demonstrate that the degree of glycemic control in these two populations remains insufficient throughout the age span, even for those patients routinely followed in university centers. Optimizing treatment modalities, deliveries, and education to achieve levels of glycemic control comparable to those obtained during the DCCT or in similar, intervention-modified, environments, requires more medical and paramedical resources, thus producing a significant long-term demand for human and financial support.

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Response to Paris et al.

Pediatricians and diabetologists

Should it be methodologically correct to compare French and Belgian patients and doctors? We thank Dr. Paris and colleagues (1) for reassuring us collectively, as pediatricians taking care of young patients with diabetes. As a young resident in endocrinology 20 years ago, I was trained in the intangible idea that the results of adult diabetologists' efforts were indisputably more efficacious in terms of glycemic control than those of pediatricians. Was it true in those times? Remember that in many countries, including France, pediatricians were advocating free diet (freedom meaning a lot of sugar) and one insulin shot daily and discouraging the use of capillary blood in favor of the good old urinary measurements.

Those times are gone, fortunately for our patients, and we can now enjoy shameless comparisons of results with our colleagues. As an echo to Dr. Paris's letter, let us regret that besides well-known intervention studies of intensive therapy in highly selected centers and patients, there are too few reports of actual glycemic results in large cohorts (mostly adult) of patients with type 1 diabetes.

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LDL Cholesterol and Troglitazone Therapy

The recent study by Tack et al. (1) reported larger average LDL size in individuals on troglitazone therapy and appears to suggest that the increases in LDL cholesterol concentration typically observed with this agent (2,3) are not ultimately harmful to patients with type 2 diabetes because they are offset by potentially beneficial changes in lipoprotein composition and susceptibility to oxidation. This conclusion must be interpreted with caution for several reasons.

First, although several observational studies have shown an association between small, dense LDL and coronary heart disease (CHD) in patients with diabetes (4,5), the effects of changing LDL size have never been directly evaluated in any patient population. In contrast, increases in LDL cholesterol concentrations have been shown to be a strong and consistent risk factor for coronary artery disease in both cross-sectional and longitudinal analyses of individuals with or without diabetes. Furthermore, lowering LDL cholesterol concentration has been proven to decrease the incidence of cardiovascular events in several interventional studies (6,7).

Neither the American Diabetes Association nor the National Cholesterol Education Program has treatment recommendations suggesting that increases in LDL concentration are ameliorated by changes in LDL size. Thus, increases in LDL size should not be construed to negate the known risk of increases in total LDL cholesterol concentrations.

Second, the data from Tack et al. (1) showed decreased susceptibility to oxidation in the LDL after troglitazone therapy. This is consistent with the change in size because small, dense LDL are known to be more susceptible to oxidation in vitro (8). Although there are a large number of in vitro studies pointing to the importance of LDL oxidation in the atherosclerotic process, there are no clinical trials showing that changes in LDL oxidation can alter CHD risk. Manipulations that decrease oxidization ability might be of potential benefit; until clinical trial data are available, however, such manipulations associated with increasing LDL cholesterol concentrations cannot be assumed to be beneficial.

Finally, the study reported by Tack et al. (1) was not conducted in patients with diabetes. Individuals with diabetes are known to have increases in several CHD risk factors and to be at significantly increased risk for cardiovascular disease (CVD). Thus, any therapeutic regimen that results in increased LDL cholesterol concentrations in patients with diabetes must be viewed as increasing the risk of atherosclerosis in affected indi-