Mohammad Hassan, Orthop Muscul Syst 2014, 3:2
http://dx.doi.org/10.4172/2161-0533.S1.016

OMICS Group Conferences
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3rd International Conference and Exhibition on
Orthopedics & Rheumatology
July 28-30, 2014 DoubleTree by Hilton Hotel San Francisco Airport, USA

MicroRNA 23a~27a~24-2 cluster regulation of bone formation

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MicroRNAs (miRNAs) are single-stranded non-coding RNAs of ~22 nucleotides which repress gene expression to provide a refined level of gene regulation in a broad spectrum of biological processes and disease. Inhibition of mRNA stability or the translation by miRNAs has emerged as a key developmental switch for the commitment of mesenchymal cells to the osteoblast lineage, growth, and differentiation and post natal bone homeostasis in the adult skeleton. In this study it was identified a cluster of 3 microRNAs (miR-23a, 27a and 24-2) from preosteoblast MC3T3-E1 cells, located on mouse chromosome 8, that inhibits osteogenesis by targeting SATB2 and Runx2. Current understanding of this emerging concept of bone-regulating miRNAs is lacking *in vivo* mouse genetic studies of their function. To fill in this gap of knowledge a miR-23a~27a~24-2 cluster knockout (PuroΔTK) and a knockdown (TRE-MIRZIP) mouse model was created. Heterozygous knockout mouse has developed a high bone mass phenotype. It has been seen that miR-cluster promotes growth of preosteoblasts by controlling RB1 phosphorylation, cMYC induction and activated Wnt signaling. Similar to our findings, metastatic osteosarcoma cell line have demonstrated a significantly higher level miR-23a, and 27a expression. Immunoprecipitation of the miR cluster-induced silencing complex (RISC) aided in the identification of several HoxA class factors (HoxA5, A10 & A11) as potential targets to inhibit *in vitro* differentiation. These RNP-IP studies also discovered several key chromatin remodeling factors all involved in stage and tissue specific epigenetic regulation. When taken together, our findings lead us to conclude that miR cluster -23a~27a~24-2 controls osteoblast growth, differentiation and post natal bone homeostasis. The studies will significantly add to the current understanding of the novel role of miR-cluster function to regulate bone formation and could translate to potential therapeutics for bone regeneration, skeletal disorders and osteosarcoma.

Biography

Mohammad Hassan obtained his PhD from Indian Institute of Chemical Biology, Kolkata, India, in gene expression of protozoan parasites and then carried out postdoctoral research at the University of Massachusetts Medical School, Worcester, USA. He is currently an assistant professor in the department of Oral and Maxillofacial Surgery, School of Dentistry at the University of Alabama, Birmingham, USA. He discovered several key mechanisms of miRNA function in bone formation and homeostasis. His lab is currently focused to study the *in vivo* regulation of miR-23a~27a~24-2 cluster in bone and craniofacial tissue formation. He is well recognized in the field of bone biology and serves the editorial board of several basic and bone biology journals.

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