

not improve overall diabetic control further emphasized that the glycemic index should be used in combination with other food attributes (for example, macro- and micronutrient content and overall calories) rather than as the sole criterion for planning diets for diabetics. More recent work, such as the useful studies of Collier and O'Dea showing marked responses of insulin (4) and gastric inhibitory polypeptide (5) to fat, have served to strengthen this position. On the positive side, we see an important function of the glycemic index in allowing identification of starchy carbohydrate foods that may be incorporated into the higher carbohydrate diets now being recommended in the treatment of diabetes. Such diets have as their goal the reduction of fat intake. With foods that have a low glycemic index, this may be achieved without increasing the postprandial glycemia. Even when diets include very high levels of fat (46.5 grams of butter per 75 grams of carbohydrate), the original glycemic index approach is useful, as demonstrated by the studies of Collier and O'Dea. Thus, despite the addition of fat, lentils, a food with a low glycemic index, still produced an appreciably lower glycemic response than potatoes, a food with a higher glycemic index (5).

Our previous work with dietary fiber suggested that a reduction in blood glucose coincided with a reduced rate of carbohydrate absorption (6). Our studies

with foods and those of O'Dea and coworkers have confirmed that rate of digestion may be a major factor in determining the glucose (7) and insulin (5, 8)response to starchy foods. Study of the effects on the endocrine response of adding fat and protein to meals is important. However, such studies are complementary to extensive glycemic index testing. This is urgently needed to get an overall picture of the glycemic responses to the many foods that have not been tested and to enable selection of specific foods for more detailed testing and, at a later stage, possible incorporation into therapeutic diets.

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## References

- 1. P. A. Crapo and J. M. Olefsky, New Engl. J. Med. 309, 44 (1983).
- D. J. A. Jenkins et al., Am. J. Clin. Nutr. 34, 362 (1981)
- 3. R. H. Taylor et al., Proc. Nutr. Soc. 39, 56A (1980).
- 4. G. Collier and K. O'Dea, Am. J. Clin. Nutr. 37, 941 (1983)
- 5. G. Collier, A. McLean, K. O'Dea, Diabetologia, in press. 6. D. J. A. Jenkins et al., Brit. Med. J. 1, 1392
- (1978).
- (1978).
  D. J. A. Jenkins et al., Diabetologia 22, 450 (1982); K. O'Dea, P. Snow, P. J. Nestel, Am. J. Clin. Nutr. 34, 1991 (1981); D. J. A. Jenkins et al., Brit. Med. J. 2, 14 (1980).
  D. J. A. Jenkins et al., Am. J. Clin. Nutr. 35, 1339 (1982).

*Erratum*: In the report "Monoclonal antibodies in the lymphatics: Selective delivery to lymph node metastases of a solid tumor" by J. N. Weinstein *et al.* (28 Oct., p. 423), figure 2 was printed incorrectly. In the bar graph on the left, the captions under N = 13 and N = 4 were interchanged. The correct figure is printed helow

