Symposium: Nutritional Implications of Dietary Protein Restriction in Diabetes Mellitus

Protein Consumption and Diabetes Mellitus: An Overview

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Two developments have stimulated recent interest in protein metabolism in human diabetes mellitus. The first is the accumulation of clinical evidence that dietary protein restriction can delay the progression of chronic renal failure, an important complication of diabetes. The second—and the stimulus for the four papers published in this supplement to The Journal of Nutrition—is our increasingly sophisticated, but incomplete understanding of the abnormalities of amino acid metabolism that occur in diabetes mellitus.

Although detailed interest in whole-body protein metabolism in diabetes is a relatively recent phenomenon, this was not always the case. As documented in the symposium paper by Charlton and Nair, abnormal weight loss with resulting protein-energy malnutrition were long recognized as cardinal features of insulin-dependent diabetes (IDDM). It is grimly ironic that before the discovery of insulin in 1922, the only treatment that delayed death due to IDDM was deliberate starvation, which reduced endogenous insulin requirements enough to temporarily match the patient’s dwindling capacity to produce endogenous insulin. During the years before 1922, the optimum protein content of the diabetic diet was researched and debated (Marsh et al. 1922). The discovery of insulin, and its near-miraculous effects, put an end to clinical concerns about protein nutrition in diabetes (Bliss 1982), even though basic investigation into the effects of insulin on protein and amino acid metabolism continued (Jefferson 1980), and despite evidence that mild biochemical abnormalities of amino acid metabolism were known to persist in conventionally treated IDDM (Felig et al. 1977, Tamborlane et al. 1979).

In 1983, Nair et al. used tracer infusions of [1-13C]leucine to confirm in a simple manner the rapid increase in amino acid catabolism that occurs in IDDM when insulin is withdrawn, while also demonstrating marked increases in whole-body proteolysis and protein synthesis. This historic study was the first of a large number of subsequent, increasingly sophisticated tracer kinetic investigations that have shed new light on the characteristics of altered whole-body and regional protein metabolism in this disease (De Feo and Haymond 1991, Nair 1992).

During the same years, clinical results accumulated, demonstrating that dietary protein restriction can slow the progression of chronic renal failure (Wylie-Rosett 1988). On the basis of these data, the American Diabetes Association (ADA), which had previously recommended that 12–20% of energy in the diabetic diet should come from protein (~1.0–1.8 g protein/kg of adult body weight), noted in 1986 that Americans in general consume too much protein and advised diabetic adults to reduce their protein intake to 0.8 g/(kg·d). This protein intake, although considerably less than customary, is regarded as safe for normal persons (American Diabetes Association 1979 and 1987, Wylie-Rosett 1988).

But is a protein level that has been determined to be safe for normal persons equally safe for those with IDDM (Hoffer 1989 and 1993, Brodsky and Robbins 1989)? In particular, is protein restriction safe for the 85% of people with IDDM who use conventional, rather than intensive insulin treatment regimens (Harris et al. 1994) and in whom residual abnormalities of amino acid metabolism frequently persist? In the ensuing years, information from large clinical trials has created some doubt about the practical effectiveness of protein restriction in chronic renal failure (Henry 1994, Klahr et al. 1994), while limited data from human metabolic studies have raised the possibility that protein restriction could entail some nutritional risk. Since 1994, the ADA has returned to its original position, now judging that there is insufficient information to recommend protein intakes in the diabetic diet that differ from the conventional 12–20% of total energy (American Diabetes Association 1994, Henry 1994).

Where do we stand now? With some qualifications, the proposition that protein restriction can protect renal function in diabetes remains valid (Pedrini et al. 1996). Moreover, while it is true that amino acid metabolism is altered in IDDM, the practical implications for IDDM patients, particularly in the insulin era, are not obvious. If there are nutritional risks, for which patients are the risks sufficiently important to preclude the benefits of protein restriction? The goal of this symposium was to review current understanding of the effects of human diabetes on protein metabolism while trying, to the extent possible, to extrapolate this understanding to the clinic or bedside and to suggest goals for future research.
Many of the findings about amino acid metabolism in diabetes described in this symposium were made possible by methodologic advances in the tracer field. These include advances in both radiotracer and stable isotope tracer concepts. The latter have been enabled by improvements in mass spectrometer technology and the increased availability of relatively inexpensive stable isotopes.

LITERATURE CITED


