

tion, and worsened glycemic control, which might be related to the cytochrome P450-mediated metabolic pathway (4,5). The results in this study are an important observation for patients with diabetes and are consistent with another report that pitavastatin, which is metabolized with little involvement of cytochrome P450 isoenzymes (4), did not show these major adverse effects (6). Because pitavastatin has been marketed for only a few years, further studies with a greater number of subjects and a longer duration are needed to establish the safety of this agent.

In conclusion, pitavastatin is effective in lowering LDL cholesterol and triglyceride levels without affecting glycemic control in patients with diabetes. We believe that this agent must also help prevent the development of macrovascular disease in diabetic patients, as has been seen with other statins, but this still requires confirmation in a controlled clinical trial.

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Twelve-Hour Glycemic Profiles With Meals of High, Medium, or Low Glycemic Load

Diets of low glycemic load may dampen the postprandial glycemic response, thereby avoiding high blood glucose concentrations that could be detrimental to health (1). We compared blood glucose profiles of two nondiabetic subjects (A and B) consuming meals of high (baguette, strawberry jam, and maltose), medium (baked potato, cheese, and Coca Cola), or low (chickpeas, tuna, vinegar, and oil) glycemic load at regular intervals throughout the day.

Three test meals contained the same calorie content but different glycemic load for each subject (A: glycemic load = 92, 49, and 19; B: glycemic load = 115, 66, and 24). For each glycemic load category, three full portions of the test meal with a 4-h interval in between and six half-portions with a 2-h interval in between were consumed on 2 different days. For each subject, six 12-h blood glucose profiles deduced from the interstitial glucose in subcutaneous abdominal tissue measured by MiniMed continuous glucose monitoring system were obtained.

A relatively stable blood glucose profile was observed throughout the day with low-glycemic load meals for both subjects (Fig. 1). Consumptions of high- and medium-glycemic load meals were usually followed by peaks of blood glucose. However, there did not appear to be an obvious dose-response effect between the actual glycemic load and the height of the peaks (either full portion versus half portion or high glycemic load versus medium glycemic load), suggesting a possible “threshold” effect (2). Nibbling diets with small frequent meals may only help avoid

hyperglycemia when the meal glycemic load is below a certain threshold level. Compared with the glucose response of the first meal, some of those triggered by each subsequent but identical meal appeared to be lower. This apparently greater “breakfast” glycemic response may be due to higher ACTH and glucocorticoid levels before awakening. Since glycemic index values of food are derived in the fasting state, the glycemic load formula may give better prediction of the postprandial glucose response for breakfast than those for lunch or dinner.

Calculated meal glycemic load may deviate from the actual glycemic response of food combinations. Potential limitations of the continuous glucose monitoring system also need to be considered when interpreting our glycemic profiles (3). In this study, 25% of the sensor-deduced blood glucose concentrations deviated by $\geq 15\%$ from the corresponding fingerstick glucometer values (for calibration) among 48 paired values.

Our pilot study suggests that a stable blood glucose profile can be maintained by consuming a low-glycemic load diet. However, meal glycemic load may need to be below a certain threshold to be of benefit. Identical meals may produce different blood glucose responses at different times of the day, indicating that the glycemic load formula may not predict the postprandial glucose response for meals eaten in the nonfasting state.

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