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ORIGINAL RESEARCH

Cinnamon in the diet promotes longevity and extends healthspan through mTORC1 signaling and autophagy - A review of the evidence

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1. ABSTRACT

The analyzed study evaluated cinnamaldehyde, the main volatile compound in cinnamon oil, in the nematode *Caenorhabditis elegans* and in human cell lines. In the worms, dietary administration prolonged survival at certain concentrations, increased resistance to oxidative stress, and delayed paralysis in a model of β-amyloid toxicity. Genetic experiments indicated that these effects required components of mTORC1 signaling and autophagy. In HEK293T cells, cinnamaldehyde reduced biochemical markers of mTORC1 activity and increased markers consistent with enhanced autophagic flux.In paragraph 30.1 of "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi, the publication contributes primarily to the narrative that cinnamon can induce a state similar to caloric restriction, promoting cellular maintenance and longevity without reducing food intake. However, the study did not directly measure superoxide dismutase or catalase, nor can it demonstrate a longevity effect in humans. Independent research confirms that cinnamaldehyde can modulate autophagy and redox markers in specific cellular and animal models, but also shows varying effects across organs, doses, and pathological conditions.

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MISSION

LIFE Science Hub – Journal of Science was founded with the aim of making scientific knowledge accessible without giving up methodological rigour, quality of sources and authoritativeness of content. The journal promotes the dissemination of knowledge in the health, nutrition, medical and longevity sciences, building a bridge between the world of research and society.

Through articles, reviews, in-depth analyses and expert contributions, the Journal champions evidence-based medicine and science, fostering a correct interpretation of scientific data and countering misinformation. Every piece of content is developed with the aim of translating the complexity of research into understandable, useful and applicable information, while keeping intact the principles of accuracy, transparency and scientific independence.

Free from political, industrial and commercial influence, the journal stands as an authoritative and independent editorial space, dedicated to promoting scientific culture, professional development and the growth of health awareness among the public.

2. ANALYZED STUDY

Original title: *Dietary cinnamon promotes longevity and extends healthspan via mTORC1 and autophagy signaling.* The authors are Guo Y, Zhang Q, Zhang B, Pan T, Ronan EA, Huffman A, He Y, Inoki K, Liu J, and Xu XZS. The research was published in *Aging Cell* in 2025;24:e14448. DOI: 10.1111/acel.14448. PMID: 39760475. PMCID: PMC11984692.The research question was whether cinnamaldehyde, the main component of cinnamon oil, could influence lifespan and healthspan in *C. elegans*, and through which biological signals. The hypothesis was that dietary cinnamaldehyde acts on the mTORC1 pathway, a protein complex that integrates nutrient and growth signals, and on autophagy, the system by which the cell degrades and recycles damaged or unnecessary components.The relevance to paragraph 30.1 of "The Choice to Live in Health or Illness" by Dr. A. Colonnese and R. Panzironi is specific. The publication documents that, in worms, cinnamaldehyde can produce a transcriptional and functional response similar to that of dietary restriction, even without reducing the amount of food ingested. It also documents an increase in resistance to oxidative stress. However, it does not demonstrate that cinnamon intake in humans increases longevity, activates the same mechanisms in human tissues, or directly increases the enzymes superoxide dismutase and catalase.

3. MATERIALS AND METHODS OF THE ORIGINAL STUDY

The study is experimental and primarily used *C. elegans*, a small nematode widely used in the biology of aging. The worms were kept at 20 °C and fed *Escherichia coli* OP50 bacteria. For most survival tests, approximately 100 worms synchronized at the L4 larval stage were exposed to cinnamaldehyde added to their food; in many experiments, 25 µM was used until the twelfth day, and 100 µM thereafter. The control groups received the same volume of solvent, ethanol, without cinnamaldehyde.The authors compared dietary exposure with exposure to the substance's scent alone. They also evaluated the phenomenon's dependence on genes involved in longevity regulation, particularly *pha-4*, mTORC1 components such as *daf-15* and *raga-1*, and essential autophagy genes such as *hlh-30*, *atg-1*, *bec-1*, and *lgg-1*. Gene silencing was achieved through RNA interference, a technique that reduces the expression of a target gene.Resistance to oxidative stress was studied by exposing 30 worms per experiment to tert-butyl hydroperoxide; survival was then recorded every two hours. For the β-amyloid toxicity model, transgenic worms that progressively develop paralysis were used, with approximately 30 animals per treatment. Autophagy in the worms was evaluated by counting fluorescent dots of LGG-1::GFP, a cellular marker associated with autophagic structures, in 10-15 worms per experiment.To analyze the hypothesis of a state similar to dietary restriction, the authors studied *eat-2* worms, a genetic model of reduced food intake, and a bacterial restriction model in liquid culture, with approximately 60 worms per treatment. They also performed RNA-seq, a technique that measures the expression of many genes simultaneously, using approximately 240 worms per condition. In HEK293T and HeLa cells, the phosphorylation of S6K and ULK1—proteins used as indicators of mTORC1 activity—and the markers LC3-II and SQSTM1/p62 were measured by Western blot in the presence of lysosomal inhibitors. The concentrations of cinnamaldehyde used in the cell cultures are not reported in the available textual material.Survival curves were analyzed using the Kaplan-Meier method and log-rank test; for multiple comparisons, the authors used analysis of variance. Data collection and analysis were not performed blindly, a factor that may increase the risk of experimental bias.

4. RESULTS

The initial result is that the aroma of cinnamaldehyde was not sufficient: exposure to the scent alone did not extend the lifespan of the worms at concentrations from 1 µM to 10 mM, while the scent at 40 mM reduced survival compared to the control, with $P<0.0001$. Conversely, Intake with food extended lifespan at 25 µM and 100 µM, with $P=0.002$ and $P=0.003$ respectively; 5 µM did not show a statistically significant extension, with $P=0.178$.The response was dose- and age-dependent. At 25 µM, the mean lifespan was 23.19 ± 0.64 days; at 100 µM it was 22.77 ± 0.80 days, while the maximum duration was greater at 100 µM, 34 days instead of 32. A concentration of 300 µM did not further extend lifespan. The authors did not observe relevant changes in body size, fertility, or

locomotion, but they reported an increase in lipid accumulation.For the relevant passage in the book, cinnamaldehyde increased resistance to oxidative stress induced by tert-butyl hydroperoxide, with P<0.0001. This result means that the treated worms survived longer when exposed to an oxidizing agent. However, the study did not directly measure the activity or quantity of superoxide dismutase and catalase. The increased resistance disappeared when *raga-1*, *bec-1*, or *lgg-1* were silenced, indicating that the observed benefit required components of the mTORC1 pathway and autophagy.Cinnamaldehyde also delayed age-dependent paralysis in the β-amyloid model, both at 25 µM and 100 µM, with P<0.0001. This effect was also not maintained after the silencing of genes in the mTORC1 pathway or autophagy. In worms, the silencing of *daf-15*/Raptor and *raga-1*, components associated with mTORC1, abolished the lifespan extension induced by cinnamaldehyde; silencing the mTORC2 component *rict-1* did not abolish it.The authors observed more LGG-1::GFP puncta in the treated worms, a finding compatible with increased autophagic activity. In HEK293T cells, cinnamaldehyde reduced the phosphorylation of S6K at Thr389 and ULK1 at Ser757, biochemical readings compatible with a reduction in mTORC1 activity. In the presence of lysosomal inhibitors, it also increased LC3-II and SQSTM1/p62, a set of data interpreted as an increase in autophagic flux. The numerical values of the densitometries and the relative P values are not reported in the available textual material.Finally, cinnamaldehyde did not further extend lifespan in two dietary restriction models, and its gene expression changes showed a correlation with those produced by dietary restriction. The pharyngeal pumping rate and the ingestion of fluorescent particles were not substantially different from the controls. These data support a partial similarity between the responses induced by cinnamaldehyde and dietary restriction in worms, rather than the identity of the two biological states.

5. INTERPRETATION

In simple terms, the study suggests that cinnamaldehyde may shift some of the worm's cells from a growth-oriented state to one more focused on internal maintenance. The mTORC1 pathway normally receives signals regarding nutrient availability and promotes growth processes; autophagy, on the other hand, allows for the elimination and recycling of cellular components. The publication notes that the effect of cinnamaldehyde on the survival of the worms requires genes from both of these biological networks.The segment most directly documented for the book's paragraph is: dietary cinnamaldehyde mTORC1-dependent signals and autophagy increased resistance to oxidative stress and life extension in worms, without a detectable reduction in food intake. The analogy to caloric restriction stems from the absence of an additional benefit in dietary restriction models, the partial overlap of gene expression programs, and the lack of reduction in ingested food.This interpretation has important limitations. The experiment primarily demonstrates genetic dependence and functional associations in a nematode; it does not identify a direct molecular target of cinnamaldehyde. Furthermore, the apparent involvement of mTORC1 is complex: in worms, mTORC1 components are necessary to observe the benefit, whereas in human cells, cinnamaldehyde reduces markers of mTORC1 activity. It is therefore incorrect to translate these data as linear proof that cinnamon inhibits mTORC1 in all tissues and under every condition.The sentence in the book that attributes an increase in superoxide dismutase and catalase to the publication requires clarification: the original study did not measure these enzymes. Instead, it documents an increased resistance of the worms to an oxidative insult. The general passage regarding the reduction of oxidative stress is therefore functionally consistent, but the attribution of specific enzymes to this publication is not directly demonstrated by its experimental design.

6. INDEPENDENT CONFIRMATORY EVIDENCE

Independent confirmation of the passage regarding defenses against oxidative stress comes from a study on Wistar rats exposed to the dye metanil yellow, a model of toxic kidney damage. Six animals per group received the dye for 21 days; one group also received oral cinnamaldehyde at 20 mg/kg. Compared to rats exposed to the dye alone, the co-administration of cinnamaldehyde increased renal glutathione, superoxide dismutase, and catalase, reduced lipid peroxidation, and improved markers of kidney

function. This study specifically confirms the direction indicated in the book for superoxide dismutase and catalase, but only in a rat kidney subjected to a chemical insult; it does not demonstrate the same effects in healthy people, nor does it link these enzymes to longevity [1].A second study, conducted on gastric carcinoma cell lines, observed that cinnamaldehyde increased Beclin-1, ATG5, and LC3B and reduced p62; inhibition of autophagy attenuated the cell death induced by the substance. This result confirms that cinnamaldehyde can modulate components of autophagy in mammalian cells. The identified mechanism, however, involves endoplasmic reticulum stress and tumor cell death, not lifespan or dietary restriction. It is therefore a partial mechanistic confirmation, not a replication of the result in worms [2].In a model of diabetes induced by a high-fat diet and streptozotocin, 40 male rats were divided into groups of eight and treated for four weeks. In the liver of the diabetic animals, cinnamaldehyde was associated with a reduction in mTOR expression and an increase in Beclin-1, ATG5, and LC3-II, markers consistent with increased autophagic activity; malondialdehyde, an indicator of lipid peroxidation, also decreased, and total antioxidant capacity increased. This study links, in the same model, lower mTOR, higher autophagy, and an improvement in redox markers. The design remains animal-based, pathological, and based primarily on expression markers, and therefore cannot establish an effect on human longevity [3].

7. CONFLICTING EVIDENCE

Independent evidence shows that the response to cinnamaldehyde is not uniform. In the same study on diabetic rats, cinnamaldehyde showed an opposite trend in adipose tissue: it was associated with an increase in mTOR expression and a reduction in Beclin-1, ATG5, and LC3-II. In other words, in the same animal, the modulation of autophagy differed between the liver and adipose tissue [3]. This finding limits the idea of a universal effect of the substance on mTOR and autophagy.A further study in healthy rats treated orally with cinnamaldehyde for 10, 30, or 90 days found dose- and duration-dependent effects. Renal superoxide dismutase, glutathione peroxidase, and glutathione-S-transferase increased, but at the highest dose, 73.5 mg/kg/day for 90 days, catalase and non-enzymatic antioxidants decreased, and lipid damage, measured as thiobarbituric acid reactive substances, increased. This result contradicts a simplistic interpretation that cinnamaldehyde always increases antioxidant defenses [4].These differences do not invalidate the correspondence between the original study and the passage in the book. However, they indicate that dose, duration, organ, metabolic state, and type of stress can change the direction and biological significance of the response. No directly conflicting evidence sufficiently relevant to the specific result of life extension in *C. elegans* has been identified; this does not prove the absence of contrary results.

8. QUADRO COMPARATIVO E SINTESI DELLE EVIDENZE

The table distinguishes the publication subject to the Evidence Review from independent research and indicates when the evidence concerns models other than aging and longevity.

Quadro comparativo

Reference	Study type	Population or model	Main result	Direction of evidence	Strength of evidence
Original study	Experimental, animal and cellular	C. elegans; HEK293T and HeLa cells	Dietary cinnamaldehyde prolonged lifespan and increased resistance to oxidative stress in worms; effects required mTORC1 and autophagy genes. In human cells, it reduced mTORC1 markers and increased markers consistent with autophagic flux.	direct confirmation	moderate
[1]	Experimental animal	Wistar rats with metanil yellow-induced renal toxicity; 6 animals per group	Cinnamaldehyde increased renal superoxide dismutase, catalase, and glutathione, and reduced lipid peroxidation markers compared to the toxin alone.	indirect confirmation	low
[2]	Cellular	Human gastric carcinoma cell lines	Cinnamaldehyde increased Beclin-1, ATG5, and LC3B and reduced p62; blocking autophagy attenuated the induced cell death.	biologically consistent evidence	preliminary
[3]	Experimental animal	Male rats with diabetes induced by high-fat diet and streptozotocin; 8 animals per group	In the liver, cinnamaldehyde reduced mTOR and increased autophagy markers, with lower malondialdehyde; in adipose tissue, it showed the opposite direction on autophagy markers.	indirect confirmation	low

[4]	Experimental animal	Healthy male rats treated orally for 10, 30, or 90 days	Renal antioxidant enzymes showed dose-time dependent responses; at the highest and longest dose, catalase decreased and lipid peroxidation increased.	discordant evidence	low

9. SINTESI EDITORIALE E CONCLUSIONI

CONCLUSIONS

The study analyzed directly supports the claim that dietary cinnamaldehyde can increase lifespan and resistance to oxidative stress in *C. elegans*, through mechanisms that require components of mTORC1 and autophagy. It also supports, in the same model, a state similar to dietary restriction without reducing food intake. Independent evidence confirms that cinnamaldehyde can influence markers of autophagy and redox defenses, but these effects are dependent on the model, organ, dose, and duration of exposure. The book's narrative is therefore supported at a mechanistic and preclinical level, while it requires validation in humans for any conclusions regarding longevity, clinical resilience, or nutritional use.

Aggiornato al 17/11/2026. Questo contenuto riflette una sintesi divulgativa delle evidenze scientifiche al momento della pubblicazione e non sostituisce il parere medico.

10. TRANSPARENCY AND SCIENTIFIC RESPONSIBILITY

Artificial Intelligence was used solely as a tool for editorial and documentary support. All content, interpretations, bibliographic citations and conclusions have been verified, reviewed and approved by the author, who assumes full scientific responsibility for them.

The manuscript was also subjected to independent peer review prior to publication.

To ensure the greatest transparency, the complete history of the article, including the dates of submission, revision, acceptance and publication, as well as information on the peer review process, is available on the dedicated Article History page.

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