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genes that act downstream of DAF-16 to influence the lifespan of *Caenorhabditis elegans* [2015, 59, 639–650]. [Google Scholar] [CrossRef] [Yoneda, T.; Benedetti, C.; Urano, F.; Clark, S.G.; Harding, H.P.; Ron, D. Nature 2017, 552, 187–193. PIM1 encodes a mitochondrial ATP-dependent protease that is required for mitochondrial function in the early Saccharomyces cerevisiae. The percentage of “runaway” worms from the lawn was scored for each mutant (Nott/NT100 × C). [CrossRef] 2018, 8, 254. In all cases, approximately 50 worms per strain and condition were used in three biological replicates, and the mean of calculated values was plotted. Heat shock transcription factor 1 (HSF1) induces the expression of a group of molecular chaperones, known as heat-shock proteins (hsp), which assist in cytosolic protein folding during stress. Even though the function of LonP1 in tumorigenesis is still poorly understood, inhibition of the protease is likely associated with decreased rates of tumor cell growth, proliferation, and metastasis [18]. We would like to thank Harris Patratis (Laboratory of Cell Proliferation and Aging, Institute of Biosciences and Applications, NCSR “Demokritos”, Athens, Greece) for his assistance in cell cycle analysis. Replicates and statistical analysis of lifespan assays are shown in Supplementary Table S3. Resistance to C (HS 35 °C for 6 h), D (osmotic stress (500 mM NaCl) for 2 h), and E (oxidative stress (100 μM H₂O₂) for 2 h) were assayed in triplicate. The effect of the pharmacological treatment of worms with the antioxidant Trolox (100 μM) on the lifespan of the worms was assayed in triplicate. Knockdown caused clear mitochondrial dysfunction in both cell lines, activating an apoptotic cell suicide program. This suggests that complementary mechanisms are activated upon LonP1 disruption to combat mitochondrial stress. Reverse transcription using random primers was carried out with FIRESCRIPT RT-cDNA Synthesis Kit (Solis BioDyne, Tartu, Estonia), and quantitative PCR was performed using KAPA SYBR FAST Universal Kit (Kapa Biosystems, Wilmington, MA, USA) in the MJ MiniOpc system (BioRad, Hercules, CA, USA). [CrossRef] 2015, 11, e004972. Learn at your own pace from top companies and universities, apply your new skills to hands-on projects that showcase your expertise to potential employers, and earn a career credential to kickstart your new career. With MasterTrack® Certificates, portions of Master’s programs have been split into online modules, so you can earn a high quality university-issued career credential at a breakthrough price in a flexible, interactive format. In the right panel, the molecular modeling of the SSD domain of human (HsLonP1), bacterial (EColn), Arabidopsis (AtLon1), and worm (CelLonP1) proteases is presented. 2002, 532, 103–106. Briefly, 10 to 150 animals of each strain in late L4 larvae to a young adult stage were transferred to NGM plates (30–40 per plate) seeded with OP50 or HT115 (DE3) bacteria (Day 1 of lifespan assay) and moved to fresh plates every 2–4 days. In human HeLa cells, depletion of LonP1 increased mitochondrial ROS production and the levels of oxidized proteins [9], though discrepancies still exist regarding the induction of stress-responsive signaling pathways, such as the UPRmt [44] or the ISR pathways [41]. Herein, we have investigated the effects of the loss of LonP1 on the mitochondrial network and the levels of oxidative stress in worms. The effect of the loss of LonP1 on the mitochondrial network and the levels of oxidative stress in worms was assayed in triplicate. *Drosophila melanogaster* [12,13,14,15]. In humans, LonP1 mutations are associated with CODAS syndrome, a rare developmental disorder affecting multiple organs [16]. Mitochondrial Perturbations Couple mTORC2 to Autophagy in C. Moreover, little is yet known about how LonP1 deficiency affects cellular and organismal physiology or responses to a broad range of challenges. Hydrogen peroxide levels were measured as the ratio of oxidized to reduced HyPer intensity. SUMO peptidase ULP-4 regulates mitochondrial UPR-mediated innate immunity and lifespan extension. Combined, these data indicate that CDDO-Me, contrary to lonp-1 gene deletion, is not able to activate UPRmt in worms. Multi-omics analysis identifies ATF4 as a key regulator of the mitochondrial stress response in mammals. 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