

Elucidating the bone developmental defect in Werner Syndrome using stem cells



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Abstract

Werner Syndrome (WS) is an autosomal recessive genetic disorder characterized by premature aging. This disease is caused by mutations in the WRN gene. The first sign of WS is short stature. Individuals with WS have an abnormally slow growth rate, and growth stops at puberty. As a result, affected individuals have short stature. However, the mechanism is still not clear. To find out the cause of short stature in WS, the author reprogrammed the WS patients' fibroblasts and the isogenic normal control into mesenchymal stem cells and compared the transcriptome by RNA-Seq. Among the top ten candidates, SHOX (short stature homeobox) was noted. SHOX plays an important role in chondrogenesis. SHOX deficiency is a frequent cause of short stature. So, what is the role of SHOX in WS pathogenesis? To answer this question, the author induced hESCs towards chondrocytes and found that the loss of WRN and SHOX blocked the chondrocyte differentiation. In summary, they found that the down regulation of SHOX in WS inhibited chondrogenesis at the early developmental stage, which may account for the short stature.

Biography

Tian Yuyao is a PhD pursuing candidate in The Chinese University of Hong Kong, School of Biomedical Sciences Hong Kong. She has completed her master study from Peking University.



3rd International Conference on Stem Cells and Regenerative Medicine, June 29-30, 2020

Citation: Tian Yuyao, Elucidating the bone developmental defect in Werner syndrome using stem cells, Stem Cell Congress 2020, 3rd International Conference on Stem Cells and Regenerative Medicine, June 29-30, 2020, 06