Nutrients play a critical role in the promotion of normal health and prevention of disease. It is, therefore, not surprising that malnutrition can be directly related to significant alterations in organ structure and function [1]. However, the effects of malnutrition appear to be greater in the fetus and during early infancy than later on in life.

Studies have suggested that growth is 'pre-programmed' to occur at a certain time or 'critical' epoch which, if missed, may not be recoverable [2]. Even short periods of nutritional deprivation may not only affect somatic but also brain growth and development [2]. However, poor fetal growth has also been related to altered 'metabolic programming' and the development of insulin resistance and metabolic syndrome X, an important consideration in these high-risk infants.

Concerns in term are greater in preterm infants. Forty percent of preterm infants are small-for-gestational age (SGA) at birth compared to 10% of term infants. Nutritional requirements are greater and nutritional deficiency is also more likely. The net effect is that up to a 100% of very-low-birthweight infants are SGA at hospital discharge [3]. Infants are also neurologically more vulnerable to the effects of perinatal ischemia and inflammation, therefore the development of periventricular-intraventricular hemorrhage and periventricular leukomalacia.

Fetal-postnatal malnutrition not only affects somatic growth but also organ growth, structure and function. In the study of Myers et al. [4], a 30% reduction in body weight was paralleled by an 8% reduction in brain weight but a ≥35% reduction in lung, liver, pancreatic and spleen weights. The brain is 'spared' at the expense of other organ systems which indirectly, e.g., through the development of chronic lung disease, sepsis, etc., may further limit brain growth by reducing intake and/or altering requirements.
Studies in preterm infants have indicated a direct relationship between poorer early growth and poorer development. Studies also indicate that it is the course of postnatal rather than prenatal growth that correlates best with developmental outcome [5]. Even short periods of nutrient supplementation during early life have been related to better neurodevelopment at 18 months and 7.5–8.0 years of age [6]. More aggressive nutritional support has also been shown to increase head and corticospinal growth in term and preterm infants after perinatal brain injury [7].

While better growth may be related to better development, concern has been expressed that ‘catch-up’ growth in preterm infants may be associated with the development of insulin resistance and metabolic syndrome X [8]. However, ‘catch-up’ cannot be prevented, it is a physiologic phenomenon that occurs after a period of growth failure in all preterm infants. Data from our group (to be presented) indicate that while the rate and extent of ‘catch-up’ is greater in preterm infants fed a nutrient-enriched compared to term infant formula after hospital discharge, it is not paralleled by an increased or altered adiposity but increased non-fat mass during the first year of life.

References