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NUTRITION AND CHILD HEALTH
PERSPECTIVES FOR THE 1980s

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To those who love little children.

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Introduction

The last year of the 1970s was declared by the United Nations as the Year of the Child. World-wide, the greatest problem in childhood is inappropriate nutrition. While the children of less advantaged countries suffer the dire consequences of widespread undernutrition, the children of affluent countries tread the path of degenerative diseases through overnutrition, another form of *mal*nutrition. As editors of the Proceedings of the 21st Annual Meeting of the American College of Nutrition, we present the efforts of leading pediatric investigators in the area of Nutrition and Child Health. We trust that the sense of history, as conveyed by authors such as our 1980 American College of Nutrition Awardee, Dr. Harold Harrison, will leave us with a correct perspective of where we are and the debt we owe to past investigators; and that the taste of the future, as exemplified by our Grace A. Goldsmith Awardee Dr. Dennis Bier, will serve to stimulate young and new investigators into this fertile and critical area of research in the 1980s.

Dr. Lifshitz and colleagues begin the symposium with a reminder to us about the major global problem of malnutrition. They bring to us specifically their experience with the biochemical, physiologic, and morphologic effects of malnutrition and diarrhea on the intestine. They draw on human and animal data to present their thesis that the absorptive, protective, and homeostatic functions of the intestine are impaired in malnutrition.

The next three papers deal specifically with the problems of "overnutrition."

Drs. James and Sahakian present a stimulating discussion of the energetic basis of obesity. The authors challenge the "fat infant to fat adult" theory and present arguments against the thesis that increased fat cell numbers in childhood result in adult obesity. They remind us of the importance of heredity in the cause of obesity, especially citing twin studies. Finally the authors argue that the pre-obese child may have decreased energy requirements and that hyperphagia may be secondary to the energy imbalance.

Dr. Berenson and co-workers present the work that they have performed on 5,000 children from birth through adolescence. They identify a consistent ranking or tracking of serum lipids and lipoproteins, especially beta lipoprotein, and remind us of the importance of examination of the "lipid hypothesis" from childhood.

Dr. Forbes, as befits his role as Editor-in-Chief of a pediatric journal, enters into a scholarly discussion of body composition in adolescence. After a concise description of methodology, he describes the sex differences in fat composition, the lean body mass of adolescents and the effecercise on lean body mass. In particular, Dr. Forbes highlights the deficiencies in our understanding of body composition during a critical period of growth.

A historical perspective is given by Dr. Harold Harrison. Dr. Harrison has woven together the "Story of Vitamin D," a story that intimately involves Helen and Harold Harrison and spans from the identification of vitamin D deficiency as the cause of rickets, to the discovery of the vitamin D metabolic pathways. As Dr. Harrison so aptly puts it, "the vitamin D story was thought to be finished" in the 1950s. But as

history unfolds itself, we are carried along by Dr. Harrison through the work of Deluca, Fraser, and Kodicek, and Norman, to 1980 and a capsule view of the vitamin D story as it stands today. For the distinguished accomplishments of his lifetime, Dr. Harrison was awarded the American College of Nutrition Award.

The current trend of emphasis on breast milk feeding for infants has re-awakened tremendous interest in the physiology of breast milk. The Nichols' have produced a comprehensive review of human milk, summarizing the work of past decades and allowing the reader to recognize the gaps that exist in our understanding of human lactation. Renewed scientific interest in breast milk comes at a time when renewed interest in nutrition is occurring in all public sectors, and the 1980s should see the fruits of intensive research into this age-old and tremendous nutritional resource.

Dr. Lars Hanson's exciting studies of secretory IgA in breast milk, spanning the last two decades, have been critical in the understanding of neonatal defenses against infection. Dr. Hanson presents the remarkable information regarding the "homing" feature of lymphocytes produced in the maternal intestinal Peyer's patches. These lymphocytes, through an enteromammaric circulation home to the breast, and provide the secretory IgA specific for the defense needs of the infant. The various other protective factors against infection that are present in breast milk provide a backdrop for the role of secretory IgA. New information is presented regarding the potential value of vaccination of the mother to stimulate the production of antibodies for the breast milk and indirectly for the infant. Finally Dr. Hanson reminds us of the dangerous period of weaning in developing countries when breast milk feeding is stopped, and when the infant is exposed to microorganisms and resultant infection, which lead to compromised nutrition.

One exciting area of future research in the 1980s involves the use of stable isotopes to determine nutrient kinetics in human infants. Dr. Dennis Bier, recipient of the American College of Nutrition Grace A. Goldsmith Award for young investigators in nutrition research, has had a pioneer role in the use of stable isotopes for children. In this Symposium, Dr. Bier and colleagues describe the utility of stable isotopes in the study of energy intake on whole body protein dynamics. They stress the excellent precision and relatively low intrasubject variation that is possible with stable isotope-labelled leucine in the estimation of protein turnover. Data are presented in support of the thesis that increasing energy intake from non-protein sources results in increased protein synthesis.

What do the 1980s hold for nutrition in child health? Will the problems of undernutrition be overcome in the "underdeveloped" countries? Will the problems of overnutrition and atherosclerosis in "overdeveloped" countries be tackled through preventive measures instituted in childhood? Will basic studies of age-old nutritional themes (vitamin D, breast milk) continue to provide us stimulating insights into the physiology of the human body? Will new methodologies such as the use of stable isotopes in pregnancy and infancy, afford us a deeper understanding of the metabolism of the human body, especially for mothers and infants? The impetus begun in the 1970s gives us hope that the answers will be yes for most of these questions. We predict that the 1980s will be exciting years for nutrition research and understanding.

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Contents

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Perspectives for the 1980s, pages 1–24

MALNUTRITION AND THE INTESTINE

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I. INTRODUCTION

Malabsorption in kwashiorkor and marasmus is one aspect of a vicious cycle often affecting children living in societies based on a subsistence economy and under substandard hygienic conditions. Due to the rapid turnover of the small intestinal mucosa, loss of absorptive function and mucosal atrophy are likely to ensue when protein synthesis is diminished by a limited supply of essential amino acids (Ghadimi et al 1973, Waldman 1966). The syndrome of protein energy malnutrition is almost always complicated by diarrhea (McLaren 1974). In the early 1900's,a curious disease known as "culebrilla" was described (Correa 1908). In this disease, which later became known as kwashiorkor, diarrhea was the chief problem and it has remained a prominent feature of all subsequent descriptions. On the other hand, when there is no increased incidence of diarrhea, an inadequate dietary intake rarely leads to severe malnutrition and malabsorption (Fagundes-Neto 1980). Therefore, the interactions between malnutrition and diarrhea have to be kept in mind to understand the effects of malnutrition on the intestine.

This paper reviews the effects of malnutrition on the intestine. An attempt is made to elucidate the effects of malnutrition itself on the intestine of experimental animals, in order to better understand the complex derangements of intestinal function associated with human malnutrition. In addition, we describe experimental data suggesting that the

small intestine of malnourished rats has an increased susceptibility to injury, which facilitates the onset of diarrhea and increases its severity.

II. INTESTINAL FUNCTIONS

The gastrointestinal tract is classically considered the organ system for nutrient absorption. An alteration in this function is a well recognized facet of the clinical picture of malnutrition in infancy (Herskovic 1969, James 1971, Viteri and Torun 1980). However, the intestine performs other important functions which could be altered in malnutrition; these include protection from the outside world and provision of a homeostatic-metabolic exchange surface.

The gastrointestinal tract constitutes the largest uninterrupted contact area between the environment and the organism; its total area is estimated to be over 200 times the body surface area. The functional and structural integrity of the intestine is vital to protect the individual from the outside world. Malnourished children have an increased susceptibility to intestinal infections which lead to diarrheal disease and endotoxemia (Klein et al 1977). The pathophysiological factors which may allow the process of infection need to be studied further.

Limited information is available regarding intestinal immunologic function during malnutrition (Gross and Newberne 1980). A reduction in the size of Peyer patches and the appendix, with a depletion of the paracortical areas and germinal center are found in all gut associated lymphoid tissues (Smythe et al 1971). Although serum immunoglobulin concentrations appear normal or even increased in human malnutrition, functional studies indicate that the system of local mucosal humoral immunity primarily involving sIgA may be impaired. Although the exact biological significance of this change is unknown, it may result in increased frequency of infections, endotoxemia and a predisposition to food allergies (Chandra 1975).

The intestine also constitutes a homeostatic-metabolic exchange surface. For example, the intestine plays an important role in maintaining water and acid base balance. Dehydration and metabolic acidosis only take place when the

homeostatic capacity of the intestine is exhausted (Lugo de Rivera et al 1972). The intestinal route may account for a large proportion of bicarbonate losses even in conditions primarily afflicting other organs such as the kidney (Schoeneman and Lifshitz 1974). Therefore, disturbances in the homeostatic role of the intestine may lead to diarrhea, and, vice versa, diarrhea may alter homeostasis.

III. MALNUTRITION AND MALABSORPTION

1. Gastrointestinal malfunction in malnourished patients

The abnormalities in the digestive-absorptive function reported in human malnutrition are a consequence of deprivation and life in a contaminated environment. It is now well established that infectious processes play a major role in the pathophysiology of the malabsorption which is seen in children with protein-energy malnutrition. These perpetuate and worsen the abnormal nutritional status of the patient and are associated with diarrhea (Lifshitz in press, Scrimshaw 1977). In contrast, in anorexia nervosa, a self-inflicted form of food deprivation, there is often constipation rather than diarrhea (Bruch 1965).

The alterations of intestinal absorptive functions in malnourished patients are summarized in Table 1. Malabsorption of nutrients is primarily related to pancreatic insufficiency which rapidly develops when protein-energy intake is not adequate (Barbezat and Hansen 1968). This results in an impairment of the digestion and absorption of protein, carbohydrates, lipids, minerals and lipid soluble vitamins (Lebenthal et al 1976, McCance et al 1970, Vitale 1974) with an increase in protein excretion through the intestinal lumen (Schneider and Viteri 1974). The alterations in pancreatic function seem to be transitory and return to normal as soon as nutritional recovery begins (Schneider and Viteri 1974). Fat malabsorption is also impaired in children with protein energy malnutrition because the intraduodenal capacity to form fat micelles is markedly reduced due to bile salt insufficiency.

TABLE 1

GASTROINTESTINAL MALFUNCTION IN MALNOURISHED PATIENTS

1. Nutrient Malabsorption
   -- Pancreatic and bile salt insufficiency
   -- Gastrointestinal alterations:
      a) decreased absorptive capacity
      b) mucosal enzyme deficiencies
      c) morphological abnormalities
      d) gastric atrophy and hypochlorhydria
      e) bacterial overgrowth
      f) hypotonia and hypomotility

2. Decreased Protection from Outside World

3. Altered Homeostatic - Metabolic Exchange Surface

The type of fat intake as well as the presence of intestinal alterations may also contribute to steatorrhea. Vegetable fats are better tolerated than animal lipids (Dean 1952), and unsaturated fatty acids are well tolerated even in high quantities by malnourished children (Ashworth et al 1968). In addition, a variable portion of the stool fat excretion may be endogenous and derived from an accumulation of fat within the intestine (Underwood et al 1967). It has been suggested that this is due to a defect in β lipoprotein synthesis during protein malnutrition (Isselbacher and Budz 1963). However, this remains to be confirmed (Allen et al 1971). Fat malabsorption may also result from carbohydrate intolerance associated with diarrhea and malnutrition (Lifshitz and Holman 1964). This occurs when there is dilution of bile acids to concentrations below the critical micellar level due to the osmotic effects of unabsorbed carbohydrates (Ringrose et al 1972). A fall in fecal fat excretion when disaccharides are eliminated from the diet of malnourished children has been documented (Bowie et al 1963).

Malnourished patients have deficiencies in intestinal disaccharidases (Bowie et al 1963, 1965) and a diminished capacity to absorb dietary carbohydrates (James 1971). The disaccharidase deficiencies in these children may be due to deficient production of enzymes and/or tissue damage

(Hirschhorn et al 1968, Lifshitz and Holman 1964, Sunshine and Kretchmer 1964). These deficiencies may persist even after the acute stage of malnutrition. Lactase, sucrase and maltase activities are greater in patients with kwashiorkor than with marasmus (James 1971), and the incidence of lactase deficiency has been correlated with the extent of mucosal damage (Brunser et al 1968, Stanfield et al 1965). During the last decade it has been shown that feedings of maltose and sucrose can regulate sucrase and maltase activity in the small intestine of normal human volunteers (Rosensweig and Herman 1968, Rosensweig 1980). The regulation of enzyme activity by substrate has not been studied in malnutrition. Some of the intestinal oligodisaccharidase deficiencies reported in marasmus and kwashiorkor could also be due to lack of intake of the appropriate dietary substrate.

The deleterious effect of lactose in diarrheal disease among malnourished children has long been recognized (Bowie et al 1963, 1965, Dean 1952). Carbohydrate malabsorption contributes to diarrhea in malnourished patients (James 1971, Lifshitz et al 1971) and elimination of lactose and other disaccharides from the diet has resulted in improvement (Bowie et al 1963, 1965, Lifshitz et al 1971). A deficient absorption of monosaccharides may also be an important feature of chronic malnutrition and diarrheal disease (Lifshitz et al 1970). Since carbohydrates provide 50% of dietary calories it must be assumed that carbohydrate malabsorption plays an important role in the development and maintenance of malnutrition (Lifshitz in press).

Malabsorption of nitrogen and protein during malnutrition is well documented (Thompson and Trowell 1952). Approximately one third of the nitrogen intake is wasted (Waterlow et al 1960). A severe reduction in nitrogen absorption usually occurs when there is severe diarrhea (Robinson et al 1957) or disaccharide intolerance (Bowie et al 1967). The absorption of specific amino acids as well as di- and tripeptides may also be altered in malnutrition (Adibi and Allen 1970, Matthews and Adibi 1976). Malnourished chilren and adults show a significant decrease in several intestinal peptide hydrolases (Gjessing et al 1977, Hazuria et al 1974, Kumar et al 1971) and this decrease persists even after nutritional recovery and adequate protein intake. These enzyme deficiencies are correlated with morphological alterations of the intestinal mucosa (Gjessing et al 1977).

Atrophy of the gastric mucosa and hypochlorhydria have also been reported in children with severe protein-energy malnutrition (Herbst et al 1969). The intestinal wall may become thin and hypotonic and there is also decreased intestinal motility (James 1970). Ileal function is also defective in severe protein-energy malnutrition as demonstrated by an impairment of vitamin B_{12} absorption and a great loss of bile salts in the stools (Alvarado et al 1973).

A striking feature of protein-energy malnutrition is bacterial overgrowth in the small intestine (Mata et al 1972). Gastric hypochlorhydria, intestinal hypotonia and hypomotility (James 1970) and immunological deficiencies (Bell et al 1976) are factors that may facilitate the colonization of the small bowel. These may occur simultaneously with the ingestion of large quantities of fecal bacteria, because of poor sanitary conditions (Mata et al 1971).

The most common problem associated with nonspecific bacterial proliferation of fecal and colonic bacteria in the upper segments of the small bowel is diarrheal disease (Burke and Danks 1966, Coello-Ramirez and Lifshitz 1972, and Ramos-Alvarez and Olarte 1964). Although these enteric microbial populations may be the cause of the diarrhea, usually bacterial overgrowth is a secondary alteration (Lifshitz 1977). Enteric bacterial proliferation in diarrhea and malnutrition may also result from the presence of free carbohydrates in the intestinal lumen (Coello-Ramirez and Lifshitz 1972). Whatever the reason, small bowel colonization with enteric microflora may worsen the alterations in intestinal function (Burke and Danks 1966, Coello-Ramirez and Lifshitz 1972 and Lifshitz et al 1970). These bacteria, mainly anaerobes such as Bacteroides and Veilonella, are capable of causing deconjugation and 7-alpha dehydroxylation of primary bile salts in the intestinal lumen, and they may alter food-stuffs producing hydroxy fatty acids, short chain organic acids and alcohol, all of which could be injurious to the bowel. These injurious factors in elevated concentrations, in the jejunal lumen may induce glucose malabsorption, sodium and water secretion and also morphological abnormalities of the intestinal mucosa (Donaldson 1965, and Gracy et al 1971). However, proliferation of intestinal microflora in upper segments of the bowel has been reported to occur in malnourished children even when they have no diarrhea (Mata et al 1972). Therefore, further studies should be done to determine the quantitative and qualitative