

Double Burden of Malnutrition at the Individual Level

The frequent co-occurrence of undernutrition and nutrition-related cardiometabolic risk

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Key messages

- > The phenomenon of the double burden of undernutrition and overnutrition reflects a particularly rapid nutrition transition in low-income countries, with compounded health consequences.
- > The term ‘dysnutrition’ could be used instead of under- and overnutrition to encompass all forms of malnutrition, since for the general population and even for professionals ‘malnutrition’ usually means global undernutrition, while overnutrition conveys the notion of overeating, which is not always the case.
- > The main phenotype of the double burden is the co-occurrence of child stunting/underweight and adult overweight/obesity, at country or household level.
- > The double nutritional burden also exists at the individual level in the form of overweight/obesity combined with stunting or micronutrient deficiencies, or else, nutrition-related cardiovascular risk factors other than obesity combined with stunting or micronutrient deficiencies.
- > The double nutritional burden needs to be understood and addressed through a life course continuum, and improving diet quality at all ages is crucial.

Background: main features of the double burden of malnutrition

The double burden of malnutrition is defined by the World Health Organization (WHO) as the coexistence of undernutrition along with overweight/obesity or other nutrition-related noncommunicable diseases (NCDs). ‘Undernutrition’ encompasses stunting, wasting or thinness, as well as specific micronutrient deficiencies. ‘Overnutrition’ refers primarily to overweight or obesity. Overnutrition-related conditions or cardiometabolic risk factors other than obesity include high blood pressure, hyperglycemia and diabetes and at-risk blood lipid profile. These conditions cluster as the metabolic syndrome.

The dichotomy of undernutrition- and overnutrition-related conditions is no longer relevant. Nutrition-related NCDs such as diabetes and cardiovascular disease (CVD) are rapidly rising everywhere while undernutrition is still highly prevalent, particularly in low-income countries or regions. Urbanization as a major driver of the nutrition transition is occurring most rapidly in low-income countries, and this very rapid nutrition transition is largely responsible for the double burden now observed.¹ Food system and environmental changes are undoubtedly a major culprit.

“Urbanization as a major driver of the nutrition transition is largely responsible for the double burden now observed”

Undernutrition- and overnutrition-related conditions are not as antithetical as they may appear. There is a biological link between undernutrition *in utero* or in infancy and the risk of obesity, diabetes and hypertension later on in life, according



A busy street in Ghana

to the theory of developmental origins of chronic diseases that evolved from the Barker hypothesis.² The theory provides one explanation for the co-occurrence of, say, stunting and obesity within individuals. Similarly, at household level, mothers are more prone to be overweight or obese in an obesogenic environment, particularly if they themselves had stunted growth, which in turn restricts fetal growth and may result in stunting in their progeny.

Instead of speaking of the double burden of 'malnutrition,' we advocate the use of the double burden of 'dysnutrition' to encompass both undernutrition and overnutrition³ for two main reasons:

1. For the general population, and even for professionals, 'malnutrition' usually means global undernutrition. For instance, the strategy of community management of acute malnutrition (CMAM) only refers to undernutrition; and
2. 'Overnutrition' is a misnomer, since excess intake of some macronutrients is often combined with inadequate intake of micronutrients and since nutrition-related NCD risk is not forcibly linked with overeating and obesity, while this is conveyed by the term 'overnutrition.'

The double nutritional burden is observed at country, household and individual level. The principal phenotypes that have been studied are the following:

- > Overnutrition and undernutrition at country level, usually in the form of a ratio, whether considering all age groups or only obesity in adults and undernutrition in children.^{4,5} Global ratios are of the order of 4, and as expected, the ratios are much lower in low-income compared to middle- or higher-income countries, ranging from 1.1 to 15.6.
- > Overweight/obesity in adults and stunting or underweight in under-5 children, at household level. The combination of an overweight mother with an undernourished child (stunted or underweight) has been most studied.⁶
- > Obesity and anemia at individual level in adults, primarily in women. Three components of the double burden in households and individuals of sub-Saharan Africa were examined in the light of Demographic and Health Survey (DHS) data, anemia and overweight in women and stunting in under-5 children.¹ Both types of double burden were more prevalent in urban and peri-urban areas, although the odds of maternal anemia and of child stunting were higher in rural areas.

A growing issue is now the 'triple threat' of stunting, anemia and obesity, whether at the population or individual level, as revealed in a preview of the 2018 Global Nutrition Report.^a Perhaps with the exception of obesity combined with anemia in women, the double nutritional burden at the individual level has been little studied and is less well understood than that ob-

served at country or household level. The present paper focuses on the double nutritional burden within individuals.

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“A growing issue is now the ‘triple threat’ of stunting, anemia and obesity”

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The double burden at the individual level

Various phenotypes may be observed at the individual level, including the co-occurrence of obesity with stunting or micronutrient deficiencies and the combination of undernutrition (stunting, underweight or micronutrient deficiencies) with markers of CVD risk other than obesity.

Co-occurrence of stunting and obesity

This phenotype of the double nutritional burden has been studied particularly among children.⁷ It was first reported in Latin America and in Chile, where it was observed that between 1987 and 2002, the association of child stunting with obesity increased faster than that of child tallness and obesity, reflecting the nutrition transition.⁸

Children may be at the same time stunted and overweight/obese for several reasons. Their diet may be deficient in those nutrients required for growth in height, for instance the type 2 nutrients according to Golden (e.g., protein and zinc), while providing excessive amounts of energy. Another explanation is that stunting dates back to undernutrition *in utero* or in early infancy without catch-up growth before the age of two years. It is now well established that early undernutrition increases subsequent risk of obesity, particularly abdominal obesity and NCDs.⁹

Even during childhood and not only during adolescence and adulthood, stunting was associated with higher blood pressure, as shown in Brazilian children.¹⁰ Stunted children have proportionally more body fat and less muscle mass, possibly owing to impaired fat oxidation.¹¹

Childhood undernutrition and adult ‘overnutrition’ are to be understood as a continuum, the former programming the latter. A review of cohort studies in low- and middle-income countries (LMICs) showed that a higher birthweight combined with higher linear growth in the first two years of life resulted in significant adult height gains and conferred some protection against NCDs.¹² Conversely, lower birthweight and poor neonatal growth are associated with lower lean body mass in adulthood.¹⁴ This suggests that suboptimal lean body mass associated with undernutrition early in life may predispose to fat accretion, and it can explain why higher birthweight is associated with lower CVD risk.

Obesity associated with micronutrient deficiencies

Obesity alters a number of metabolic pathways, and it is therefore not surprising that it is associated with poor status in several micronutrients. Obesity and anemia are causally related, but this may not be the case for other obesity-associated micronutrients. Obesity-linked inflammation impairs iron absorption, through its stimulation of the synthesis of hepcidin, which regulates iron absorption. In Mexico, for example, based on a national nutrition survey, it was shown that obese women and children were at increased risk of iron-deficiency anemia owing to inflammation; iron intake of non-obese and obese subjects was not different.¹⁴ The within-subject double burden of obesity and anemia exacerbates the gender inequalities, as both conditions are more highly prevalent in women than in men, as shown in North and West Africa.¹⁵⁻¹⁷

Obesity has been reportedly associated with other micronutrient deficiencies, including zinc, vitamin A and vitamin D.⁷ Relative to vitamin A deficiency, obesity would lead to reduction of tissue but not circulating levels of the vitamin.¹⁸ As regards vitamin D status, a graded and inverse relationship with body mass index (BMI) has been reported.¹⁹ This is not unrelated to unfavorable metabolic phenotypes (insulin resistance, type 2 diabetes and CVD) that have been observed in vitamin D deficiency or suboptimal status as evidenced by low serum 25(OH)D concentrations. It has been suggested that vitamin D is sequestered in body fat compartments, leading to its reduced utilization.¹⁹ In populations where these nutrients are in short supply, obesity may exacerbate the deficiencies.

Undernutrition and diabetes

WHO used to recognize a distinct type of diabetes associated with undernutrition, but this category no longer exists. However, reports from Ethiopia²⁰ and India²¹ showed that undernutrition was present in a high proportion of young insulin-dependent diabetic subjects and called for reopening the case of malnutrition-related diabetes. It should also be remembered here that fetal or infancy undernutrition is associated with a higher risk of diabetes in adulthood, owing to the developmental origins of chronic disease.²

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Micronutrient deficiencies such as vitamin E, zinc and vitamin D have been postulated to contribute to, or be associated with, diabetes. However, whether the deficiency or suboptimal status is causally related to the disease or is only associated with it is not as yet unraveled. Supplements of vitamin E²² and zinc^{23,24} improved the metabolic control of diabetes, and zinc supplements also improved the lipid profile and blood pressure. Vitamin B₁₂ deficiency has also been suspected, but there is only very limited evidence that this deficiency predisposes to diabetes or CVD.²⁵

Co-occurrence of stunting and cardiometabolic risk other than obesity

Without corrupting the concept, the double nutritional burden may also take other forms in which undernutrition combines with nutrition-related cardiometabolic disease or risk markers, but in the absence of obesity. This is so for undernutrition-related diabetes. As mentioned above, even mild stunting was associated with higher blood pressure in children and adolescents of a low-income region of Brazil.¹⁰ The stunted subjects also had higher levels of body fat than non-stunted subjects.²⁶ In urban Burkina Faso, our group showed that the double nutritional burden was widespread even among adults exempt from a prior diagnosis of diabetes or CVD. More than 20% of the subjects presented at least one deficiency sign and one cardiometabolic risk marker.¹⁷ The undernutrition signs were low BMI, iron deficiency or anemia and low serum retinol; the nutrition-related CVD risk markers were those of the metabolic syndrome. Of concern, the double burden was more frequent in women than in men, and in subjects of lower rather than upper socioeconomic status.

Poor antioxidant nutrient status and CVD

Food antioxidants, which include vitamins C, D and E, as well as other phytoactive substances not considered as vitamins such as carotenoids and flavonoids, increase serum antioxidant activity and decrease oxidative stress, thereby protecting against cancer and CVD. Not all antioxidants have the status of nutrients, however, which requires that they be defined with a dietary reference intake.²⁷ Further research is needed to uncover the optimal intake and circulating levels of antioxidants to act against CVD and other NCDs so that they acquire the nutrient status.

Supplements of various antioxidants have been shown to reduce CVD risk. However, the positive effect of supplements does not mean that the study subjects were deficient: low status for a given micronutrient is distinct from a deficiency status. Optimal micronutrient intake, supplement dosage, or circulating levels may still be undefined.

It is puzzling that the positive effects of antioxidants are primarily observed when these are ingested in foods or beverages, in particular fruits and vegetables, while supplements have gen-

erally not proved effective.²⁸ Low intake of fruits and vegetables could be regarded as a deficiency state of a sort.

The association of vitamin C with CVD risk has been the subject of several studies.²⁹ A better vitamin C status was linked in some studies to improvements in lipid profiles, arterial stiffness and endothelial function, while other studies have not confirmed these results. However, dietary but not supplementary vitamin C was inversely related to CVD. Similarly, in the CARDIA longitudinal study, dietary and plasma vitamin C were inversely related to high blood pressure while supplemental vitamin C was not.³⁰

A low Omega-3 Index (blood levels of EPA + DHA in erythrocytes) has also been found to be associated with CVD risk.³¹ However, the effects of supplements and the connection of dietary omega-3 fatty acids with blood levels have been inconsistent, owing primarily to variations in bioavailability and baseline status.

Similarly, low vitamin D status was found to be associated with CVD risk and with the metabolic syndrome, but the effects of supplements were not conclusive.^{32,33}

Deficiencies of vitamins involved in one-carbon metabolism and CVD

DNA methylation is the principal epigenetic mechanism through which environmental factors such as diet may impact or prevent CVD.³⁴ S-adenosyl methionine is the methyl donor molecule the production of which is affected by several nutrients including amino acids and vitamins, particularly folate, vitamin B₁₂ and vitamin B₆. Folate and vitamin B₁₂ are involved in the conversion of homocysteine to methionine. Homocysteine is also converted to cysteine and vitamin B₆ is required as coenzyme. It has therefore been speculated that a deficiency in one of these nutrients would lead to high circulating homocysteine, and affect DNA methylation and thereby CVD.³⁵ In vitamin B₁₂ deficiency with adequate folate status, folate becomes trapped in 5-methyl tetrahydrofolate (5-MTHF) and cannot participate in further reactions. High levels of circulating homocysteine (and low folate) have been suspected of contributing to CVD and have been observed in prediabetics.³⁶

The principal phenotypes of the double nutritional burden at the individual level that were described above are summarized in **Table 1**.

Conclusion: Implications for action

Not all individuals respond similarly to similar diets and lifestyles given the influence of genetic and epigenetic factors, as well as the microbiota. Nonetheless, diet quality is at the core of obesity and the dual burden of malnutrition,³⁷ and therefore improving diet quality appears crucial in all age groups. The interrelationships of dietary components with epigenetic changes and CVD risk require further research, however.

TABLE 1: Principal phenotypes of the double nutritional burden at the individual level

Undernutrition, suboptimal nutrient status	Combined with:
Undernutrition: stunting	> Obesity > High blood pressure
Undernutrition: stunting or underweight	> Diabetes
Iron-deficiency anemia	> Obesity
Other micronutrient deficiencies	
Low status for antioxidant nutrients	> Higher CVD risk – metabolic syndrome
Low status for vitamins involved in one-carbon metabolism	



Schoolchildren involved in the NFSI program in Cotonou, Benin

“Diet quality is at the core of obesity and the dual burden of malnutrition, and therefore improving diet quality appears crucial in all age groups”

Much like for understanding the double nutritional burden, a life-course approach is required for appropriate action, with interventions targeting all age groups. Strategies to address the double burden, including WHO’s ‘double duty,’ were discussed in other papers. The fact that higher birthweights and improved linear growth during the first two years of life are associated with gains in adult height (and schooling) and confer some protection against NCDs¹² supports the present focus on the first 1,000 days, from conception to the age of two years. School-age children are also a priority target group, and the Nutrition-Friendly School Initiative (NFSI) advocated by WHO

as a means of preventing the double nutritional burden is also highly relevant, as we showed in capital cities of Burkina Faso and Benin, although on a small scale.^{38,39}

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Notes

- ^a www.devex.com/organizations/food-and-agriculture-organization-of-the-united-nations-fao-44117