

Resource Summary Report

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Interventions Testing Program

RRID:SCR_008266

Type: Tool

Proper Citation

Interventions Testing Program (RRID:SCR_008266)

Resource Information

URL: <http://www.nia.nih.gov/research/dab/interventions-testing-program-itp>

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Description: NIA's ITP is a multi-institutional study investigating treatments with the potential to extend lifespan and delay disease and dysfunction in mice. Priority consideration will be given to the treatments that are easily obtainable, reasonably priced, and can be delivered in the diet (preferred) or water. Interventions that require labor intensive forms of administration, such as daily injections or gavage, are not feasible within the design of the ITP. Treatments currently under study include: - Pharmaceuticals - Nutraceuticals - Foods - Diets - Dietary supplements - Plant extracts - Hormones - Peptides - Amino acids - Chelators - Redox agents - Other agents or mixtures of agents. Although the mice involved in this study will be housed at the University of Michigan, the Jackson Laboratories, and the University of Texas Health Sciences Center at San Antonio, the project is designed to involve collaborations with investigators at any university, institute, or other organization that has ideas about pharmacological interventions that might decelerate aging and wishes to test these in a lifespan study of mice. Sponsors: This program is supported by the National Institute of Aging.

Synonyms: ITP

Resource Type: research forum portal, disease-related portal, portal, topical portal, data or information resource

Keywords: dysfunction, extract, food, gavage, acid, administration, agent, amino, chelator, diet, dietary, disease, hormone, injection, intervention, lifespan, mixture, mouse, nutraceutical, peptide, pharmaceutical, plant, redox, supplement, treatment, water

Related Condition: Aging

Resource Name: Interventions Testing Program

Resource ID: SCR_008266

Alternate IDs: nif-0000-23305

Old URLs:

<http://www.nia.nih.gov/ResearchInformation/ScientificResources/InterventionsTestingProgram.htm>

Record Creation Time: 20220129T080246+0000

Record Last Update: 20260731T042753+0000

Ratings and Alerts

No rating or validation information has been found for Interventions Testing Program.

No alerts have been found for Interventions Testing Program.

Data and Source Information

Source: [SciCrunch Registry](#)

Usage and Citation Metrics

We found 31 mentions in open access literature.

Listed below are recent publications. The full list is available at [dkNET](#).

Jiang N, et al. (2025) Deciphering the timing and impact of life-extending interventions: temporal efficacy profiler distinguishes early, midlife, and senescence phase efficacies. Nature communications, 16(1), 10164.

Harrison DE, et al. (2024) Astaxanthin and meclizine extend lifespan in UM-HET3 male mice; fisetin, SG1002 (hydrogen sulfide donor), dimethyl fumarate, mycophenolic acid, and 4-phenylbutyrate do not significantly affect lifespan in either sex at the doses and schedules used. GeroScience, 46(1), 795.

Ashiqueali SA, et al. (2024) Early life interventions metformin and trodusquemine metabolically reprogram the developing mouse liver through transcriptomic alterations. Aging cell, 23(9), e14227.

Jiang N, et al. (2024) The Gehan test identifies life-extending compounds overlooked by the log-rank test in the NIA Interventions Testing Program: Metformin, Enalapril, caffeic acid phenethyl ester, green tea extract, and 17-dimethylaminoethylamino-17-demethoxygeldanamycin hydrochloride. GeroScience, 46(5), 4533.

Watanabe K, et al. (2023) Lifespan-extending interventions induce consistent patterns of fatty acid oxidation in mouse livers. Communications biology, 6(1), 768.

Song J, et al. (2023) Age-associated adipose tissue inflammation promotes monocyte chemotaxis and enhances atherosclerosis. Aging cell, 22(2), e13783.

Garratt M, et al. (2022) Lifespan extension in female mice by early, transient exposure to adult female olfactory cues. *eLife*, 11.

Sato T, et al. (2022) Aging is associated with increased brain iron through cortex-derived hepcidin expression. *eLife*, 11.

Li X, et al. (2022) Cap-independent translation of GPLD1 enhances markers of brain health in long-lived mutant and drug-treated mice. *Aging cell*, 21(9), e13685.

Wink L, et al. (2022) Rapamycin, Acarbose and 17 β -estradiol share common mechanisms regulating the MAPK pathways involved in intracellular signaling and inflammation. *Immunity & ageing : I & A*, 19(1), 8.

Harrison DE, et al. (2021) 17- α -estradiol late in life extends lifespan in aging UM-HET3 male mice; nicotinamide riboside and three other drugs do not affect lifespan in either sex. *Aging cell*, 20(5), e13328.

Shen Z, et al. (2021) Cap-independent translation: A shared mechanism for lifespan extension by rapamycin, acarbose, and 17 β -estradiol. *Aging cell*, 20(5), e13345.

Miller RA, et al. (2020) Canagliflozin extends life span in genetically heterogeneous male but not female mice. *JCI insight*, 5(21).

Herrera JJ, et al. (2020) Acarbose has sex-dependent and -independent effects on age-related physical function, cardiac health, and lipid biology. *JCI insight*, 5(21).

Harrison DE, et al. (2019) Acarbose improves health and lifespan in aging HET3 mice. *Aging cell*, 18(2), e12898.

Cheng CJ, et al. (2019) Genetically heterogeneous mice exhibit a female survival advantage that is age- and site-specific: Results from a large multi-site study. *Aging cell*, 18(3), e12905.

Garratt M, et al. (2018) Male lifespan extension with 17- β estradiol is linked to a sex-specific metabolomic response modulated by gonadal hormones in mice. *Aging cell*, 17(4), e12786.

Ashbrook DG, et al. (2018) Post-genomic behavioral genetics: From revolution to routine. *Genes, brain, and behavior*, 17(3), e12441.

Folch J, et al. (2018) Experimental Models for Aging and their Potential for Novel Drug Discovery. *Current neuropharmacology*, 16(10), 1466.

Garratt M, et al. (2017) Sex differences in lifespan extension with acarbose and 17- β estradiol: gonadal hormones underlie male-specific improvements in glucose tolerance and mTORC2 signaling. *Aging cell*, 16(6), 1256.