Mitochondrial hormesis links low-dose arsenite exposure to lifespan extension.

Schmeisser S, Schmeisser K, Weimer S, Groth M, Priebe S, Fazius E, Kuhlow D, Pick D, Einax JW, Guthke R, Platzer M, Zarse K, Ristow M (2013) Mitochondrial hormesis links low-dose arsenite exposure to lifespan extension. *Aging Cell* 12(3), 508-517.

Details

Abstract

Arsenite is one of the most toxic chemical substances known and is assumed to exert detrimental effects on viability even at lowest concentrations. By contrast and unlike higher concentrations, we here find that exposure to low-dose arsenite promotes growth of cultured mammalian cells. In the nematode C. elegans, low-dose arsenite promotes resistance against thermal and chemical stressors and extends lifespan of this metazoan, whereas higher concentrations reduce longevity. While arsenite causes a transient increase in reactive oxygen species (ROS) levels in C. elegans, co-exposure to ROS scavengers prevents the lifespan-extending capabilities of arsenite, indicating that transiently increased ROS levels act as transducers of arsenite effects on lifespan, a process known as mitohormesis. This requires two transcription factors, namely DAF-16 and SKN-1, which employ the metallothionein MTL-2 as well as the mitochondrial transporter TIN-9.1 to extend lifespan. Taken together, low-dose arsenite extends lifespan, providing evidence for nonlinear dose-response characteristics of toxin-mediated stress resistance and longevity in a multicellular organism.

Involved units

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Leibniz-HKI-Authors



Reinhard Guthke

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Steffen Priebe

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Topics

RNA-Seq data analysis and modeling

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