The Impact of Outpatient Diabetes Management on Serum Lipids in Urban African-Americans With Type 2 Diabetes

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OBJECTIVE — Treating dyslipidemia in diabetic patients is essential, particularly among minority populations with increased risk of complications. Because little is known about the impact of outpatient diabetes management on lipid outcomes, we examined changes in lipid profiles in urban African-Americans who attended a structured diabetes care program.

RESEARCH DESIGN AND METHODS — A retrospective analysis of initial and 1-year follow-up lipid values was conducted among patients selected from a computerized registry of an urban outpatient diabetes clinic. The independent effects of lipid-specific medications, glycemic control, and weight loss on serum total cholesterol, LDL cholesterol, HDL cholesterol, and triglyceride levels were evaluated by analysis of covariance and multiple linear regression.

RESULTS — In 345 patients (91% African-American and 95% with type 2 diabetes), HbA1c decreased from 9.3% at the initial visit to 8.2% at 1 year (P < 0.001); total and LDL cholesterol and triglyceride levels were significantly lower, and HDL cholesterol was higher. After stratifying based on use of lipid-specific therapy, different outcomes were observed. In 243 patients not taking dyslipidemia medications, average total cholesterol, LDL cholesterol, and triglyceride concentrations at 1 year were similar to initial values, whereas in 102 patients receiving pharmacotherapy, these lipid levels were all lower at 1 year relative to baseline (P < 0.001). Mean HDL cholesterol increased regardless of lipid treatment status (P < 0.001). After adjusting for other variables, changes in LDL cholesterol concentration were associated only with use of lipid-specific agents (P = 0.003), whereas improved HbA1c and weight loss had no independent effect. Lipid therapy, improved glycemic control, and weight loss were not independently related to changes in HDL cholesterol and therefore could not account for the positive changes observed. Use of lipid-directed medications, improvement in glycemic control, and weight loss all resulted in significant declines in triglyceride levels but only improved HbA1c and weight loss had an independent effect.

CONCLUSIONS — Among urban African-Americans, diabetes management led to favorable changes in HDL cholesterol and triglyceride levels, but improved glycemic control and weight loss had no independent effect on LDL cholesterol concentration. Initiation of pharmacologic therapy to treat high LDL cholesterol levels should be considered early in the course of diabetes management to reach recommended targets and reduce the risk of cardiovascular complications in this patient population.

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Abbreviations: ADA, American Diabetes Association; ANCOVA, analysis of covariance; CVD, cardiovascular disease; HMG, hydroxymethylglutaryl.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

Diabetes is recognized as an independent risk factor for cardiovascular disease (CVD) (1), and cardiovascular outcomes are less favorable than in those without diabetes (2–6). However, little progress has been made in reducing heart disease mortality in diabetic patients when compared with the nondiabetic U.S. population (7). The pathogenesis of heart disease in individuals with diabetes is complex, but serum lipids are frequently abnormal and likely contribute to the increased risk of CVD (8,9). Because use of medications that improve lipid profiles can also reduce CVD risk among individuals with diabetes (10–12), aggressive intervention is recommended when dyslipidemia is detected (13).

Recent data indicate that recommended clinical targets for lipids are not being achieved among patients at high risk for CVD (14). Structured diabetes programs have been shown to improve glycemic control, but there is actually little information about the impact of such programs on the severity of dyslipidemia in type 2 diabetes, particularly in populations enriched in ethnic groups at increased risk of diabetes-related CVD mortality (15–19).

In our setting, which serves primarily African-Americans with type 2 diabetes, patients typically have LDL cholesterol levels that fall outside of American Diabetes Association (ADA) clinical targets, with an average value of 140 mg/dl at presentation (20). Low HDL cholesterol is less common, and only a low percentage of patients have hypertriglyceridemia (20). Therefore, interventions directed at lowering LDL cholesterol and raising HDL cholesterol should take priority in decisions about the choice of therapy for dyslipidemia. To determine the impact of diabetes care on serum lipids in this population, we examined the independent effects of lipid-directed pharmacotherapy, glycemic control, and weight loss on lipid outcomes after 1 year of treatment.
RESEARCH DESIGN AND METHODS

Overview of treatment program
The diabetes treatment program has been described previously (17,18). Briefly, intensive education in lifestyle modification, self-management training, and diet, coupled with intensification of medical therapy when needed to control hyperglycemia, have been integral parts of our structured care program. The overall treatment approach has been shown to result in significantly lower HbA1c levels (17,18).

Patient selection
Patients who initially presented to the program between 1991 and 1998 were selected from a computerized registry if they had a 1-year (52 ± 10 weeks) follow-up visit and if serum total cholesterol, HDL cholesterol, LDL cholesterol, and triglyceride levels were measured at both the initial (baseline) and 1-year visits. We chose a 1-year follow-up because we believed this would be a sufficient amount of time to expect life-style and pharmacologic interventions to take effect or be initiated. Data on lipid-specific medications for these patients were obtained from the computerized Pharmacy and Drug Information system maintained by the health system.

Laboratory studies and measurements
Serum total cholesterol, HDL cholesterol, and triglyceride levels were determined on fasting blood samples using standard techniques as described previously (21). Because LDL cholesterol levels were determined using the Friedewald equation, patients with triglyceride levels ≥400 mg/dl were not included in the analysis (22); only ~4% of African-American patients in our setting have triglyceride levels >400 mg/dl (20).

Analyses
Patients were stratified into individuals receiving medical therapy for dyslipidemia at 1 year (pharmacotherapy group) and individuals not receiving lipid-directed therapy (nonpharmacotherapy group), and lipid outcomes were examined separately for each category. Pharmacotherapy patients included individuals in whom medication was maintained or increased during the 1-year follow-up as well as individuals for whom therapy was initiated. Statistical differences between groups were tested on log-transformed data using either paired or unpaired Student’s t tests, and χ2 analysis was used for proportions.

To evaluate the impact of care on reaching lipoprotein targets, we examined LDL cholesterol, HDL cholesterol, and triglyceride distributions using ADA clinical guidelines applicable to the time encompassed by the data set. Before 1998, the recommended goal for LDL cholesterol was <130 mg/dl, and drug treatment was suggested for values ≥160 mg/dl. The proportion of patients meeting the ADA definition of an HDL cholesterol level at high risk for CVD was also determined, using sex-specific criteria (<35 mg/dl in men and <45 mg/dl in women). Finally, we determined the percentage of patients at goal (<200 mg/dl) for triglyceride levels (23).

We next determined the independent effects of lipid-directed therapy, improved glycemic control, and weight loss on changes in LDL cholesterol, HDL cholesterol, and triglyceride concentrations using both analysis of covariance (ANCOVA) and multiple linear regression. For the ANCOVA, patients were stratified according to lipid therapy status (therapy versus no therapy), whether they had a reduction in HbA1c level (HbA1c improved versus not improved), and by weight loss (weight loss versus no weight loss). HbA1c was defined as “improved” if there was a decrease of >0.5% at 1 year; the “HbA1c not improved” group was expected to include individuals in whom HbA1c values were similar or higher than baseline. Weight loss was defined as a decrease of >1.0 kg at 1 year; it was anticipated that the “no weight loss” category would incorporate individuals whose weight was either unchanged or increased compared with their initial visit. A change in lipoprotein concentration relative to baseline was considered significant if the 95% CI did not cross zero. Statistical differences in the changes in lipid levels between categories were tested after adjusting for patient age, duration of diabetes, sex, race, baseline lipid levels, mode of treatment for hyperglycemia, and year of initial visit.

Using multiple linear regression models, we separately evaluated the independent effect of lipid therapy, weight change, and HbA1c change on change in LDL cholesterol, HDL cholesterol, and triglyceride levels (all defined as year minus baseline values). Analyses were adjusted as for the ANCOVA.

RESULTS

Patient characteristics
We identified 345 patients in whom total cholesterol, LDL cholesterol, HDL cholesterol, and triglyceride levels were measured at the initial and 1-year visits. At presentation, the mean age of this study group was 57 years, mean weight was 89.6 kg, and mean BMI was 32.6 kg/m2; 68% were women, 91% were African-American, and 95% had type 2 diabetes. The mean duration of diabetes was 5.4 years. Average HbA1c declined from 9.3 to 8.2% (P < 0.001) by 1 year in the study group. Patients not included in the analyses (n = 5,728) were slightly younger (52 years, P < 0.001) at presentation, had a similar duration of diabetes (5.4 years), had a comparable weight (88.0 kg) but slightly lower BMI (31.6 kg/m2, P = 0.019), and similar HbA1c (9.3%) relative to those studied. The proportion of women and African-Americans were similar.

Among study patients, there were decreases in total cholesterol (219 to 212 mg/dl, P < 0.001), LDL cholesterol (148 to 139 mg/dl, P < 0.001), and triglyceride (148 to 142 mg/dl, P = 0.007) concentrations and an increase in HDL cholesterol level (47 to 50 mg/dl, P < 0.001). Compared with the study patients at the initial visit, those not analyzed had lower total cholesterol, LDL cholesterol, and HDL cholesterol values (207, 136, and 46 mg/dl, respectively; P < 0.04) and higher but not significantly different triglyceride levels (167 mg/dl, P = 0.94). Subsequent analyses focused on those in the study group.

Lipid profiles in nonpharmacotherapy patients
The mean HbA1c level of nonpharmacotherapy patients (n = 243) declined significantly (P < 0.001) relative to the initial visit, with an average decrease of 1.0% (Table 1). Despite the improvement in HbA1c, the total cholesterol, LDL cholesterol, and triglyceride concentrations at 1 year were comparable to levels found at the initial visit (all P ≥ 0.28).

LDL cholesterol distributions of non-
pharmacotherapy patients are shown in Fig. 1. The LDL cholesterol distribution improved only minimally at 1 year, with a slight increase in the proportion of individuals with levels from 100 to 129 mg/dl category and a modest decrease in the percentage with levels from 130 to 159 mg/dl (Fig. 1). In addition, 25% had an LDL cholesterol value ≥160 mg/dl (pre-1998 treatment threshold) at 1 year versus 26% at the initial visit; 48% had 1-year levels <130 mg/dl (pre-1998 goal) compared with 43% at baseline.

Mean HDL cholesterol increased in nonpharmacotherapy individuals at 1 year (P < 0.001, Table 1). The HDL cholesterol distribution also improved; the proportion with a high-risk HDL cholesterol concentration decreased from 41% at baseline to 30% at 1 year. For triglycerides, 82% had levels <200 mg/dl at both time points (data not shown).

**Lipid profiles in pharmacotherapy patients**

Compared with the nonpharmacotherapy group, pharmacotherapy patients (n = 102) were older (59 vs. 56 years, P = 0.011) and had a longer duration of diabetes (6.8 vs. 4.7 years, P = 0.03). Most patients (94%) were using hydroxymethylglutaryl (HMG)-CoA reductase inhibitors (average dose 22 mg). There were no differences between lipid therapy classes in the percentage who were women, who had type 2 diabetes, or who were African-American.

There was a significant decrease in HbA1c in those on drug therapy, and the average decline (1.3%) was similar (P = 0.40) to that detected in the nonpharmacotherapy group (Table 1). At the initial visit, pharmacotherapy patients had a higher HbA1c level (P = 0.017) than nonpharmacotherapy individuals and similar weight (P = 0.99). In contrast to the nonpharmacotherapy patients, the pharmacotherapy group experienced no significant weight change. Pharmacotherapy patients had higher total cholesterol, LDL cholesterol, and triglyceride levels (P < 0.001) at baseline compared with nonpharmacotherapy patients. Use of lipid therapy led to significant decreases in all three lipoprotein concentrations by 1 year (all P < 0.001).

A shift to a more favorable LDL cholesterol distribution was evident among pharmacotherapy patients (Fig. 2). The proportion with LDL cholesterol levels ≥160 mg/dl decreased from 60% at the initial visit to 30% at 1 year, whereas the number with values <130 mg/dl increased from 21 to 39%.

Like the nonpharmacotherapy group, HDL cholesterol concentration increased in pharmacotherapy patients (P < 0.001) at 1 year (Table 1), and the percentage with levels in the high-risk category decreased from 37% at the initial visit to 24% at 1 year. Finally, 82% had triglyceride levels of <200 mg/dl at both the initial and 1-year visits (data not shown).

**Evaluating the independent effects of lipid therapy, glycemic control, and weight loss**

Of the 345 patients, 56% had improved HbA1c levels after 1 year; among these individuals, HbA1c decreased an average of 3.1% (from 10.5 to 7.4%, P < 0.001). For individuals who did not show improvement in glycemic control, HbA1c increased an average of 1.6% (from 7.7 to 9.3%, P < 0.001). A total of 29% of patients experienced a >1-kg weight loss, with an average decrease of 5.6 kg (from 92.5 to 86.9 kg, P < 0.001); patients with no weight loss gained an average of 4.0 kg (from 86.6 to 90.5 kg, P < 0.001).

We used ANCOVA to determine whether lipid-directed therapy, improved glycemic control, and weight loss had independent effects on change in individual

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**Table 1—HbA1c, weight, and lipid profiles, according to dyslipidemia therapy status**

<table>
<thead>
<tr>
<th></th>
<th>No pharmacotherapy (n = 243)</th>
<th>Pharmacotherapy (n = 102)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Initial 1 Year</td>
<td>Initial 1 Year</td>
</tr>
<tr>
<td>HbA1c (%)</td>
<td>9.2 (2.7)</td>
<td>9.8 (2.5)*</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>89.6 (21.8)</td>
<td>89.5 (22.7)</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>32.8 (8.1)</td>
<td>32.6 (7.6)</td>
</tr>
<tr>
<td>Lipids (mg/dl)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total cholesterol</td>
<td>208 (47)</td>
<td>247 (55)*</td>
</tr>
<tr>
<td>LDL</td>
<td>138 (43)</td>
<td>174 (50)*</td>
</tr>
<tr>
<td>HDL</td>
<td>48 (16)</td>
<td>47 (13)</td>
</tr>
<tr>
<td>Triglycerides</td>
<td>141 (69)</td>
<td>164 (71)*</td>
</tr>
</tbody>
</table>

Data are means (SD). *Significant difference between 1-year and initial visit; †significant difference between therapy groups at initial visit.

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**Figure 1—LDL cholesterol distribution for patients not on lipid pharmacotherapy at 1 year.**
lipid levels (Table 2). Use of lipid-directed therapy was associated with a significant decline in LDL cholesterol level, which was statistically greater than that detected in individuals not given lipid-directed therapy \((P = 0.003)\). Improved HbA\(_{1c}\) was also associated with a significant decrease in LDL cholesterol, but this was not different from that in individuals with no improvement in HbA\(_{1c}\) \((P = 0.47)\). Losing \(\geq 1\) kg body weight was not associated with a significant decrease in LDL cholesterol concentration, and changes were comparable between weight-loss categories \((P = 0.86)\). Therefore, only use of lipid-directed therapy was independently associated with decrease in LDL cholesterol levels.

HDL cholesterol concentrations increased significantly in most categories, but changes were similar regardless of lipid therapy status \((P = 0.80)\), HbA\(_{1c}\) outcome \((P = 0.44)\), or weight loss \((P = 0.82)\). Lipid therapy, improvement in HbA\(_{1c}\), and weight loss were all associated with significant decreases in triglyceride levels, but only weight loss demonstrated an independent effect \((P = 0.035)\) (Table 2). Results of ANCOVA analyses were comparable if stricter definitions were used for improvement in HbA\(_{1c}\) \((\geq 1.0\%\) or weight loss \((\geq 2.5\) kg) (not shown).

Using multiple linear regression, results mostly agreed with the ANCOVA. Lipid therapy was significantly associated \((P = 0.028)\) with change in LDL cholesterol, whereas change in HbA\(_{1c}\) and weight had no independent effect \((P = 0.43)\); HbA\(_{1c}\), use of lipid therapy, and weight did not significantly impact HDL cholesterol \((P > 0.50)\). Weight change was related to change in triglycerides \((P = 0.0085)\), whereas use of lipid therapy had no effect \((P = 0.76)\). The only exception to the ANCOVA results was that change in HbA\(_{1c}\) was significantly related to change in triglyceride level \((P = 0.027)\).

After 1994, our program became more aggressive in intensifying therapy for hyperglycemia, which resulted in better HbA\(_{1c}\) outcomes compared with earlier years \((24)\). Therefore, we performed the above analyses on the subset of patients seen between 1995 and 1998. The 1-year HbA\(_{1c}\) outcome for 1995–1998 was 7.9\%, compared with 8.6\% for 1991–1994 \((P = 0.013)\). Despite the better HbA\(_{1c}\) outcome for 1995–1998, repeat ANCOVA and multiple linear regression analyses of this patient subset showed the relationships between HbA\(_{1c}\), weight, and lipid medications with lipid levels the same as for the whole study period of 1991–1998 (data not shown).

**CONCLUSIONS** — The 345 patients in this study had significant reductions in levels of HbA\(_{1c}\), total cholesterol, LDL cholesterol, and triglycerides as well as an increase in HDL cholesterol, which is evidence that the diabetes management program improved both glycemic control and serum lipids. However, lipid outcomes were not affected uniformly by traditional program components such as glycemic control, weight loss, and lipid-directed pharmacotherapy.

We detected significant reductions in LDL cholesterol only in patients given lipid-directed pharmacotherapy. Patients

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### Table 2: Effect of lipid therapy, glycemic control, and weight loss on LDL cholesterol, HDL cholesterol, and triglyceride changes (mg/dl)

<table>
<thead>
<tr>
<th></th>
<th>Change in LDL</th>
<th>Change in HDL</th>
<th>Change in triglycerides</th>
</tr>
</thead>
<tbody>
<tr>
<td>No</td>
<td>(-2 (-7 to 3))</td>
<td>+3 (1–5)(\dagger)</td>
<td>(-1 (-9 to 8))</td>
</tr>
<tr>
<td>Yes</td>
<td>(-27 (-38 to -16))(\ddagger)</td>
<td>+2 (0–5)</td>
<td>(-17 (-31 to -3))(\dagger)</td>
</tr>
<tr>
<td>HbA(_{1c}) at 1 year</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Not improved</td>
<td>(-6 (-13 to 0))</td>
<td>+2 (1–4)</td>
<td>(1 (-9 to 12))</td>
</tr>
<tr>
<td>Improved ((\geq 0.5%))</td>
<td>(-12 (-20 to -5))(\dagger)</td>
<td>+3 (1–5)(\dagger)</td>
<td>(-11 (-20 to -1))(\dagger)</td>
</tr>
<tr>
<td>Weight change at 1 year</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>No weight loss</td>
<td>(-11 (-17 to -4))</td>
<td>+2 (1–4)(\dagger)</td>
<td>(-1 (-10 to 8))</td>
</tr>
<tr>
<td>Lost weight ((\geq 1.0) kg)</td>
<td>(-8 (-15 to 0))</td>
<td>+3 (1–6)(\dagger)</td>
<td>(-14 (-26 to -3))(\dagger)</td>
</tr>
</tbody>
</table>

Data are means (95% CI). Changes in lipids defined as 1 year minus initial values. Negative changes denote a decrease and positive changes an increase in lipid concentration compared with initial visit. Analyses were conducted by ANCOVA and were adjusted for other variables in the table plus patient age, duration of diabetes, race (African-American versus other), sex, hyperglycemia treatment (diet, oral agents, insulin), year of initial visit, and initial lipid values. *Significantly different from those not on therapy \((P = 0.003)\); †significantly different from baseline \((CI\) does not cross zero); ‡significantly different from those without weight loss \((P = 0.035)\).
classified as “improved HbA1c” had a substantial decrease in HbA1c and achieved an average level of 7.4% after 1 year and a value close to national standards (13). Individuals classified as “not improved” had an increase in HbA1c, but the change in LDL cholesterol was comparable regardless of final glycemic status. Similarly, patients with “weight loss” lost an average of 5.6 kg, whereas individuals classified as “no weight loss” gained 4.0 kg, and changes in LDL cholesterol were also comparable between groups. This is consistent with findings from another study, in which intensive versus nonintensive lifestyle modification resulted in similar declines in LDL cholesterol among African-American patients with type 2 diabetes (25). Our results suggest that although glycemic control and weight management are cornerstones of diabetes therapy, neither intervention may play a major role in improving LDL cholesterol in populations comparable to ours. Lifestyle modification must continually be emphasized and reinforced in overall diabetes care and may lower cardiovascular risk through factors not examined here. Our finding that LDL cholesterol declined even among individuals who did not experience HbA1c improvement or weight loss may be explained by other management elements such as lifestyle modification (changes in diet composition and/or physical activity). Because we do not capture data on exercise or dietary habits, we are unable to retrospectively assess the effects of these interventions on lipid profiles. Because the decrease in LDL cholesterol was highly associated with use of lipid-specific pharmacotherapy, our results indicate that early introduction of lipid-modifying agents into the treatment program should be given strong consideration for patients with high LDL cholesterol levels.

Recent data indicate that detection and treatment of high LDL cholesterol often falls short of national recommendations (14, 26, 27). Although this study was not designed to examine practice patterns, our data also indicate that providers were not sufficiently aggressive in their management when high LDL cholesterol concentrations were found. Guidelines operating during 1998 and earlier suggested institution of pharmacotherapy for an LDL cholesterol value \( \geq 160 \text{ mg/dl} \) (23), but in our study, 25% of the untreated patients had levels higher than this threshold and remained without therapy at 1 year. Moreover, nearly one-third of treated patients still had LDL cholesterol levels \( \geq 160 \text{ mg/dl} \) at 1 year, implying delayed or insufficient intensification of therapy. Since 1998, a more stringent clinical target for LDL cholesterol in diabetes, with a goal of <100 mg/dl, has been suggested by the ADA, and it is a goal now recommended by the National Cholesterol Education Program (28). It is not yet known whether practitioners have responded to the recommended lower targets for LDL cholesterol with more aggressive use of medication therapy.

We have defined previously clinical inertia as a failure to intensify therapy when such action is needed and not otherwise contraindicated. Studying the basis for clinical inertia and implementing measures to overcome it can result in additional improvement in glycemic control of a population (24). Successful diabetes management requires attention to multiple metabolic parameters. When practitioners are focused on a single health problem, other processes of care may not be delivered (29). It is possible that the intense concentration on treating hyperglycemia may have detracted from aggressively managing dyslipidemia. Examination of potential barriers in the management of dyslipidemia requires further investigation and is underway (30).

Although the diabetes care program led to an increase in HDL cholesterol concentrations, an encouraging observation indicating the benefit of the program on this lipoprotein, definite variables that accounted for this change were not identified. The therapies examined here (glycemic control, weight loss, and lipid pharmacotherapy) were not independently associated with changes in HDL cholesterol. Other components of the education and treatment paradigms may have resulted in higher HDL cholesterol. Nevertheless, despite improvement in the HDL cholesterol profile after 1 year of management, a substantial proportion of patients still had levels that would confer increased cardiovascular risk. In this analysis, there was a predominant use of HMG-CoA reductase inhibitors, which typically have only a modest effect on HDL cholesterol levels (13, 23). These observations suggest that HDL cholesterol may have been underemphasized as an independent target of intervention when practitioners made therapeutic decisions and indicates a potential need to increase awareness of the benefits of increasing HDL cholesterol (31) during the course of diabetes care.

Although use of lipid medication significantly reduced triglyceride concentration relative to baseline, this effect was statistically similar to the change seen among patients not on lipid therapy. The decrease in triglyceride level with improved HbA1c was also significant compared with baseline. Although the results of the ANCOVA indicated that improved HbA1c did not have an independent effect on triglyceride change, multiple linear regression confirmed the effect of glycemic control on this lipoprotein. Weight loss also independently affected triglyceride levels; the average change in the weight-loss category was significantly greater than in the no-weight-loss group and indicates that even the modest weight decrease detected here would likely be beneficial in the management of hypertriglyceridemia.

Certain limitations to the study must be noted. Because LDL cholesterol levels tended to be high and triglyceride levels tended to be low in our patient population, LDL cholesterol was of primary interest to us. Therefore, we retrospectively selected patients according to whether levels of this lipoprotein were available. This led to statistical differences in some characteristics between the study population and those not included in the analyses. Although the clinical significance of these small differences is unclear, the study sample size, although still informative about the results of care, may not reflect eventual changes in the lipid profiles of the larger patient base. A recent study supports findings from the Diabetes Control and Complications Trial that achieving glycemic control improves lipid status among patients with type 1 diabetes (32, 33). Studies examining the relationship between achieving normal blood glucose levels and the effect it has on serum lipids among patients with type 2 diabetes have yielded inconsistent results (23, 34). For instance, in contrast to other data, our educational program resulted in a significant improvement in HDL cholesterol levels (25). The findings here might be specific to our clinic population, which is enriched in African-Americans, who typically have a lipid profile different from this population.
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from that of Caucasians (20). Additional studies are required to determine whether our observations can be generalized to other clinical settings and whether individual lipoproteins remain at target over longer follow-up periods. Studies focusing on lipid levels rather than just glycemic control as primary outcomes of care are needed in diabetes.

Our analysis shows how serum lipids are likely to respond within the context of routine diabetes care. Standard approaches to managing diabetes will likely benefit HDL cholesterol and triglyceride levels, even without use of lipid-directed medications, but glycemic control and weight management may provide little benefit in reducing high LDL cholesterol levels. In accordance with what is becoming recognized increasingly (35), our results indicate that patients with elevated LDL cholesterol concentrations may need early introduction of lipid-specific agents into their treatment programs rather than wait a long period to observe effects of nonpharmacologic approaches. Moreover, education programs aimed at lipid-related decision-making by providers may be needed to increase attention to management of all aspects of diabetic dyslipidemia—beyond improving LDL cholesterol alone. Establishing the most effective methods of achieving recommended lipid targets will be needed to reduce the morbidity and mortality of CVD in diabetic patients at high risk.

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