Cruikshanks et al. (3) in the early 1980s generally confirmed these findings. That study also noted only modest differences in lipid levels between adolescent IDDM patients and nondiabetic control subjects with the major differences occurring in younger girls with IDDM.

At this point, we can only speculate what specific role diet plays in the modest differences in lipid and lipoprotein levels noted in some IDDM patient groups when compared with nondiabetic control subjects. In the DCCT, associations between lipid levels and dietary variables such as total calories, fat, carbohydrate, and cholesterol intake (derived from baseline diet histories) were very weak ($r^2 < 0.04$). Schober notes that the modest differences in lipid levels between IDDM campers and nondiabetic control subjects disappeared after 4 wk of camping—with the diabetic children eating a relatively high-fat diet. Because the authors did not obtain any measure of baseline diets, it is conceivable that the camp diet, albeit high in fat, had a lower fat content than the childrens' usual diet. Moreover, other variables, such as tighter metabolic control or changed exercise patterns, may explain the change in lipids while at summer camp. Until more data become available from the DCCT and other studies, explanations for the altered lipid and lipoprotein profiles in IDDM remain speculative.

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DCCT, Diabetes Complications and Control Trial; IDDM, insulin-dependent diabetes mellitus; TG, triglyceride.

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Stability of Mailed and Couriered Capillary HbA_{1c} Samples

We previously evaluated the accuracy and stability of the Bio-Rad capillary HbA_{1c} collection system (Hercules, CA) for samples collected and assayed at a single location (1). The Bio-Rad system is designed specifically for the Diamat HPLC and requires no pretreatment when assayed ≥24 h after collection. Although other capillary HbA_{1c} collections have been used with affinity chromatography (2) and colorimetric assays (3), they are not readily adapted to HPLC. We undertook this study to substantiate the stability of mailed or couriered capillary HbA_{1c} samples using the Bio-Rad collection system.

Three simultaneous capillary HbA_{1c} samples were obtained from 100 pediatric patients attending diabetes outreach clinics in rural Minnesota. One set of samples was mailed from the collection site. Another set of samples was stored at ambient temperatures (couriered) for up to 4 days before being delivered to the laboratory. The reference samples were stored on ice for up to 4 days before being delivered to the laboratory. We instructed 52 of the patients to collect a sample (home) during the following 24 h and to mail it to the laboratory. All samples were sent to Park Nicollet Medical Center Laboratory.

The study was conducted in April 1992, during which the low and high daily temperatures throughout Minnesota averaged 31 and 48°F and ranged from a daily low of 12–53°F and a daily high of 28–90°F. The capillary HbA_{1c} values ranged from 4.7 to 15.9% with an average value of 9.1% (reference range 4.2–5.8%). All chromatographs were normal in appearance.

The maximum difference from the reference for each collection method was 0.4%, with >90% within 0.2%. No significant difference was observed between the reference and couriered samples and between the reference and home samples using a paired Student's *t* test. Although the average of the reference samples exceeded the average of the mailed samples by 0.067 (SE = 0.013, $P < 0.001$), this difference is not clinically significant.

Mailing of capillary HbA_{1c} samples offers an important service to patients in rural areas. Collection kits could be sent to patients for collection before a clinic visit; the results would then be available at the time of the clinic visit. We have shown that capillary HbA_{1c} samples collected with the Bio-Rad capillary HbA_{1c} collection system can be mailed or couriered, at ambient temperatures, without a loss in accuracy or stability. Likewise, samples can be collected by the patient at home and mailed to the laboratory.

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HPLC, high-performance liquid chromatography.

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Comment on Consistency

A careful reading of the Garber et al. (1) commentary for clinicians leaves me feeling uncomfortable. This stems from several internal inconsistencies that, although acceptable in comparing individual researchers' results, ought not creep into a practical commentary for clinicians.

To take a few notable examples, in Table 1, if a fasting plasma glucose >200 mg/dl is poor or high and 140 mg/dl is normal or desirable, what is a fasting plasma glucose between 141 and 199 mg/dl? Similarly, what is a 2-h postprandial plasma glucose between 200 and 235 mg/dl? More reasonable would be normal or desirable listed as ≤115 and 140 mg/dl, respectively; 115–140 and 140–200 mg/dl as acceptable or borderline, and >140 and >200 mg/dl as poor or high.

This principle is properly applied to the fasting plasma cholesterol values in Table 1 but not to the triglyceride levels, where levels between 150 and 200 mg/dl are in limbo, and the acceptable level is listed at 200–250 mg/dl. Why not 150–250 mg/dl? The plasma LDLs also pose a problem in the range from ≤160 to ≥190 mg/dl—are these acceptable or borderline or are they poor or high? Or are they to be ignored?

Furthermore, after characterizing triglyceride levels of >1.69 mM (150 mg/dl) as "elevated triglycerides" in the text on several occasions, the authors recommend that ". . . in hypertriglyceridemic (>2.82–3.39 mM [250–300 mg/dl]) patients, resins are not effective in lowering total cholesterol and cause increases in triglyceride levels . . ." This definition of hypertriglyceridemia (or isn't hypertriglyceridemia equated with elevated triglycerides?) is a different one than previously given (although presumably the one used in ref. 48).

The bottom line is consistency and practicality. Unfortunately, this commentary may confuse more than it clarifies.

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LDL, LOW-DENSITY LIPOPROTEIN.

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Response to Dr. Matz

We thank Dr. Matz for his comments on our commentary, *Detection and Management of Lipid Disorders in Diabetic Patients.*

With regard to his concerns on the classification of normal, acceptable, and poor values in our Table, "Suggested standards for biochemical indexes of metabolic control," he is correct in that we show a single rather than a range of values for most indexes, including fasting plasma glucose and 2-h postprandial plasma glucose. However, values for these (and other indexes) were taken directly from prior publications and standards of the ADA (as noted in the Table). To reformulate or reclassify these values would be to revise the existing recommendations of the ADA, which was not our intent. The purpose of our commentary was to note that many physicians do not adhere to ADA recommendations and to reiterate for clinicians' benefit, the already published standards for control as they exist in the literature.

His second comment concerned differing values characterizing elevated triglycerides and hypertriglyceridemia, and that in some cases, elevated triglycerides are defined as >150 mg/dl and in other cases >250–300 mg/dl (as in reference 48). Definitions of elevated triglycerides differ between publications and guidelines. A prime example is the discrepancy between the National Cholesterol Education Program (hypertriglyceridemia defined as >250 mg/dl) and the ADA (>150 mg/dl); to complicate matters, the International Committee for the Evaluation of Hypertriglyceridemia as a Vascular Risk Factor considers >200 mg/dl to be elevated. In all cases, we used the original values rather than presuming that hypertriglyceridemia is >150 mg/dl (as in the ADA). While this may have added some confusion, it also adds veracity and authority to the commentary. It reflects existing inconsistencies in the literature.

We suggest that his comments be forwarded to the next American Diabetes Association Consensus Development Conference in this area.

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