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Abstract:

Bone cells can sense physical forces and convert mechanical stimulation conditions into biochemical signals that lead to expression of mechanically sensitive genes and proteins. However, it is still poorly understood how genes and proteins in bone cells are orchestrated to respond to mechanical stimulations. In this research, we applied integrated proteomics, statistical, and network biology techniques to study proteome-level changes to bone tissue cells in response to two different conditions, normal loading and fatigue loading. We harvested ulna midshafts and isolated proteins from the control, loaded, and fatigue loaded Rats. Using a label-free liquid chromatography tandem mass spectrometry (LC-MS/MS) experimental proteomics technique, we derived a comprehensive list of 1,058 proteins that are differentially expressed among normal loading, fatigue loading, and controls. By carefully developing protein selection filters and statistical models, we were able to identify 42 proteins representing 21 Rat genes that were significantly associated with bone cells' response to quantitative changes between normal loading and fatigue loading conditions. We further applied network biology techniques by building a fatigue loading activated protein-protein interaction subnetwork involving 9 of the human-homolog counterpart of the 21 rat genes in a large connected network component. Our study shows that the combination of decreased anti-apoptotic factor, Raf1, and increased pro-apoptotic factor, PDCD8, results in significant increase in the

number of apoptotic osteocytes following fatigue loading. We believe controlling osteoblast differentiation/proliferation and osteocyte apoptosis could be promising directions for developing future therapeutic solutions for related bone diseases.

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