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MicroRNA MIR146A regulates osteogenic differentiation in human mesenchymal stem cells

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Chromatin remodeling influences the differentiation of stem cells. Histone methyltransferase enhancer of zeste homolog 2 (EZH2) and histone demethylase jumonji domain containing 3 (JMJD3; KDM6B) modulate levels of histone H3 lysine 27 trimethylation (H3K27me3) that impact chromatin, but the regulation of these epigenetic modulators in stem cells is poorly defined. The precise mechanisms that control the expression of EZH2 and JMJD3 during stem cell differentiation are not clear. Using the osteogenic differentiation of human mesenchymal stem cells (hMSCs) as a model system, we identified microRNA MIR146A to be a negative regulator of JMJD3. hMSC differentiation toward osteoblasts increased JMJD3 expression but decreased H3K27me3 and MIR146A levels. Overexpression of MIR146A in differentiating hMSCs downregulated JMJD3 and osteogenic marker runt-related transcription factor 2 (RUNX2), but upregulated H3K27me3. We hypothesize that MIR146A inhibits JMJD3 in uncommitted, multipotent hMSCs to prevent the initiation of key chromatin remodeling events that occur during osteogenic differentiation. Modulating MIR146A expression in hMSCs could optimize their therapeutic potential by influencing the long-term maintenance of multipotency and by directing their differentiation in the osteoblastic lineage.

Biography

Christopher J Payne received his PhD in Cell Biology from the Oregon Health & Science University, USA, in 2003. He continued his training with Postdoctoral fellowships at the Fred Hutchinson Cancer Research Center, the University of Washington and the Jackson Laboratory, USA, from 2004 through 2009. Since 2009, he is an Assistant Professor in Human Molecular Genetics at Northwestern University School of Medicine in Chicago. He has published 26 papers in journals such as Cell, Science, Stem Cells and Development. In 2012, he received the Clinical Science Award from the American College of Embryology. He serves on the Editorial Board of *Andrology - Open Access*.

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