ministered daily. Our observation would have been more important if the relationship of Lp(a) was with circulating free insulin levels. Notwithstanding these drawbacks, we believe this observation is important and should be pursued further with prospective studies in both diabetic and nondiabetic subjects. We appreciate the input of Couper et al.

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Lp(a), lipoprotein(a); IDDM, insulin-dependent diabetes mellitus, BMI; body mass index.

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Fitness Levels in IDDM Adolescents

Austin et al. (1) speculate that higher physical fitness levels in adolescents with IDDM may decrease the risk of cardiovascular disease through modulating lipid levels. This hypothesis is supported by recent reports of a greater role for lipids in endurance exercise than was originally thought (2) and by adaptive hyperlipogenesis associated with a certain genetic vigor underlying coronary artery disease and obesity (3).

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IDDM, insulin-dependent diabetes mellitus.

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Assessing Risk Factors When Screening for Diabetes Mellitus

Even though an estimated 6 million cases of diabetes mellitus in the U.S. are undiagnosed, the advantages and disadvantages of screening programs to identify these individuals are controversial (1–5). In its position statement, the American Diabetes Association has proposed that risk factors for diabetes should be assessed and only those individuals with ≥1 risk factors be screened for diabetes (6). These risk factors include *1*) a family history of diabetes mellitus; *2*) obesity; *3*) an at-risk race (American Indian, Hispanic, or black); *4*) previously identified impaired glucose tolerance; *5*) hypertension or significant hyperlipidemia; and *6*) in nonpregnant women, a history of gestational diabetes mellitus or delivery of babies >9 lbs.

To determine the usefulness of these recommendations, we measured both the risk factors for diabetes by a verbal questionnaire and the blood glucose concentrations by a reflectance meter of 575 self-selected participants in a diabetes screening program. Blood glucose was tested regardless of the answers to the questionnaire.

Of the participants, 34% (193) had no risk factors for diabetes mellitus, and 8.9% (51) had abnormal screening glucose concentrations. Of the participants with abnormal screening glucose concentrations, 31% (16) had no risk factors for diabetes mellitus. Overall, 8.3% of the self-selected participants without risk factors for diabetes mellitus had abnormal screening tests, compared with 9.2% of those with ≥1 risk factors (NS). No differences were observed in the abnormal screening glucose concentrations of participants without risk factors for diabetes compared with those with ≥1 risk factors. A history of prior, borderline diabetes was the only risk factor predictive of an abnormal screening test (OR 5.6, 95% CI 2.1–14.6) as was the presence of ≥3 risk factors for diabetes (OR 4.5, 95% CI 1.7–11.4). A follow-up of all participants with abnormal screening tests an average of 1.8 yr after screening (range: 1.1–2.6 yr) revealed that 41% had diabetes mellitus: 6 from the group with no risk factors and 15 from those with ≥1 risk factor (NS).

Assessing diabetes risk factors to identify individuals appropriate for blood glucose screening imposes an administrative burden on those conducting the screening program. This burden is cost-effective only if it aids in selecting

individuals most likely to have an abnormal screening test (and ultimately the diagnosis of diabetes mellitus) and does not exclude these individuals. Our data demonstrate that in a group of self-selected participants in a screening program for diabetes mellitus, assessing risk factors for diabetes mellitus does not increase the proportion of participants with abnormal screening glucose concentrations or with the ultimate diagnosis of diabetes mellitus. Excluding individuals with no risk factors for diabetes from diabetes screening would exclude a significant number of individuals who would have an abnormal screening test. Therefore, the recommendation of screening only individuals with ≥1 risk factors for diabetes should be reconsidered.

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OR, odds ratio; CI, confidence interval.

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Intraperitoneal Insulin Affects Insulin-like Growth Factor Binding Protein-1 in a Well-Controlled Type I Diabetic Patient

The GH/IGF-I axis has been implicated in the pathogenesis of diabetic complications, particularly retinopathy. Insulin administered via the peritoneal cavity is almost entirely absorbed into the portal circulation and would appear as the most physiological method of delivering insulin (1). Two studies have looked at basal GH levels on intraperitoneal insulin regimens and found a decrease along with improved blood glucose profiles (2,3).

IGFBP-1 is produced mainly by the liver in the nonpregnant adult and is inversely regulated by insulin. IGFBP-1 regulation is disordered in type I diabetes, and it has been suggested that elevated levels of IGFBP-1 disturb the IGF-I feedback control of GH secretion (4).

We studied a 19-yr-old woman with type I diabetes on her standard subcutaneous insulin regimen and again 1 wk after the insertion of an intraperitoneal cannula to assess the effects of intraperitoneal insulin delivery on the GH/IGF-I axis. The patient was well controlled with an HbA_1 of 7.5% (5.5–8.5%) and no evidence of complications. Her weight was 58.4 kg, and her BMI was 21.4 kg/m^2. Her diet remained constant in terms of energy, protein, and carbohydrate intake. Activity was limited on both occasions because of the frequent (30 min) sampling.

Her total daily dose of subcutaneous insulin was 72 U divided into 34 U pork Insulatard (protamine) at 0800 and 25 U at 1700 with 10 U of pork Velosulin (soluble) insulin at 0800 and 3 U at 1700. On intraperitoneal insulin the patient received a total of 80 U on the day of the study, 50 U as a variable basal infusion of Actrapid (soluble) insulin, and 30 U as meal and snacktime boluses.

GH was measured by a double antibody IRMA kit (North East Thames Regional Immunoassay Unit, St. Bartholomew's Hospital, London, England). Serum IGF-I was measured by RIA after acid-ethanol cryoprecipitation, using rabbit antiserum 861/5 kindly provided by Professor P. D. Gluckman, Auckland, New Zealand. IGFBP-1 was measured using an IRMA (Medix Biochemica, Kaunianen, Finland). The peak amplitudes of GH were compared between regimens using the Mann-Whitney test; and glucose, IGF-I, and IGFBP-1 were compared using paired Student's *t* tests.

The mean ± SE plasma glucose was 6.7 ± 0.4 mM on subcutaneous insulin and 7.0 ± 0.4 mM on intraperitoneal insulin (n = 48, P = 0.6). On the intraperitoneal insulin regimen the mean serum IGFBP-1 decreased from 9.2 ± 1.9 to 4.4 ± 0.7 μg/L; (n = 24, P = 0.03) (Fig. 1*A* and *B*). A significant decrease in GH peak amplitude (median subcutaneous 38.9 vs. intraperitoneal 28.2 mIU/L; P = 0.03) was observed (Fig. 1*A* and *B*). Total serum IGF-I did not change significantly over the 24-h periods studied (subcutaneous 418 ± 11 vs. intraperitoneal 390 ± 9 ng/ml; n = 24, P = 0.07).

GH regulation has been shown to be disturbed in type I diabetes even when good metabolic control is obtained. Our patient had and maintained good blood glucose control during both studies, thus eliminating the effect that improving blood glucose control has on decreasing GH secretion (5).

A negative-feedback loop on GH release by IGF-I is thought to occur either through the suppression of GH-releasing hormone on GH secretion or by stimulating somatostatin release in the