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The Association between Serum Endogenous Secretory Receptor for Advanced Glycation End Products and Vertebral Fractures in Type 2 Diabetes
(*Endocrinol Metab* 2012;27:289-94, Cheol Ho Lee et al.)

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Recently, it has been suggested that there is a close link between bone and energy metabolism through osteocalcin and the Wnt/β-catenin signaling pathway. In clinical settings, accumulating evidence has shown an association between type 2 diabetes and osteoporosis. However, patients with type 2 diabetes have high fracture rates despite the absence of low bone mineral density [1].

In this context, and with great interest, Lee et al. [2] suggests an alternative link between bone and energy metabolism. In this study, participants with a lower concentration of serum endogenous secretory receptors for advanced glycation end products (esRAGE) had a higher risk of moderate to severe vertebral fractures than those with higher serum esRAGE concentrations. This association was independent of age, sex, duration of diabetes, alcohol consumption, smoking status, body mass index, history of stroke and coronary artery occlusive disease, and the use of thiazolidinedione. This report is quite valid and suggests a novel mechanism to explain the cause of higher fracture rates in patients with type 2 diabetes.

Although the authors have very clearly described their study and their results, in my opinion, some points in this manuscript need to be emphasized. First, to determine the independent association between serum esRAGE concentrations and the risk for vertebral fractures in patients with type 2 diabetes, the authors used a multivariate logistic regression model with adjustment for other risk factors for osteoporotic fracture including age, sex, body mass index, alcohol consumption, smoking status, and the use of thiazolidinedione. However, other well-established risk factors for osteoporotic fracture such as previous fracture history, family history of fracture, and bone mineral density were not included in the model [3]. Thus, the association between serum esRAGE concentrations and the risk for vertebral fracture may be more clearly confirmed with additional adjustment for these risk factors. Second, as the authors mention, the small sample size and low prevalence of vertebral fracture limits any definite conclusions. In addition, measurement of bone mineral density and bone turnover markers would help in the interpretation of the study results and help clarify the pathophysiologic mechanism of high fracture rates in patients with type 2 diabetes.

I hope that my comments add value to this well-written manuscript by Lee et al. [2], which reveals the association between serum esRAGE concentrations and vertebral fractures in type 2 diabetes.

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CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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