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## Comparison of the Effects of Vitamins and/or Mineral Supplementation on Glomerular and Tubular Dysfunction in Type 2 Diabetes

Response to Farvid et al.

Recently, Farvid et al. (1) reported on the effects of dietary supplementation of physiologic doses of vitamins and/or minerals on urinary albumin excretion rate (UAER)/urinary protein excretion rate (UPER), blood pressure, and lipid profile. Although not mentioned in the article, data on blood pressure and lipids have previously been reported elsewhere (2,3).

The main finding is a significant reduction in UAER of ~66% in the group receiving both minerals and vitamins. Although Farvid et al. claimed this to be the primary end point, it was only measured once as albumin-to-creatinine ratio in morning spot urine at baseline and after 3 months. Repeated measurements (usually at least three) are always required to obtain valid data and correct diagnosis of persistent micro- and macroalbuminuria due to a coefficient of variation of 30–50%. There is a marked discrepancy be-

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## The Effect of Rosiglitazone on Overweight Subjects With Type 1 Diabetes

Response to Orchard

Our report (1) on the effect of rosiglitazone on overweight subjects with type 1 diabetes showed that rosiglitazone-treated subjects with a BMI  $\geq 30$  kg/m<sup>2</sup> experienced significantly greater improvements in HbA<sub>1c</sub> (A1C) levels than those with a BMI  $< 30$  kg/m<sup>2</sup> (–1.4 vs. –0.4%,  $P = 0.032$ ). In addition, regression analysis showed that BMI, total daily insulin dose, and total, LDL, and HDL cholesterol levels were predictors of improvement in A1C (1). In his letter (2), Orchard raises the intriguing possibility that an estimate of insulin sensitivity (eGDR), which is based on waist-to-hip ratio, hypertension status, and A1C (3), could be

an identifier of type 1 diabetic individuals who might benefit from thiazolidinedione therapy. We calculated eGDR in our subjects and found that in the rosiglitazone-treated subjects, eGDR was significantly related to change in A1C level ( $P = 0.003$ ,  $r = 0.575$ ). No such relationship was found in the placebo-treated subjects. However, a regression analysis incorporating BMI; total daily insulin dose; total, LDL, and HDL cholesterol; and eGDR showed that eGDR was not a significant predictor of improvement in A1C ( $P = 0.155$ ) in the rosiglitazone-treated subjects.

Waist-to-hip ratios were the same in both the rosiglitazone and placebo groups at baseline ( $0.91 \pm 0.06$ ) and at the end of the study ( $0.93 \pm 0.06$ ), which is consistent with the observation that weight gain with thiazolidinediones is mainly peripheral rather than central. Orchard (2) noted that blood pressure but not lipids improved in our rosiglitazone-treated type 1 diabetic subjects. This result was somewhat surprising since we had observed the opposite results in our studies of troglitazone in combination with insulin in type 2 diabetic subjects (4,5). It is important to keep in mind that these studies were not designed to evaluate the effect of thiazolidinedione therapy on blood pressure; all of our subjects were treated with antihypertensive medications in an effort to normalize blood pressure levels. In addition, baseline blood pressure and history of hypertension were not related to change in A1C and were not significant predictors of improvement in A1C level in our rosiglitazone-treated type 1 diabetic subjects. Triglyceride levels also were not related to change in A1C. On the other hand, markers of insulin resistance in the type 1 diabetic subjects, such as BMI, total daily insulin dose, and cholesterol levels, were related to improvement in glycemic control when rosiglitazone treatment was used. Therefore, we do not believe that we can draw any firm conclusions from our data about the relative linkage of blood pressure versus lipid levels to insulin resistance.

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