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Perspective

The Interaction between *NFATC1* and *FOS* Methylation and Post-Menopausal Osteoporosis

Toyonobu Oshita*

Department of Osteoporosis, Sichuan University, Chengdu, Sichuan, China

DESCRIPTION

Genetic and epigenetic factors have a vital role throughout the development of osteoporosis. RANK/RANKL pathway is vital for the bone transforming and *NFATC1* and *C-FOS* are the downtargets of this pathway. Here, we have a tendency to report methylation standing of NFATC1 and *C-FOS* genes in post and premenopausal cases. During this 35 pre-menopausal and 40 post-menopausal cases were enclosed. MS-HRM was used for identification of *NFATC1* and *C-FOS* metyhylation. *NFATC1* was methylated in 12 of the 40 post-menopausal women and *C-FOS* was methylated in 7 of the postmenopausal women. Here, we have a tendency to found statistically important association between unmethylation of *NFATC1* and post-menopausal status. This result explains the epigenetic regulation of osteoclasts throughout the menopausal transition and our results are often used for epigenetic explanation of post-menopausal osteoporosis for the first time [1]. Therefore, our results showed great worth of epigenetic profile of post-menopausal women.

Nuclear issue of Activated T-cell gene family contains five members; that area unit *NFATC1*, *NFATC2*, *NFATC3*, *NFATC4* and *NFATC5* one and that they regulated by the calcium signal pathway. This pathway plays a vital role throughout the regulation of the various systems, for instance; system, skeletal system and cardiovascular system. *NFATC1* regulates bone cell specific genes to be able to regulate osteoclast differentiation, like TRAP, cathepsin K, thyrocalcitonin receptor and *C-FOS*. Bone cell differentiation triggers *NFATC1* expression and this pathway was in check of RANKL (Receptor activator of nuclear factor-kappa B (NF-κB) ligand), NF-κB and c-Fos signaling. RANK/RANKL is that the necessary pathway for the bone remodeling. RANKL-RANK activates *C-FOS*, which was triggers *NFATC1* expression. The interaction between *C-FOS* and bone cell differentiation ascertained in mice and researchers showed the mice whose *C-Fos* knockout they developed osteoporosis.

Due to the genetic and epigenetic factors, osteoporosis reported as a complex multifactorial disease. Many candidate gene association studies had been done however the etiology and molecular mechanism of disease isn't clearly explained. Throughout the menopause, the deficiency of estrogen hormone causes enlarged level of FSH that accelerates bone loss. Differential gene expression of bone cells has been done to solve this advanced interaction. Wu used circulating monocytes for genome wide differential gene expression analysis in osteoporotic and non-osteoporotic postmenopausal Caucasian females and that they identified differential gene expression in circulating monocytes [2]. Generally the result of estrogens, corticosteroids and oxidative stress on osteoblast cells and showed the gene expression profile or polymorphisms of premenopausal and primary osteoporosis cases.

Based on our data, there aren't any epigenetic studies of *NFATC1* and *C-FOS* in post-menopausal women. During this study, we have a tendency to aim to analyse methylation status of *NFATC1* and *C-FOS* genes in post and premenopausal cases and to point out their association between menopausal osteoporosis [3,4]. This can be the primary epigenetic study on postmenopausal women that shows the post-menopausal osteoporosis and *NFATC1* interaction. RAS signal pathway is vital for the bone cell survival and triggers downstream gene activations. *NFATC1* and *C-FOS* are the downtargets of RAS pathway that they need an important role throughout the osteoclastogenesis. *NFATC1* regulates osteoclast specific genes throughout the regulation of bone cell differentiation and c-FOS is one in all the genes that *NFATC1* triggers their expression. *NFATC*-1 is one in the entire necessary transcription factor throughout the differentiation of osteoclast precursors.

Decreased level of estrogen hormone triggers many physiological and hormonal problems and postmenopausal osteoporosis is one in all the results of decreased level of estrogen hormone. The result of steroid hormone on bone cell differentiation showed lack of the estrogen causes failure of apoptosis and inhibits activation of *NFATC1* and c-Src. There is no significant association between the *C-FOS* gene methylation and menopause. However we have a tendency to found important association of unmethylation of *NFATC1* and post-menopausal status. *NFATC1* and bone cell formation interaction has been shown by the *in vitro* studies. In this analysis, we are able to conclude that the epigenetic silencing of the *NFATC1* gene are

Correspondence to: Toyonobu Oshita, Department of Osteoporosis, Sichuan University, Chengdu, Sichuan, China, E-mail: oshita.tb@om.asahi-kasei.co.cn

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often raise osteoclast activity and it's directly connected with post-menopasusal osteoporosis. These results can shed lightweight any epigenetic studies in postmenopausal cases [5]. This study is the initial epigenetic study that investigated the *NFATC1* and *FOS* methylation in post-menopausal cases and shows the interaction between epigenetics and post-menopausal osteoporosis. Though the restricted variety of sample size in our study and lack of epigenetic studies during this field proves our results crucial and so, our results showed magnitude of epigenetic profile of post-menopausal women.

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