Rapamycin proves both good and bad for aging in human cells

Norihiko Sasaki
Yoko Itakura
Masashi Toyoda

Video Byte

Keywords: Cell Communication and Signaling, Tokyo Metropolitan Institute of Gerontology, rapamycin, senescence-associated secretory phenotype, endothelial cells, stress-induced premature senescence, endothelial-mesenchymal transition, autophagy, anti-aging, blood vessels, oxidative stress, hydrogen peroxide, coronary artery, polymer chain reaction, intercellular adhesion molecule-1, ganglioside GM1, leukocytes, adhesion assay, immunostaining, TGF-β pathway

Posted Date: April 29th, 2020

DOI: https://doi.org/10.21203/rs.3.rs-26255/v1

Abstract

Rapamycin is a promising candidate as an anti-aging drug. Normally used to fight cancer, rapamycin has been shown to extend the lifespan of mice. But what about humans? To find out, researchers monitored the effect of rapamycin on test tubes of human coronary artery endothelial cells. These cells are integral to the structure and homeostasis of blood vessels in the heart but tend to harden with age, posing a looming threat to older individuals. Experiments showed that while rapamycin suppressed the expression of certain senescence-related proteins, it actually promoted endothelial cell differentiation into mesenchymal cells through morphological changes by activating autophagy, causing a functional modification. Because heart function is supported by the vascular network, induction of the endothelial–mesenchymal transition can drive the pathogenesis of heart disease. These findings serve as a healthy warning for clinical studies: Should rapamycin be used for its anti-aging properties, it would best be combined with drugs that limit its negative side effects.