

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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The authors would like to thank the reviewers for their time and constructive comments concerning our manuscript. This study analyzed the relationship between vertebral fracture and endogenous secretory receptor for advanced glycation end products (esRAGE) in type 2 diabetes. We verified that patients in the lowest tertile of esRAGE had a higher risk of moderate or severe vertebral fractures than patients in the highest tertile.

It is well known that type 2 diabetes patients had a higher risk of hip or vertebral fractures although they had a similar or slightly higher bone mineral density (BMD) compared to healthy individuals [1,2]. This suggests that reduced bone quality is more profoundly associated with the increase in fracture risk, rather than decreased BMD in type 2 diabetes patients. Previous studies have indicated an increase in advanced glycation end products (AGEs) due to elevated blood glucose, and a decrease in esRAGE interrupting signal transmission within the cells of AGEs, as potential causal factors for deteriorating BMD [3,4].

As we mentioned, this study has statistical limitations. Some risk factors including previous fracture history, family history of osteoporotic fracture, and BMD were not included in multiple regression analysis of the relationship between esRAGE and vertebral fractures. Previous history of fracture was ex-

cluded from the analysis since it was known in only 50% to 60% of the subjects. The major limitation of our study is the lack of BMD measurements, which is an important risk factor for vertebral fractures. In addition, small sample size makes it difficult to draw conclusions. We feel that a prospective study is needed to further investigate the effects of low esRAGE levels on the increased risk of fractures in type 2 diabetes. Thanks again for your interest and comprehensive comments in our paper.

CONFLICTS OF INTEREST

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

REFERENCES

1. Janghorbani M, Van Dam RM, Willett WC, Hu FB. Systematic review of type 1 and type 2 diabetes mellitus and risk of fracture. *Am J Epidemiol* 2007;166:495-505.
2. Yamamoto M, Yamaguchi T, Yamauchi M, Kaji H, Sugimoto T. Diabetic patients have an increased risk of vertebral fractures independent of BMD or diabetic complica-

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- tions. *J Bone Miner Res* 2009;24:702-9.
3. Saito M, Fujii K, Mori Y, Marumo K. Role of collagen enzymatic and glycation induced cross-links as a determinant of bone quality in spontaneously diabetic WBN/Kob rats. *Osteoporos Int* 2006;17:1514-23.
 4. Ogawa N, Yamaguchi T, Yano S, Yamauchi M, Yamamoto M, Sugimoto T. The combination of high glucose and advanced glycation end-products (AGEs) inhibits the mineralization of osteoblastic MC3T3-E1 cells through glucose-induced increase in the receptor for AGEs. *Horm Metab Res* 2007;39:871-5.