1. INTRODUCTION

The basic cause of malnutrition (protein-energy malnutrition [PEM] and micronutrient malnutrition) is poverty. Although lack of care, health, and environmental conditions are important components in the etiology of malnutrition, lack of access to food is still one of the major underlying causes of malnutrition. There is strong correlation between a country’s gross national product (GNP) and its level of malnutrition. However, the challenge facing nutritionists and other professionals in the field of health is to identify strategies to prevent malnutrition that are economically feasible.

Although originally protein was considered the main factor in the etiology of malnutrition (see Chapter 16), lack of energy was later understood to be as important as the underlying cause. In the last two decades, much work has been carried out in the field of micronutrient deficiencies. The role of vitamin A deficiency in children had a major impact on the role and implications of micronutrient deficiencies. The poor strata in developing countries have a lack of purchasing power and spend a large percentage of their income on staple food. Animal product and fruits that are important sources of micronutrients are often more expensive and unaffordable, and therefore, it can be expected that, multiple micronutrient deficiencies rather than singular deficiencies are common in these settings (1–3). The micronutrient deficiencies of concern are vitamin A, vitamin B complex, vitamin C, iron (FE), iodine, and zinc (Zn). Although reduced energy intake remains a problem in many settings, sub-optimal intakes of several micronutrients are more widespread and may be present even when energy needs are met.

Micronutrients can affect a variety of health and disease outcomes, for example, child growth and development via direct independent effects (e.g.: Zn, folic acid); and indirect effects through interactions with each other (e.g.: Vit A, Zn and Fe), and promoting appetite, which leads to increased food intake and thereby higher intake of other macro and micronutrients.

The conceptual framework that describes the mechanisms by which multiple micronutrient malnutrition can affect health and disease outcomes is shown in Fig. 1. Poor dietary quality and inadequate intakes are the underlying determinants of several
micronutrient deficiencies. For many micronutrients, bio-availability is affected by the mix of foods eaten, the presence of inhibitors, mode of preparation, and drug-diet interactions (4,5). Exceptions would be trace elements such as iodine and selenium; the concentrations of these nutrients in foods depend on the soil and/or water content of the region where they are grown or harvested. Infections such as diarrhea and parasitic infestations, which are common in these settings, can also contribute to multiple micronutrient malnutrition by influencing nutrient absorption, utilization, and excretion (6,7). However, these infections are also exacerbated by micronutrient deficiencies.

At the physiological and metabolic level, there is considerable evidence of several micronutrient interactions that may have the potential to affect outcomes such as child growth and development. The interactions are more important when nutrient intakes are inadequate. There is some evidence that suggests that multiple micronutrient deficiencies result in decreased appetite and anorexia, and these conditions disappear as soon as a dietary balance is restored (8,9).

Micronutrients can also interact at the level of nutrient absorption. For example, vitamin C is a known enhancer of iron absorption, whereas zinc and calcium can interfere with the absorption and availability of iron (10). The interrelationship between iron and zinc may also be bi-directional (11). In terms of nutrient utilization, studies have shown that vitamin A affects both iron and zinc metabolism (12,13). For example, zinc is required for the synthesis of retinol binding protein, which is required to transport vitamin A in the body. This interaction has been extensively studied in animal models and to a certain extent in young children (14–16). Vitamin A deficiency also inhibits iron