

Webinar

Linking cellular stress responses to systemic physiology: mechanisms and Consequences

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All cells possess conserved mechanisms to counteract the toxic effects of stress. Heat shock response (HSR) is one of the most conserved mechanisms that enables the cells to protect the integrity of their proteome under high temperature stress. HSR is traditionally characterised by robust and transient synthesis of heat shock proteins (HSPs) due to stress-induced activation of the conserved transcription factor, heat shock factor 1 (HSF1). However, the effects of activating stress response pathways beyond protecting the cellular proteome remained unknown for long time. My work using the nematode Caenorhabditis elegans as a model organism showed that the neurotransmitter serotonin, released by the maternal neurons upon stress, initiates a conserved transcriptional program in C. elegans germline that protects the future progeny from heat stress. Furthermore, activation of HSF-1 in maternal germline confers enhanced stress resilience to the progeny by establishing a heritable epigenetic 'memory' of stress. This study, therefore, uncovers novel functions of HSF-1 and describes the effect of cellular stress response on organismal physiology and proteostasis.

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