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Micronutrient Malnutrition, Obesity, and Chronic Disease in Countries Undergoing the Nutrition Transition: Potential Links and Program/Policy Implications

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Abstract

Background: The nutrition transition occurring in many developing countries may invite the misconception that diets are moving entirely away from undernutrition toward problems of excess. But despite the sufficiency of energy in these countries, diet quality is poor and micronutrient deficiencies often remain. In this context, micronutrient deficiencies may actually contribute to the development and severity of diet-related chronic diseases.

Objectives: This paper discusses the potential long-term effects of micronutrient malnutrition in early childhood on obesity and related disease outcomes. The links between early micronutrient malnutrition, stunting, and subsequent short adult stature—emerging risk factors for obesity and associated chronic diseases—are reviewed. This paper also explores recent literature linking micronutrient malnutrition in adults to increased risk and severity of chronic disease. Finally, this paper discusses the program and policy implications of these relationships.

Methods: Literature searches on the topics of interest were conducted in Medline. This paper is not the result of a systematic literature review, but rather discusses relevant literature to bring attention to links between under- and over-nutrition that have not been widely considered.

Conclusions: In children, micronutrient malnutrition is a cause of stunting and may be accompanied by metabolic adaptations that increase the risk of later obesity and related disease. In adults, deficiencies in key micronutrients may promote oxidative stress, folate deficiency may increase risk for heart disease, and zinc deficiency may be exacerbated in the presence of diabetes while also affecting glucose transport. Low fruit and vegetable consumption may additionally increase the risk of cardiovascular disease (CVD) and cancer through a variety of mechanisms. The importance of supporting

programs and policies that address the spectrum of malnutrition, including micronutrient malnutrition and emerging obesity together, is stressed.

Key words: diet quality, micronutrient malnutrition, obesity, chronic disease, cancer, adults, children

Contents

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1. Introduction

In many developing countries affected by rapid globalization, industrialization, and urbanization, obesity and diet-related chronic diseases such as diabetes and cardiovascular disease (CVD) are emerging as important health concerns. For example, the combined prevalence of overweight and obesity in Mexico rose by 78 percent from 1980 to 1998, and the mortality rates from diabetes mellitus and high blood pressure rose 62 percent and 55 percent, respectively, over the same period (Rivera et al. 2002). The disability-adjusted life years lost to noncommunicable disease risk factors (such as high blood pressure, overweight, and sedentary lifestyles) in the Americas, excluding the United States and Canada, are now almost three times the amount lost to childhood and maternal undernutrition (Jacoby 2004). Similar situations exist in other areas of the world as well, including countries such as China, Egypt, and South Africa (Gu et al. 2005; Galal 2002; Puoane et al. 2002). The World Health Organization estimates that 66 percent of deaths due to chronic disease worldwide now occur in developing countries and that obesity is a primary risk factor in these contexts (WHO 2004). And within such countries, the poor are increasingly affected as development progresses (Monteiro et al. 2004).

This move toward obesity and related chronic diseases in many developing countries invites the misconception that diets are shifting entirely away from problems of constraint toward problems of excess. However, the nutrition transition occurring in such countries, while associated with diets higher in energy, is also marked by poor diet quality. Thus, while energy may be plentiful, it increasingly comes from energy-dense sources such as added sugars and edible oils, while intakes of complex carbohydrates, fiber, and micronutrient-rich foods such as fruits and vegetables and animal-source foods are often low (Bermudez and Tucker 2003; Murphy and Allen 2003; Drewnowski and Popkin 1997).

In fact, even as wasting and underweight are replaced by rising rates of obesity, affecting women first and later children and men as development progresses,

micronutrient malnutrition remains a significant public health problem in many transitional countries undergoing the nutrition transition. For example, the prevalences of iron and zinc deficiency in a 1998 nationally representative sample of Mexican women were 40 percent and 30 percent, respectively, based on serum concentrations, while the prevalence of overweight and obesity (BMI ≥ 25) was greater than 50 percent (Rivera et al. 2002; Villalpando et al. 2003). The prevalence of anemia (an indicator of iron-deficiency) in Peru and Egypt from nationally representative data in 2000 was 32 percent and 28 percent, respectively. At the same time, the prevalence of overweight and obesity was greater than 50 percent in Peru and was greater than 77 percent in Egypt (DHS 2005).

It is likely that these micronutrient deficiencies coexist with overweight and obesity for the following reasons. First, obese adults and stunted children live within the same households in a variety of transitional countries (Garrett and Ruel 2005). This suggests that micronutrient deficiencies, which are strongly associated with childhood stunting, remain, at least among children, even while energy is clearly available in excess. Second, there is evidence that obese individuals, particularly those living in poverty, may simultaneously be affected by iron deficiency in both developed and developing countries contexts. For example, an analysis of the National Health and Nutrition Examination Survey III (1988-1994) in the United States found that children (2-16 years of age) who were overweight or at risk of being overweight were about twice as likely to be iron-deficient as children who were not overweight (Nead et al. 2004). And an analysis of Andhra Pradesh from the nationally representative National Family Health Survey (1998/1999) in India found that, while the prevalence of anemia was lower in overweight than in underweight and normal weight women, the rate of anemia in the overweight group was still quite high at 41 percent (Bentley and Griffiths 2003). Third, the association of food insecurity[1] with obesity has been demonstrated in several studies,

[1] Food insecurity is defined as the "limited or uncertain availability to acquire acceptable foods in socially acceptable ways," a concept that includes problems of insufficient diet quality (Carlson, Andrews, and Bickel 1999; Alaimo et al. 1998).

primarily in the United States (Adams, Grummer-Strawn, and Chavez 2003; MMWR 2003; Oh and Hong 2003; Townsend et al. 2001), and recent research in the United States demonstrates that micronutrient-poor obesogenic diets may be common among the poor because they are more affordable than diets of high quality (Drewnowski and Darmon 2005; Drewnowski and Specter 2004). More research is needed to verify these connections in developing country contexts, but the overlap of stunting and overweight/obesity within households and the coexistence of micronutrient malnutrition and overweight/obesity within individuals in developing countries provides strong preliminary evidence that obesity does occur in the presence of food insecurity. And finally, since micronutrient deficiencies tend to cluster within individuals, people who are both overweight and anemic are likely to be deficient in other micronutrients as well.

Micronutrient malnutrition in the context of the nutrition transition presents unique concerns. While the most commonly cited consequences (such as stunting, impaired cognitive development, increased morbidity and mortality in children, and increased maternal morbidity) remain, micronutrient malnutrition may have additional deleterious health consequences when combined with obesity and related chronic diseases.

This review paper discusses several links between micronutrient deficiency, obesity, and obesity-related chronic diseases. First, we consider the potential long-term effects of micronutrient malnutrition in early childhood. We focus on the links between early micronutrient malnutrition, stunting, and subsequent short adult stature—emerging risk factors for obesity and associated chronic diseases in the nutrition transition context. Second, we link micronutrient malnutrition in adults to increased risk and severity of diabetes, CVD, and cancer. We discuss links between deficiencies in antioxidant micronutrients and chronic disease, the potential for zinc deficiency to exacerbate diabetes due to its structural relationship with insulin, the relationship between folate deficiency and increased homocysteine levels—an established risk factor for CVD, and the association between low fruit and vegetable intakes and increased risk for CVD and cancer. We conclude by discussing the public health and policy implications of these

links in the context of developing countries undergoing the nutrition transition, and recommend directions for future research.

This paper is not the culmination of a systematic literature review, but was written as a conceptual piece to draw attention to a variety of potential links between under- and over-nutrition in the context of the nutrition transition. The literature presented was identified through a search of pertinent topics on Medline, and papers were selected due to content relevant to the ideas presented.

2. Micronutrient Malnutrition in Childhood May Indirectly Increase Risk of Later Obesity and Chronic Disease via Stunting

Micronutrient malnutrition contributes to stunting

Along with established risk factors such as frequent infection and poor weaning foods, there is strong evidence that several micronutrients (primarily zinc, iron, and vitamin A) play important roles in linear growth, and that deficiencies in these key nutrients may result in stunting (Rivera et al. 2003). Zinc directly influences the growth hormone and insulin-like growth factor-I systems (Dorup and Clausen 1991), affects bone metabolism (Nishi 1996), and is involved in DNA synthesis (Clausen and Dorup 1998; Bunce 1994). Zinc and vitamin A affect immune function (Black 2003; Black and Sazawal 2001; Black 1998; Underwood and Arthur 1996; West et al. 1991), and thus risk of morbidity and associated growth faltering. And, zinc and iron deficiencies can result in anorexia, leading to decreased intakes of all nutrients, which can also limit growth (Clausen and Dorup 1998; Lawless et al. 1994). A meta-analysis of zinc supplementation trials found positive linear growth effects in 25 out of 33 studies reviewed, indicating strong evidence that zinc deficiency results in linear growth faltering (Brown et al. 2002). Reviews of iron and vitamin A supplementation trials did not reveal consistent results, but did show significant positive effects on linear growth in the context of severe deficiencies (Rivera et al. 2003). Because multiple micronutrient deficiencies often exist within individuals, it has been suggested that inconsistent results with regard to growth

effects in single micronutrient supplementation trials may be due to the presence of other growth-limiting micronutrient deficiencies in the study populations rather than a true absence of effect (Allen 1994). Indeed, a meta-analysis of multiple micronutrient supplementation trials does show consistent effects on linear growth (Ramakrishnan et al. 2004).

Macronutrient deficiencies in protein and energy may also affect linear growth. However, energy supplementation trials among stunted children have had inconsistent effects, which again may be because growth-restricting micronutrient deficiencies are likely to remain in poor populations despite the provision of supplemental energy (Allen 1994). While some protein supplementation trials have had small effects on linear growth, it is difficult to distinguish whether the benefits in these cases were related strictly to the increased intake of protein, or were due to concomitant increases in intakes of growth-promoting micronutrients, such as zinc and vitamin A, that are common components of protein-rich foods (Allen 1994). Because stunted linear growth remains widespread in countries undergoing the nutrition transition, where energy supply is generally not constrained, it is reasonable to assume that micronutrient deficiencies are likely to be the main growth-limiting nutritional influences in these settings.

Early childhood stunting may increase the risk of later obesity

Obesity rates are rising rapidly in countries undergoing the nutrition transition despite continued high prevalences of childhood stunting, an indicator of chronic undernutrition. While it may seem paradoxical, there is growing evidence that undernutrition in early life may predispose individuals toward later obesity. An association between stunting and overweight/obesity has been demonstrated in children in low-income urban population in Brazil (Sawaya et al. 1995), and in nationally representative surveys from Russia, China, Brazil, and South Africa (Popkin, Richards, and Montiero 1996). The association of short adult stature, a result of childhood stunting, and obesity was also seen among women in urban Brazil (Florencio et al. 2001). While

several other studies did not find this association, a study in Guatemala observed a link between childhood stunting and adult abdominal fatness, a risk factor for chronic disease (Schroeder, Martorell, and Flores 1999), and another in Jamaica noted the association of both childhood stunting and low birthweight with increased central fat distribution in adolescence (Walker et al. 2002). In a review of early nutrition and later adiposity, Martorell, Stein, and Schroeder (2001) noted the possibility that stunting might predispose individuals to later obesity, but that this association may only be expressed if individuals are exposed to the energy-dense diets that are seen in countries undergoing the nutrition transition.

The premise for the observed relationship between stunting and obesity is the early origins hypothesis, originally put forward by Barker (1990), which suggests that nutrient deficiencies in early childhood "program" the body to make permanent metabolic and other physiological adaptations in response to nutrition constraints, and that this can lead to obesity in the context of later abundant energy availability (McMillen and Robinson 2005). Several specific physiological mechanisms have been suggested and tested among a group of children in Brazil, revealing that stunted children have impaired fat oxidation—a predictor of obesity, that they may have increased susceptibility to weight-gain effects from high-fat diets, and that they may not properly regulate food intake (Hoffman et al. 2000a, 2000b; Sawaya et al. 1998).

Early childhood stunting and related short adult stature may increase the risk of developing chronic disease

The process of stunting takes place during early childhood when nutrient deficiencies impair linear growth during the period of maximum growth velocity, and after which recovery is rarely seen (Martorell, Khan, and Schroeder 1994). Thus, stunting in early childhood generally leads to short adult stature (Rivera et al. 1995). Short adult stature has been associated with CVD in a variety of studies in developed world populations (Rich-Edwards et al. 1995; Kannam et al. 1994; Hebert et al. 1993). Evidence from countries undergoing the nutrition transition is also emerging. In Brazil,

women in the lowest quartile of height had significantly higher odds of hypertension than women in the highest quartile, controlling for other risk factors (Sichieri et al. 2000). A study in Bangladesh identified short stature and high body mass index as independent risk factors for glucose intolerance in both men and women (Sayeed et al. 1997), and a study in Mexico found that short stature was associated with a higher prevalence of both Type II Diabetes and arterial hypertension in men and women after adjusting for potential confounders (Lara-Esqueda et al. 2004). There is also some evidence for the early emergence of chronic disease risk factors among stunted children. For example, a study among Jamaican children showed that stunting during the first two years of life was associated with an increased risk of high systolic blood pressure by 7-8 years of age, controlling for confounders (Gaskin et al. 2000). Again, stunting and subsequent short stature are thought to be associated with chronic diseases such as diabetes and hypertension because they are indicators of childhood malnutrition, which may trigger changes in physiological development that increase chronic disease risk factors (McMillen and Robinson 2005). Short stature may also increase the risk of hypertension due to the physics of resistance to blood flow in short vessels (Sichieri et al. 2000).

Thus, micronutrient malnutrition may be indirectly contributing to the risk of overweight/obesity and chronic diseases via its relationship with childhood stunting and subsequent short adult stature, which, in turn, increase the risk for overweight/obesity and chronic disease in the context of the nutrition transition.

3. Micronutrient Malnutrition in Adulthood May Increase the Risk and Severity of Diabetes, CVD, and Cancer

Micronutrient malnutrition may contribute to Type II Diabetes

In order to better understand the links between micronutrient malnutrition and Type II Diabetes, it is important to first review the pathophysiology of the disease. In non-diabetic normal individuals, insulin is produced and stored in the pancreatic beta cells and is released in response to glucose entering the blood. Insulin then binds to cell

receptors and facilitates the uptake of glucose across cell membranes. In the initial phases of Type II Diabetes, the pancreatic islet cells that produce insulin function normally, but there is failure of the intracellular events that activate glucose transport following signal by the insulin/receptor complex. This results in hyperglycemia. The pancreas responds by producing increasing amounts of insulin, which results in down-regulation of insulin receptors, exacerbating the problem. The beta cells of the pancreas, in their inadequate efforts to create enough insulin to offset the high amount of glucose in the blood, weaken as the disease progresses, resulting in even greater hyperglycemia (Chausmer 1998).

There is ample evidence that oxidative stress is increased with diabetes, and it is implicated in the progression of virtually all of the complications of diabetes (Thompson and Godin 1995; Baynes and Thorpe 1999) such as atherosclerosis, retinopathy, neuropathy, and vascular complications affecting the heart (Thompson and Godin 1995; Halliwell and Gutteridge 1990; Pirart 1977). Oxidative stress may be increased among diabetics for several reasons. Because glucose is vulnerable to oxidation, hyperglycemia, the hallmark of diabetes, may lead to increased production of harmful reactive oxidative species (ROS) such as peroxides and free radicals (Hunt, Smith, and Wolff 1990; Wolff, Jiang, and Hunt 1991). The disposal and deactivation of these ROS is essential and occurs through the interaction of enzymes and non-enzymatic compounds (Thompson and Godin 1995). A variety of micronutrients play a role in these processes, thus deficiencies in these micronutrients may contribute to the development and progression of disease complications (Wolff and Dean 1987). While many micronutrients, including vitamin C and vitamin E, function as antioxidants, we focus our discussion on zinc, due to the high prevalence of zinc deficiency in the developing world context and due to additional unique links between zinc and diabetes.

Copper-Zinc superoxide dismutase is a zinc-containing antioxidant enzyme that works in the cytoplasm to break down superoxide ions (Thompson and Godin 1995). In addition to breaking down superoxide ions as an enzymatic compound, zinc also inhibits ROS production through competition with iron and copper for key binding sites on cell

membranes and some proteins, making free iron and copper more available for binding to ferritin and metallothionein, respectively, thus incapacitating their oxidative capacities. Zinc also binds to specific sites on proteins and directly protects those areas from oxidation (Bettger 1993). Dietary zinc deficiency in rats does not seem to affect levels of superoxide dismutase because this enzyme can function using other metal ions in place of zinc. However, increased oxidative and peroxidative damage has been noted in zinc-deficient rats due to low dietary intakes, perhaps due to some of the other antioxidant activities of zinc (Bettger 1993). Thus, it is possible that zinc deficiency could contribute to increased oxidative stress and associated increases in the complications from Type II Diabetes.

Zinc status and diabetes share several additional links beyond the antioxidant connection. The metabolism of zinc is compromised in the presence of diabetes. Hyperzincuria and hypozincemia are commonly seen, although the mechanism for the urinary loss is not fully understood (Chausmer 1998). Hypozincemia may reflect urinary losses, and may also be due to decreased absorption of zinc in the gut among diabetics (Kinlaw et al. 1983). Thus, zinc deficiency and its pro-oxidant effects may arise as a result of diabetes rather than acting as initiators of disease development (Chausmer 1998), making the causal pathway between zinc status and diabetes difficult to distinguish.

However, a mechanism by which prior zinc deficiency may contribute to the development and progress of diabetes has also been suggested. Zinc has a unique structural relationship with insulin. In the beta cells of the pancreas, zinc forms crystals with insulin for storage and release (Chausmer 1998) and free zinc also helps with glucose metabolism. The development and diagnosis of Type II Diabetes is generally preceded by hyperinsulinemia, with secretion of insulin high due a diminishing ability of insulin to adequately transport and deliver glucose from the blood to the cells. When free zinc levels are low, even more insulin is secreted to compensate, depleting cellular zinc in the pancreas as it departs in the form of insulin crystals. The hyperzincuria that comes with the development of diabetes further lowers the availability of zinc for re-uptake into

the pancreas cells. As a result of having to produce and release greater and greater amounts of insulin, the beta cells may become exhausted, and with cellular zinc depleted, may have increased vulnerability to oxidative damage, resulting in beta cell damage and eventual inability to supply sufficient insulin in later stages of the disease. Thus, it has been hypothesized that preventing zinc deficiency may contribute to protecting against the development and progress of diabetes, although research to test this hypothesis is needed (Chausmer 1998; Sprietsma and Schuitemaker 1994).

Micronutrient malnutrition may contribute to CVD and cancer

Folate, a micronutrient found largely in fruits and vegetables, appears to play a unique role in the development of heart disease. Folate is a co-substrate in homocysteine metabolism (Eichholzer et al. 2001), thus folate deficiency leads to high levels of blood homocysteine, an important risk factor for coronary heart disease (Verhoef, Stampfer, and Rimm 1998). In addition, folate has anti-inflammatory actions and enhances the synthesis of nitric oxide, which, when only present at reduced levels, is associated with impaired vasodilation and atherosclerosis (Das 2003). In fact, some now estimate that 10-25 percent of deaths from heart disease and stroke worldwide may be due to folate deficiency (UNICEF/The Micronutrient Initiative 2004).

In addition to playing a role in the development and complications from diabetes, oxidative stress is also associated with CVD. It has been proposed that oxidized low-density lipoprotein (LDL) cholesterol contributes to the development of atherosclerosis through a variety of mechanisms, including leading to loss of endothelieal integrity and enhancing the uptake of lipoproteins that lead to foam cell formation, thus supporting fatty streak and lesion formation (Stocker and Keaney 2004; Steinberg et al. 1989). However, clinical supplementation trials of antioxidant vitamins, such as vitamin E and vitamin C, have not resulted in consistent evidence of reduced CVD morbidity or mortality (Stocker and Keaney 2004; Madamanchi, Vendrov, and Runge 2005; Sachidanandam, Fagan, and Ergul 2005; Gaziano 2004). There are several different

theories for the lack of observed effect in these studies. For example, some propose that oxidative stress may not be a causal factor in the development of atherosclerosis, but that atherosclerosis and oxidative stress may both be consequences of inflammatory processes. It has also been suggested that clinical trials have focused too narrowly on only a few antioxidants, such as vitamin E, which only offer protection from certain types of oxidation, and that further studies are needed to explore the independent and joint effects of a wider range of antioxidants and other beneficial dietary components (Stocker and Keaney 2004).

While studies of single micronutrients have not provided consistent results, a large body of evidence shows that fruit and vegetable consumption is highly protective against the development of both CVD and cancer (Reddy 2002; Bazzano, Serdula, and Liu 2003; Liu et al. 2000; van't et al. 2000; IARC 2003). In fact, the World Health Organization estimates that low fruit and vegetable intake is responsible for 31 percent of ischemic heart disease and 11 percent of strokes, and ranks it as the sixth main risk factor for mortality worldwide (WHO 2002). Fruits and vegetables are rich in a wide variety of micronutrients and other dietary components, such as fiber and phytoestrogens, further suggesting that the joint effects of multiple nutrients may be more important than the actions of single nutrients. Fruits and vegetables contain high amounts folate and antioxidants, such as vitamin C, which have already been discussed in terms of CVD protection. Antioxidants may also modulate the endogenous formation of carcinogenic factors such as free radicals and reactive nitrogen species and thus modify cancer risk as well. A variety of other mechanisms and associations may further account for the protective effects of fruit and vegetable consumption. For example, Phase I and Phase II enzymes catalyze the formation of oxidative species, and catalyze conjugation reactions to improve their excretion, respectively. Thus these enzymes provide a balance between carcinogen activation and detoxification, and dietary components found in fruits and vegetables may act on these enzymes to tip the balance toward increased cancer protection (IARC 2003). Specific dietary components have also been found to have protective effects against specific types of cancer. For example, lycopene, found in

tomatoes, is associated with reduced risk of prostate cancer (Heber 2004). Thus, a wide variety of micronutrients and other dietary components, when ingested together via diets rich in fruits and vegetables, provide important protection against the development of CVD and various forms of cancer.

4. Discussion

This review highlights several potential links between micronutrient malnutrition and increased risk for obesity and related chronic diseases. In children, micronutrient malnutrition is a cause of stunting and may be accompanied by metabolic adaptations in response to nutrient deficiencies that increase the risk of later obesity and related chronic disease. In adults, deficiencies in key antioxidant micronutrients may promote oxidative stress, creating an environment in which the incidence and severity of diseases such as diabetes, CVD, and cancer are increased. Folate deficiency in adults may increase serum levels of homocysteine, an established risk factor for heart disease. In the presence of diabetes, disordered handling of key micronutrients, such as zinc, may worsen existing deficiencies, while the unique structural relationship of zinc with insulin may have specific implications for the development and severity of diabetes in the presence of zinc deficiency. Low fruit and vegetable consumption may additionally increase the risk of CVD and cancer through a variety of mechanisms linked with micronutrients and other dietary components.

These links are particularly salient in the context of developing countries undergoing the nutrition transition, where micronutrient deficiencies remain widespread, stunting is common, fruit and vegetable consumption is low, and obesity and related chronic diseases such as diabetes and heart disease are taking over as the leading causes of morbidity and mortality. Further research in several areas is needed to better understand the nature of the relationships between micronutrient malnutrition, overweight/obesity and chronic disease, and to improve interventions and policies to redirect the nutrition transition toward healthier outcomes in developing countries.

The mechanisms behind the association between childhood stunting and increased risk for overweight/obesity and chronic disease need to be elucidated

While many have noted that stunted children and subsequently short adults appear to be at increased risk for overweight/obesity and related chronic diseases, only a few have attempted to isolate the metabolic reasons for this link. Many studies focusing on micronutrient malnutrition, undernutrition, and stunting have shown that stunting is largely irreversible after the first 2-3 years of life (Martorell, Khan, and Schroeder 1994), which has focused efforts to prevent or reduce stunting on the periods of intrauterine and early growth. However, it is not clear whether, even if short stature endures, the metabolic changes that have been suggested as the cause of increased risk for later obesity and related disease are also irreversible. A greater understanding of the types of metabolic changes that occur with childhood stunting and which may contribute to future obesity and disease risk would help in designing effective life-cycle approaches to countering the epidemic of overweight/obesity and related diseases in countries undergoing the nutrition transition. Ideally, stunting should be prevented via good diet quality throughout the life cycle. However, if the metabolic changes that accompany stunting are not as permanent as short stature, then new interventions aimed specifically at reducing obesity and chronic disease might still be effective even after linear growth has slowed or ended, providing an additional window of opportunity.

The extent of micronutrient malnutrition and overweight/obesity overlap among adults, and the implications of this confluence need to be clarified

This paper has argued that micronutrient malnutrition and overweight/obesity are likely to overlap among adults and has suggested that this convergence may be exacerbating the rise in associated chronic disease in countries undergoing the nutrition transition. While studies of dietary patterns in transitional countries suggest that the overlap of these conditions is likely, little research has been done to show this

relationship. Forthcoming research using data from three countries at different stages of the nutrition transition shows that, in Mexico, for example, overweight/obese women are equally likely to be anemic as other women (Eckhardt et al. under review), but there is little other work that has been done on this issue. Research documenting the extent to which overweight/obesity overlaps with a variety of micronutrient deficiencies within individuals would provide additional insight into the state of diet quality in countries undergoing the nutritional transition. Additional analyses showing how the pattern of overlapping micronutrient malnutrition and overweight/obesity differs with respect to sociodemographic characteristics and diet patterns would help identify the sector of the population at risk both for undernutrition with respect to micronutrients and for overnutrition with respect to energy-dense obesogenic diets.

Established evidence for the overlap of micronutrient deficiencies and overweight/obesity and related chronic disease within individuals would also open other lines of research. This paper has described several ways in which micronutrient deficiencies might contribute to the development and severity of chronic diseases such as diabetes, CVD, and cancer. However, some of these relationships, such as the potential contribution of zinc deficiency toward the development and severity of diabetes, are still theories that need further substantiation. And, even for those more established relationships, such as that between folate deficiency and CVD, the magnitude of the micronutrient deficiency contribution, relative to genetic and other risk factors, is unclear. In the context of the nutrition transition where micronutrient malnutrition remains a serious public health problem along with emerging obesity and related chronic disease, the potential for links between the two could have widespread implications. At the very least, the coexistence of these two types of malnutrition in the same population presents a challenge to public health workers and policymakers. At the most, the presence of micronutrient malnutrition could be contributing in a meaningful way to the increase in overweight/obesity and related disease.

On a separate note, because diabetes causes disordered handling of several micronutrients such as zinc and vitamin C, which are commonly deficient in transitional

populations due to poor diet quality, the prevalence of deficiencies in these micronutrients may be influenced by concurrent disease in countries undergoing the nutrition transition. In Mexico, for example, where the prevalence of zinc deficiency is 30 percent among women (Villalpando et al. 2003), and where diabetes is on the rise and is often undiagnosed, it could be important to determine whether underlying diabetes may be contributing to zinc deficiency and to quantify the magnitude of this effect relative to low dietary intakes.

If the contribution of micronutrient malnutrition to chronic disease is significant, research into interventions to address this link would be warranted. For example, perhaps zinc supplementation, a common strategy for combating linear growth retardation in children, could be extended to include diabetic adults. Indeed, some studies of zinc supplementation in adults with Type II Diabetes have shown benefits, including significantly improved glucose disposal and decreased lipid peroxidation (Anderson et al. 2001; Marchesini et al. 1998). With regard to folate, pregnant women who are folate deficient are at increased risk of having babies with neural tube defects such as spina bifida. This known risk has prompted some countries to adopt programs for fortifying flour with folate. The role of folate in reducing homocysteine levels and potentially preventing heart disease and stroke, however, may not be as widely recognized. Many transitional countries only have voluntary, rather than mandatory, programs (such as the Dominican Republic and Kazakhstan), while others have no fortification program in place (such as Egypt) (UNICEF/The Micronutrient Initiative 2004). Governments and advocates of fortification may want to consider the effects of folate deficiency on the development of CVD in addition to neural tube defects as motivation for developing and sustaining fortification programs, and may also want to consider chronic disease outcomes in addition to the incidence of neural tube defects when attempting to measure program impacts.

The concept of diet quality must be adopted as the firm goal of all nutrition

Micronutrient malnutrition and overweight/obesity are not conditions occurring at opposite ends of the malnutrition spectrum. They are related conditions both with regards to cause (poor quality diets) and with regard to effects (the promotion of chronic disease). We strongly stress that the most important strategy for reducing all forms of malnutrition in countries undergoing the nutrition transition is the promotion of programs and policies that promote sustainable strategies to enhance diet quality among all people throughout the life cycle.

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