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The inactivation of tumor suppressor genes by increased H3K9me3 drives spontaneous transformation of rat MSCs

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Rodent mesenchymal stromal cells (MSCs) have been demonstrated to spontaneously undergo tumorigenic transformation after long term *ex vivo* culture. The mechanism leading to the MSCs spontaneous transformation is unclear. To investigate the role of H3K9me3 for the spontaneously transformation of MSCs, the pre-senescent and transformed MSCs were prepared according to the criterion described previously. H3K9me3 target genes were evaluated with ChIP-on-chip arrays. The expression of tumor suppressor genes (*CDKN2B, CDKN2C, CDKN1C* and *PTEN*) was evaluated with RT-qPCR, these gene-associated H3K9me3 were quantified with chromatin immunoprecipitation (ChIP) and the DNA methylation levels were analyzed with bisulfite DNA sequencing (BSP). We found that there were 1277 genes in pre-senescent MSCs and 2519 genes in transformed MSCs targeted by H3K9me3 (Cutoff: FDR≤0.05). The genes associated with H3K9me3 are related to the catalogs of cell differentiation, development and nucleotide and protein metabolism. The expression of *CDKN2B, CDKN2C, CDKN1C* and *PTEN* were obviously decrease in transformed MSCs, with an up-regulation in genes associated H3K9me3 and CpG sites methylation. These results demonstrate that an H3K9me3 enhanced DNA methylation contributes a crucial role in the spontaneous transformation of MSCs.

Biography

Yong Zheng is presently studying the mechanisms underlying these changes of epigenetic modification. He has his research interests in understanding the molecular mechanisms of tumorigenesis. He has identified the key stages during the spontaneous of rat mesenchymal stem cells and his previous study identified, for the first time, an Ezh2/H3K27me-independent and H3K9me enhanced aberrant DNA methylation of the *p16* gene, which might be an epigenetic signature for MSC spontaneous transformation.

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