RESEARCH PAPER

Ginger Extract Modestly Extends Lifespan in Wild-Type Caenorhabditis elegans but Not in daf-2 Mutants

Ali H. Alghamdi[1]

[1]Department of Biology, Faculty of Science, Al-Baha University, Al-Baha, Saudi Arabia, 65779 | Email: aayfan@bu.edu.sa

ABSTRACT

Natural plant-derived compounds are widely investigated for their potential effects on aging and stress resistance using Caenorhabditis elegans as a simple whole-organism model. Ginger (Zingiber officinale) contains several bioactive compounds, including gingerols and shogaols, that have been associated with antioxidant and stress-modulating activities. In this study, we examined the effects of ginger extract on lifespan in wild-type N2 C. elegans and in daf-2 mutant worms using nematode growth medium plates with FUdR to prevent progeny production. Ginger extract was tested at 25, 50, and 100 µg/mL. In wild-type N2 worms, ginger extract increased mean lifespan from 21.03 days in the control group to 23.01 days at 25 µg/mL and 22.98 days at 100 µg/mL, representing approximate increases of 9.4% and 9.3%, respectively. The 50 µg/mL treatment increased mean lifespan to 22.17 days, but this effect was not marked as statistically significant. In contrast, ginger extract did not further extend lifespan in daf-2 mutants at any tested concentration. Mean lifespan in the daf-2 control group was 30.20 days, compared with 29.47, 29.64, and 30.75 days at 25, 50, and 100 µg/mL, respectively. These findings suggest that ginger extract can extend lifespan in wild-type worms, but this effect is lost in the daf-2 mutant background. The results are consistent with previous evidence that ginger extract may act through, or converge on, the insulin/IGF-1-like signaling pathway involving DAF-2-regulated longevity mechanisms. However, additional confirmation using downstream mutants such as daf-16 and skn-1 would strengthen the mechanistic interpretation.

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Introduction

Aging is a complex biological process influenced by genetic, metabolic, dietary, and environmental factors (1,2). The nematode Caenorhabditis elegans is widely used as an experimental model for studying aging (3,4) because it has a short lifespan, a well-characterized genome, conserved stress-response pathways, and simple methods for measuring survival under controlled laboratory conditions (5,6). Although findings from *C. elegans* cannot be directly translated to humans, the model is useful for screening dietary compounds and identifying conserved molecular pathways that influence longevity and stress resistance (3,7–9) Plant-derived bioactive compounds have received increasing attention in aging research because many of them can affect oxidative stress, metabolism, proteostasis, and stress-response signaling (10–12). Previous work using *C. elegans* has shown that dietary compounds can influence lifespan and health-related outcomes, but these effects often depend on dose, developmental timing, diet composition, and genetic background (3). For example, Many compounds rich in polyphenols and antioxidants have been shown to positively affect lifespan and aging-related outcomes in *C. elegans*, supporting the use of this model for screening dietary and plant-derived bioactive compounds (8,9,13–16). One such compound, ginger, has long been a commonly consumed spice and medicinal plant that contains several bioactive compounds, especially gingerols and shogaols. These compounds have been linked to antioxidant and stress-modulating effects in different biological systems (17–19). In *C. elegans*, ginger extract has been reported to extend lifespan, improve movement, reduce lipofuscin accumulation, enhance stress tolerance, and activate stress-related transcriptional regulators including DAF-16 and SKN-1(20,21). One study reported that 60 µg/mL ginger extract extended *C. elegans* lifespan by 23.16% and suggested that the effect was mainly associated with the insulin/IGF-1 signaling pathway (20). Studies on isolated ginger compounds have also shown lifespan- and stress-related effects. For example, 6-gingerol increased stress resistance and was associated with increased SOD-3 and HSP-16.2 expression, while 6-shogaol showed dose-dependent lifespan extension and increased stress-tolerance markers in *C. elegans* (22).

The insulin/IGF-1-like signaling pathway is one of the most important longevity pathways in *C. elegans*. In this pathway, *DAF-2* functions as an insulin/IGF-1 receptor-like protein that regulates downstream signaling and influences the activity of transcription factors such as DAF-16/FOXO. Reduced *DAF-2* signaling is associated with lifespan extension, largely through increased activity of protective stress-response and longevity programs (23,24). Therefore, testing lifespan effects in *daf-2* mutants can help determine whether a

compound acts through the same pathway or whether it may extend lifespan through a DAF-2-independent mechanism.

In the present study, we examined the effects of ginger extract at 25, 50, and 100 µg/mL on lifespan in wild-type N2 *C. elegans* and *daf-2* mutant worms. The purpose was not only to determine whether ginger extract extends lifespan under the present experimental conditions, but also to assess whether the effect is maintained or abolished in the *daf-2* mutant background. Based on previous reports linking ginger extract to DAF-16/SKN-1 activation and insulin/IGF-1-like signaling, we expected that ginger extract would extend lifespan in wild-type worms but show reduced or absent effects in *daf-2* mutants if the mechanism overlaps with DAF-2-regulated longevity signaling.

Methodology

Preparation of Ginger Extract

Ginger extract was prepared from Zingiber officinale and used to evaluate its effect on lifespan in Caenorhabditis elegans. The extract was dissolved in dimethyl sulfoxide (DMSO) or an appropriate solvent to prepare a stock solution. Working concentrations were freshly prepared by diluting the stock solution into the treatment medium to obtain final concentrations of 25, 50, and 100 µg/ml. The same final concentration of solvent was used in the control group to ensure that any observed effects were due to ginger extract and not to the solvent.

C. elegans Strains and Maintenance

Wild-type N2 *C. elegans* and *daf-2* mutant worms were used in this study. Worms were maintained on nematode growth medium (NGM) plates seeded with Escherichia coli OP50 as the food source. Cultures were maintained under standard laboratory conditions at 20°C. Worms were transferred regularly to fresh OP50-seeded NGM plates to maintain healthy synchronized populations.

Lifespan Assay

Lifespan assays were conducted on NGM plates seeded with OP50. Ginger extract was added to the plates to achieve final concentrations of 25, 50, and 100 µg/ml. Control plates received the same amount without ginger extract. To prevent progeny production during the lifespan assay, FUdR was added to the NGM plates. For each treatment group, synchronized worms were transferred to treatment plates at L4 stage. Worms were monitored regularly throughout the experiment. Animals were scored as dead when they failed to respond to gentle touch with a platinum wire or worm pick. Worms that crawled off the plate, ruptured, bagged, or died due to handling-related damage were censored and excluded from death-event analysis according to standard lifespan assay criteria.

The lifespan assay included the following treatment groups:

Control group
Ginger extract 25 µg/ml
Ginger extract 50 µg/ml
Ginger extract 100 µg/ml

These groups were tested in both wild-type N2 worms and *daf-2* mutant worms.

Statistical Analysis

Lifespan data were analyzed using JASP. Survival analysis was performed using the Kaplan–Meier method to compare survival patterns between the control and ginger extract-treated groups. Mean lifespan values and descriptive statistics were also calculated for each treatment group. Differences among treatment groups were analyzed using analysis of variance (ANOVA), followed by appropriate post hoc comparisons when required. Graphs were prepared using GraphPad Prism. Statistical significance was set at $p < 0.05$, and significant differences compared with the control group were indicated in the lifespan summary tables.**Results**

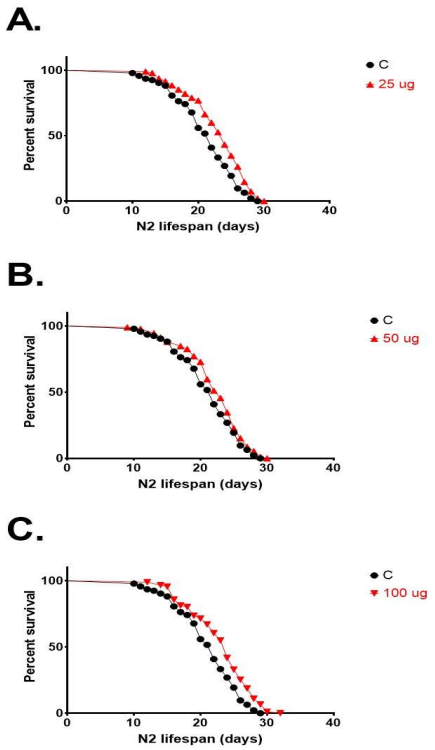

Figure 1: Kaplan–Meier survival curves of wild-type N2 *C. elegans* treated with ginger extract. Panel A shows the effect of ginger extract at 25 µg/ml, Panel B shows the effect at 50 µg/ml, and Panel C shows the effect at 100 µg/ml, compared with the control. Survival differences were assessed using Kaplan–Meier survival analysis.

In this experiment, we first examined the effect of ginger extract on lifespan in wild-type N2 *C. elegans*. Compared with the control, ginger extract treatment produced a visible shift in the survival curves at 25 µg/ml and 100 µg/ml, while the effect

at 50 µg/ml was less pronounced (Figure 1). In Figure 1A, worms treated with 25 µg/m ginger extract showed improved survival compared with the control group, and a similar pattern was observed at 100 µg/ml in Figure 1C. In contrast, Figure 1B showed greater overlap between the 50 µg/ml treatment and the control group, suggesting a weaker response at this concentration.

Treatments	Mode	Median	Mean	SEM	Confidence %95 Interval Mean Lower	Upper
C	21	22	21.03	0.482	20.07	21.99
25 ug	25.7	24	23.01*	0.456	22.11	23.92
50 ug	24.5	23	22.17	0.467	21.25	23.1
100 ug	24.65	24	22.98*	0.494	21.99	23.96

Descriptive Summary of Lifespan Data (*) indicates significant differences compared to the DMSO control

Table 1: Descriptive summary of lifespan data in wild-type N2 *C. elegans* treated with ginger extract. The descriptive lifespan data in Table 1 supported these observations. The mean lifespan of the control group was 21.03 days, whereas treatment with 25 µg/ml increased mean lifespan to 23.01 days, corresponding to an approximate 9.4% increase. Treatment with 100 µg/ml increased mean lifespan to 22.98 days, corresponding to an approximate 9.3% increase. Both 25 and 100 µg/ml were marked as significantly different from the DMSO control. The 50 µg/mL treatment increased mean lifespan to 22.17 days, equivalent to an approximate 5.4% increase, but this difference was not marked as statistically significant. Together, these results show that ginger extract produced a modest lifespan extension in wild-type N2 worms, although the response was not clearly dose-dependent.

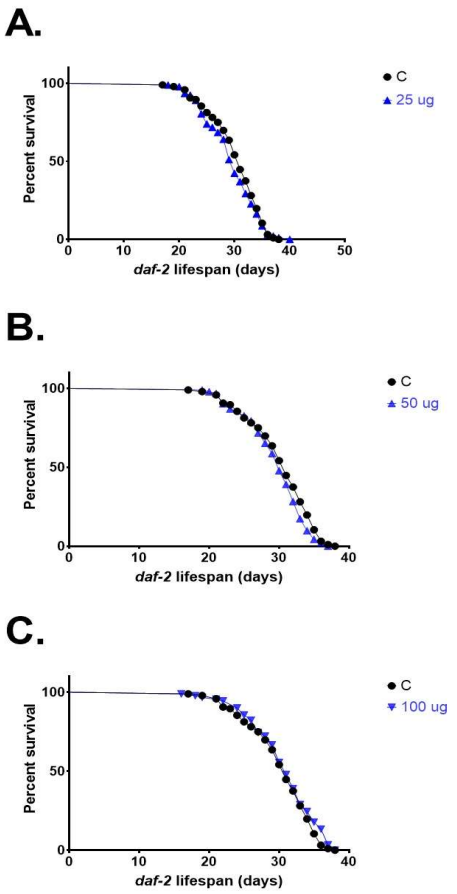

Figure 2: Kaplan–Meier survival curves of *daf-2* mutant *C. elegans* treated with ginger extract. Panel A shows the effect of ginger extract at 25 µg/ml, Panel B shows the effect at 50 µg/ml, and Panel C shows the effect at 100 µg/ml, compared with the control. Survival differences were assessed using Kaplan–Meier survival analysis.

We next examined whether the effect of ginger extract on lifespan was maintained in the *daf-2* mutant background. Unlike the pattern observed in wild-type N2 worms, ginger extract did not produce a clear rightward shift in the survival curves of *daf-2* mutants (Figure 2). In Figure 2A, the survival curve for 25 µg/ml was close to the control curve and did not show improved survival. Similarly, Figure 2B showed substantial overlap between the 50 µg/ml treatment and the control group, while Figure 2C showed that the 100 µg/ml treatment followed a survival pattern similar to the control group, with only minor differences across the lifespan curve.

Treatments	Mode	Median	Mean	SEM	Confidence Interval %95 Mean Lower	Upper
C	32.69	31	30.2	0.478	29.25	31.15
25 ug	30.16	30	29.47	0.487	28.5	30.44
50 ug	31.68	30	29.64	0.438	28.77	30.51
100 ug	31.45	31	30.75	0.498	29.76	31.74

Descriptive Summary of Lifespan Data (*) indicates significant differences compared to the DMSO control

Table 2: Descriptive summary of lifespan data in *daf-2* mutant *C. elegans* treated with ginger extract.

This pattern was consistent with the descriptive data in Table 2. The mean lifespan of the *daf-2* control group was 30.20 days, compared with 29.47 days at 25 µg/ml, 29.64 days at 50 µg/ml, and 30.75 days at 100 µg/ml. None of the ginger extract treatments were marked as significantly different from the DMSO control. These results indicate that, under the present experimental conditions, ginger extract did not further extend lifespan in the *daf-2* mutant background.

Overall, ginger extract showed a modest lifespan-extending effect in wild-type N2 worms at selected concentrations, particularly 25 µg/ml and 100 µg/ml, but this effect was not observed in *daf-2* mutant worms. The wild-type data showed significant increases in mean lifespan at two concentrations, whereas the *daf-2* data showed no significant improvement at any tested concentration. This difference between the two genetic backgrounds suggests that the lifespan response to ginger extract was present in wild-type worms but absent in the *daf-2* mutant background.

Discussion

In the present study, we evaluated the effect of ginger extract on the lifespan of wild-type Bristol N2 and *daf-2* mutant *C. elegans*. Treatment of N2 worms with ginger extract at 25 and 100 µg/ml increased mean lifespan by 9.4% and 9.3%, respectively, relative to the DMSO-treated control, whereas the intermediate concentration of 50 µg/ml produced a smaller and non-significant increase. In *daf-2* mutants, none of the three tested concentrations significantly altered mean lifespan, and the survival curves of treated and control animals largely overlapped. The lifespan-extending effect of ginger extract was therefore present in the wild-type background but absent in the *daf-2* mutant background, a pattern that is consistent with a requirement for an intact insulin/IGF-1 signaling (IIS) pathway (20).

The modest lifespan extension observed in wild-type N2 worms fits within the broader biological profile of ginger as a source of bioactive phenolic compounds. Gingerols and shogaols, the major pungent constituents of ginger, have been widely associated with antioxidant, anti-inflammatory, and metabolic effects, all of which are relevant to aging biology because oxidative stress, chronic inflammation, and metabolic dysregulation contribute to age-associated functional decline (25–28). These effects are biologically important because aging is not driven by a single mechanism, but by the gradual accumulation of cellular and physiological changes that affect stress resistance, mitochondrial function, inflammatory balance, and metabolic homeostasis (29,30). Ginger-derived compounds have been reported to influence several of these processes, including the regulation of oxidative-stress responses, reduction of inflammatory signaling, and improvement of metabolic parameters in different experimental and clinical contexts (31,32). In human studies, ginger supplementation has been investigated mainly for inflammatory, oxidative-stress, gastrointestinal, and metabolic outcomes rather than for direct lifespan extension, with some studies suggesting beneficial effects on inflammatory and oxidative-stress biomarkers (26,33–35). In experimental aging models, ginger and its major bioactives have also been associated with improved stress resistance and healthspan-related phenotypes, including lifespan extension, reduced lipofuscin accumulation, and improved movement in *C. elegans* (20,21,27). Therefore, the present findings are consistent with the broader view that ginger-derived compounds can influence biological processes linked to aging, although direct translation to mammalian lifespan or human healthspan should remain cautious.

The magnitude of the effect observed in N2 worms is smaller than the approximately 23% extension previously reported for whole ginger extract at 60 µg/ml, where the response was attributed to gingerols and shogaols and shown to depend on DAF-16 and SKN-1 (20). Differences of this kind are commonly observed across studies of plant-derived interventions in *C. elegans* and are likely to reflect variation in extract composition, solvent, OP50 batch, and assay conditions, all of which are known to influence the absolute magnitude of lifespan effects (36,37). The qualitative direction of our wild-type result is nevertheless in agreement with prior reports describing positive effects of ginger and its principal bioactives on lifespan and stress resistance in this organism (20,21). The lifespan response in N2 worms did not show a clear dose-dependent pattern. Although 25 and 100 µg/ml significantly increased lifespan, the intermediate concentration of 50 µg/ml produced only a smaller, non-significant effect. This result should therefore be interpreted with caution. The pattern may be related to the complex chemical composition of ginger extract, but it may also reflect normal variability between lifespan assay replicates. For

this reason, the present data are not sufficient to support a hormetic mechanism, and additional concentrations and biological replicates would be needed to test this possibility.

The most informative observation of the present study is the absence of detectable lifespan extension in *daf-2* mutants. Because *daf-2* mutants are already long-lived due to reduced insulin/IGF-1-like signaling and increased activation of downstream longevity programs (38,39), the failure of ginger extract to further extend lifespan in this background is consistent with involvement of the IIS pathway (20). Previous evidence shows that ginger extract extends lifespan in *C. elegans* through IIS-associated mechanisms involving DAF-16 and SKN-1 (20). Thus, the present findings do not establish a new mechanism, but they support and extend the existing model by showing that ginger extract does not further increase lifespan when DAF-2 signaling is already disrupted.

The absence of an additional lifespan effect in *daf-2* mutants does not demonstrate that ginger extract acts directly on DAF-2; rather, it indicates that the response is not additive when DAF-2 signaling is already reduced. Therefore, the present data should be interpreted as supportive of IIS involvement, in agreement with previous evidence implicating DAF-16 and SKN-1(20), rather than as proof of the precise molecular target of ginger extract. Future work using additional pathway mutants, transcription-factor localization assays, and stress-response reporters would help clarify where ginger extract acts within, or alongside, the IIS network. In addition, chemical standardization of the extract, for example by HPLC quantification of gingerols and shogaols, would improve comparison of dose–response effects across studies.

Beyond questions of pathway architecture, the interpretation of ginger's lifespan effect should also consider experimental context. The present study tested ginger extract using a single L4-onset exposure window on a standard OP50 diet. Because lifespan responses in *C. elegans* can be influenced by diet, timing of exposure, bacterial food conditions, and genotype, it remains possible that the magnitude or pattern of the response would differ under earlier exposure, altered glucose availability, or modified feeding conditions. Future studies using different exposure windows and dietary backgrounds would help define how robust the ginger effect is across experimental contexts.

Finally, although the IIS pathway is broadly conserved across metazoans, the present findings should not be overextended beyond the *C. elegans* model. The absence of additional lifespan extension in *daf-2* mutants supports the involvement of insulin/IGF-1-like signaling in the worm response, in agreement with previous evidence implicating DAF-16 and SKN-1 in ginger-induced longevity(20). However, the present data do not directly address mammalian lifespan, healthspan, or metabolic outcomes. Existing human studies on ginger have focused mainly on metabolic, gastrointestinal, inflammatory, and body-weight-related endpoints rather than lifespan or aging biomarkers (26,33,40,41) . Translational claims should therefore remain cautious and should be tested through dedicated mammalian, cellular, or clinically relevant models rather than inferred from worm lifespan data alone.

Conclusion

In conclusion, ginger extract modestly but significantly extended lifespan in wild-type N2 *C. elegans*, whereas no extension was observed in *daf-2* mutants. These findings support the involvement of DAF-2-mediated insulin/IGF-1 signaling and complement previous evidence implicating DAF-16 and SKN-1 in ginger-induced longevity. Although the response was moderate and not clearly dose-dependent, the study adds further evidence that ginger-derived compounds can modulate aging-related pathways in *C. elegans*. Future work using standardized extracts and additional mechanistic readouts will help define the robustness and translational relevance of this effect.

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