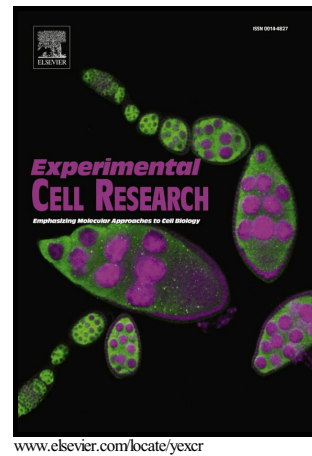


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The calcium transient characteristics induced by fluid shear stress affect the osteoblast proliferation.

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Abstract

Ca²⁺ signaling is essential for bone metabolism. Fluid shear stress (FSS), which can induce a rapid release of calcium from endoplasmic reticulum (ER) to produce calcium transients, plays a significant role in osteoblast proliferation and differentiation. However, it is still unclear of how calcium transients induced by FSS activating a number of downstream signals which subsequently regulate cell functions. In this study, we performed a group of Ca²⁺ transients models, which were induced by FSS to investigate the effects of different magnitudes of Ca²⁺ transients in osteoblast proliferation. Further, we performed a global proteomic profile of MC3T3-E1 cells in different Ca²⁺ transients models stimulated by FSS. GO enrichment and KEGG pathway analysis revealed that the TCA cycle was activated in the proliferating process. The activation of TCA needed mitochondrial Ca²⁺ uptake which were influenced by the amplitude of Ca²⁺ transients induced by FSS. Our work elucidate that osteoblast proliferation induced by FSS was related to the magnitude of calcium transients, which further activated energetic metabolism signaling pathway. This work revealed further understanding the mechanism of osteoblast proliferation induced by mechanic loading and help us to design new methods for osteoporosis therapy.

Keyword: shear stress, proteomic, TCA cycle, calcium transients, mitochondrial calcium uptake

1 Introduction

Fluid shear stress (FSS) affected both proliferation and differentiation of osteoblasts.

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