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Pot1a regulates self-renewal activity of hematopoietic stem cells

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Aging or repeated cell division induces the accumulation of DNA damage which impairs hematopoietic stem cell (HSC) function. Protection of telomeres 1 (Pot1), a component of shelter in contributes to the suppression of unnecessary DNA damage response (DDR) at telomeres. We identified that high levels of Pot1a was expressed in HSCs and that this expression decreased shapely with age. Knockdown of Pot1a increased telomeric DDR and the frequency of symmetric differentiation divisions in cultures and significantly reduced long-term reconstitution (LTR) activity. In contrast, overexpression of Pot1a or treatment with exogenous Pot1a protein Pot1a protein prevented telomeric DDR and maintained symmetric self-renewing divisions and LTR activity in HSCs indicating that Pot1a rejuvenated stem cell activity of HSCs. Human POT1 protein also increased the number of cord blood HSCs. These data suggest that the protection of telomeric DNA from DDR signaling is critical for sustained self-renewal of HSCs and that Pot1a is a novel target for *ex vivo* expansion of HSCs.

Biography

Fumio Arai has completed his PhD from Meikai University and Postdoctoral studies from Keio University School of Medicine. He is a Professor of Department of stem cell biology and Medicine, Graduate School of Medical Sciences, Kyushu University. His research interest is in studying the mechanisms of the cell fate regulation of HSCs at the single cell level for the establishment of the system that is able to expand HSCs.

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