Memory of temperature perception controls peroxide resistance in *C. elegans*

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Background

Organisms use sensory information to adjust their development, metabolism, lifespan, and heat defenses. *Caenorhabditis elegans* is an excellent model to study these phenomena due to a simple nervous system, short lifespan, strict developmental progression, and a fully sequenced genome. Recently, we have shown that worms use sensory information to coordinate resistance to peroxides using a collection of stains in which sensory neurons are genetically ablated using cell-specific caspases (Fig. 1). Temperature sensing neurons are overrepresented, which suggests a role for temperature perception in the coordination of peroxide resistance. We investigated the extent to which temperature and temperature sensation regulates peroxide resistance. We found that the worm's history of temperature experiences modulates the animal's peroxide resistance dependent upon thermosensory machinery within the AFD neurons that signal to DAF-16/FOXO and SKN-1/NRF to control peroxide resistance. We propose that the AFD neurons are essential components of a sensory network that integrates distinct environmental information to mount a coordinated peroxide resistance response that is most appropriate to current environmental conditions. Understanding how sensory information controls peroxide resistance in *C. elegans* may provide a template for understanding how more complex animals coordinate their cellular stress responses.

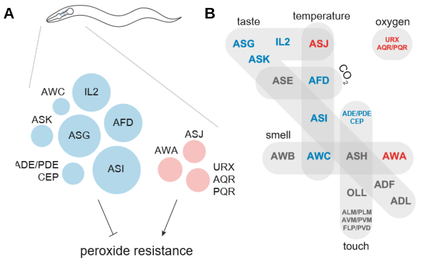
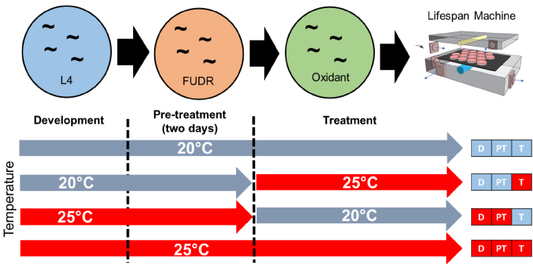


Figure 1. Many sensory neurons regulate resistance to oxidative stress. A) Individual pairs or subsets of sensory neurons were genetically ablated with cell-specific caspases and survival to 6 mM *tert*-butyl hydroperoxide at day two of adulthood was determined using the Lifespan Machine. The size of the circle corresponds to the percent difference in survival compared to wild type. B) A diagram of the sensory neurons organized by major function. Thermosensory neurons are overrepresented. Blue, red, and grey correspond to an increase, decrease or no effect of survival respectively.

Methods



Temperature history controls peroxide resistance

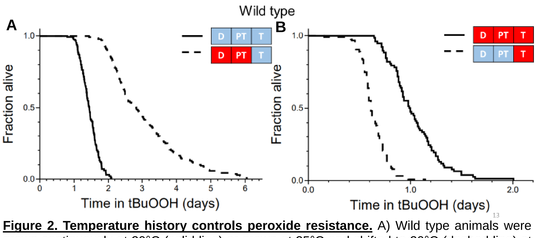


Figure 2. Temperature history controls peroxide resistance. A) Wild type animals were grown continuously at 20°C (solid line) or grown at 25°C and shifted to 20°C (dashed line) at day two of adulthood and survival to 6 mM *tert*-butyl hydroperoxide was determined using the lifespan machine. B) Wild type animals were grown continuously at 25°C (solid line) or grown at 20°C and shifted to 25°C (dashed line) at day two of adulthood and survival to to 6 mM *tert*-butyl hydroperoxide was determined using the lifespan machine.

The AFD neurons, DAF-16/FOXO, and SKN-1/NRF are required for temperature history to control peroxide resistance

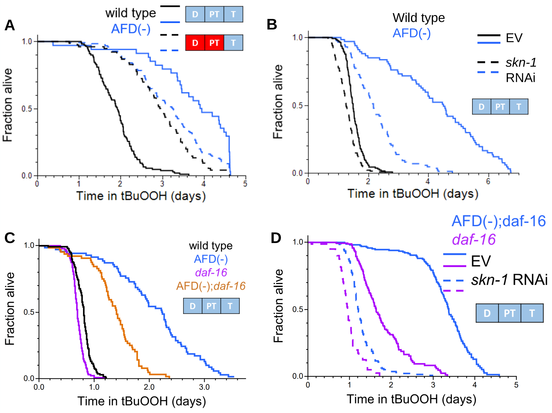


Figure 3. Temperature history requires AFD, DAF-16/FOXO, and SKN-1/NRF to control peroxide resistance. A) Wild type and AFD-ablated animals were grown continuously at 20°C or grown at 25°C and shifted to 20°C at day two of adulthood and survival to 6 mM *tert*-butyl hydroperoxide was determined using the lifespan machine. B) Wild type and AFD-ablated animals were grown at 20°C with empty vector of *skn-1* RNAi and survival to 6 mM *tert*-butyl hydroperoxide was determined at day two of adulthood using the lifespan machine. C) Wild type, AFD-ablated, *daf-16(mu86)*, and AFD-ablated;*daf-16(mu86)* animals were grown at 20°C and survival to 6 mM *tert*-butyl hydroperoxide was determined using the lifespan machine. D) *daf-16(mu86)* mutants and AFD(-);*daf-16(mu86)* animals were grown at 20°C with empty vector or *skn-1* RNAi and survival to 6 mM *tert*-butyl hydroperoxide was determined at day two of adulthood using the lifespan machine.