and low-pathogen settings, conversely, lower infant mortality means that mothers can maximise fitness by producing fewer offspring, and investing more in each during early life. This strategy allows each offspring to divert more energy to maintenance and early growth, promoting life-long homoeostasis and health, and probably a lengthier reproductive period.

This perspective helps explain why in high-income countries with long-term efforts to reduce infectious disease, economic development has induced prolonged secular increases in height and lifespan,[135] whereas in many LMICs where poor quality diets and high infectious burdens persist, economic development is more strongly associated with trends in BMI,[88] and an escalating burden of non-communicable disease. Secular trends in stature in LMICs remain weak;[136,137] instead, a secular decline in menarchal age has occurred, especially in urban settings (appendix pp 43–44).[57] The mechanisms of developmental plasticity described in panel 1 might play a key role in orchestrating such life history trade-offs in association with ecological conditions.

Trade-offs between biological functions might prove especially rewarding targets for interventions aiming to reduce malnutrition and associated adverse health effects. Whether efforts to reduce the risk of non-communicable disease in adults in LMICs are successful might be contingent on reducing both early undernutrition and the burden of infection. This proposition is supported by evidence that, independent of nutritional supply, childhood vaccination benefits linear growth.[138]

Conclusion

Examining the DBM from the perspective of individual health is very different from approaching it at the population level. Beyond the common driving factors,[4] undernutrition and overweight show multiple physiological connections and interactions. As LMICs undergo economic development and nutrition transition, the resulting DBM is exposing growing numbers of individuals to various forms of ill health, including growth retardation, dysbiosis, inflammation, obesity, non-communicable diseases, and childbirth complications. Recognising these interconnections might reveal new, shared opportunities to improve metabolic health.

The evolutionary perspective helps explain why the DBM is so harmful to health. Exposure to malnutrition during early critical periods results in the body reducing its valuation of the future, diverting energy from growth and health to survival and potential reproduction. If the only substantial environmental change through the life course is increased dietary energy, these trade-offs might simply intensify. This scenario could explain why some programmes aiming to prevent undernutrition have inadvertently increased obesity and the risk of non-communicable disease.[79] High-energy diets that are low in protein, fibre, and micronutrients might drive over-consumption of fat and carbohydrate.[139] High burdens of infectious disease also constrain linear growth and favour inflammation, which is further exacerbated by overweight.

The programme of treatment for childhood malnutrition in Brazil (panel 2) highlights where efforts should be directed to break this cycle.[39] The programme improves dietary quality while also cutting the burden of infection. This composite improvement makes the long-term future more attainable, and the body responds by promoting linear growth rather than adiposity, while restoring metabolism to lower the risk of non-communicable disease.

To be effective, the double-duty actions proposed in the third paper in this Series[5] should achieve two goals. First, the actions need to affect each generation early in life, so that the trajectory of development can be shifted beneficially through the physiological mechanisms listed in panel 1. This approach highlights the importance of optimising nutrition among adolescents, whose metabolism constitutes the developmental niche for the next generation. Secondly, interventions need to be sufficiently comprehensive to affect the functional trade-offs that we have outlined. Successful interventions might need to begin before conception and continue through pregnancy and lactation. A cautionary note is that these interventions still need to balance health benefits and costs. For example, research is required to optimise the balance between promoting fetal growth and maintaining maternal metabolic health during pregnancy. Such interventions should be supported by the sustained provision of healthy complementary foods, and effective reductions in the burden of infection during childhood.

No single intervention can solve the DBM, and efforts must also be sustained over decades to realise their full benefits. Even if stunting is reduced, adults already overweight will bear additional health consequences if they were undernourished during development. On the positive side, effective double-duty actions[5,6] might benefit health across the lifespan and into the next generation. To achieve these goals, major societal shifts are required regarding nutrition and public health. Ultimately, the global DBM reflects many adverse trends through which individuals are disempowered and their nutritional status and health undermined. The fourth paper in this Series[6] shows that specific interventions to reduce the DBM can be cost-effective, but substantial progress requires that this approach be scaled up into the entire global food system, while also meeting the need for human food systems to maintain planetary health.[140]

Contributors

This paper was conceptualised by AD, JCW, and CSY, and its development steered by JCW, CSY, ALS, and AD. Literature reviews were done by JCW, RW, and MM. Data extraction and coding was done by MSP. Statistical analysis was done by RW and JCW. Summaries of research in India and Brazil were prepared by CSY and ALS, respectively. The conceptual diagram was developed by JCW and RW. JCW wrote the first draft of the manuscript, and all authors contributed to revising it and approved the final version.

Declaration of interests

We declare no competing interests.

Acknowledgments

Funding for the preparation of the Series was provided by WHO, through a grant from the Bill & Melinda Gates Foundation. The funder had no role in the analysis and interpretation of the evidence or in writing the paper and the decision to submit for publication.

References

1 Doak CM, Adair LS, Bentley M, Monteiro C, Popkin BM. The dual burden household and the nutrition transition paradox. *IntJ Obes (Lond)* 2005; **29:** 129–36.
2 Popkin BM, Corvalan C, Grummer-Strawn LM. Dynamics of the double burden of malnutrition and the changing nutrition reality. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32497-3.
3 Wells JCK, Wibaek R, Poullas M. The dual burden of malnutrition increases the risk of cesarean delivery: evidence from India. *Front Public Health* 2018; **6:** 292.
4 Swinburn BA, Kraak VI, Allender S, et al. The global syndemic of obesity, undernutrition, and climate change: the *Lancet* Commission report. *Lancet* 2019; **393:** 791–846.
5 Hawkes C, Ruel MT, Salm L, Sinclair B, Branca F. Double-duty actions: seizing programme and policy opportunities to address malnutrition in all its forms. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32506-1.
6 Nugent R, Levin C, Hale J, Hutchinson B. Economic effects of the double burden of malnutrition. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32473-0.
7 Harika R, Faber M, Samuel F, Kimiywe J, Mulugeta A, Eilander A. Micronutrient status and dietary intake of iron, vitamin A, iodine, folate and zinc in women of reproductive age and pregnant women in Ethiopia, Kenya, Nigeria and South Africa: a systematic review of data from 2005 to 2015. *Nutrients* 2017; **9:** e1096.
8 Kuper H, Taylor A, Krishna KV, et al. Is vulnerability to cardiometabolic disease in Indians mediated by abdominal adiposity or higher body adiposity. *BMC Public Health* 2014; **14:** 1239.
9 WHO Expert Consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* 2004; **363:** 157–63.
10 Sommer F, Anderson JM, Bharti R, Raes J, Rosenstiel P. The resilience of the intestinal microbiota influences health and disease. *Nat Rev Microbiol* 2017; **15:** 630–38.
11 Khoruts A. Targeting the microbiome: from probiotics to fecal microbiota transplantation. *Genome Med* 2018; **10:** 80.
12 Winick M, Noble A. Cellular response in rats during malnutrition at various ages. *J Nutr* 1966; **89:** 300–06.
13 Crispi F, Miranda J, Gratacos E. Long-term cardiovascular consequences of fetal growth restriction: biology, clinical implications, and opportunities for prevention of adult disease. *Am J Obstet Gynecol* 2018; **218:** S869–79.
14 Latini G, De Mitri B, Del Vecchio A, Chitano G, De Felice C, Zetterstrom R. Foetal growth of kidneys, liver and spleen in intrauterine growth restriction: "programming" causing "metabolic syndrome" in adult age. *Acta Paediatr* 2004; **93:** 1635–39.
15 Martin-Gronert MS, Ozanne SE. Metabolic programming of insulin action and secretion. *Diabetes Obes Metab* 2012; **14** (suppl 3)**:** 29–39.
16 Heijmans BT, Tobi EW, Stein AD, et al. Persistent epigenetic differences associated with prenatal exposure to famine in humans. *Proc Natl Acad Sci USA* 2008; **105:** 17046–49.
17 Dominguez-Salas P, Moore SE, Baker MS, et al. Maternal nutrition at conception modulates DNA methylation of human metastable epialleles. *Nat Commun* 2014; **5:** 3746.
18 de Zegher F, Diaz M, Lopez-Bermejo A, Ibanez L. Recognition of a sequence: more growth before birth, longer telomeres at birth, more lean mass after birth. *Pediatr Obes* 2017; **12:** 274–79.
19 Wojcicki JM, Heyman MB, Elwan D, Lin J, Blackburn E, Epel E. Early exclusive breastfeeding is associated with longer telomeres in Latino preschool children. *Am J Clin Nutr* 2016; **104:** 397–405.
20 Smith MI, Yatsunenko T, Manary MJ, et al. Gut microbiomes of Malawian twin pairs discordant for kwashiorkor. *Science* 2013; **339:** 548–54.
21 Victora CG, Adair L, Fall C, et al. Maternal and child undernutrition: consequences for adult health and human capital. *Lancet* 2008; **371:** 340–57.
22 Adair LS, Fall CH, Osmond C, et al. Associations of linear growth and relative weight gain during early life with adult health and human capital in countries of low and middle income: findings from five birth cohort studies. *Lancet* 2013; **382:** 525–34.

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

23 Fleming TP, Watkins AJ, Velazquez MA, et al. Origins of lifetime health around the time of conception: causes and consequences. *Lancet* 2018; **391:** 1842–52.

24 Mei Z, Grummer-Strawn LM, Thompson D, Dietz WH. Shifts in percentiles of growth during early childhood: analysis of longitudinal data from the California Child Health and Development Study. *Pediatrics* 2004; **113:** e617–27.

25 Wells JC. Maternal capital and the metabolic ghetto: an evolutionary perspective on the transgenerational basis of health inequalities. *Am J Hum Biol* 2010; **22:** 1–17.

26 Yajnik CS. Nutrient-mediated teratogenesis and fuel-mediated teratogenesis: two pathways of intrauterine programming of diabetes. *Int J Gynaecol Obstet* 2009; **104** (suppl 1): S27–31.

27 Victora CG, de Onis M, Hallal PC, Blossner M, Shrimpton R. Worldwide timing of growth faltering: revisiting implications for interventions. *Pediatrics* 2010; **125:** e473–80.

28 Schoenbuchner SM, Dolan C, Mwangome M, et al. The relationship between wasting and stunting: a retrospective cohort analysis of longitudinal data in Gambian children from 1976–2016. *Am J Clin Nutr* 2019; **110:** 498–507.

29 Ceesay SM, Prentice AM, Cole TJ, et al. Effects on birth weight and perinatal mortality of maternal dietary supplements in rural Gambia: 5 year randomised controlled trial. *BMJ* 1997; **315:** 786–90.

30 Kramer MS, Kakuma R. Energy and protein intake in pregnancy. *Cochrane Database Syst Rev* 2003; **4:** CD000032.

31 Devakumar D, Fall CH, Sachdev HS, et al. Maternal antenatal multiple micronutrient supplementation for long-term health benefits in children: a systematic review and meta-analysis. *BMC Med* 2016; **14:** 90.

32 Barquera S, Peterson KE, Must A, et al. Coexistence of maternal central adiposity and child stunting in Mexico. *Int J Obes (Lond)* 2007; **31:** 601–07.

33 Hemachandra AH, Klebanoff MA. Use of serial ultrasound to identify periods of fetal growth restriction in relation to neonatal anthropometry. *Am J Hum Biol* 2006; **18:** 791–97.

34 Ong KK, Ahmed ML, Emmett PM, Preece MA, Dunger DB. Association between postnatal catch-up growth and obesity in childhood: prospective cohort study. *BMJ* 2000; **320:** 967–71.

35 Baird J, Fisher D, Lucas P, Kleijnen J, Roberts H, Law C. Being big or growing fast: systematic review of size and growth in infancy and later obesity. *BMJ* 2005; **331:** 929.

36 Kuzawa CW, Hallal PC, Adair L, et al. Birth weight, postnatal weight gain, and adult body composition in five low and middle income countries. *Am J Hum Biol* 2012; **24:** 5–13.

37 Dulloo AG. Regulation of body composition during weight recovery: integrating the control of energy partitioning and thermogenesis. *ClinNutr* 1997; **16** (suppl 1): 25–35.

38 Hales CN, Barker DJ. The thrifty phenotype hypothesis. *Br Med Bull* 2001; **60:** 5–20.

39 Martins VJ, de Albuquerque MP, Sawaya AL. Endocrine changes in undernutrition, metabolic programming, and nutritional recovery. In: Preedy VR, Patel VB, eds. Handbook of famine, starvation, and nutritional deprivation. Switzerland: Springer International Publishing AG 2017: 1–21.

40 Florencio TT, Ferreira HS, Cavalcante JC, Stux GR, Sawaya AL. Short stature, abdominal obesity, insulin resistance and alterations in lipid profile in very low-income women living in Maceio, north-eastern Brazil. *Eur J Cardiovasc Prev Rehabil* 2007; **14:** 346–48.

41 Hoffman DJ, Sawaya AL, Verreschi I, Tucker KL, Roberts SB. Why are nutritionally stunted children at increased risk of obesity? Studies of metabolic rate and fat oxidation in shantytown children from Sao Paulo, Brazil. *Am J Clin Nutr* 2000; **72:** 702–07.

42 Said-Mohamed R, Bernard JY, Ndzana AC, Pasquet P. Is overweight in stunted preschool children in Cameroon related to reductions in fat oxidation, resting energy expenditure and physical activity? *PLoS One* 2012; **7:** e39007.

43 Pomeroy E, Stock JT, Stanojevic S, Miranda JJ, Cole TJ, Wells JC. Stunting, adiposity, and the individual-level "dual burden" among urban lowland and rural highland Peruvian children. *Am J Hum Biol* 2014; **26:** 481–90.

44 Fabiansen C, Yameogo CW, Iuel-Brockdorf AS, et al. Effectiveness of food supplements in increasing fat-free tissue accretion in children with moderate acute malnutrition: a randomised 2×2×3 factorial trial in Burkina Faso. *PLoS Med* 2017; **14:** e1002387.

45 Wells JC, Chomtho S, Fewtrell MS. Programming of body composition by early growth and nutrition. *Proc Nutr Soc* 2007; **66:** 423–34.

46 Dodds R, Denison HJ, Ntani G, et al. Birth weight and muscle strength: a systematic review and meta-analysis. *J Nutr Health Aging* 2012; **16:** 609–15.

47 Lelijveld N, Seal A, Wells JC, et al. Chronic disease outcomes after severe acute malnutrition in Malawian children (ChroSAM): a cohort study. *Lancet Glob Health* 2016; **4:** e654–62.

48 von Dadelszen P, Magee LA. Fall in mean arterial pressure and fetal growth restriction in pregnancy hypertension: an updated metaregression analysis. *J Obstet Gynaecol Can* 2002; **24:** 941–45.

49 Garcia OP, Long KZ, Rosado JL. Impact of micronutrient deficiencies on obesity. *Nutr Rev* 2009; **67:** 559–72.

50 Galley JD, Bailey M, Kamp Dush C, Schoppe-Sullivan S, Christian LM. Maternal obesity is associated with alterations in the gut microbiome in toddlers. *PLoS One* 2014; **9:** e113026.

51 Cnattingius S, Villamor E, Lagerros YT, Wikstrom AK, Granath F. High birth weight and obesity–a vicious circle across generations. *Int J Obes (Lond)* 2012; **36:** 1320–24.

52 Carslake D, Pinger PR, Romundstad P, Davey Smith G. Early-onset paternal smoking and offspring adiposity: further investigation of a potential intergenerational effect using the HUNT study. *PLoS One* 2016; **11:** e0166952.

53 Isganaitis E, Suehiro H, Cardona C. Who's your daddy?: paternal inheritance of metabolic disease risk. *Curr Opin Endocrinol Diabetes Obes* 2017; **24:** 47–55.

54 Rachmi CN, Agho KE, Li M, Baur LA. Stunting, underweight and overweight in children aged 2·0–4·9 years in Indonesia: prevalence trends and associated risk factors. *PLoS One* 2016; **11:** e0154756.

55 Global Burden of Metabolic Risk Factors for Chronic Diseases Collaboration. Cardiovascular disease, chronic kidney disease, and diabetes mortality burden of cardiometabolic risk factors from 1980 to 2010: a comparative risk assessment. *Lancet Diabetes Endocrinol* 2014; **2:** 634–47.

56 Wells JC. The thrifty phenotype: an adaptation in growth or metabolism? *Am J Hum Biol* 2011; **23:** 65–75.

57 Wells JC. The metabolic ghetto: an evolutionary perspective on nutrition, power relations and chronic disease. Cambridge: Cambridge University Press, 2016.

58 Li Y, Ley SH, Tobias DK, et al. Birth weight and later life adherence to unhealthy lifestyles in predicting type 2 diabetes: prospective cohort study. *BMJ* 2015; **351:** h3672.

59 Li Y, Ley SH, VanderWeele TJ, et al. Joint association between birth weight at term and later life adherence to a healthy lifestyle with risk of hypertension: a prospective cohort study. *BMC Med* 2015; **13:** 175.

60 Mitanchez D, Chavatte-Palmer P. Review shows that maternal obesity induces serious adverse neonatal effects and is associated with childhood obesity in their offspring. *Acta Paediatr* 2018; **107:** 1156–65.

61 Diaz M, Garcia C, Sebastiani G, de Zegher F, Lopez-Bermejo A, Ibanez L. Placental and cord blood methylation of genes involved in energy homeostasis: association with fetal growth and neonatal body composition. *Diabetes* 2017; **66:** 779–84.

62 El Hajj N, Schneider E, Lehnen H, Haaf T. Epigenetics and life-long consequences of an adverse nutritional and diabetic intrauterine environment. *Reproduction* 2014; **148:** R111–20.

63 Tan Q, Frost M, Heijmans BT, et al. Epigenetic signature of birth weight discordance in adult twins. *BMC Genomics* 2014; **15:** 1062.

64 Metcalfe NB, Monaghan P. Compensation for a bad start: grow now, pay later? *Trends Ecol Evol* 2001; **16:** 254–60.

65 Yang TO, Reeves GK, Green J, et al. Birth weight and adult cancer incidence: large prospective study and meta-analysis. *Ann Oncol* 2014; **25:** 1836–43.

66 Green J, Cairns BJ, Casabonne D, et al. Height and cancer incidence in the Million Women Study: prospective cohort, and meta-analysis of prospective studies of height and total cancer risk. *Lancet Oncol* 2011; **12:** 785–94.

67 Bhargava SK, Sachdev HS, Fall CH, et al. Relation of serial changes in childhood body-mass index to impaired glucose tolerance in young adulthood. *N Engl J Med* 2004; **350:** 865–75.

68 Rao S, Yajnik CS, Kanade A, et al. Intake of micronutrient-rich foods in rural Indian mothers is associated with the size of their babies at birth: Pune Maternal Nutrition Study. *J Nutr* 2001; **131:** 1217–24.

69 Yajnik CS, Fall CH, Coyaji KJ, et al. Neonatal anthropometry: the thin-fat Indian baby. The Pune Maternal Nutrition Study. *Int J Obes Relat Metab Disord* 2003; **27:** 173–80.

70 Bavdekar A, Yajnik CS, Fall CH, et al. Insulin resistance syndrome in 8-year-old Indian children: small at birth, big at 8 years, or both? *Diabetes* 1999; **48:** 2422–29.

71 Yajnik CS, Katre PA, Joshi SM, et al. Higher glucose, insulin and insulin resistance (HOMA-IR) in childhood predict adverse cardiovascular risk in early adulthood: the Pune Children's Study. *Diabetologia* 2015; **58:** 1626–36.

72 Kale SD, Kulkarni SR, Lubree HG, et al. Characteristics of gestational diabetic mothers and their babies in an Indian diabetes clinic. *J Assoc Physicians India* 2005; **53:** 857–63.

73 Kale SD, Yajnik CS, Kulkarni SR, et al. High risk of diabetes and metabolic syndrome in Indian women with gestational diabetes mellitus. *Diabet Med* 2004; **21:** 1257–58.

74 Villar J, Cheikh Ismail L, Victora CG, et al. International standards for newborn weight, length, and head circumference by gestational age and sex: the Newborn Cross-Sectional Study of the INTERGROWTH-21st Project. *Lancet* 2014; **384:** 857–68.

75 Leon DA, Koupilova I, Lithell HO, et al. Failure to realise growth potential in utero and adult obesity in relation to blood pressure in 50 year old Swedish men. *BMJ* 1996; **312:** 401–06.

76 Smith ER, Shankar AH, Wu LS, et al. Modifiers of the effect of maternal multiple micronutrient supplementation on stillbirth, birth outcomes, and infant mortality: a meta-analysis of individual patient data from 17 randomised trials in low-income and middle-income countries. *Lancet Glob Health* 2017; **5:** e1090–100.

77 Hawkesworth S, Walker CG, Sawo Y, et al. Nutritional supplementation during pregnancy and offspring cardiovascular disease risk in The Gambia. *Am J Clin Nutr* 2011; **94** (suppl): 1853–60S.

78 Stein AD, Wang M, Ramirez-Zea M, et al. Exposure to a nutrition supplementation intervention in early childhood and risk factors for cardiovascular disease in adulthood: evidence from Guatemala. *Am J Epidemiol* 2006; **164:** 1160–70.

79 Ford ND, Behrman JR, Hoddinott JF, et al. Exposure to improved nutrition from conception to age 2 years and adult cardiometabolic disease risk: a modelling study. *Lancet Glob Health* 2018; **6:** e875–84.

80 Victora CG, Bahl R, Barros AJ, et al. Breastfeeding in the 21st century: epidemiology, mechanisms, and lifelong effect. *Lancet* 2016; **387:** 475–90.

81 Bajaj H, Ye C, Hanley AJ, et al. Prior lactation reduces future diabetic risk through sustained postweaning effects on insulin sensitivity. *Am Journal Physiol Endocrinol Metab* 2017; **312:** e215–23.

82 Samuel TM, Thomas T, Thankachan P, Bhat S, Virtanen SM, Kurpad AV. Breast milk zinc transfer and early post-natal growth among urban South Indian term infants using measures of breast milk volume and breast milk zinc concentrations. *Matern Child Nutr* 2014; **10:** 398–409.

83 Charbonneau MR, O'Donnell D, Blanton LV, et al. Sialylated milk oligosaccharides promote microbiota-dependent growth in models of infant undernutrition. *Cell* 2016; **164:** 859–71.

84 Whitaker KM, Marino RC, Haapala JL, et al. Associations of maternal weight status before, during, and after pregnancy with inflammatory markers in breast milk. *Obesity (Silver Spring)* 2017; **25:** 2092–99.

85 Dugas C, Perron J, Kearney M, et al. Postnatal prevention of childhood obesity in offspring prenatally exposed to gestational diabetes mellitus: where are we now? *Obes Facts* 2017; **10:** 396–406.

86 NCD Risk Factor Collaboration (NCD-RisC). Worldwide trends in body-mass index, underweight, overweight, and obesity from 1975 to 2016: a pooled analysis of 2416 population-based measurement studies in 128·9 million children, adolescents, and adults. *Lancet* 2017; **390:** 2627–42.

87 Wamani H, Astrom AN, Peterson S, Tumwine JK, Tylleskar T. Boys are more stunted than girls in sub-Saharan Africa: a meta-analysis of 16 demographic and health surveys. *BMC Pediatr* 2007; **7:** 17.

88 NCD Risk Factor Collaboration (NCD-RisC). Trends in adult body-mass index in 200 countries from 1975 to 2014: a pooled analysis of 1698 population-based measurement studies with 19·2 million participants. *Lancet* 2016; **387:** 1377–96.

89 Traissac P, El Ati J, Gartner A, Ben Gharbia H, Delpeuch F. Gender inequalities in excess adiposity and anaemia combine in a large double burden of malnutrition gap detrimental to women in an urban area in North Africa. *Public Health Nutr* 2016; **19:** 1428–37.

90 Intapad S, Ojeda NB, Dasinger JH, Alexander BT. Sex differences in the developmental origins of cardiovascular disease. *Physiology (Bethesda)* 2014; **29:** 122–32.

91 Wells JC. Ethnic variability in adiposity, thrifty phenotypes and cardiometabolic risk: addressing the full range of ethnicity, including those of mixed ethnicity. *Obes Rev* 2012; **13** (suppl 2): 14–29.

92 D'Angelo S, Yajnik CS, Kumaran K, et al. Body size and body composition: a comparison of children in India and the UK through infancy and early childhood. *J Epidemiol Community Health* 2015; **69:** 1147–53.

93 Chandalia M, Lin P, Seenivasan T, et al. Insulin resistance and body fat distribution in South Asian men compared to Caucasian men. *PLoS One* 2007; **2:** e812.

94 Prendergast AJ, Rukobo S, Chasekwa B, et al. Stunting is characterized by chronic inflammation in Zimbabwean infants. *PLoS One* 2014; **9:** e86928.

95 Choi J, Joseph L, Pilote L. Obesity and C-reactive protein in various populations: a systematic review and meta-analysis. *Obes Rev* 2013; **14:** 232–44.

96 Visser M, Bouter LM, McQuillan GM, Wener MH, Harris TB. Elevated C-reactive protein levels in overweight and obese adults. *JAMA* 1999; **282:** 2131–35.

97 Merrill RD, Burke RM, Northrop-Clewes CA, et al. Factors associated with inflammation in preschool children and women of reproductive age: Biomarkers Reflecting Inflammation and Nutritional Determinants of Anemia (BRINDA) project. *Am J Clin Nutr* 2017; **106** (suppl 1): 348–58S.

98 McDade TW, Ryan C, Jones MJ, et al. Social and physical environments early in development predict DNA methylation of inflammatory genes in young adulthood. *Proc Natl Acad Sci USA* 2017; **114:** 7611–16.

99 Pedersen JM, Budtz-Jorgensen E, Mortensen EL, et al. Late midlife C-reactive protein and interleukin-6 in middle aged Danish men in relation to body size history within and across generations. *Obesity (Silver Spring)* 2016; **24:** 461–68.

100 Tzoulaki I, Jarvelin MR, Hartikainen AL, et al. Size at birth, weight gain over the life course, and low-grade inflammation in young adulthood: northern Finland 1966 Birth Cohort study. *Eur Heart J* 2008; **29:** 1049–56.

101 Blanton LV, Barratt MJ, Charbonneau MR, Ahmed T, Gordon JI. Childhood undernutrition, the gut microbiota, and microbiota-directed therapeutics. *Science* 2016; **352:** 1533.

102 Dinh DM, Ramadass B, Kattula D, et al. Longitudinal analysis of the intestinal microbiota in persistently stunted young children in south India. *PLoS One* 2016; **11:** e0155405.

103 Le Chatelier E, Nielsen T, Qin J, et al. Richness of human gut microbiome correlates with metabolic markers. *Nature* 2013; **500:** 541–46.

104 Neuman H, Debelius JW, Knight R, Koren O. Microbial endocrinology: the interplay between the microbiota and the endocrine system. *FEMS Microbiol Rev* 2015; **39:** 509–21.

105 de la Cuesta-Zuluaga J, Corrales-Agudelo V, Carmona JA, Abad JM, Escobar JS. Body size phenotypes comprehensively assess cardiometabolic risk and refine the association between obesity and gut microbiota. *Int J Obes (Lond)* 2017; **42:** 424–32.

106 Grosicki GJ, Fielding RA, Lustgarten MS. Gut microbiota contribute to age-related changes in skeletal muscle size, composition, and function: biological basis for a gut-muscle axis. *Calcif Tissue Int* 2017; **102:** 433–42.

107 Wells JC. The new "obstetrical dilemma": stunting, obesity and the risk of obstructed labour. *Anat Rec (Hoboken)* 2017; **300:** 716–31.

108 Alonso BD, Silva F, Latorre M, Diniz CSG, Bick D. Caesarean birth rates in public and privately funded hospitals: a cross-sectional study. *Rev Saude Publica* 2017; **51:** 101.

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

109 Betran AP, Temmerman M, Kingdon C, et al. Interventions to reduce unnecessary caesarean sections in healthy women and babies. *Lancet* 2018; **392:** 1358–68.
110 Rodriguez-Ramirez S, Munoz-Espinosa A, Rivera JA, Gonzalez-Castell D, Gonzalez de Cosio T. Mexican children under 2 years of age consume food groups high in energy and low in micronutrients. *J Nutr* 2016; **146:** 1916–23S.
111 Rynders CA, Blanc S, DeJong N, Bessesen DH, Bergouignan A. Sedentary behaviour is a key determinant of metabolic inflexibility. *J Physiol* 2018; **596:** 1319–30.
112 Torres SJ, Nowson CA. Relationship between stress, eating behavior, and obesity. *Nutrition* 2007; **23:** 887–94.
113 Bjorntorp P. Do stress reactions cause abdominal obesity and comorbidities? *Obes Rev* 2001; **2:** 73–86.
114 Marphatia AA, Cole TJ, Grijalva-Eternod CS, Wells JC. Associations of gender inequality with child malnutrition and mortality across 96 countries. *Glob Health Epidemiol Genom* 2016; **1:** e6.
115 Wells JC, Marphatia AA, Cole TJ, McCoy D. Associations of economic and gender inequality with global obesity prevalence: understanding the female excess. *Soc Sci Med* 2012; **75:** 482–90.
116 Rosset PM. Food is different: why we must get the WTO out of agriculture. London: Zed Books, 2006.
117 Paul H, Steinbrecher R. Hungry corporations: transnational biotech companies colonise the food chain. London: Zed Books, 2003.
118 Ruel MT, Alderman H, Maternal and Child Nutrition Study Group. Nutrition-sensitive interventions and programmes: how can they help to accelerate progress in improving maternal and child nutrition? *Lancet* 2013; **382:** 536–51.
119 Wang Y, Beydoun MA. The obesity epidemic in the United States—gender, age, socioeconomic, racial/ethnic, and geographic characteristics: a systematic review and meta-regression analysis. *Epidemiol Rev* 2007; **29:** 6–28.
120 Kshatriya GK, Acharya SK. Triple burden of obesity, undernutrition, and cardiovascular disease risk among Indian tribes. *PLoS One* 2016; **11:** e0147934.
121 Hudda MT, Nightingale CM, Donin AS, et al. Reassessing ethnic differences in mean BMI and changes between 2007 and 2013 in English Children. *Obesity (Silver Spring)* 2018; **26:** 412–19.
122 Nightingale CM, Rudnicka AR, Owen CG, et al. Influence of adiposity on insulin resistance and glycemia markers among United Kingdom children of South Asian, black African-Caribbean, and white European origin: Child Heart and Health Study in England. *Diabetes Care* 2013; **36:** 1712–19.
123 Paciorek CJ, Stevens GA, Finucane MM, Ezzati M, Nutrition Impact Model Study Group (Child Growth). Children's height and weight in rural and urban populations in low-income and middle-income countries: a systematic analysis of population-representative data. *Lancet Glob Health* 2013; **1:** e300–09.
124 Hernandez AV, Pasupuleti V, Deshpande A, Bernabe-Ortiz A, Miranda JJ. Effect of rural-to-urban within-country migration on cardiovascular risk factors in low- and middle-income countries: a systematic review. *Heart* 2012; **98:** 185–94.
125 Tyrovolas S, Koyanagi A, Olaya B, et al. The role of muscle mass and body fat on disability among older adults: a cross-national analysis. *Exp Gerontol* 2015; **69:** 27–35.
126 McEniry M, McDermott J. Early-life conditions, rapid demographic changes, and older adult health in the developing world. *Biodemography Soc Biol* 2015; **61:** 147–66.
127 Tzioumis E, Adair LS. Childhood dual burden of under- and overnutrition in low- and middle-income countries: a critical review. *Food Nutr Bull* 2014; **35:** 230–43.
128 Caleyachetty R, Thomas GN, Kengne AP, et al. The double burden of malnutrition among adolescents: analysis of data from the Global School-Based Student Health and Health Behavior in School-Aged Children surveys in 57 low- and middle-income countries. *Am J Clin Nutr* 2018; **108:** 414–24.
129 Huffman SL, Piwoz EG, Vosti SA, Dewey KG. Babies, soft drinks and snacks: a concern in low- and middle-income countries? *Matern Child Nutr* 2014; **10:** 562–74.
130 Stearns SC. The evolution of life histories. Oxford: Oxford University Press, 1992.
131 Wells JC, Nesse RM, Sear R, et al. Evolutionary public health: introducing the concept. *Lancet* 2017; **390:** 500–09.
132 Wells JC, Cole TJ, Cortina-Borja M, et al. Low maternal capital predicts life history trade-offs in daughters: why adverse outcomes cluster in individuals. *Front Public Health* 2019; **7:** 206.
133 Talmor A, Dunphy B. Female obesity and infertility. *Best Pract Res Clin Obstet Gynaecol* 2015; **29:** 498–506.
134 Okin D, Medzhitov R. Evolution of inflammatory diseases. *Curr Biol* 2012; **22:** R733–40.
135 Crimmins EM, Finch CE. Infection, inflammation, height, and longevity. *Proc Natl Acad Sci USA* 2006; **103:** 498–503.
136 NCD Risk Factor Collaboration. A century of trends in adult human height. *Elife* 2016; **5:** e13410.
137 Penuelas J, Janssens IA, Ciais P, et al. Increasing gap in human height between rich and poor countries associated to their different intakes of N and P. *Sci Rep* 2017; **7:** 17671.
138 Anekwe TD, Kumar S. The effect of a vaccination program on child anthropometry: evidence from India's Universal Immunization Program. *J Public Health (Oxf)* 2012; **34:** 489–97.
139 Gosby AK, Conigrave AD, Raubenheimer D, Simpson SJ. Protein leverage and energy intake. *Obes Rev* 2014; **15:** 183–91.
140 Willett W, Rockström J, Loken B, et al. Food in the Anthropocene: the EAT–*Lancet* Commission on healthy diets from sustainable food systems. *Lancet* 2019; **393:** 447–92.

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

Double Burden of Malnutrition 3

Double-duty actions: seizing programme and policy opportunities to address malnutrition in all its forms

Corinna Hawkes, Marie T Ruel, Leah Salm, Bryony Sinclair, Francesco Branca

Actions to address different forms of malnutrition are typically managed by separate communities, policies, programmes, governance structures, and funding streams. By contrast, double-duty actions, which aim to simultaneously tackle both undernutrition and problems of overweight, obesity, and diet-related non-communicable diseases (DR-NCDs) have been proposed as a way to effectively address malnutrition in all its forms in a more holisitic way. This Series paper identifies ten double-duty actions that have strong potential to reduce the risk of both undernutrition, obesity, and DR-NCDs. It does so by summarising evidence on common drivers of different forms of malnutrition; documenting examples of unintended harm caused by some undernutrition-focused programmes on obesity and DR-NCDs; and highlighting examples of double-duty actions to tackle multiple forms of malnutrition. We find that undernutrition, obesity, and DR-NCDs are intrinsically linked through early-life nutrition, diet diversity, food environments, and socioeconomic factors. Some evidence shows that programmes focused on undernutrition have raised risks of poor quality diets, obesity, and DR-NCDs, especially in countries undergoing a rapid nutrition transition. This Series paper builds on this evidence to develop a framework to guide the design of double-duty approaches and strategies, and defines the first steps needed to deliver them. With a clear package of double-duty actions now identified, there is an urgent need to move forward with double-duty actions to address malnutrition in all its forms.

Published Online December 15, 2019 https://doi.org/10.1016/S0140-6736(19)32506-1

This is the third in a **Series** of four papers about the double burden of malnutrition

Centre for Food Policy, City, University of London, London, UK (Prof C Hawkes PhD); **International Food Policy Research Institute, Washington, DC, USA** (M T Ruel PhD, L Salm MSc); **World Cancer Research Fund International, London, UK** (B Sinclair MPH); **and World Health Organization, Geneva, Switzerland** (F Branca PhD)

Correspondence to: Prof Corinna Hawkes, Centre for Food Policy, City, University of London, London EC1R 1UW, UK **corinna.hawkes@city.ac.uk**

Introduction

Most countries, at all levels of development, experience multiple forms of malnutrition.[1] The coexistence of nutritional deficiencies and overweight or obesity and associated diet-related non-communicable diseases (DR-NCDs)—ie, the double burden of malnutrition (DBM), which is observed within communities, households, and individuals.[2] In high-income countries, where overweight and obesity affects more than half of the population, food insecurity among people with low incomes manifests as low-quality diets often dominated by high consumption of foods, snacks, and beverages high in energy, sugar, fat, and salt.[3] These diets lead to excessive intakes of energy, DR-NCDs, and deficiencies in protein and essential micronutrients such as iron, folate, vitamins B6, B12, C, D, and calcium.[4,5] At the other extreme, low-income and middle-income countries (LMICs) still struggling with persistent problems of maternal undernutrition, child stunting and wasting, and widespread micronutrient deficiencies are having a rapid rise in overweight and obesity at lower levels of national income than previously seen.[2]

The DBM presents new challenges for policy and programming. In LMICs, national nutrition policies and donor funding have historically focused on undernutrition. Yet there is no longer just undernutrition, but also overweight, obesity, and DR-NCDs to deal with. There has been increasing global recognition that all types of malnutrition need to be addressed (panel 1). Target 2.2 of the Sustainable Development Goals is to "end malnutrition in all its forms"[12] and the *Lancet* Commission on the global syndemic of obesity, undernutrition, and climate change highlights the need to tackle these interconnected problems simultaneously.[19]

Nevertheless, actions to address the different manifestations of malnutrition are still isolated from each other and implemented through different governance and funding mechanisms (panel 1). Studies over a decade

Key messages

- Actions to address undernutrition, and overweight and obesity have historically been developed and delivered separately from one another. Some evidence shows that programmes addressing undernutrition have unintentionally increased risks for obesity and diet-related non-communicable diseases (DR-NCDs) in low-income and middle-income countries where food environments are changing rapidly. Yet policies and interventions to address undernutrition typically fail to consider these risks.
- By contrast, double-duty actions aim to simultaneously prevent or reduce the risk of both nutritional deficiencies leading to underweight, wasting, stunting or micronutrient deficiencies, and obesity or DR-NCDs, with the same intervention, programme, or policy.
- Double-duty actions are based on the rationale that all forms of malnutrition share common drivers that can be leveraged for double impact. These drivers include early life nutrition, diet diversity, food environments, and socioeconomic factors.
- The available evidence indicates that there are ten strong candidates for double-duty actions across different sectors. These actions include interventions delivered through health services, social safety nets, educational settings, agriculture, food systems, and food environments
- Putting a double-duty approach into operation involves assessing the potential harm of existing actions and redesigning programmes and policies with a focus on double-duty actions. Changes in governance, financing, and capacity building will be needed to put the approach to use.
- Double-duty actions are urgently needed as part of a holistic approach to ending malnutrition in all its forms by 2030.

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

Panel 1: **The pathway to double duty**

International and national policy response to the double burden of malnutrition (DBM)

In 1992, in the World Declaration on Nutrition, 159 countries pledged to reduce all forms of malnutrition.[6] However, the nutrition plans developed by low-income and middle-income countries following the Declaration still tended to focus on undernutrition. Nevertheless, at the global level, the existence of the DBM continued to be discussed and recognised. In 2000 the Commission on the Nutrition Challenges of the 21st Century proposed what it termed a new paradigm of a DBM.[7] The UN System Standing Committee on Nutrition (UNSCN) hosted a conference on the DBM in 2005, and UNSCN News published a special issue dedicated to the topic.[8] In 2012, the member states of WHO endorsed a Comprehensive Implementation Plan for Maternal, Infant and Young Child Nutrition, which introduced six global nutrition targets (stunting, wasting, overweight in children younger than 5 years, breastfeeding, low birthweight, and anaemia in women of reproductive age).[9] This plan was the first time that childhood overweight had been included as a global goal in an international strategy also including undernutrition.

The development of strategies focused on obesity and non-communicable diseases took a separate path. In 2004, the member states of WHO endorsed The Global Strategy on Diet and Physical Activity and an NCD Global Monitoring Framework in 2013,[10] introducing nine reduction targets for non-communicable diseases, including one on no increase in the prevalence of overweight and obesity or diabetes in adolescents and adults. The first Global Action Plan for the Prevention and Control of NCDs was adopted in 2013.[11]

In 2014, the Rome Declaration on Nutrition emerging from the second International Conference on Nutrition became the first to use the term malnutrition in all its forms.[12] The Global Nutrition Report then brought the Maternal, Infant and Young Child Nutrition targets and diet-related non-communicable diseases targets from the NCD Monitoring Framework together in its tracking.[13] Ending malnutrition in all its forms became target 2.2 of the Sustainable Development Goal[14] and central to the work programme of the UN Decade of Action on Nutrition 2016–25.[15]

During this period, nation states increasingly recognised the importance of addressing the DBM. For example, 42% of countries (from a sample of 162 countries) now have between six and eight nutrition targets, including both undernutrition and obesity-related targets and 84% of countries now have targets for adult overweight or obesity.[1] Nevertheless, in their policies and interventions, countries continue to take disparate approaches to different forms of malnutrition.[16] For example, in Bangladesh, Indonesia, and Guatemala (three countries identified in the first paper in this Series[2] as having a high DBM) all acknowledged the existence of the DBM. Yet these countries have separate strategies and different actions to tackle different forms of malnutrition. The Tanzania National Multisectoral Nutrition Action Plan (NMNAP) published in 2016 is a rare example of a strategy that explicitly states it is a double-duty action plan but nevertheless lists separate actions for different forms of malnutrition.[17]

In this context, the Global Nutrition Report 2015[13] noted a gap in efforts to combat both undernutrition, and obesity and non-communicable diseases at once, with its authors calling for a package of what they termed double-duty actions "that address both undernutrition and unhealthy diets in an internationally agreed-upon package".[18]

See Online for appendix

ago raised the concern that taking a siloed approach to tackling food insecurity and undernutrition could do harm to obesity,[20] and miss opportunities to use the same platforms for shared action.[16]

The objective of this Series paper is to explore the potential for a more holistic approach to address the DBM. Double-duty actions, a term coined in the 2015 Global Nutrition Report,[13,18] are interventions, programmes, and policies that simultaneously prevent or reduce the risk of both nutritional deficiencies leading to underweight, wasting, stunting and micronutrient deficiencies, and problems of obesity and DR-NCDs. Instead of narrowly focusing on one problem at a time, these actions aim to maximise the benefits of taking action on one form of nutrition for another, and minimise the risks of any form of malnutrition.[21,22] The term triple duty has also been used to refer to actions that address additional development problems, such as climate change.[18,23]

This Series paper answers the call to identify priority double-duty actions.[18] It does so, firstly, by setting out the rationale for double-duty actions (that different forms of malnutrition share common drivers) and using this evidence to develop a simple framework of the factors that need to be considered when designing actions to address more than one form of malnutrition. Second, using this framework as a guide, we review the literature to identify existing evidence that actions focused on undernutrition introduce risks or cause harm for obesity and DR-NCDs (see appendix pp 2–4 for method); and third, we identify the opportunities to retrofit existing, established actions focused on undernutrition to also address obesity and non-communicable diseases.[21] The Series paper ends by setting out the next steps for using the double-duty approach and identifying research priorities.

Rationale: common drivers of the DBM

The common drivers of different forms of malnutrition have been identified as biology, epigenetics, early-life nutrition, diets, socioeconomic factors, food environments and food systems, and governance.[13,21,23,24] The first[2] and second[24] papers in this Series provide evidence that biological and epigenetic factors, and global food systems policies are common drivers, and the *Lancet* Commission on the global syndemic of obesity, undernutrition, and climate change identifies shared systems drivers.[19]

Four intermediate (and modifiable) drivers for which there is evidence of influence on multiple forms of malnutrition are early-life nutrition, diet quality, food environments, and socioeconomic factors. The evidence shows that actions that promote healthy growth in early life and nutritious diets throughout the life course, combined with healthy food environments, adequate income and education, and the knowledge and skills that support these goals have the potential to benefit multiple forms of malnutrition. The figure provides a simple depiction of how interventions could leverage these common drivers to deliver on multiple forms of malnutrition.

Early-life nutrition

Nutrition in mothers during pregnancy and lactation, and in infants and young children during their first few years of life, has profound implications for malnutrition

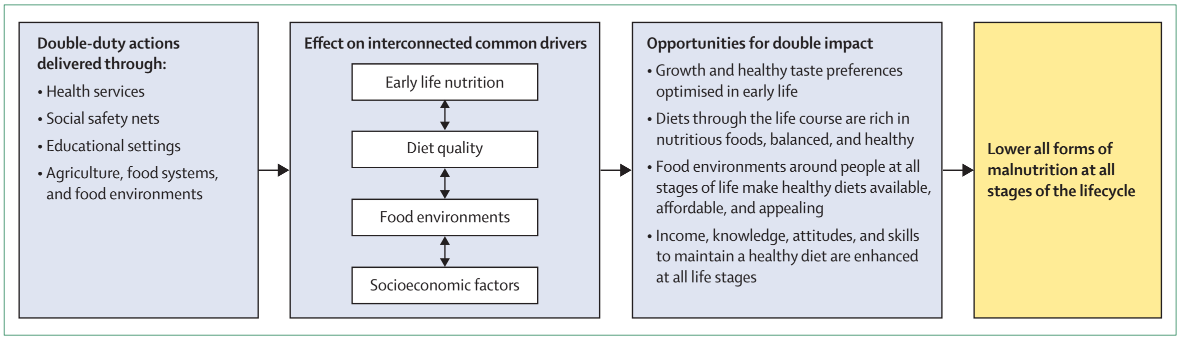

Figure: **Framework to assess opportunities for double-duty effect of nutrition actions**

in all its forms throughout the life course. Inadequate nutrient intake in early life not only leads to undernutrition among infants but also predisposes them to a more central distribution of body fat if they gain weight later in life. This early undernutrition increases the so-called toxicity of obesity—ie, compared with adults who did not experience early undernutrition, DR-NCDs manifest at lower body-mass index (BMI) thresholds for those who did.[24] This finding might explain, at least in part, the recent explosion of DR-NCDs in LMICs as they continue to develop. Extensive evidence shows that rapid weight gain during early life (which might occur in response to interventions aimed at treating or preventing undernutrition) increases the risk of adult obesity and DR-NCDs.[24] Another way early life is important is through the tastes that infants are exposed to, because a varied exposure has been shown to facilitate acceptance of nutritious foods both at the time and in later life.[25–27] Promotion of good nutrition during early life is thus a unique opportunity to tackle all forms of malnutrition.

Diet quality

High-quality diets reduce the risk of malnutrition in all its forms by promoting healthy growth, development, and immunity, and preventing obesity and DR-NCDs at all stages of the lifecycle. The components of healthy diets are: optimal breastfeeding practices in the first 2 years; a diversity and abundance of fruits and vegetables, wholegrains, fibre, nuts, and seeds; modest amounts of animal source foods; and minimal amounts of processed meats, and foods high in energy, free sugar, saturated fat, trans fat, and salt.[28,29] A diverse diet combining starchy staples, vitamin A-rich and other fruits and vegetables, and animal source foods is associated with lower levels of stunting,[30] and diets containing plenty of wholegrains, nuts, vegetables, and fruits, and modest amounts of animal source foods along with low levels of salt can make substantial contributions to reducing the burden of diet-related diseases.[31] Conversely, inadequate consumption of fruits and vegetables is a risk for both micronutrient deficiencies and DR-NCDs. High consumption of fast foods and sugary drinks is associated with increased risks of obesity in children, adolescents, and adults and with gestational diabetes in pregnant women in high-income countries.[32–36] Evidence on the association between the consumption of foods, snacks, and beverages high in energy, sugar, fat, and salt and undernutrition is still emerging. Studies from LMICs indicate that high consumption of these foods is associated with lower micronutrient intake, micronutrient deficiencies in children, lower length-for-age Z scores, and the coexistence of child stunting and maternal overweight.[37–41] Actions that reduce intake of these foods while promoting fruits, vegetables, wholegrains, nuts, seeds, and adhering to recommended levels of animal source foods, therefore provide an opportunity to tackle multiple forms of malnutrition.

Food environments

The foods available to people, the cost of these foods, and how they are marketed and promoted (often termed food environments) emerge as a common driver of the DBM owing to their role in shaping what people eat. Evidence shows that healthier food environments are associated with greater intake of nutritious foods.[42] Yet as described in the first paper in this Series,[2] worldwide availability of unhealthy processed foods, snacks, and beverages high in energy, sugar, fat, and salt has soared since 2004. Sales of breastmilk substitutes, including follow on formulae, are also growing at an unprecedented pace.[1] Manufacturers, supermarkets, food vendors, and restaurants make these foods easily accessible and affordable, often using aggressive marketing techniques.[43] Heavy promotion of breastmilk substitutes and follow-on formulas, and of inappropriate complementary foods, snacks, and sweetened beverages targeted to children influences consumption.[44,45] Companies promote foods such as biscuits, snacks, instant noodles, sugary breakfast cereals, and drinks fortified with micronutrients as healthy by including a nutrient claim, or other suggestive indicators on the food packaging.[46–48] Very young children in LMICs are regularly consuming these snacks and foods high in fat, added sugar, and salt, and little nutritional value.[45,49–53] Acting on food environments to ensure that they make healthy diets available, affordable, and

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

appealing and discourage promotion and marketing is thus a shared opportunity to prevent malnutrition in all its forms.

Socioeconomic factors

Income and education are important drivers for the risk of both undernutrition, and obesity and DR-NCD. Rises in income per capita are associated with reductions in child stunting.[54,55] Wealth, however, is a double edged sword for malnutrition since its effects on increasing overweight or obesity are larger than its effects on reducing childhood stunting.[56] The first paper in this Series[2] describes how the effects of wealth on different forms of malnutrition differ by the countries' economic development. Education is closely associated with income and wealth and generally has positive influences on nutrition[57] Enhancing both education and income while mitigating the risks associated with the latter will be a key element of addressing malnutrition in all its forms.

The evidence: what are the opportunities and risks of undernutrition-focused actions for obesity and DR-NCDS?

We now present evidence on how interventions already designed to address undernutrition through multiple sectors—health, social safety nets, education, agriculture, food systems, and food environments—could be designed to take account of the four reviewed drivers to leverage opportunities and manage risks to do no harm.

Health services

Opportunities

The table summarises the basic preventive health interventions targeting undernutrition delivered through health service facilities and networks of community-based health workers at different stages of the lifecycle.[58,59] Since most interventions target maternal and early-life nutrition, they offer a prime opportunity to prevent and treat malnutrition in all its forms especially given that they require regular contact between health workers and caregivers.

Antenatal care during pregnancy is a key intervention designed to support optimal growth of the fetus and positive birth outcomes. The 2017 WHO antenatal care recommendations include a focus on dietary interventions to promote healthy diets and prevent both undernutrition and obesity, making these interventions a double-duty action (panel 2).[61]

For lactating mothers and their infants, one very widely adopted intervention around the world is the protection, promotion, and support of optimal breastfeeding practices.[62] Evidence shows that breastfeeding helps to prevent undernutrition and stimulates immunity and cognitive development, while also reducing the risk of overweight and obesity in childhood, obesity and DR-NCDs later in life, and, for the mother, delays future pregnancies and reduces the risk of breast cancer.[63–66] Scaling up efforts to promote and protect optimal breastfeeding practices is thus a second, unequivocal opportunity for a double-duty action, providing benefits (and no risks) to both mother and child in both the short and long term (panel 2). Proven interventions to promote breastfeeding through the health system include social behavior change communication strategies combining facility-based and community-based nutrition counselling interventions and mass media.[67,68]

Promotion of complementary feeding practices is also a widespread intervention in LMICs,[69] for which well designed strategies for social behaviour change communication (with or without food and micronutrient supplementation) have been shown to be effective.[70–72] The timely introduction of nutritious, diverse fresh foods in sufficient quantity and quality at 6 months not only fosters children's growth and cognitive development but can also prevent overweight and obesity during early childhood, and obesity and DR-NCDs at adulthood.[11] However, guidance on complementary feeding has tended to focus on undernutrition and ignored the emergence of unhealthy food environments that promote the consumption of processed sweet and salty snacks among young children. A third double-duty action is thus to redesign complementary feeding guidance and actions to ensure that they include not only the foods that should be consumed, but also those to be avoided (panel 2).

Another primary health-care programme designed originally to address undernutrition is growth monitoring and promotion.[73] The main purpose of growth monitoring is to identify children who are failing to thrive by regularly measuring their weight and then provide nutrition and health counselling to promote optimal growth. WHO recommends some modifications of growth monitoring and promotion programmes to include detection of overweight and related counselling, making it a fourth option for a specific double-duty action (panel 2). The feasibility of adding these components should be carefully assessed, given the well documented operational challenges and inconclusive evidence of effectiveness of growth monitoring and promotion programmes on child growth.[74–76]

	Intervention
Mothers during pregnancy and postnatal period	Promotion of and support for healthy maternal diets; supplementation with food or micronutrients in food insecure environments
Lactating mothers and their infant or young child (0–24 months)	Promotion of optimal breastfeeding and complementary feeding practices (including food or micronutrient supplementation for children aged 0–24 months; promotion of healthy maternal diets)
Infants and young children (aged <5 years)	Growth monitoring and promotion
Infants and young children (aged <5 years)	Detection and treatment of acute malnutrition

***Table:* Health system interventions to promote and support maternal and child nutrition during the first 1000 days**

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

Panel 2: Ten priority candidates for double-duty actions

Health services

1. Scale up new WHO antenatal care recommendations

New WHO antenatal care recommendations[60] focus on:

- Counselling about healthy eating and keeping physically active during pregnancy to stay healthy and prevent excessive weight gain
- In undernourished populations, behaviour change communication on increasing daily energy and protein intake is recommended to reduce risk of low birthweight
- In undernourished populations, balanced energy and protein dietary supplementation is recommended to reduce risk of stillbirths and neonates who are small for gestational age; cash or food vouchers might be tested to improve maternal diets

An additional recommendation for double-duty actions:

- Carefully monitor targeting of protein and energy supplements (or cash or food vouchers) to prevent unintended excess weight gain during pregnancy

2. Scale up programmes to protect, promote, and support breastfeeding

- Scale up interventions to promote and support breastfeeding initiation, exclusive breastfeeding for 6 months and continued breastfeeding up to age 24 months or beyond
- Eliminate the promotion of breastmilk substitutes (infant formula and follow-on formula)

3. Redesign guidance for complementary feeding practices and related indicators

- Incorporate messages to emphasise healthy and diverse diets, including daily intake of vegetables and fruits
- Include recommendations to avoid feeding young children foods, snacks, and beverages high in energy, sugar, fat, and salt
- Include specific guidance on the selection of healthy snacks
- Revisit guidance on energy density in complementary foods taking into account the risks of excessive energy density, especially in countries and regions where energy intake is not limited in the diet
- Include new training curricula for primary health-care workers to provide double-duty nutrition counselling

4. Redesign existing growth monitoring (GMP) programmes

For ongoing GMP programmes in contexts where childhood overweight is, or is becoming, a problem:

- Include the measurement of child weight and height or length in primary care centres, if feasible*
- Use the weight-for-height or weight-for-length (or body-mass-index-for-age) indicators and growth charts to diagnose the risk of overweight and obesity, if feasible*, alongside wasting
- Include referral and appropriate counselling on healthy diets and snacks to address all types of malnutrition

5. Prevent undue harm from energy-dense and micronutrient-fortified foods and ready to use supplements

- Promote healthy diets as the default measure to prevent undernutrition
- Establish clear criteria on when the distribution of energy-dense and micronutrient-fortified foods and supplements targeted to mothers during pregnancy and lactation, and children aged up to 24 months is justified; and establish targeting guidelines based on household food insecurity and individual nutritional status
- Include nutrition counselling on healthy diets and snacks for mothers and young children in all supplement distribution programmes
- Ensure careful choice and targeting of high-energy, micronutrient-fortified foods and supplements provided to treat moderate and severe acute malnutrition or to prevent stunting or wasting
- Manage duration of food supplementation to avoid excessive or rapid weight gain beyond needed for recovery or prevention of moderate or severe acute malnutrition; limit sharing of food supplements with siblings; and incorporate nutrition counselling on healthy diets and snacks as components of prevention programmes for undernutrition

Social safety nets

6. Redesign cash and food transfers, subsidies, and vouchers

- Include strong education and behaviour change communication focused on healthy diets, physical activity, and preventive use of health services
- Include regular health check-ups for all household members and early detection of overweight or obesity, and DR-NCDs
- For subsidies or food vouchers, focus on and link to retailers providing nutritious foods; exclude foods, snacks, and beverages high in energy, sugar, fat, and salt
- Introduce rewards for transfers or vouchers spent on nutritious foods
- Implement complementary measures to rebalance food environments towards healthier food choices and outcomes, such as restrictions on marketing, taxes, and nutrition labelling

Educational settings

7. Redesign school feeding programmes and devise new nutritional guidelines for food in and around educational institutions

- Ensure that guidelines for school feeding programmes and food provided by the commercial sector in day care, preschools, and schools meet energy and nutrient needs and restrict foods, snacks, and beverages high in energy, sugar, fat, and salt
- Involve parents and children in planning meals and food in and around schools

(Continues on next page)

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

(Panel 2 continued from previous page)

- Eliminate the promotion and sale of foods, snacks, and beverages high in energy, sugar, fat, and salt around schools
- Build knowledge and skills to create awareness, shape tastes, and motivate consumption of healthy diets through education, school gardens, and mainstreaming food throughout the curriculum
- Incorporate the promotion of nutritious foods and healthy diets using innovative communication tools tailored to youth

Agriculture, food systems, and food environments

8. Scale up nutrition-sensitive agriculture programmes

- Promote diversity in food production and consumption among poor households living in remote areas with little access to markets
- Include approaches to empower women in agricultural programmes
- Provide counselling and behaviour change communication focused on nutritious foods and healthy diets for all household members; for small producers of nutritious foods, educate on benefits of saving some of the production for own consumption or purchasing other nutritious foods
- Carefully design and support urban and periurban agriculture to promote and support the growing demand for nutritious foods in urban areas

9. Design new agricultural and food system policies to support healthy diets

- Refocus agriculture towards the production of nutritious foods such as fruits, vegetables, nuts, legumes, and wholegrains, and making these foods more affordable for all
- Align actions through the food system to ensure that diversity of nutritious foods produced by agriculture reach consumers through the value chain

10. Implement policies to improve food environments from the perspective of malnutrition in all its forms

In addition to the actions in 1–9 that aim to improve food environments, implement policies such as:

- Eliminate the promotion of breastmilk substitutes and follow-on formula and reduce the marketing of foods, snacks, and beverages high in energy, sugar, fat, and salt, including those which are fortified
- Monitor and restrict nutrition and health claims on foods, snacks, and beverages high in energy, sugar, fat, and salt
- Use well targeted taxes on foods, snacks, and beverages high in energy, sugar, fat, and salt and subsidies for nutritious foods
- Improve the nutritional quality of the food supply through incentives to community food production, fortification, biofortification, and reformulation
- Set incentives and rules for retailers and traders to ensure a healthier community food environment

*The operational feasibility of these changes should be tested, given the added complexity of incorporating length measures and messaging on overweight and obesity.

Risks

Supplementation with energy, protein, and micronutrients is another action with proven benefits on maternal or child micronutrient status, birth outcomes, and child growth, especially in food insecure environments.[59,77–80] A study of food supplementation during pregnancy and early childhood in Guatemala found that supplementation improved early child nutrition and growth and had long-term positive effects on many outcomes later in life, including height, cognitive development, schooling achievement, economic productivity, and reproductive health in women, and significantly lowered the risks of diabetes at adulthood.[81] However, the study showed that the group who received the high energy supplement with protein, had greater BMI, body fat, and central adiposity at adulthood (37–54 years) than did a group that received a low energy supplement without protein in early life.[82] The study signals the potentially negative effects of food (energy) supplementation in populations who have poverty and food insecurity in early life but who might be exposed to rapidly changing and increasingly obesogenic food environments as they move into adult life in countries undergoing rapid income growth and an accelerated nutrition transition.

Concerns have also been raised about food supplements designed to treat and prevent acute malnutrition. Ready-to-use therapeutic foods (a type of lipid-based nutrient supplement high in energy, fat, and sugar, high-quality protein, and micronutrients) is a proven life-saving treatment for severe acute malnutrition.[83,84] Other lipid-based nutrient supplement products with lower concentrations of energy, fat, and sugar are used in small doses as a preventive measure to improve nutrition and growth in young children in food insecure areas, or to treat children with moderate acute malnutrition. Four concerns have been raised, which are still to be fully substantiated, about the potential risks associated with the intake of these products. First, rapid weight gain during early childhood, which these products might trigger, might lead to excess adiposity, and metabolic syndrome later in life, especially in countries undergoing a rapid nutrition transition.[85–88] Second, intake of these supplements over several months might affect the gut microbiome of the recipient and might also influence their taste preferences and later life consumption patterns.[88,89] Third, potential mis-targeting of supplements because of errors in the detection of moderate acute malnutrition or severe acute malnutrition in children, or sharing with siblings, might lead to excess energy intakes among children who are not energy deficient or acutely malnourished.[85] Fourth, the distribution of supplements might displace nutrition counselling programmes aimed at promoting optimal complementary feeding practices and healthy diets.

Research on the long-term effects of the regular use of these different products in early childhood is needed to better evaluate the risks. However, no alternative product that is equally safe, convenient, and effective for use at the community level is available for treating severe acute malnutrition. A fifth double-duty action would thus be to continue treatment for severe acute malnutrition but to establish clear criteria and manage the potential long-term risks of energy-dense micronutrient-fortified foods and supplements used for prevention and treatment of different forms of undernutrition (panel 2).

Social safety nets

Opportunities

Social safety net programmes include income support (cash transfers and benefits or welfare programmes) and food transfers or subsidy programmes (providing vouchers or subsidised prices on select foods). The goal of these programmes is generally to reduce poverty among poor and marginalised groups and reduce food insecurity. Some, particularly the conditional cash transfer programmes, target women and promote the use of health, nutrition, and education services as conditions for receipt of income, in an effort to build human capital.[90]

Social safety net programmes have had positive effects on undernutrition outcomes. As described in detail in the appendix (pp 5–7), conditional cash transfer and food transfer or subsidy programmes in Mexico, Egypt, and the USA improve elements of diet quality, food insecurity, poverty and undernutrition outcomes and, in some cases, the use of health and education services.[91–94] These programmes reach millions of poor people and provide cash that can be spent on nutritious foods, increase access to education on healthy eating, and provide direct food subsidies or packages. Therefore, they present an important opportunity for a sixth double-duty action to enhance diets, education, and resources that could reduce the risk of obesity and non-communicable diseases while also improving undernutrition outcomes (panel 2).

Risks

From the evidence presented earlier, social safety net programmes are clearly an important and effective tool to reduce poverty and food insecurity. However, despite their benefits, some programmes have had unintended negative effects on some aspects of diet quality and the risk of obesity and DR-NCDs (appendix pp 5–11).[95–98] These effects appear to be either because the programme directly provided or subsidised foods, snacks, and beverages high in energy, sugar, fat, and salt, or because they provided income that could be used to purchase these types of foods, which had become readily available and affordable in transitioning food environments. For example, the conditional cash transfer programme in Mexico (Oportunidades) was associated with excessive weight gain among women in urban areas who were already overweight or obese before entering the programme,[99,100] and the PAL programme increased total energy intake in a population that already consumed excess energy at baseline (appendix p 5).[92,101] Similar evidence from Guatemala shows that a food assistance programme that provided food rations to mothers and children during the first 1000 days reduced child stunting by 11% but increased women's weight (by 600 g) at 24 months post partum in a population in which more than 42·5% of women (non-pregnant or non-lactating) were overweight or obese at baseline.[102] Further evidence of harm comes from non-experimental evaluations of conditional cash transfer programmes in Brazil and Colombia and various food assistance programmes in Peru (appendix pp 8–11).

For food subsidy programmes, in Egypt, mothers in urban areas receiving food rations under the national food subsidy programme (providing bread and flour, and a targeted ration card that provided subsidies for rice, sugar, cooking oil, and black tea) had higher BMI and their children were more likely to be stunted or obese than non-beneficiaries (appendix p 6).[103] Beneficiaries in urban areas also had poorer diet diversity and lower frequencies of vegetable, meat, and fish consumption than did non-beneficiaries. The evidence, therefore, suggests that the subsidy programme might have caused double harm by increasing both chronic undernutrition and overweight in children and exacerbating the existing problem of overweight and obesity in women.

Despite their documented unintended negative effects, these safety net programmes also provide prime examples of how redesigning programmes can leverage opportunities for double duty. For example, the Mexico conditional cash transfer programme incorporated a new health component designed to track both child undernutrition, and overweight and obesity; regular check-ups for the detection of diabetes, hypertension, overweight, and obesity in adults; and a revamped social behaviour change communication strategy that includes counselling on healthy diets to prevent the risk of obesity and DR-NCD (appendix p 5).[104] In Egypt, the government reform of the programme in 2014, expanded the variety of subsidised foods to include micronutrient-rich foods such as lentils, fava beans, meat, chicken, fish, milk, and cheese, and restricted the bread subsidy to ration-card holders (appendix p 6). Enhancements have also been made to the PAL programme in Mexico and the SNAP programmes in the USA to reduce the risk of exacerbating obesity and DR-NCD (appendix p 5 and p 7). These examples confirm the large potential of social safety net programmes to serve as a double-duty approach if they are designed to address malnutrition in all its forms (panel 2).

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

Educational settings

Opportunities

School feeding programmes that offer meals, snacks, or take-home rations exist in at least 150 countries, serving 368 million children.[105,106] In LMICs, these programmes are established to improve nutrition, cognitive and psychosocial development, and dietary behaviours.[107,108] In higher-income countries, direct provision of nutritious foods or standards to limit the availability of foods, snacks, and beverages high in energy, sugar, fat, and salt has been shown to improve targeted dietary behaviours.[42]

By giving the opportunity to provide a healthy diet directly to children, combined with the possibility of school-based food and nutrition education, healthy school meals emerge as a seventh opportunity for double-duty action.[109] However, this opportunity has yet to be fully leveraged. Nutritional guidelines for schools in LMICs rarely appear to consider malnutrition in all its forms, having been developed either for contexts where undernutrition historically dominates or those with high prevalence of obesity.[110,111]

Risks

Providing food or meals in schools becomes a risk if it increases the accessibility of unhealthy snacks and foods high in fat, added sugar, and salt and provides little nutritional value. There is surprisingly little information on the quality of school meals in LMICs but some evidence shows that foods eaten in schools and sold in the vicinity are of poor nutritional quality. Evidence from Brazil, Iran, Mexico, Haiti, Guatemala, India, South Africa, and the Philippines shows that foods sold by vendors in and outside of schools include chips, cookies, crackers, ice cream, fried foods, sugary drinks, hamburgers, pizza, and confectionary.[47,112–119] A review of school food policies in eight countries in Latin America also reported widespread availability of these foods in kiosks in and out of schools.[120] Studies also found that substantial proportions of students consume snacks and sugar sweetened beverages on and off school property,[117,121] and that promotion of snack foods and drinks inside schools is widespread,[47,121] such as signage boards with the school's name advertising a food or beverage.[122] Double-duty actions for schools thus need to consider not only the quality of the food available through official channels in schools, but also the unhealthy food vending practices in and around schools.

The review identified one example in which schools had taken the opportunity to retrofit an established programme to a double-duty approach. The National Nursery Schools Council Program (JUNJI) in Chile is a free day-care programme that provides two meals and a snack to children younger than 6 years from low-income backgrounds. Concerned by the high rates of obesity, the programme reduced the energy content of the meals by 100 kcal.[123] The intervention was unsuccessful in reducing obesity, but a follow-up pilot study tested a new approach involving parents and focusing on improving diets at home and at school. Significant reductions in energy and fat intakes and snack consumption were achieved, as well as increases in fruit and vegetable intakes and physical activity.[124] The example emphasises the potential of using educational platforms for double-duty action by focusing on both home and school environments (panel 2).

Agriculture, food systems, and food environments

Opportunities

In the past decade, there has been a concerted effort in LMICs to build nutrition goals into agricultural development programmes. Such programmes (often termed nutrition sensitive agriculture) include biofortification, homestead food production, aquaculture, livestock and dairy programmes, agriculture extension services, nutrition-sensitive value chains, and irrigation interventions.[125] The aim of these programmes is typically to promote diversity in production of nutritious foods for direct consumption and possibly for income from the sale of surplus production. A recent review found that these programmes consistently improve food environments by enhancing household access to nutritious foods, thereby leading to increased quality of mothers' and young children's diets.[125] Thus, these agricultural development programmes have the potential to promote nutritious diets that benefit multiple forms of malnutrition, making the scale-up of these programmes an eighth candidate for double-duty actions.

School gardens also have the potential to shape attitudes and behaviours of school aged children around diet and indirectly by influencing attitudes at home, and improving food environments.[126–128] In cities, agricultural programmes such as urban agriculture and direct farm-consumer markets could also play a role in improving food environments and food security if provided with sustained support.[129]

Risks

The review by Ruel and colleagues[125] identified no risks from nutrition-sensitive agricultural programmes, although the programmes reviewed were implemented in extremely poor rural communities. One potential risk of these types of programmes for obesity and DR-NCDs is their potential effect on increasing income from the sale of agricultural products. If this additional income is used to purchase foods, snacks, and beverages high in energy, sugar, fat, and salt available in food environments, the programmes could inadvertently increase the risks of obesity.[130]

Larger agricultural development investments have typically been implemented without any specific nutrition objectives, and their historical focus has been on delivering enough dietary energy to prevent hunger and food insecurity.[131] Historically, policies have incentivised the production of grains, oilseeds, and sugar.[132] Breeding programmes designed to increase yield of staple crops initially funded in the 1940s took off in Latin America and Asia to become the so-called Green Revolution. Still today, the Consultative Group of International Agricultural Research Centers (CGIAR) allocates about half of its resources to research on rice and maize.[133] The concern has been voiced that the narrow focus on dietary energy has created risks for other aspects of diets.[134] For example, the Green Revolution is credited with boosting overall energy consumption from basic cereals (rice, wheat); however, it did little to improve dietary diversity and micronutrient intake, and might have even worsened trends.[135] Overall, increasing productivity of cereals and oilseeds provided cheap feed for livestock and inputs for processed foods, arguably introducing a risk for obesity and DR-NCDs by providing low-cost ingredients used by manufacturers in industrially processed foods.[132,134] A ninth double-duty action is thus to explore how agriculture and food systems policies can incentivise larger scale shifts to transform the dynamics of the food supply that underpins food environments.[133]

Food environments are also a key component of food systems. The evidence presented on double-duty actions 1–9 show that healthy food environments are crucial to any double-duty approach. Unhealthy food environments that make foods, snacks, and beverages high in energy, sugar, fat, and salt, readily available, affordable, appealing, and aspirational undermine the benefits of providing adequate incomes through social safety nets, making nutritious foods more available through educational settings and agricultural programmes, and offering the provision of guidance and counselling to promote healthy diets. Yet, advice on how to avoid these foods is rarely featured in guidance, education, or counselling in health service programmes. A tenth double-duty action, cross-cutting actions 1–9, therefore relates to policies to reduce the availability, affordability, and appeal of foods, snacks, and beverages high in energy, sugar, fat, and salt in food environments, and vice versa for nutritious foods (panel 2). To date, such policies have typically been proposed and implemented to address obesity, notably the taxation of sugar-sweetened beverages. The evidence in this Series paper shows that double-duty food environment policies should be designed to address malnutrition in all its forms, including the risks of low protein and micronutrient intake that are caused by high consumption of foods, snacks, and beverages high in energy, sugar, fat, and salt. Such policies introduced by governments to tackle obesity now need to be adapted to promote diets that benefit all forms of malnutrition. Research to better understand the effects of these foods on undernutrition is also urgently needed (panel 3).[136]

Next steps: putting a double-duty approach into operation

The evidence presented in this Series paper indicates that continuing with business as usual with existing nutrition programmes and policies is not fit for purpose in the new nutrition reality. The ten identified double-duty actions are a means of leveraging shared opportunity and reducing risks of established programmes and policies currently addressing undernutrition (panel 2). Two steps are needed: designing a double-duty strategy; and then delivering it.

Designing a double-duty strategy

The design of a double-duty strategy should include the three following processes.[21] First, existing programmes and policies targeting undernutrition should be reviewed to assess whether they are presenting risks or doing harm, and what opportunities they provide to be retrofitted as double-duty actions. The framework in the figure provides a starting point for how this assessment

Panel 3: Research priorities to advance the double-duty agenda

Diets, food environments, and food systems

- Development of simpler tools to measure dietary intake; and design and validation of indicators of diet quality that capture both risks of undernutrition and of obesity and diet-related non-communicable diseases (DR-NCDs) for use across populations
- Detailed assessment of dietary intake of individuals at different stages of the lifecycle in populations living in different contexts including urban and rural areas, and low-income and middle-income countries
- Analyses of the association between consuming foods, snacks, drinks high in energy, sugars, fat, and salt and undernutrition, and concomitant association with obesity and DR-NCDs
- Analysis of the role of food environments in dietary intake and patterns in the context of other drivers (eg, socioeconomic status) in different contexts and their influence on different forms of malnutrition
- Assessment of how food systems processes and policies influence both sides of the double burden of malnutrition, including the role of different system sectors (eg, agricultural production, trade, processing, retail) and environmental aspects such as climate change

Impact assessments

- Assessment of short-term and long-term effects of interventions focused on undernutrition during early life (eg, food supplements, fortified foods and products [eg, lipid-based nutrient supplements and ready-to-use therapeutic foods], follow-on formula) on diets, undernutrition, and obesity and DR-NCD outcomes
- Assessment of the effects of redesigned or newly designed double-duty actions in health, social safety nets, education, and agriculture on short-term and long-term diets, undernutrition, and obesity and DR-NCD outcomes at different stages of the lifecycle
- Assessment of the effects of food environment policy innovations to improve access to healthier diets on changes in diets, undernutrition, and DR-NCDs

Operational issues

- Assessment of the feasibility and quality of implementation of redesigned or newly designed double-duty actions in health, social safety nets, education, and agriculture to identify bottlenecks that might prevent efficiency and effectiveness given increased complexity
- Assessment of cost and staff workload of redesigned or newly designed double-duty actions
- Assessment of operational issues related to scale-up of double-duty actions

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

could be done. Following the framework, this assessment should explore how existing actions are influencing—or failing to influence—the common drivers, as a means of identifying the risks and opportunities created. Second, existing programmes and policies should be redesigned to take a double-duty approach using the ideas laid out in panel 2. Third, new actions should be designed, as needed, purposively to tackle malnutrition in all its forms at all stages of the lifecycle and especially for women during pregnancy and lactation, infants, preschoolers, school age children, and adolescents. Evaluations should be built into the design and redesign of double-duty actions to ensure their effects on outcomes related to the DBM can be assessed, including possible unintended consequences (panel 3).

Delivering a double-duty strategy

To enable the delivery of the double-duty strategy, more fundamental changes will be needed in governance, funding, capacity, and research. Signs that countries are improving the governance of nutrition are encouraging, with an increasing number of countries having created a nutrition coordination mechanisms in high governmental offices, many of which are in the president or prime minister's office.[1] These actions now need to incorporate malnutrition in all its forms and one minister or ministry must be made responsible for all.

The stimulus for a change in governance is unlikely unless there are changes in funding. Financing of action for nutrition is still largely channelled to undernutrition programmes, and obesity is typically excluded from global estimates of the cost of eliminating malnutrition.[137] At the national level, if and how actions designed to address overweight and obesity are costed in nutrition plans in countries with the DBM is unclear.[138] In countries with costed nutrition plans that include overweight and obesity and DR-NCDs, funding does not appear to be available to deliver these actions.[1] Double-duty actions provide an opportunity for donors to continue with existing programming while building in considerations for the new nutrition reality.[23] This approach will require new strategic alignments by donors towards malnutrition in all its forms along with different funding streams. Understanding the costs of double-duty actions, as well as their cost-effectiveness, could help to inform this process, as addressed in the fourth paper in this Series.[139]

Given the entrenched nature of existing approaches, individual and institutional capacity strengthening will be needed to change mindsets and enable action. For example, educational institutions and professional bodies should teach the knowledge and build the skills needed to tackle malnutrition in all its forms simultaneously. Policy makers (nutrition policy leads and those working in other ministries responsible for relevant programmes) and implementers, such as health workers delivering nutrition counselling, will also need training on the double-duty approach. The capacity to deliver double-duty actions does not yet exist and will need to be built and appropriately funded. To guide and justify the allocation of resources, research will also be needed to assess what works and at what cost, and how capacity can be most effectively built, as indicated in panel 3.

To accelerate progress, the nutrition community needs to take ownership of the double-duty agenda and adopt a new paradigm and mindset that favours a more holistic approach to designing actions to simultaneously address the whole spectrum of malnutrition problems. The evidence presented in this Series paper highlights the urgency of moving forward with double-duty actions if the world is to have any hope of attaining the sustainable development goal of ending malnutrition in all its forms.

Contributors

CH led the conception and preparation of the paper, bringing in the different authors, leading the preparation of the manuscript, and structuring and restructuring after receiving the reviewers comments. MTR led the literature search and write up on programmes that caused harm and how they were modified, and contributed to the literature search, tables and figures, study design, data interpretation, writing, and addressing reviewers comments. LS contributed to literature searching, synthesis of information, write up of paper, and editing final draft. BS contributed to conceptualising the paper, reviewed the scientific literature on the effect of healthy diets on both sides of the DBM, contributed to the section on common drivers of the DBM, and reviewed and commented on drafts and the final paper. FB contributed to conceptualising the paper, provided an analysis of country policy documents and costed action plans on the DBM, and reviewed and commented on drafts and the final paper.

Decleration of interests

We declare no competing interests.

Acknowledgments

Funding for the preparation of the Series was provided by WHO, through a grant from the Bill & Melinda Gates Foundation. The funder had no role in the analysis and interpretation of the evidence or in writing the paper and the decision to submit for publication. The CGIAR Research Program on Agriculture for Nutrition and Health (A4NH) led by the International Food Policy Research Institute (IFPRI) also provided funding. We thank Madeleine Coste for her help in reviewing the literature

References

1 Development Initiatives. 2018 Global Nutrition Report: shining a light to spur action on nutrition. 2018. https://globalnutritionreport.org/reports/global-nutrition-report-2018/ (accessed Oct 19, 2019).
2 Popkin BM, Corvalan C, Grummer-Strawn LM. Dynamics of the double burden of malnutrition and the changing nutrition reality. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32497-3.
3 Drewnowski A. The economics of food choice behavior: why poverty and obesity are linked. In: Drewnowski A, Rolls B, eds. Obesity treatment and prevention: new directions. Nestlé Nutrition Institute Workshop Series. Basil: Nestec Ltd, Vevey/S Karger AG, 2012: 95–112.
4 Ritchie H, Roser M. Micronutrient Deficiency. OurWorldInData.org. 2018. https://ourworldindata.org/micronutrient-deficiency' (accessed May 4, 2018).
5 Martínez Steele E, Raubenheimer D, Simpson SJ, Baraldi LG, Monteiro CA. Ultra-processed foods, protein leverage and energy intake in the USA. *Public Health Nutr* 2018; **21:** 114–24.
6 FAO/WHO. World Declaration and Plan of Action for Nutrition. Rome: FAO, 1992 http://www.fao.org/3/u9260e/u9260e00.pdf (accessed Oct 19, 2019).

7 The Commission on Nutrition. Ending malnutrition by 2020: an agenda for change in the millennium, final report to the ACC/SCN by the Commission on Nutrition Challenges of the 21st Century. 2000. https://www.unscn.org/uploads/web/news/2000-FEB-Ending-Malnutrition-by-2020-Agenda-for-Change-in-the-Millennium-Report.pdf (accessed Oct 19, 2019).

8 UNSCN (UN Standing Committee on Nutrition). Tackling the Double Burden of Malnutrition. *SCN News* 2016; **32:** 38–45.

9 WHO. World Health Assembly Resolution 65.6. Agenda item 13.3: Maternal, infant and young child nutrition. Geneva, 2012. http://apps.who.int/gb/DGNP/pdf_files/A65_REC1-en.pdf (accessed Oct 20, 2019).

10 WHO. NCD targets and indicators, 2013. https://www.who.int/nmh/global_monitoring_framework/en (accessed Oct 20, 2019).

11 WHO. Global action plan for the prevention and control of noncommunicable diseases 2013-2020. Geneva, Switzerland: World Health Organization (WHO), 2013. https://www.who.int/nmh/events/ncd_action_plan/en/ (accessed Oct 20, 2019).

12 FAO/WHO. Second International Conference on Nutrition - Conference Outcome Document: Rome Declaration on Nutrition. Rome, Italy, 2014. http://www.fao.org/3/a-ml542e.pdf (accessed Oct 20, 2019).

13 IFPRI. Global Nutrition Report 2015: Actions and accountability to advance nutrition and sustainable development. International Food Policy Research Institute, 2015. https://www.ifpri.org/publication/global-nutrition-report-2015 (accessed Oct 20, 2019).

14 United Nations General Assembly. Transforming our world: The 2030 agenda for sustainable development. 2015. https://sustainabledevelopment.un.org/post2015/transformingourworld/publication (accessed Oct 20, 2019).

15 United Nations. Work programme of the United Nations Decade of Action on Nutrition (2016-2025). 2017. https://www.who.int/nutrition/decade-of-action/workprogramme-2016to2025/en/ (accessed Oct 20, 2019).

16 Corvalan C. Latin America: Avoiding the nutrition transition 'trap'. 10th Dr Abraham Horwitz Lecture. UN Standing Committee on Nutrition. Tackling the Double Burden of Malnutrition. https://www.unscn.org/layout/modules/resources/files/scnnews32.pdf (accessed Oct 29, 2019).

17 United Republic of Tanzania. (2016) Prime Minister's Office. United Republic of Tanzania (2016): National Multisectoral Nutrition Action Plan (NMNAP) for the period July 2016 – June 2021. Dar es Salaam, 2016. https://mafiadoc.com/united-republic-of-tanzania-prime-ministers-office-report-on-the-2016-_599419761723ddcb690dafda.html (accessed Oct 20, 2019).

18 Hawkes C, Haddad L. The challenge to nutrition researchers everywhere: identify a set of actions that reduce malnutrition in all its forms. *Lancet* Global Health Blog, 2015. https://marlin-prod.literatumonline.com/pb-assets/Lancet/langlo/TLGH_Blogs_2013-2018.pdf (accessed Oct 20, 2019).

19 Swinburn BA, Kraak VI, Allender S, et al. The global syndemic of obesity, undernutrition, and climate change: The *Lancet* Commission report. *Lancet* 2019; **393:** 791–846.

20 Uauy R, Kain J. The epidemiological transition: need to incorporate obesity prevention into nutrition programmes. *Public Health Nutr* 2002; **5:** 223–29.

21 WHO. Double-duty actions. Policy brief. Geneva: World Health Organization (WHO), 2017.https://www.who.int/nutrition/publications/double-duty-actions-nutrition-policybrief/en/ (accessed Oct 20, 2019).

22 Hawkes C. From What to How: The role of double-duty actions in addressing the double burden. *Sight Life* 2018; **32.** https://sightandlife.org/wp-content/uploads/2018/12/17_SALMZ_0218_Perspectives_04.pdf (accessed Oct 29, 2019).

23 DI. Global Nutrition Report 2017: Nourishing the SDGs. Development Initiative, 2017. https://nutritionconnect.org/resource-center/2017-global-nutrition-report-nourishing-sdgs (accessed Oct 20, 2019).

24 Wells JC, Sawaya AL, Wibaek R, et al. The double burden of malnutrition: aetiological pathways and consequences for health. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32472-9.

25 De Cosmi V, Scaglioni S, Agostoni C. Early taste experiences and later food choices. *Nutrients* 2017; **9:** 107.

26 Mennella JA. Ontogeny of taste preferences: basic biology and implications for health. *Am J Clin Nutr* 2014; **99:** 704–11S.

27 Nicklaus S. The Role of Dietary Experience in the Development of Eating Behavior during the First Years of Life. *Ann Nutr Metab* 2017; **70:** 241–45.

28 WHO. Healthy diets. 2015. http://www.who.int/news-room/fact-sheets/detail/healthy-diet (accessed Oct 20, 2019).

29 Willett W, Rockström J, Loken B, et al. Food in the Anthropocene: the EAT-*Lancet* Commission on healthy diets from sustainable food systems. *Lancet* 2019; **393:** 447–92.

30 Arimond M, Ruel MT, Dietary diversity is associated with child nutritional status: evidence from 11 demographic and health surveys. *J Nutr* 2004; **134:** 2579–85.

31 Afshin A, Sur PJ, Fay KA, et al. Health effects of dietary risks in 195 countries, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. *Lancet* 2019; **393:** 1958–72.

32 Fraser LK, Clarke GP, Cade JE, Edwards KL. Fast food and obesity: a spatial analysis in a large United Kingdom population of children aged 13–15. *Am J Prev Med* 2012; **42:** e77–85.

33 Taveras EM, Berkey CS, Rifas-Shiman SL, et al. Association of consumption of fried food away from home with body mass index and diet quality in older children and adolescents. *Pediatrics* 2005; **116:** e518–24.

34 Niemeier HM, Raynor HA, Lloyd-Richardson EE, Rogers ML, Wing RR. Fast food consumption and breakfast skipping: predictors of weight gain from adolescence to adulthood in a nationally representative sample. *J Adolesc Health* 2006; **39:** 842–49.

35 Malik VS, Pan A, Willett WC, Hu FB. Sugar-sweetened beverages and weight gain in children and adults: a systematic review and meta-analysis. *Am J Clin Nutr* 2013; **98:** 1084–102.

36 Te Morenga L, Mallard S, Mann J. Dietary sugars and body weight: systematic review and meta-analyses of randomised controlled trials and cohort studies. *BMJ* 2012; **346:** e7492.

37 Louzada MLC, Baraldi LG, Steele EM, et al. Consumption of ultra-processed foods and obesity in Brazilian adolescents and adults. *Prev Med* 2015; **81:** 9–15.

38 Lander R, Enkhjargal TS, Batjargal J, et al. Poor dietary quality of complementary foods is associated with multiple micronutrient deficiencies during early childhood in Mongolia. *Public Health Nutr* 2010; **13:** 1304–13.

39 Anderson VP, Cornwall J, Jack S, Gibson RS. Intakes from non-breastmilk foods for stunted toddlers living in poor urban villages of Phnom Penh, Cambodia, are inadequate. *Matern Child Nutr* 2008; **4:** 146–59.

40 Aitsi-Selmi A. Households with a stunted child and obese mother: trends and child feeding practices in a middle-income country, 1992–2008. *Matern Child Health J* 2015; **19:** 1284–91.

41 Pries AM, Rehman AM, Filteau S, Sharma N, Upadhyay A, Ferguson EL. Unhealthy snack food and beverage consumption is associated with lower dietary adequacy and length-for-age z-Scores among 12–23-month-olds in Kathmandu Valley, Nepal. *J Nutr* 2019; published online July 16. DOI:10.1093/jn/nxz140.

42 Micha R, Karageorgou D, Bakogianni I, et al. Effectiveness of school food environment policies on children's dietary behaviors: A systematic review and meta-analysis. *PLoS One* 2018; **13:** e0194555.

43 Popkin BM, Reardon T. Obesity and the food system transformation in Latin America. *Obes Rev* 2018; **19:** 1028–64.

44 Pries AM, Huffman SL, Mengkheang K, et al. High use of commercial food products among infants and young children and promotions for these products in Cambodia. *Matern Child Nutr* 2016; **12** (suppl 2): 52–63.

45 Huffman SL, Piwoz EG, Vosti SA, Dewey KG. Babies, soft drinks and snacks: a concern in low- and middle-income countries? *Matern Child Nutr* 2014; **10:** 562–74.

46 Nair Ghaswalla A. Fortified biscuits: Britannia bakes a plan for a healthy business. *The Hindu* Sep 18, 2014. https://www.thehindubusinessline.com/companies/Fortified-biscuits-Britannia-bakes-a-plan-for-a-healthy-business/article20868419.ece.

47 Reeve E, Thow AM, Bell C, et al. Implementation lessons for school food policies and marketing restrictions in the Philippines: a qualitative policy analysis. *Global Health* 2018; **14:** 8.

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

48 Verrill L, Wood D, Cates S, Lando A, Zhang Y. Vitamin-fortified snack food may lead consumers to make poor dietary decisions. *J Acad Nutr Diet* 2017; **117:** 376–85.

49 Jaime PC, Prado RRD, Malta DC. Family influence on the consumption of sugary drinks by children under two years old. *Rev Saude Publica* 2017; **51** (suppl 1)**:** 13s.

50 Kavle JA, Mehanna S, Saleh G, et al. Exploring why junk foods are 'essential' foods and how culturally tailored recommendations improved feeding in Egyptian children. *Matern Child Nutr* 2015; **11:** 346–70.

51 Brown K, Henretty N, Chary A, et al. Mixed-methods study identifies key strategies for improving infant and young child feeding practices in a highly stunted rural indigenous population in Guatemala. *Matern Child Nutr* 2016; **12:** 262–77.

52 Pries AM, Huffman SL, Champeny M, et al. Consumption of commercially produced snack foods and sugar-sweetened beverages during the complementary feeding period in four African and Asian urban contexts. *Matern Child Nutr* 2017; **13** (suppl 2)**:** e12412.

53 Pries AM, Huffman SL, Mengkheang K, et al. High use of commercial food products among infants and young children and promotions for these products in Cambodia. *Matern Child Nutr* 2016; **12** (suppl 2)**:** 52–63.

54 Webb P, Block S. Support for agriculture during economic transformation: impacts on poverty and undernutrition. *Proc Natl Acad Sci USA* 2012; **109:** 12309–14.

55 Headey D, Hoddinott J, Park S. Drivers of nutritional change in four South Asian countries: a dynamic observational analysis. *Matern Child Nutr* 2016; **12** (suppl 1)**:** 210–18.

56 Ruel MT, Alderman H. Nutrition-sensitive interventions and programmes: how can they help to accelerate progress in improving maternal and child nutrition? *Lancet* 2013; **382:** 536–51.

57 Alderman H, Headey DD. How Important is Parental Education for Child Nutrition? *World Dev* 2017; **94:** 448–64.

58 Kerber KJ, de Graft-Johnson JE, Bhutta ZA, Okong P, Starrs A, Lawn JE. Continuum of care for maternal, newborn, and child health: from slogan to service delivery. *Lancet* 2007; **370:** 1358–69.

59 Bhutta ZA, Das JK, Rizvi A, et al. Evidence-based interventions for improvement of maternal and child nutrition: what can be done and at what cost? *Lancet* 2013; **382:** 452–77.

60 WHO. WHO recommendations on antenatal care for a positive pregnancy experience. Geneva, Switzerland: World Health Organization, 2016. https://www.who.int/reproductivehealth/publications/maternal_perinatal_health/anc-positive-pregnancy-experience/en/ (accessed Oct 20, 2019).

61 The World Health Organization. Assessing and managing children at primary health-care facilities to prevent overweight and obesity in the context of the double burden of malnutrition Updates for the Integrated Management of Childhood Illness (IMCI). Geneva, Switzerland: World Health Organization, 2017.

62 World Health Organization. Essential nutrition actions. improving maternal, newborn, infant and young child health and nutrition. Geneva, 2013. https://www.who.int/nutrition/publications/infantfeeding/essential_nutrition_actions.pdf (accessed Oct 20, 2019).

63 Victora CG, Bahl R, Barros AJD, et al. Breastfeeding in the 21st century: epidemiology, mechanisms, and lifelong effect. *Lancet* 2016; **387:** 475–90.

64 Horta BL, Loret de Mola C, Victora CG. Long-term consequences of breastfeeding on cholesterol, obesity, systolic blood pressure and type 2 diabetes: a systematic review and meta-analysis. *Acta Paediatr* 2015; **104:** 30–37.

65 Yan J, Liu L, Zhu Y, Huang G, Wang PP. The association between breastfeeding and childhood obesity: a meta-analysis. *BMC Public Health* 2014; **14:** 1267.

66 World Cancer Research Fund, American Institute for Cancer Research. Diet, nutrition, physical activity and breast cancer. 2017 http://www.aicr.org/continuous-update-project/reports/breast-cancer-report-2017.pdf (accessed Oct 20, 2019).

67 Bhutta ZA, Das JK, Rizvi A, et al. Evidence-based interventions for improvement of maternal and child nutrition: what can be done and at what cost? *Lancet* 2013; **382:** 452–77.

68 Menon P, Nguyen PH, Saha KK, et al. Impacts on Breastfeeding Practices of At-Scale Strategies That Combine Intensive Interpersonal Counseling, Mass Media, and Community Mobilization: Results of Cluster-Randomized Program Evaluations in Bangladesh and Viet Nam. *PLoS Med* 2016; **13:** e1002159.

69 WHO–PAHO. Guiding principles for complementary feeding of the breastfed child. Washington, DC: Pan American Health Organization, 2003. https://www.paho.org/hq/index.php?option=com_content&view=article&id=5666:2011-principios-orientacion-alimentacion-complementaria-nino-amamantado-2003&Itemid=4069&lang=en (accessed Oct 20, 2019).

70 Nguyen PH, Kim SS, Nguyen TT, et al. Exposure to mass media and interpersonal counseling has additive effects on exclusive breastfeeding and its psychosocial determinants among Vietnamese mothers. *Matern Child Nutr* 2016; **12:** 713–25.

71 Menon P, Nguyen PH, Saha KK, et al. Combining intensive counseling by frontline workers with a nationwide mass media campaign has large differential impacts on complementary feeding practices but not on child growth: results of a cluster-randomized program evaluation in Bangladesh. *J Nutr* 2016; **146:** 2075–84.

72 Panjwani A, Heidkamp R. complementary feeding interventions have a small but significant impact on linear and ponderal growth of children in low- and middle-income countries: a systematic review and meta-analysis. *J Nutr* 2017; **147:** 2169–78S.

73 King MH, Savage King F, Martodipoero S, World Health Organization. Primary child care: a manual for health workers. Oxford: Oxford University Press, 1979.

74 Mangasaryan N, Arabi M, Schultink W. Revisiting the concept of growth monitoring and its possible role in community-based nutrition programs. *Food Nutr Bull* 2011; **32:** 42–53.

75 Roberfroid D, Kolsteren P, Hoerée T, Maire B. Do growth monitoring and promotion programs answer the performance criteria of a screening program? A critical analysis based on a systematic review. *Trop Med Int Health* 2005; **10:** 1121–33.

76 Ashworth A, Shrimpton R, Jamil K. Growth monitoring and promotion: review of evidence of impact. *Matern Child Nutr* 2008; **4** (suppl 1)**:** 86–117.

77 Eichler K, Wieser S, Rüthemann I, Brügger U. Effects of micronutrient fortified milk and cereal food for infants and children: a systematic review. *BMC Public Health* 2012; **12:** 506.

78 Ramakrishnan U, Nguyen P, Martorell R. Effects of micronutrients on growth of children under 5 y of age: meta-analyses of single and multiple nutrient interventions. *Am J Clin Nutr* 2009; **89:** 191–203.

79 Bhutta ZA, Ahmed T, Black RE, et al. What works? Interventions for maternal and child undernutrition and survival. *Lancet* 2008; **371:** 417–40.

80 Haider BA, Bhutta ZA. Multiple-micronutrient supplementation for women during pregnancy. *Cochrane Database Syst Rev* 2015; **4:** CD004905.

81 Martorell R. Improved nutrition in the first 1000 days and adult human capital and health. *Am J Hum Biol* 2017; **29:** e22952.

82 Ford ND, Behrman JR, Hoddinott JF, et al. Exposure to improved nutrition from conception to age 2 years and adult cardiometabolic disease risk: a modelling study. *Lancet Glob Health* 2018; **6:** e875–84.

83 WHO. Guideline: Updates on the management of severe acute malnutrition in infants and children. Geneva, Switzerland: World Health Organization (WHO), 2013. https://www.ncbi.nlm.nih.gov/books/NBK190328/pdf/Bookshelf_NBK190328.pdf (accessed Oct 20, 2019).

84 Lenters LM, Wazny K, Webb P, Ahmed T, Bhutta ZA. Treatment of severe and moderate acute malnutrition in low- and middle-income settings: a systematic review, meta-analysis and Delphi process. *BMC Public Health* 2013; **13** (suppl 3)**:** S23.

85 Sridhar SB, Darbinian J, Ehrlich SF, et al. Maternal gestational weight gain and offspring risk for childhood overweight or obesity. *Am J Obstet Gynecol* 2014; **211:** e1–8.

86 Lucas K, James P, Choh AC, et al. The positive association of infant weight gain with adulthood body mass index has strengthened over time in the Fels Longitudinal Study. *Pediatr Obes* 2018; **13:** 476–84.

87 Adair LS, Fall CH, Osmond C, et al. Associations of linear growth and relative weight gain during early life with adult health and human capital in countries of low and middle income: findings from five birth cohort studies. *Lancet* 2013; **382:** 525–34.

88 Bazzano AN, Potts KS, Bazzano LA, Mason JB. The life course implications of ready to use therapeutic food for children in low-income countries. *Int J Environ Res Public Health* 2017; **14:** 403.

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

89 Luque V, Escribano J, Closa-Monasterolo R, et al. Unhealthy dietary patterns established in infancy track to mid-childhood: the EU childhood obesity project. *J Nutr* 2018; **148:** 752–59.

90 Levy S. Progress against Poverty: Sustaining Mexico's Progresa-Oportunidades program. Washington, DC: Brookings Institution Press, 2006.

91 Parker SW, Todd PE. Conditional Cash Transfers: The Case of Progresa/Oportunidades. *J Econ Lit* 2017; **55:** 866–915.

92 Leroy JL, Gadsden P, Rodríguez-Ramírez S, de Cossío TG. Cash and in-kind transfers in poor rural communities in Mexico increase household fruit, vegetable, and micronutrient consumption but also lead to excess energy consumption. *J Nutr* 2010; **140:** 612–17.

93 Abdalla M, Al-Shawarby S. The Tamween food subsidy system in Egypt: evolution and recent implementation reforms. In: Alderman H, Gentilini U, Yemtsov R, eds. The 1.5 billion people question: food, vouchers, or cash transfers? Washington: World Bank, 2016: 107–47.

94 Oliveira V, Tiehen L, Prell M, Smallwood D. Evolution and implementation of the Supplemental Nutrition Assistance Program in the United States. In: Alderman H, Gentilini U, Yemtsov R, eds. The 1.5 billion people question: Food, vouchers, or cash transfers? Washington, DC: World Bank, 2018: 209–63.

95 Saldiva SRDM, Silva LFF, Saldiva PHN. Avaliação antropométrica e consumo alimentar em crianças menores de cinco anos residentes em um município da região do semiárido nordestino com cobertura parcial do programa bolsa família. *Rev Nutr* 2010; **23:** 221–29.

96 Forde I, Chandola T, Garcia S, Marmot MG, Attanasio O. The impact of cash transfers to poor women in Colombia on BMI and obesity: prospective cohort study. *Int J Obes* 2012; **36:** 1209–14.

97 Carrillo-Larco RM, Miranda JJ, Bernabé-Ortiz A. Impact of food assistance programs on obesity in mothers and children: a prospective cohort study in Peru. *Am J Public Health* 2016; **106:** 1301–07.

98 Chaparro MP, Bernabe-Ortiz A, Harrison GG. Association between food assistance program participation and overweight. *Rev Saude Publica* 2014; **48:** 889–98.

99 Neufeld LM, Garcia-Guerra A, Leroy J, Flores López ML, Fernández Gaxiola AC, Rivera-Donmarco JA. Impacto del programa Oportunidades en nutrición y alimentación en zonas urbanas de México. Cuernavaca, Mexico: National Institute of Public Health, 2005.

100 Fernald LCH, Gertler PJ, Neufeld LM. Role of cash in conditional cash transfer programmes for child health, growth, and development: an analysis of Mexico's Oportunidades. *Lancet* 2008; **371:** 828–37.

101 Leroy JL, Gadsden P, González de Cossío T, Gertler P. Cash and in-kind transfers lead to excess weight gain in a population of women with a high prevalence of overweight in rural Mexico. *J Nutr* 2013; **143:** 378–83.

102 Leroy JL, Olney DK, Ruel MT. PROCOMIDA, a food-assisted maternal and child health and nutrition program, contributes to postpartum weight retention in Guatemala: a cluster-randomized controlled intervention trial. *J Nutr* 2019; published online Aug 2. DOI:https://doi.org/10.1093/jn/nxz175.

103 Ecker O, Al-Riffai P, Breisinger C, El-Batrawy R. Nutrition and economic development: Exploring Egypt's exceptionalism and the role of food subsidies. Washington DC, 2016 DOI:10.2499/9780896292383.

104 Bonvecchio A, González W, eds. Estrategia Integral de Atención a la Nutrición (EsIAN) para población beneficiaria de PROSPERA Programa de Inclusión Social. Mexico: Manual para la Unidad de Salud.

105 Global Panel on Agriculture and Food Systems for Nutrition. Healthy meals in schools: policy innovations linking agriculture, food systems and nutrition. Policy Brief 2015. https://www.gcnf.org/wp-content/uploads/2016/03/GloPan-healthy-meals-in-schools-2015-November.pdf (accessed Oct 20, 2019).

106 World Food Programme. 2013 - State of School Feeding Worldwide. 2013. https://www.wfp.org/publications/state-school-feeding-worldwide-2013 (accessed Oct 20, 2019).

107 Drake L, Meena F, Aurina E, et al. School feeding programs in middle childhood and adolescence. In: Bundy D, de Silva N, Horton SE, Jamison D, Patton G, eds. Child and Adolescent Health and Development. Washington, DC: World Bank, 2017: 79–98.

108 Kristjansson EA, Gelli A, Welch V, et al. Costs, and cost-outcome of school feeding programmes and feeding programmes for young children. Evidence and recommendations. *Int J Educ Dev* 2016; **48:** 79–83.

109 UNSCN. School as a system to improve nutrition. A new statement for school-based food and nutrition interventions. Discussion Paper. Geneva, Switzerland: United Nations Standing Committee on Nutrition, 2017. https://www.unscn.org/uploads/web/news/document/School-Paper-EN-WEB-8oct.pdf (accessed Oct 20, 2019).

110 Wijesinha-Bettoni R, Orito A, Löwik M, Mclean C, Muehlhoff E. Increasing fruit and vegetable consumption among schoolchildren: efforts in middle-income countries. *Food Nutr Bull* 2013; **34:** 75–94.

111 Aliyar R, Gelli A, Hamdani SH. A review of nutritional guidelines and menu compositions for school feeding programs in 12 countries. *Front Public Health* 2015; **3:** 148.

112 Bonvecchio-Arenas A, Theodore FL, Hernández-Cordero S, et al. La escuela como alternativa en la prevención de la obesidad: la experiencia en el sistema escolar mexicano. *Rev Española Nutr Comunitaria* 2010; **16:** 13–16.

113 Morshed AB, Becker HV, Delnatus JR, Wolff PB, Iannotti LL. Early nutrition transition in Haiti: linking food purchasing and availability to overweight status in school-aged children. *Public Health Nutr* 2016; **19:** 3378–85.

114 Pehlke EL, Letona P, Hurley K, Gittelsohn J. Guatemalan school food environment: impact on schoolchildren's risk of both undernutrition and overweight/obesity. *Health Promot Int* 2016; **31:** 542–50.

115 Rathi N, Riddell L, Worsley A. Food environment and policies in private schools in Kolkata, India. *Health Promot Int* 2017; **32:** 340–50.

116 Rathi N, Riddell L, Worsley A. What influences urban Indian secondary school students' food consumption? - A qualitative study. *Appetite* 2016; **105:** 790–97.

117 Azeredo CM, de Rezende LFM, Canella DS, et al. Food environments in schools and in the immediate vicinity are associated with unhealthy food consumption among Brazilian adolescents. *Prev Med* 2016; **88:** 73–79.

118 Yazdi Feyzabadi V, Keshavarz Mohammadi N, Omidvar N, Karimi-Shahanjarini A, Nedjat S, Rashidian A. Factors Associated With Unhealthy Snacks Consumption Among Adolescents in Iran's Schools. *Int J Health Policy Manag* 2017; **6:** 519–28.

119 Faber M, Laurie S, Maduna M, Magudulela T, Muehlhoff E. Is the school food environment conducive to healthy eating in poorly resourced South African schools? *Public Health Nutr* 2014; **17:** 1214–23.

120 FAO and Governo Federal Brasil. School feeding and possibilities of direct purchasing from family farming: case studies from eight countries. Santiago, 2014. http://www.fao.org/3/a-i3413e.pdf (accessed Oct 20, 2019).

121 Godin KM, Chacón V, Barnoya J, Leatherdale ST. The school environment and sugar-sweetened beverage consumption among Guatemalan adolescents. *Public Health Nutr* 2017; **20:** 2980–87.

122 de Villiers A, Steyn NP, Draper CE, et al. "HealthKick": formative assessment of the health environment in low-resource primary schools in the Western Cape Province of South Africa. *BMC Public Health* 2012; **12:** 794.

123 Corvalán C, Uauy R, Flores R, Kleinbaum D, Martorell R. Reductions in the energy content of meals served in the Chilean National Nursery School Council Program did not consistently decrease obesity among beneficiaries. *J Nutr* 2008; **138:** 2237–43.

124 Salazar G, Vasquez F, Concha F, et al. Pilot nutrition and physical activity intervention for preschool children attending daycare centres (JUNJI): primary and secondary outcomes. *Nutr Hosp* 2014; **29:** 1004–12.

125 Ruel MT, Quisumbing AR, Balagamwala M. Nutrition-sensitive agriculture: what have we learned so far? *Glob Food Sec* 2018; **17:** 128–53.

126 Berezowitz CK, Bontrager Yoder AB, Schoeller DA. School gardens enhance academic performance and dietary outcomes in children. *J Sch Health* 2015; **85:** 508–18.

127 Utter J, Denny S, Dyson B. School gardens and adolescent nutrition and BMI: Results from a national, multilevel study. *Prev Med* 2016; **83:** 1–4.

128 Davis JN, Spaniol MR, Somerset S. Sustenance and sustainability: maximizing the impact of school gardens on health outcomes. *Public Health Nutr* 2015; **18:** 2358–67.

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

129 Warren E, Hawkesworth S, Knai C. Investigating the association between urban agriculture and food security, dietary diversity, and nutritional status: A systematic literature review. *Food Policy* 2015; **53:** 54–66.

130 Pullar J, Allen L, Townsend N, et al. The impact of poverty reduction and development interventions on non-communicable diseases and their behavioural risk factors in low and lower-middle income countries: A systematic review. *PLoS One* 2018; **13:** e0193378.

131 World Bank. Learning from World Bank history: Agriculture and food-based approaches for addressing malnutrition. Agriculture and environmental services Discussion Paper Series 10. Washington, DC, 2014 http://documents.worldbank.org/curated/en/497241468168227810/pdf/887400NWP0Box30ning0from0WB0History.pdf.

132 Anand SS, Hawkes C, de Souza RJ, et al. Food Consumption and its Impact on Cardiovascular Disease: Importance of Solutions Focused on the Globalized Food System: A Report From the Workshop Convened by the World Heart Federation. *J Am Coll Cardiol* 2015; **66:** 1590–614.

133 Global Panel on Agriculture and Food Systems for Nutrition. Food systems and diets: Facing the challenges of the 21st century. London, UK: Global Panel on Agriculture and Food Systems for Nutrition, 2016.

134 Qaim M. Globalisation of agrifood systems and sustainable nutrition. *Proc Nutr Soc* 2017; **76:** 12–21.

135 Pingali PL. Green revolution: impacts, limits, and the path ahead. *Proc Natl Acad Sci USA* 2012; **109:** 12302–08.

136 Pries AM, Filteau S, Ferguson EL. Snack food and beverage consumption and young child nutrition in low- and middle-income countries: A systematic review. *Matern Child Nutr* 2019; **15** (suppl 4)**:** e12729.

137 Shekar M, Kakietek J, Dayton Eberwein J, Walters D. An investment framework for nutrition: reaching the global targets for stunting, anemia, breastfeeding, and wasting. The World Bank, 2017.

138 Scaling Up Nutrition. Planning and costing for the acceleration of actions for nutrition: experiences of countries in the Movement for Scaling Up Nutrition. Geneva, Switzerland, 2014.

139 Nugent R, Levin C, Hale J, Hutchinson B. Economic effects of the double burden of malnutrition. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32473-0.

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

Double Burden of Malnutrition 4

Economic effects of the double burden of malnutrition

Rachel Nugent, Carol Levin, Jessica Hale, Brian Hutchinson

Observations from many countries indicate that multiple forms of malnutrition might coexist in a country, a household, and an individual. In this Series, the double burden of malnutrition (DBM) encompasses undernutrition in the form of stunting, and overweight and obesity. Health effects of the DBM include those associated with both undernutrition, such as impaired childhood development and greater susceptibility to infectious diseases, and overweight, especially in terms of increased risk of added visceral fat and increased risk of non-communicable diseases. These health effects have not been translated into economic costs for individuals and economies in the form of lost wages and productivity, as well as higher medical expenses. We summarise the existing approaches to modelling the economic effects of malnutrition and point out the weaknesses of these approaches for measuring economic losses from the DBM. Where population needs suggest that nutrition interventions take into account the DBM, economic evaluation can guide the choice of so-called double-duty interventions as an alternative to separate programming for stunting and overweight. We address the evidence gap with an economic analysis of the costs and benefits of an illustrative double-duty intervention that addresses both stunting and overweight in children aged 4 years and older by providing school meals with improved quality of diet. We assess the plausibility of our method and discuss how improved data and models can generate better estimates. Double-duty interventions could save money and be more efficient than single-duty interventions.

Published **Online** December 15, 2019 https://doi.org/10.1016/S0140-6736(19)32473-0

This is the fourth in a **Series** of four papers about the double burden of malnutrition

RTI International, Seattle, WA, USA (R Nugent PhD, J Hale, B Hutchinson)**; and University of Washington Department of Global Health, Seattle, WA, USA** (R Nugent, C Levin PhD)

Correspondence to: Dr Rachel Nugent, RTI International, Seattle, WA 98104, USA. **rnugent@rti.org**

Introduction

The double burden of malnutrition (DBM) in low-income and middle-income countries was recognised by economists as important by 2001, but has not been extensively studied. The DBM is now receiving greater attention as it appears to be more permanent and widespread than earlier perceived, which implies greater economic effects.[1] More than two decades ago, Popkin and colleagues[2] identified costs of 1% and 2% of gross domestic product (GDP) from undernutrition, overweight, and associated non-communicable diseases in China and India, respectively. Popkin projected that the cost could reach 9% of GDP in China by 2025.[2] The World Bank estimated economic costs of 2–3% of GDP in Indonesia in 2012.[3] Yet major challenges remain in assessing the economic effects of the DBM, and the return on investments in reducing it.

In this Series paper, we examine methods for doing economic evaluations of the DBM, identify gaps, and recommend improvements to economic modelling while additional empirical data are awaited, and provide an example of how to evaluate the costs and benefits of an intervention designed to address the DBM in three developing countries. The cost–benefit analysis is intended purely as an illustration of what can be learned by doing economic evaluations of actual double-duty interventions, which is not yet feasible with available evidence. Our primary goal is to consider the challenges to economic modellers of estimating the effects of the DBM. Our secondary goal is to draw attention to the need for better programmatic evidence for so-called double-duty interventions that can address the DBM.

Measuring the economic costs of malnutrition

Reasons for the few studies on the economic effects of the DBM include challenges in harmonising different long-term outcomes for chronic undernutrition and obesity, diffuse and not easily measured health and economic effects of poor nutrition, scarcity of data on all forms of malnutrition within the same population, and limited economic modelling for nutrition. A subtle but important consequence of this point is that the DBM falls between distinct and largely separate undernutrition and obesity advocacy and expert communities and is not the priority of either community. Therefore, differences exist in the methods used by nutrition economists to measure economic effects of multiple forms of malnutrition.

Key messages

- Existing economic models for nutrition are not designed to measure the effects of the double burden of malnutrition
- Adding up the separate economic effects of undernutrition, and overweight and obesity is a second-best approach to measuring economic effects of the double burden of malnutrition
- Economic models need to be enhanced to incorporate effects for both undernutrition and overweight in the same population
- Using a double-duty intervention to reduce the double burden of malnutrition might be economically advantageous compared with addressing malnutrition with separate interventions for undernutrition and overweight, but of intervention evidence is insufficient and limitations of economic models prevent a firm conclusion

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

Those studying the effects of undernutrition generally use structural models and microlevel longitudinal data to examine the cognitive and productivity effects of early-life nutritional deficiencies.[4,5] The economic effect of overweight and obesity is generally measured with a cost-of-illness approach that measures direct medical exiture for obesity treatment, or for treatment of obesity-related diseases such as cardiovascular disease and diabetes. Few studies focus on reduced productivity and earlier retirement related to overweight in low-income and middle-income countries.[6,7] Thus economists have resorted to measuring the economic burden of each form of malnutrition separately and added the individual burdens to find a combined burden. However, this method does not account for possible interactions that could either diminish or exacerbate the combined effects, compared to effects when only one form of malnutrition is present. This possible interaction is a challenge for measuring the effects of double-duty interventions for the DBM, which we note but do not resolve in our analysis.

We did a scoping review of the best-known models that provide economic outcomes of nutritional status, for both undernutrition and overweight. We assessed the key characteristics of those models for purposes of analysing the economic effects of the DBM. Although the review was not exhaustive, it was comprehensive enough to highlight the main features of and differences in models that impede economic analysis of the DBM. Appendix 1 provides selected results of that review emphasising obesity models because they are more diverse than economic models of undernutrition.

See Online for appendix 1

Models that measure the economic effects of undernutrition

Models of the economic effect of stunting take the number of children who are stunted in a given population as a starting point, and then estimate the effect of undernutrition on economic productivity and mortality via changes in linear growth. Some models also take into consideration the economic costs of chronic undernutrition on cognitive development, schooling, and earnings. Results are typically presented as either share of GDP lost to stunting or as benefit–cost ratios for investments to reduce stunting.

The Lives Saved Tool (LiST) is an epidemiological model that analyses the effect of health and nutrition interventions on maternal, newborn, and child health.[8,9] To measure the economic effect of interventions, the model draws from well-designed cohort and experimental studies with both direct measurement of the long-term economic effects of poor childhood nutrition and proxies for poor nutritional status, usually height or growth faltering, which are then linked to educational attainment or wages to measure aggregate economic effect.[4,10,11] The second paper of this Series[12] highlights the biological long-term outcomes of early malnutrition, but these are rarely linked to long-term economic outcomes.

Other econometric approaches look at stunting as a function of individual, household, and environmental factors, but these economic analyses are not amenable to integrating with the existing set of obesity micro-simulation models. These models focus only on children younger than 5 years and do not include undernutrition in other key demographic groups (adolescent girls, pregnant and lactating mothers) or associated with other risks, such as micronutrient malnutrition.

The economic effects of stunting include cognitive and other developmental deficits that affect lifetime productivity, greater incidence of infectious and parasitic disease that cause physical impairments, and greater risk of chronic diseases in adults, with their attendant high medical and indirect costs.[4,5,13–16] Horton and Steckel[17] provide a global estimate of the economic costs of chronic undernutrition using deficits in mean height due to stunting. They find GDP losses of up to 12% in some low-income and middle-income countries, and totalling 8% of global GDP during the 20th century. This study projects lower GDP losses between 2000 and 2050 because of improvements in nutrition in Latin America and Asia. Hoddinott[18] summarises the costs of malnutrition from a study of seven African countries and finds losses of 3–16% of GDP, with an average loss of 7·7% of GDP. Other country-level estimates of economic effect show decreases in earnings and household consumption from childhood stunting (1·4% lower wages in Mexico, 10% lower earnings in low-income countries, and 66% lower household consumption in Guatemala).[4,19,20]

Models that measure the economic effects of obesity

In contrast to economic measures of undernutrition, empirical studies of economic burden of overweight generally look at the cost-of-illness from obesity and related non-communicable diseases, direct medical costs and indirect costs, or productivity losses, associated with early mortality and morbidity. Sometimes other indirect costs are included, such as transportation costs from seeking treatment and human capital costs due to lower investment in education and training.[21] Caution is needed in interpreting these estimates because the economic burden from overweight is only a fraction of the cost attributable to overweight-related non-communicable diseases. The indirect costs or productivity losses from absenteeism, disability, presenteeism (working while sick), and worker's compensation in the USA and other high-income settings typically account for 70% of the total global cost of obesity.[22,23] Economic microsimulation and macrosimulation models are used to evaluate the effect of an intervention or policy on health outcomes, disability, or premature death associated with hypertension, diabetes, cancer, stroke, and other obesity-related disease.[6,24–31] The models typically simulate food intake in a given population and look at the effect of changes in policies or interventions on food security, body-mass index (BMI), diabetes risk, and mortality from cardiovascular disease. Key to most of these models is their ability to model changes in food intake on the basis of available estimates of price and income elasticities of demand. Not all models have the same features; however, they all model distal, intermediate, and proximal risk factors on disease outcomes, disability, and death. The models require substantial demographic, epidemiological, disease burden, and economic data. We describe the characteristics of some example models next.

The UK Foresight obesity model, applied in Brazil, Mexico, and other country settings, is a dynamic computer microsimulation model applied to different populations.[6,7,32] The model tracks individuals through their lifecycle and applies a probability of being overweight, obese, or normal BMI. The model runs over an extended period and makes predictions of individuals having a risk of getting a particular disease, surviving, or dying, based on their BMI. The model is able to simulate the effect of interventions to prevent disease or death and compare the effect and costs of hypothetical interventions. Similar obesity models have been developed and mostly applied in high-income and middle-income settings.

Basu and colleagues[27] developed a metabolic-epidemiological microsimulation model to assess what population level changes in calorie intake and physical activity would be required to meet US federal guidelines to reduce the prevalence of obesity. Other obesity models use economic-epidemiological approaches to assess the effects of health-related food taxes or subsidies on health outcomes related to dietary risk factors. Most of these models have been developed to assess fiscal policy options, such as taxes on saturated fat, salt, sugar, and sugar-sweetened beverages, or subsidies on fruits and vegetables.[28,29,33]

The Organisation for Economic Co-operation and Development (OECD) and WHO developed the most comprehensive microsimulation model to date that includes the causal chain of lifestyle risk factors for cancer, stroke, and ischaemic heart disease. The model captures a range of risk factors, from more distant exposures (dietary intake and physical activity) to proximate risk factors, such as hypertension and diabetes. Cecchini and colleagues[26] applied this model to seven countries exploring the effects of school-based, workplace, population based, fiscal, and regulatory interventions on health outcomes and expenditures.[31] A broader set of countries and outcomes is modelled in more recent work.[31]

Models that measure the economic effects of the DBM

Few studies have measured the economic costs of the DBM per se. Popkin and colleagues looked at the cost of diet-related non-communicable diseases from undernutrition and overweight in China and India in 1995 and 2025.[2] Later, Popkin and colleagues[2,34] proposed a model to measure the direct effects of childhood stunting on overweight and obesity, and chronic diseases. The World Bank estimated GDP losses from the DBM in Indonesia.[3] However, the focus of those studies was on cost of non-communicable diseases, rather than the DBM itself.

An exception to the non-communicable disease focus is a model developed by the Economic and Social Commission of Latin America and Caribbean (ECLAC) and World Food Programme (WFP).[35] The modelling perspective is broad, aiming to account for multiple drivers of the DBM and to reflect the transitional and lifecycle aspects of malnutrition. For that purpose, this model introduces a multicohort life stage analysis based on each country's demography and epidemiology.

The ECLAC-WFP model separately measures effects of undernutrition and obesity in the standard ways described above. The model contemporaneously measures the economic costs of the DBM, accounting for the age structure of the countries, and then projects them over the 65 year anticipated life for the entire national populations of the countries studied. The ECLAC-WFP model measures lifetime effects of childhood undernutrition through multiple pathways; notably, increased risk of multiple diseases, lower educational attainment, and reduced lifetime earnings. Economic effects of overweight and obesity include medical costs and productivity losses. These two types of economic burden are then aggregated for total cost of the DBM.

The ECLAC-WFP model results range from a total cost of 0·2% of GDP in Chile to 4·3% of GDP in Ecuador (2014). For Chile, all of the economic burden of malnutrition derives from overweight and obesity; in Ecuador, the total economic burden is derived from a 2·6% loss of GDP due to undernutrition and a 1·7% loss of GDP due to overweight and obesity; and in Mexico, the undernutrition burden is 1·7% of GDP and the overweight and obesity burden is 0·6% of GDP (2014).

The ECLAC-WFP model, unlike other studies, adds the economics costs of undernutrition and overweight to reach a total economic cost of the DBM. There are several important advances from this analysis, first, it applies consistent assumptions to measure the economic burden of both undernutrition and overweight and obesity. Second, the lifecycle analysis that connects childhood malnutrition to adult malnutrition with projections of future economic costs based on population demographics and epidemiology captures the important transitional aspect of the DBM. The result is that overweight and obesity are shown to be bigger threats to economic wellbeing over time compared with undernutrition. The economic costs of obesity and overweight are projected to range from 0·4% (Chile) to 3·1% (Ecuador) of GDP between 2015 and 2078. Additionally, undernutrition is projected to result in only a 0·03% GDP loss in Mexico and 0·06% GDP loss in Ecuador during the same period.

A weakness of the ECLAC-WFP approach, which is clearly acknowledged, is the model's inability to capture interactions between undernutrition and obesity that could affect economic impacts. Such interactions are not well established, and further research is needed to determine whether they are significant and in which direction they would influence economic effects. Authors of the ECLAC-WFP study recommend that cohort studies be conducted with the aim of measuring interactions between different forms of malnutrition. A recent cross-sectional study in Burkina Faso provided evidence of persistent micronutrient deficiencies (iron and vitamin A) concurrent with a high prevalence of overweight among women, suggesting that economic studies should be enhanced to consider such interactions when modelling the DBM.[36]

Improvements needed in economic modelling to assess the DBM effects

We found that most economic models of malnutrition evaluate costs and outcomes associated with either stunting or overweight and obesity, but not both, with the exception of examples noted earlier.[2,37] Additionally, the two bodies of literature (stunting, and overweight and obesity) use different methodologies and answer different economic questions. We did not review the many models that assess economic effects of micronutrient deficiencies as we are focused on models that can be adapted for the DBM.

Although Popkin and colleagues[2,34] have modelled the direct effects of childhood stunting on overweight and obesity and chronic diseases, none of the other obesity microsimulation models noted earlier has looked at cohorts of children younger than 5 years to incorporate both stunting and overweight in the same model or used microsimulation models to evaluate the effects of policies and interventions on food consumption, dietary intake, changes in weight or anthropometric measures (wasting, stunting, underweight), and effects on nutrition related illnesses and premature death. By contrast, most of the stunting models use epidemiological estimates of the number of stunted children for a given population and then estimate the effect of nutrition on economic productivity and mortality. Some models also take into consideration the economic costs of chronic undernutrition on cognitive development, schooling, and earnings. New models are needed that can evaluate the effects of policies and interventions on both undernutrition and overweight and obesity for continuous population cohorts from birth to adulthood, and for extended periods to predict effects on health and economic costs over time. There are no models that address the DBM along the full causal pathway from distal to proximate risk factors and to ultimate health and economic consequences.

See Online for appendix 2

Illustration of potential economic effects of a double-duty intervention to reduce theDBM

The third paper of this Series[38] shows the unintended negative consequences of undernutrition programmes on obesity and recommends leveraging common drivers and programme platforms to achieve improved outcomes in both undernutrition and obesity. The second paper of this Series[12] points to the beneficial effects that arise from biological connections between improving nutrition and educational attainment. The economic benefits of such leveraging have not been established. In the absence of evidence either from modelling or from actual measurement of the DBM-specific interventions, we separately estimate the effects on stunting and obesity by applying the same intervention on simulated populations in three countries. This example is purely illustrative and the results do not apply to any specific context.

Interventions that address the DBM

Many effective and cost-effective interventions exist to reduce undernutrition and a smaller number of proven interventions exist to reduce overweight.[39,40] Hawkes and colleagues in the third paper in this Series[38] advocate for policies and programmes to address malnutrition in all its forms and use the term double duty to characterise such policies and programmes. While double-duty interventions might simultaneously reduce undernutrition and overweight, these interventions might imply higher cost or lower efficacy than separate interventions that focus on either undernutrition or overweight.

We reviewed policy and programmatic interventions that show promise for reducing the DBM.[41–48] We sought examples of interventions that have been implemented in one or more developing countries to address both underweight and overweight and for which we could obtain costs of implementation. We found no generalisable examples of double-duty interventions with measured effects on the outcomes that met those criteria.

Instead we chose a school feeding intervention that has been shown to have positive effects on both stunting and obesity and drew the effect sizes and costs from different studies. An advantage of choosing a school feeding programme is that they exist in many countries and show strong potential to improve healthy eating and positive nutrition behaviour among those exposed to the programme through the school platform. Our search strategy for school feeding interventions is detailed in appendix 2 pp 1–2. Appendix 2 pp 3–6 shows the key features of the relevant studies we reviewed. We recognise, as Hawkes and colleagues[38] showed in the third paper in this Series, that many school feeding interventions focus only on undernutrition and might adversely affect overweight. This means potentially that our cost estimates might be underestimated compared to an intervention designed to affect both.

Modelling the costs and benefits of school feeding on the DBM

We modelled the economic effects of school feeding programmes on the DBM in developing countries. We selected the intervention because multiple studies show effectiveness of school feeding in providing health and nutrition benefits; some studies provide outcome measures translatable into economic benefits; and school feeding is well-defined, widely implemented, and applicable to any setting. Given the imprecision implied in taking effect sizes from different studies to capture the effects on both stunting and obesity, we have not tried to produce a more exacting economic model or quantify statistical uncertainty around the point estimates. We conduct a cost–benefit estimate of our selected double-duty intervention to prevent the DBM, loosely adopting the Copenhagen Consensus approach that has been applied to a wide array of development challenges, including, separately, undernutrition and non-communicable diseases.[4,49–53] The method allows for a comparison of costs and benefits, usually in the form of a benefit–cost ratio.[54] For methods and data sources, see appendix 2 pp 7–13.

Undernutrition and obesity effects of the school feeding programme

We sought high-quality illustrative evidence of the effect of school feeding on undernutrition and obesity. We were unable to locate studies measuring obesity effects from school feeding in a developing country. We found only one study by Sekiyama and colleagues[55] that examined the simultaneous effect of school feeding programmes on stunted and overweight fourth grade school children (mean age, 9 years and 6 months). For purposes of the analysis, we chose a school breakfast programme in a population of 407 second to fifth grade school children (mean age, 9 years) in Jamaica for stunting outcomes and a school breakfast programme in the USA for children in grades 1–12 for child obesity outcomes. We do not claim that the results of these studies are generalisable to all settings, especially lower-income settings but the study results are useful for the illustrative exercise we conducted.

The Jamaica study is a well-designed, randomised study of a school breakfast programme that measured height change in cm among stunted children. This outcome aligns with the primary outcome of linear growth used in the economic effect literature. The children who received breakfast each day gained 0·25 cm on average during the 8 months of the intervention, or, by extrolation, up to 0·40 cm per year compared with children in the control group.[56] Standardising this effect to a school year (consisting of 200 feeding days) resulted in a 0·3125 cm gain in school age child height. For obesity effects, we used a school breakfast programme based in the USA in which children were provided daily breakfast. This study found a decrease of 0·149 in BMI for every increase in intake of one breakfast per week.[57] We standardised this effect to a school year of 200 feeding days and found a decrease of 0·827 in BMI in 1 year.

We applied both these effect sizes to the height and BMI distributions of children aged 4–5 years in three double-burden countries: Guatemala, Indonesia, and Nigeria. We chose those countries for geographical diversity and because we could obtain country-specific cost data for school feeding programmes. We had to apply the effects to cohorts of children aged 4–5 years as a proxy for their primary school counterparts aged 6–11 years because stunting information is not available for older children, who would normally be the target population for the school breakfast intervention. We acknowledge that the effects of the programme on younger children might vary from that on school children. Specifically, we might expect the effects of a good school feeding programme to be larger for older children who have greater autonomy in their dietary choices than do younger children who generally eat within the household and whose diet quality is managed by adults.

Estimation of the economic benefits of school feeding

We modelled the effects of height and weight changes from the school breakfast programme to the child populations in our chosen countries to obtain the number of cases of stunting and obesity averted as a result of the interventions. We then translate these health outcomes into economic outcomes, placing a monetary valuation on changes in health. The figure shows the analytical approach, underlying assumptions and effect sizes used in this analysis.

The economic benefits of lower stunting arise from higher human capital. Stunted children can gain years of education from improved nutrition. Following Fink and colleagues,[10] we calculate that 1 additional year of

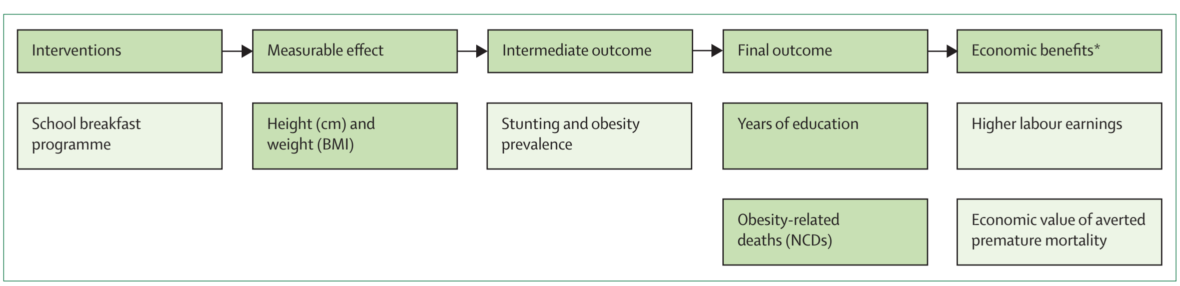

Figure: **Steps used in measuring economic effects of school feeding interventions on the DBM**
BMI=body-mass index. DBM=double burden of malnutrition. NCDs=non-communicable diseases. *Other positive outcomes result from the intervention (eg, school feeding programmes lead to higher school attendance and health-care savings from avoiding obesity).

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

education results in additional annual earnings (9·3% higher wages in Guatemala, 3·8% in Nigeria, and 5·8% in Indonesia).We apply the increased earning potential to individuals who avoid stunting, assuming that their average income is equivalent to two-thirds of GDP per capita.

The economic gains from lower obesity or overweight arise from lower premature mortality and from reducing disability. Using the before and after population-attributable fractions, we calculate the number of obesity-related deaths in the affected cohorts for each year after the cohort turns 20. We value each additional year of life at 1 times GDP per capita multiplied by a region-specific factor of the value of a statistical life-year, as obtained from Jamison and colleagues.[58]

Cost of school feeding programme

In 2008 US$, the estimated per-capita costs of school feeding programmes is $35·26 (Guatemala), $19·94 (Indonesia), and $55·82 (Nigeria).[59] The costs were specific to these countries and standardised to a 200 feeding-day programme. Using the school feeding population of 4–5 year olds, the per child costs provided in this study were multiplied by the population in each country to provide the final cost estimates. The programme was costed from 2018 to 2025.

Net economic benefits of school breakfast programmes (2018–90)

In Guatemala, Indonesia, and Nigeria the school breakfast programme intervention provides significant benefits that outweigh the costs of implementation. Net benefits range from 206 million to 3·1 billion US$, with the highest return on investment in Indonesia, driven in part by a high school attendance that allowed the programme to reach a high proportion of the cohort of children aged 4–5 years in Indonesia (table).

The benefits of the breakfast programme include the economic value of increased education (and future earnings) for children who avoid stunting and the economic value of averting premature mortality due to obesity related causes. Combining the benefits from reducing stunting and obesity, 54% of the benefits are derived from the economic value of increased education as a result of avoiding stunting, and 46% is derived from the economic value of averting premature mortality due to obesity. By measuring the effect on all forms of malnutrition, we find that the benefits from the intervention are doubled. Removing either form of malnutrition from the analysis would have lowered the return on investment for Guatemala and Indonesia and would have lowered it to below one in the case of Nigeria, changing the perception of whether the intervention should be carried out or not.

	Benefits (2016 US$)	Costs (2016 US$)	Return on investment
Guatemala	206 million	97 million	2·1
Nigeria	2·3 billion	2·1 billion	1·1
Indonesia	3·1 billion	753 million	4·2

Table: **Costs, benefits, and return on investment for a school breakfast programme in three countries**

Discussion

Earlier studies of the economic burden from stunting and obesity have shown substantial costs from both forms of malnutrition, but only a few estimates demonstrate the economic effects of the DBM. In the absence of models that can measure the economic effects of the DBM and of interventions designed to reduce the DBM we separately estimate the nutrition and health effects of a double-duty intervention. We then used an economic modelling approach to estimate the costs and benefits of a school breakfast programme to prevent the DBM in Guatemala, Indonesia, and Nigeria. We find that a school breakfast programme provides substantial benefits that outweigh the costs of implementation, with return on investment between 1·1 and 4·2.

This analysis has substantial limitations, including the absence of a model and data for analysing the economic effects of the DBM. Instead we applied a sophisticated microsimulation model for undernutrition (LiST) and a simple model to obtain the obesity effects of double-duty interventions. The intervention effect data were derived from separate populations. We made important assumptions about those populations, transferring the intervention benefits and costs from the literature for other settings and assuming a normal distribution of malnutrition in those populations. We applied the intervention to children aged 4 years and older in three DBM countries based on an effect size taken from children aged 4–5 years. As such, our results are merely illustrative of the economic benefits that could be achieved from addressing the DBM in those populations. We measure the effects of reduced mortality and acknowledge that our estimates exclude disability for which we lack measures in low-income and middle-income countries.

Our analysis was challenged by the insufficient evidence from actual DBM interventions from a range of low-income and middle-income countries because of (often highly) incomplete information and from differences in measuring outcomes and costs across sectors. These challenges hampered our ability to generate harmonised effects of our selected intervention.

To advance research in this field, several prerequisites are needed First, a standardised definition of the DBM is needed for each of the relevant levels of society such as individual, household, and national. While we now have improved epidemiological estimates of the DBM at different levels of the population (Popkin and colleagues,[60] the first paper in this Series), there is no global consensus as to which forms of malnutrition are

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

included, or what measure individually or collectively leads to the designation of the DBM. This Series focuses only on stunting and overweight and excludes micronutrient deficiencies that have been shown to have additional major economic and health costs.

Well-designed studies are needed of nutrition interventions that collect synchronous undernutrition and obesity outcomes from multiple different populations and contexts. We recommend identifying a small number of outcomes that are meaningful for multiple populations over time, such as a change in BMI, or specific nutrient intake. This approach can strengthen understanding of the emergence and persistence of the DBM in some contexts and not others.

Models that incorporate nutrition epidemiology, demographics, and economic measures should be developed to capture the costs and benefits of interventions to address the growing burden. A DBM economic model should be designed to test the proposition that a double-duty approach can achieve the same reductions in both undernutrition and overweight simultaneously at lower cost than reducing them separately.

Conclusion

This paper highlights the need to create a stronger empirical framework and suitable population-based models for guiding the economic assessment of double-duty interventions to mitigate the consequences of the DBM in low-income and middle-income countries. We demonstrate how double-duty interventions can reduce the economic effects of both forms of malnutrition. We anticipate that better definitions, combined with effect and cost data from real experience in the coming years, will input into a range of models that will provide better estimates of economic effects and the cost–benefit of employing double-duty interventions to reduce the DBM.

Existing frameworks map well the effect of nutrition-specific interventions for addressing undernutrition to a set of health outcomes related to stunting, micronutrient deficiencies, morbidity, and mortality. Similarly, frameworks exist to evaluate the effect of interventions to address overnutrition on BMI and obesity-related health outcomes, such as cardiovascular disease, stroke, and diabetes. The absence of a combined framework has hindered the development of comprehensive models to assess the economic effect of country experiences in a changing nutrition environment.[61]

We recommend the following future research to address these gaps. First, research is needed to understand the DBM, its drivers, and consequences. Second, validate a data-driven modelling tool that can project trends in the DBM (that includes both undernutrition and overweight in the same model) and related health outcomes for use by national and global policy makers. Third, identify and model cost-effective strategies to support investments to address the DBM. A starting point is to do a landscape analysis of the existing microsimulation models that are currently focused on health outcomes related to overweight and obesity, and identify models that could be modified to integrate information on underweight, stunting, and related morbidity and mortality. Finally, identify countries that are rich in epidemiological, health, agriculture, nutrition, and demographic data that can be used to populate and validate the models. These case studies should be used to introduce the framework and modelling approach to a broader set of researchers and decision makers for adoption and application in global and country settings.

Contributors

RN and CL designed the study and wrote the manuscript. BH and JH did the literature search and review. BH did economic impact calculations. All authors provided intellectual content, read, and approved the manuscript.

Declaration of interests

We declare no competing interests.

Acknowledgments

Funding for work was provided through an internal research award from RTI International. Funding for the preparation of the Series was provided by WHO, through a grant from the Bill & Melinda Gates Foundation. The funder had no role in the analysis and interpretation of the evidence or in writing the paper and the decision to submit for publication. We thank Chris Vogliano and Elizabeth Menstell for excellent research assistance.

References

1 Shrimpton R, Rokx C. The double burden of malnutrition: World Bank, 2012. http://documents.worldbank.org/curated/en/905651468339879888/The-double-burden-of-malnutrition-a-review-of-global-evidence (accessed Nov 6, 2019).
2 Popkin BM, Horton S, Kim S, Mahal A, Shuigao J. Trends in diet, nutritional status, and diet-related noncommunicable diseases in China and India: the economic costs of the nutrition transition. *Nutr Rev* 2001; **59:** 379–90.
3 Shrimpton R, Rokx C. The Double Burden of Malnutrition in Indonesia, World Bank, 2013. http://documents.worldbank.org/curated/en/955671468049836790/The-double-burden-of-malnutrition-in-Indonesia (accessed Nov 6, 2019).
4 Hoddinott J, Alderman H, Behrman JR, Haddad L, Horton S. The economic rationale for investing in stunting reduction. *Matern Child Nutr* 2013; **9** (suppl 2): 69–82.
5 Cangelosi MJ, Rodday AM, Saunders T, Cohen JT. Evaluation of the economic burden of diseases associated with poor nutrition status. *JPEN J Parenter Enteral Nutr* 2014; **38:** 35–41S.
6 Rtveladze K, Marsh T, Barquera S, et al. Obesity prevalence in Mexico: impact on health and economic burden. *Public Health Nutr* 2014; **17:** 233–39.
7 Rtveladze K, Marsh T, Webber L, et al. Health and economic burden of obesity in Brazil. *PLoS One* 2013; **8:** e68785.10.1371.
8 Johns Hopkins Bloomberg School of Public Health. LiST: Lives Saved Tool. 2016. http://www.livessavedtool.org/ (accessed June 20, 2018).
9 Walker N, Friberg, I. The Lives Saved Tool in 2017: updates, applications, and future directions. *BMC Public Health* 2017; **17** (suppl 4): 774.
10 Fink G, Peet E, Danaei G, et al. Schooling and wage income losses due to early-childhood growth faltering in developing countries: national, regional, and global estimates. *Am J Clin Nutr* 2016; **104:** 104–12.
11 Behrman J, Alderman H, Hoddinott J. Hunger and malnutrition. In: Lomborg B, ed. Global crises, global solutions. Cambridge: Cambridge University Press, 2004: 363–420.
12 Wells JC, Sawaya AL, Wibeak R, et al. The double burden of malnutrition: aetiological pathways and consequences for health. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32472-9.

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

13 Adair LS, Fall CH, Osmond C, et al. Associations of linear growth and relative weight gain during early life with adult health and human capital in countries of low and middle income: findings from five birth cohort studies. *Lancet* 2013; **382:** 525–34.

14 Victora CG, Adair L, Fall C, et al. Maternal and child undernutrition: consequences for adult health and human capital. *Lancet* 2008; **371:** 340–57.

15 Alderman H, Hoddinott J, Kinsey B. Long term consequences of early childhood malnutrition. *Oxford Economic Papers* 2006; **58:** 450–74.

16 Bloom DE, Cafiero ET, Jane-Llopis E, et al. The global economic burden of non-communicable diseases. Geneva: World Economic Forum 2011. http://www3.weforum.org/docs/WEF_Harvard_HE_GlobalEconomicBurdenNonCommunicableDiseases_2011.pdf (accessed Nov 6, 2019).

17 Horton S, Steckel RH. Malnutrition: global economic losses attributable to malnutrition 1900-2000 and projections to 2050. In: Lomborg B, ed. How much have global problems cost the earth? A scorecard from 1900 to 2050. Cambridge: Cambridge University Press, 2103: 247–72.

18 Hoddinott J. The economics of reducing malnutrition in sub-Saharan Africa, global panel working paper. London: Global Panel on Agriculture and Food Systems for Nutrition, 2016.

19 Vogl TS. Height, skills, and labor market outcomes in Mexico. *J Dev Econ* 2014; **107:** 84–96.

20 Shekar M, Heaver R, Lee Y-K, World Bank. Repositioning nutrition as central to development: a strategy for large scale action. http://documents.worldbank.org/curated/en/185651468175733998/Repositioning-nutrition-as-central-to-development-a-strategy-for-large-scale-action-overview (accessed Nov 6, 2019).

21 Trogdon JG, Finkelstein EA, Hylands T, Dellea PS, Kamal-Bahl SJ. Indirect costs of obesity: a review of the current literature. *Obes Rev* 2008; **9:** 489–500.

22 Dobbs R, Sawers C, Thompson F, et al. Overcoming obesity: an initial economic analysis: A discussion paper by the McKinsey Global Institute. https://www.mckinsey.com/industries/healthcare-systems-and-services/our-insights/how-the-world-could-better-fight-obesity (accessed Nov 6, 2019).

23 Bahia L, Coutinho ES, Barufaldi LA, et al. The costs of overweight and obesity-related diseases in the Brazilian public health system: cross-sectional study. *BMC Public Health* 2012; **12:** 440.

24 Sassi F, Cecchini M, Lauer J, Chisholm D. Improving lifestyles, tackling obesity: the health and economic impact of prevention strategies. 2009. https://www.who.int/choice/publications/d_OECD_prevention_report.pdf (accessed Nov 6, 2019).

25 Sassi F, Devaux M, Cecchini M, Rusticelli E. The obesity epidemic: analysis of past and projected future trends in selected OECD countries. 2009. http://www.oecd.org/officialdocuments/publicdisplaydocumentpdf/?doclanguage=en&cote=delsa/hea/wd/hwp(2009)3 (accessed Nov 6, 2019).

26 Cecchini M, Sassi F, Lauer JA, Lee YY, Guajardo-Barron V, Chisholm D. Tackling of unhealthy diets, physical inactivity, and obesity: health effects and cost-effectiveness. *Lancet* 2010; **376:** 1775–84.

27 Basu S, Seligman H, Bhattacharya J. Nutritional policy changes in the supplemental nutrition assistance program: a microsimulation and cost-effectiveness analysis. *Med Decis Making* 2013; **33:** 937–48.

28 Basu S, Vellakkal S, Agrawal S, Stuckler D, Popkin B, Ebrahim S. Averting obesity and type 2 diabetes in India through sugar-sweetened beverage taxation: an economic-epidemiologic modeling study. *PLoS Med* 2014; **11:** e1001582.

29 Mhurchu CN, Eyles H, Genc M, et al. Effects of health-related food taxes and subsidies on mortality from diet-related disease in New Zealand: an econometric-epidemiologic modelling study. *PLoS One* 2015; **10:** e0128477.

30 Basu S, Shankar V, Yudkin JS. Comparative effectiveness and cost-effectiveness of treat-to-target versus benefit-based tailored treatment of type 2 diabetes in low-income and middle-income countries: a modelling analysis. *Lancet Diabetes Endocrinol* 2016; **4:** 922–32.

31 OECD. The Heavy Burden of Obesity. 2019. https://www.oecd.org/health/the-heavy-burden-of-obesity-67450d67-en.htm (accessed Nov 6, 2019).

32 McPherson K, Marsh T, Brown M. Tackling obesities: future choices - modelling future trends in obesity & their impact on health. 2007. https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/295149/07-1662-obesity-modelling-trends.pdf (accessed Nov 6, 2019).

33 Cobiac LJ, Tam K, Veerman L, Blakely T. Taxes and subsidies for improving diet and population health in Australia: a cost-effectiveness modelling study. *PLoS Med* 2017; **14:** e1002232.

34 Popkin BM, Kim S, Rusev ER, Du S, Zizza C. Measuring the full economic costs of diet, physical activity and obesity-related chronic diseases. *Obes Rev* 2006; **7:** 271–93.

35 Martinez R, Fernandez A. Modelo de análisis del impacto social y económico de la desnutrición infantil en américa latina santiago, Chile: División de Desarrollo Social. CEPAL, Naciones Unidas. 2006 https://repositorio.cepal.org/handle/11362/5491 (accessed Nov 6, 2019).

36 Zeba AN, Delisle HF, Renier G, Savadogo B, Baya B. The double burden of malnutrition and cardiometabolic risk widens the gender and socio-economic health gap: a study among adults in Burkina Faso (West Africa). *Public Health Nutr* 2012; **15:** 2210–19.

37 Economic Commission for Latin America and the Caribbean (ECLAC) and World Food Programme. The cost of the double burden of malnutrition: social and economic impact. 2017. https://www.wfp.org/publications/2017-cost-double-burden-malnutrition-social-and-economic-impact (accessed Nov 6, 2019).

38 Hawkes C, Ruel MT, Salm L, Sinclair B, Branca F. Double-duty actions: seizing program and policy opportunities to address malnutrition in all its forms *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32506-1.

39 Bhutta ZA, Das JK, Rizvi A, et al. Evidence-based interventions for improvement of maternal and child nutrition: what can be done and at what cost? *Lancet* 2013; **382:** 452–77.

40 Swinburn BA, Kraak VI, Allender S, et al. The global syndemic of obesity, undernutrition, and climate change *Lancet* 2019; **393:** 791–846.

41 Shekar M, Kakietek J, Dayton Eberwein J, Walters D. An investment framework for nutrition. 2016. https://openknowledge.worldbank.org/handle/10986/26069 (accessed Nov 6, 2019).

42 Htenas AM, Tanimichi-Hoberg Y, Brown L. An overview of links between obesity and food systems: Implications for the food and agricultural global practice agenda. 2017. http://documents.worldbank.org/curated/en/222101499437276873/An-overview-of-links-between-obesity-and-food-systems-implications-for-the-agriculture-GP-agenda (accessed Nov 6, 2019).

43 WHO. The double burden of malnutrition: policy brief. 2016. https://www.who.int/nutrition/publications/doubleburdenmalnutrition-policybrief/en/ (accessed Nov 6, 2019).

44 Shrimpton R, Mbuya NV, Provo AM. The double burden of malnutrition in East Asia and the Pacific: evidence and lessons for a multisectoral response. World Bank; 2016.

45 World Cancer Research Fund International. NOURISHING framework. https://www.wcrf.org/int/policy/nourishing/our-policy-framework-promote-healthy-diets-reduce-obesity (accessed July 20, 2018).

46 High Level Panel of Experts. Nutrition and food systems. A report by the High Level Panel of Experts on Food Security and Nutrition. 2017. http://www.fao.org/3/a-i7846e.pdf (accessed Nov 6, 2019).

47 African Development Bank. Synthesis of Evidence of Multisectoral Approaches for Improved Nutrition. 2017. https://www.afdb.org/fileadmin/uploads/afdb/Documents/Generic-Documents/Banking_on_Nutrition_evidence_synthesis_advanced_copy_November_2017.pdf (accessed Nov 6, 2019).

48 WHO. Double-duty actions. Policy brief. 2017. https://www.who.int/nutrition/publications/double-duty-actions-nutrition-policybrief/en/ (accessed Nov 6, 2019).

49 Nugent R, Brouwer E, Cravioto A, Koehlmoos T. A cost-benefit analysis of a National Hypertension Treatment Program in Bangladesh. *Prev Med* 2017; **10:** S56–61.

50 Horton S, Shekar M, Ajay M. Scaling up nutrition: What will it cost? 2009. http://documents.worldbank.org/curated/en/655431468163481083/Scaling-up-nutrition-what-will-it-cost (accessed Nov 6, 2019).

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019

51 Hoddinott SHaJ. Benefits and costs of the food and nutritoin targets for the post-2015 development agenda. In: Center CC, ed. Prioritizing development: a cost benefit analysis of the United Nations' sustainable development goals. Cambridge: Cambridge University Press, 2018.

52 Jha P, Nugent R, Verguet S, Bloom D, Jamison D. Chronic disease prevention and control. In: Lomborg B, ed. Global problems, smart solutions. Cambridge: Cambridge University Press for the Copenhagen Consensus Center; 2013: 137–85.

53 Nugent R, Brouwer E. Benefits and costs of the noncommunicable disease targets for the post-2015 development agenda. In: Center CC, ed. Prioritizing development: a cost benefit analysis of the United Nations' sustainable development goals. Cambridge: Cambridge University Press; 2018.

54 Copenhagen Consensus Center. 2019. https://www.copenhagenconsensus.com/ (accessed July 29, 2019).

55 Sekiyama M, Roosita K, Ohtsuka R. Locally sustainable school lunch intervention improves hemoglobin and hematocrit levels and body mass index among elementary schoolchildren in rural west Java, Indonesia. *Nutrients* 2017; **9:** 868.

56 Powell CA, Walker SP, Chang SM, Grantham-McGregor SM. Nutrition and education: a randomized trial of the effects of breakfast in rural primary school children. *Am J Clin Nutr* 1998; **68:** 873–79.

57 Gleason PM, Dodd AH. School breakfast program but not school lunch program participation is associated with lower body mass index. *J Am Diet Assoc* 2009; **109** (suppl 2): S118–28.

58 Jamison DT, Summers LH, Alleyne G, et al. Global health 2035: a world converging within a generation. *Salud Publica Mex* 2015; **57:** 444–67.

59 Gelli A, Daryanani R. Are school feeding programs in low-income settings sustainable? Insights on the costs of school feeding compared with investments in primary education. *Food Nutr Bull* 2013; **34:** 310–17.

60 Popkin BM, Corvalan C, Grummer-Strawn LM. Dynamics of the double burden of malnutrition and the changing nutrition reality. *Lancet* 2019; published online Dec 15. https://doi.org/10.1016/S0140-6736(19)32497-3.

61 Hammond RA, Ornstein JT. A model of social influence on body mass index. *Ann NY Acad Sci* 2014; **1331:** 34–42.

Strictly embargoed until 23.30 UK time/6.30pm New York time on Sunday 15th December 2019