Effect of mechanical stretch on the proliferation and differentiation of BMSCs from ovariectomized rats

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Abstract Osteoporosis is characterized by a broken balance between bone formation and bone resorption. Mechanical stress has been considered to be an important factor in bone modeling and remodeling. However, biological responses of stromal cells in osteoporosis to mechanical stimuli remain unknown. To explore the correlation between mechanical stress and osteoblastic differentiation of bone mesenchymal stem cells (BMSCs) in osteoporosis, we built an osteoporosis model in ovariectomized (OVX) rats, and then investigated proliferation, alkaline phosphatase (ALP) activity, and the expression of osteoblastic genes in BMSCs under mechanical stress of 5 and 10 % elongation, using the Flexercell Strain system. The proliferation of BMSCs was detected using alamarBlue. The expression of osteoblastic genes was analyzed by real-time quantitative polymerase chain reaction. Protein expression was examined by Western blotting. BMSCs (OVX) and BMSCs (Sham-operated, Sham in short) proliferations were inhibited at 5 and 10 % elongation at day 3, compared with the un-stretched group, while BMSCs (OVX) proliferation was slower than BMSCs (Sham). ALP activity increased significantly at 10 % elongation in both cells, but it was less active in BMSCs (OVX) than BMSCs (Sham). At days 3 and 7, the mRNA expression of osteoblastic genes was unregulated by mechanical stretch (5 and 10 % elongation); however, osteoblastic gene expression in BMSCs (OVX) was less than that in BMSCs (Sham). The mRNA and protein expression of Runx2 showed similar trends in BMSCs (OVX) under mechanical stretch. These results indicate that the mechanical stretch stimulates osteoblastic differentiation of BMSCs (OVX); however, this differentiation was weaker than that of BMSCs (Sham).

Keywords Ovariectomized rats · Mechanical stretch · BMSCs · Runx2 · Osteoblastic differentiation

Introduction

Osteoporosis is a serious worldwide disease, characterized by decreased bone mass and a progressive erosion of the microstructure. When such disease occurs, osteoblast activity reduces while osteoclast activity increases. As a result, the balance between bone formation and bone resorption is broken, leading to a loss of bone mass and fractures. Osteoporosis may be caused by several conditions, such as hormonal imbalances, chronic diseases, medications, or prolonged gravity-free conditions.

Estrogen plays a fundamental role in skeletal growth and bone homeostasis. The deficiency of Estrogen leads to a chronic inflammatory state because of an increase of the local production of various cytokines [1, 2]. The increased cytokines will then result in an expansion of the osteoclastic pool due to increased osteoclast formation [3].

Current treatments for osteoporosis typically involve pharmacological, hormonal, and mechanical strain interventions.