Complementary Feeding And Later Health

Background

It has been well-established that diet can influence the course and prognosis of chronic diseases. Decreasing the total fat and saturated fat content and increasing the consumption of fruits and vegetables can diminish the risk of developing coronary vascular disease and lead to regression of atherosomatic plaques in individuals experiencing symptoms of coronary vascular disease. A subgroup of individuals with chronic eczema and chronic reactive arthritis disease improve when specific food allergens are removed from their diet. Other chronic diseases where a relationship between disease progression and diet exists include obesity, diabetes, and osteoporosis. It is also true that antecedents can be found in infancy or early childhood for many chronic diseases that manifest later in childhood or in adult years.

It is possible, therefore, that the diet during infancy and early childhood can effect the expression of certain chronic diseases. As a corollary, modifying the diet during infancy and early childhood might modify the expression of the chronic disease. In contrast to metabolic programming however, one assumes that to affect the cause of a chronic illness through diet, diet must be continued beyond infancy and early childhood to be effective. Unfortunately, little direct experimental evidence is available to link early eating behaviors, or specific nutrients in the diet of infants and young children, to the development of chronic illness later in life. Cohort studies examining for proximate causes of chronic illnesses can rarely be used to reliably assess the early antecedents of those illnesses.

Thus the principle research issue (or question) that the foregoing discussion highlights is the need to define the relationship between complementary feeding and later health by means of well-designed, longitudinal, prospective studies. The research issues are explored in 7 areas.

Cancer

There is no evidence that antecedents for any type of cancer, apart from those with strong hereditary or genetic components, exist in infancy or early childhood in healthy individuals not exposed to harmful doses of known carcinogens. There is also no experimental human evidence available to indicate that modifying the diet in infancy or early childhood can effect the risk of developing a malignancy later in life.

Allergy

Among the best predictors for risk of developing atopic disease is a history of atopic disease in immediate family members. Recent studies have shown that infants identified at high risk for allergy as a result of a positive family history may benefit from the delayed introduction of selected solid foods, prolonged breastfeeding, or the use of a hypoallergenic infant formula. Food allergy may manifest either as a classic IgE-associated reaction in the skin, lungs, upper respiratory tract or gastrointestinal tract or as an enteropathy involving any part of the gastrointestinal tract but not associated with IgE antibodies directed at specific foods. Such food allergy associated reactions in infancy may develop because of delayed maturation of various mechanisms, which normally lead to the development of oral tolerance to ingested antigens and potential allergens. Well-designed studies have demonstrated that dietary modifications in infants and children at high risk of developing allergy delay the expression of allergic symptoms for 18 to 60 months but ultimately there is no difference in expression of allergic symptoms beyond that age.23 These studies support recommendations for infants at high risk of allergy for exclusive breastfeeding or the use of a hypoallergenic formula for at least 6 months, and introduction of solid foods no earlier than 4 to 6 months of age with a delay in the introduction of dairy products to 1 year, eggs at 2 years, and peanuts, nuts, and fish at 3 years of age. Breastfeeding should be continued until 1 year of age or longer or a hypoallergenic formula used for the first year of life.

Diabetes

A variety of epidemiologic studies have implicated the ingestion of cows' milk as a trigger for insulin-dependent diabetes mellitus in genetically susceptible individuals.45 These observational studies are supported by other studies that identify antibodies to a 17-amino acid fragment of bovine serum albumin in a very high percentage of newly diagnosed children with type 1 diabetes.7 In 1994, the American Academy of Pediatrics recommended that infants from families at high risk for type 1 diabetes avoid cows' milk and soy protein-based formulas for the first year of life along with other dairy products during this same period of time. Since the publication of those guidelines, however, studies have been published that show the absence of cellular immunity to either bovine serum albumin or peptide fragments of bovine serum albumin in individuals with type 1 diabetes.8 Anti-bovine serum albumin or bovine serum albumin peptide antibodies have been found with equal frequency among individuals with a variety of autoimmune diseases suggesting that these antibodies are an epiphenomenon in individuals whose immune system are hyperresponsive to environmental antigens. Furthermore, no association with cows' milk, cereal, fruit, vegetable, or meat protein ingestion between 3 and 6 months of age and β-cell autoimmunity has been found.8 Prospective controlled studies to examine the relationship between diet in infancy and early childhood are currently in progress.

Obesity

During the first 2 years of life the body mass index of the parent shows a stronger correlation with the persistence and tracking of obesity than diet during this period of time.9 The relative risk of obesity persisting into early adult years is 10 times as great if both parents are obese compared with very young obese children with nonobese parents. The age at introduction of solid foods appears to correlate in infancy and early childhood with the risk for obesity. However, this loses significance as a predictor by 24 months of age.10 Clearly other factors, including childhood activity along with parents' body mass index, may be as strong or significantly stronger than diet in infancy in predicting the development of later obesity. In all however, there is a paucity of data on the specific relationship of complementary feeding to the evolution of obesity.

Hypertension

A number of dietary factors have been shown to influence blood pressure regulation at various ages. The carbohydrate, essential fatty acid, amino acid, fiber, mineral (sodium, potassium, calcium and magnesium) and trace metals composition of the diet have been shown to affect blood pressure in a large number of studies. In addition, specific foods such as garlic and onions and a variety of chemicals present in foods such as caffeine may affect clinical hypertension. Finally, nondietary factors such as obesity, smoking, and exercise have a very strong influence on both the development and course of hypertension.

Several studies have addressed the relationship of sodium intake in infancy to blood pressure in infancy and later in life. These have found no relationship between blood pressure during infancy throughout childhood up to 8 years of age and salt intake over a fairly broad range (1.9-9.3 mg/100 kcal/d).11 Furthermore, studies have failed to demonstrate a relationship between salt intake in infancy and early childhood and the development of salt preference. Finally, evidence is lacking to establish the time of onset of "diet sensitivities" in those hypertensive individuals who are "diet-sensitive" (e.g. sodium-sensitive).

Coronary Vascular Disease

Perhaps the most well-documented evolution of a chronic disease is that for coronary vascular disease. In carefully conducted autopsy studies isolated from cells have been shown to be present in the coronary intima at birth. These decrease in numbers significantly after 12 months of age and no extracellular lipid deposition in the coronary or peripheral vessels is seen until late in the first decade of life. In addition, no change in intimal smooth muscle cells or collagenization of fatty streaks occurs until late in the first decade of life and generally not until the postpubertal period.12

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Recent studies have also unequivocally demonstrated the rapid increase in prevalence of both fatty streaks and raised fibrous plaques between the ages of 15 and 30 years and have shown a strong association between the evolution of coronary vascular disease and blood lipoproteins, body mass index, hypertension, and smoking.

Thus, coronary vascular disease is clearly an evolving process with raised intimal lesions (early precursors) present between ages 8 and 10 years of age. There is however, no evidence to show that coronary vascular disease evolves in any significant way during infancy or early childhood, except in those with congenital hyperlipidemia.

Osteoporosis

The effects of diet on bone mineralization and growth have been well-established during infancy and childhood. Published studies and others currently in progress suggest that the immediate prepubertal period may be of critical importance in the evolution of osteoporosis of later adult life. There are, however, no data that relates the evolution of adult osteoporosis to diet in late infancy and early childhood.

Summary

There is at this time little or no evidence for a specific effect of complementary feeding on those chronic diseases most prevalent later in life.

Ronald E. Kleinman, MD
Department of Pediatrics
Massachusetts General Hospital
Harvard Medical School
Boston, MA 02114

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