研究業績　英文表記

和文	
表題	
著者名	
所属	
英文	
Title	**Transgenic mice overexpressing glia maturation factor－β, an oxidative stress inducible gene, show premature aging due to Zmpste24 downregulation**
Author	**Rika Imai1, Kanae Asai1, Jun－ichi Hanai2, and Masaru Takenaka1**
Affiliation	*1 Clinical Nutrition and Internal Medicine, Kobe Women's University, Kobe 654-8585, Japan;* *2 Renal Division & Division of Interdisciplinary Medicine and Biotechnology, Beth Israel Deaconess Medical Center,* *Harvard Medical School, Boston, MA 02215, USA.*
Abstract	**Glia Maturation Factor-β (GMF), a brain specific protein, is induced by proteinuria in renal tubules. Ectopic GMF overexpression causes apoptosis *in vitro* via cellular vulnerability to oxidative stress. In order to examine the roles of GMF in non-brain tissue, we constructed transgenic mice overexpressing GMF (GMF-TG). The GMF-TG mice exhibited appearance phenotypes associated with premature aging. The GMF-TG mice also demonstrated short lifespans and reduced hair regrowth, suggesting an accelerated aging process. The production of an abnormal lamin A, a nuclear envelope protein, plays a causal role in both normal aging and accelerated aging diseases, known as laminopathies.** **Importantly, we identified the abnormal lamin A (prelamin A), accompanied by a down-regulation of a lamin A processing enzyme (Zmpste24) in the kidney of the GMF-TG mice. The GMF-TG mice showed accelerated aging in the kidney, compared with wild-type mice, showing increased *TGF-β1*, *CTGF* gene and serum creatinine. The gene expression of p21/waf1 was increased at an earlier stage of life, at 10 weeks, which was in turn down-regulated at a later stage, at 60 weeks. In conclusion, we propose that GMF-TG mice might be a novel mouse model of accelerated aging, due to the abnormal lamin A.**
keyword	*glia maturation factor-β , prelamin A, Zmpste24, aging, oxidative stress*

※本データの英文表記は実際の論文上の表記とは異なります。