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ROS might just be a sideshow: Exploring simultaneous observation of mTORC1 activity and increase in ROS

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There is huge debate regarding whether reactive oxygen species (ROS) are the cause for aging. Meta-analyses indicate antioxidants have little effect, if any, on lifespan. This mandates the need for rethinking causality of ROS in senescence. In this article, high quality studies (n=20) were sorted into three categories: Basic, clinical/meta-analytic and ROS-mTOR relationship. Evidences were compared to discover inconsistencies and bigger-picture revelations. Studies showed simultaneity of mTORC1 activity and increased ROS. Basic studies suggested that ROS causes cell damage and genomic instability leading to aging. Nevertheless, meta-analyses clarify antioxidants have literally zero outcome affecting lifespan. This questions the causal role of ROS in senescence. Considering that hyperactive mTORC1 intensifies aging while decreasing ROS has little benefit, ROS could be thought of as mere chemical byproducts with no causal role and can be eliminated from the picture. This new perspective also indicates that it is the time to look for other roles for ROS rather than regarding it as the cause of senescence.

Biography

Mohammad Farahmandnia is a undergraduate Medical Doctor student at Shiraz University of Medical Science. He is a holder of Silver Medals in Biology and Basic Medical Science Olympiads. He has also co-authored 6 papers in reputed journals on stem cell research. He is currently a Member of Cell and Molecular Medicine Student Research Group\ with particular focus on systems biology of aging.

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