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Article

The effects of a high-sucrose diet on the survival of *Drosophila melanogaster* from a bacterial infection

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SUMMARY

Excess sucrose intake has been linked to a variety of health conditions. Sucrose and other dietary sugars have inflammatory effects that can negatively impact immune health. However, sucrose's influence on immune function is not fully understood, and its effects during bacterial infection may vary depending on factors such as infection method. Previous studies have used the model organism *Drosophila melanogaster* to study the effects of sucrose during bacterial infection because this species is genetically similar to humans and has a similar innate immune response. Additionally, this species is relatively simple and easy to manipulate. This study investigated the influence of sucrose on the survival of *D. melanogaster* after an oral bacterial infection with the pathogen *Serratia marcescens*. We hypothesized that a diet composed of 20% added sucrose would decrease the survival of *D. melanogaster* after *S. marcescens* infection compared to a 0% added sucrose control diet. To test this hypothesis, we orally inoculated two groups of *D. melanogaster* flies with *S. marcescens* and fed each group diets with different sucrose concentrations. Contrary to our expectations, flies given the 20% added sucrose diet had significantly longer survival times than those given the 0% added sucrose diet. It is possible that high sucrose intake negatively impacts immunity, but our results suggest that sucrose can positively impact immunity under certain conditions. More research is needed to fully elucidate which contexts make excess sucrose harmful.

INTRODUCTION

Excess dietary sugars, including sucrose, have been linked to inflammation and a wide range of diseases, including obesity, cardiovascular disease, multiple sclerosis, psoriasis, arthritis, and inflammatory bowel disease (1). Sucrose, also known as table sugar, is broken down by the digestive system into its constituent glucose and fructose hexoses (1). The impact of these dietary sugars on immunity is generally thought to be negative when consumed in excess, although they can confer important benefits when consumed in appropriate amounts (1,2). For example, glucose from digested sucrose or other sources is often stored as glycogen, which functions as a critical source of energy during immune responses (2). The immune system requires a significant amount of energy during infection, and the body will redistribute resources to support immune function (3). However, excess glucose can promote excess cytokine production, leading to unhealthy levels of inflammation (4). Excess dietary sugars can also be problematic in their ability to elevate levels of high-sensitivity C-reactive protein (hs-CRP), an inflammatory marker (5). One study showed that three different beverages containing 50 grams of either fructose, glucose, or sucrose all increased the level of hs-CRP in healthy study participants (5). Sucrose and fructose raised hs-CRP levels significantly more than glucose (5). Thus, sucrose is a relevant molecule to study when investigating how to best support immune function.

Several studies have used *D. melanogaster* to study the effects of sucrose on the immune response due to the ease of measuring its physiological parameters such as inflammation, food intake, microbiota, and life span (6). Further, approximately 60% of *D. melanogaster* genes show similarities to those of mammals, indicating extensive genetic similarities between fruit flies and humans (6). The *D. melanogaster* immune system is relatively simple, and its innate immune responses are functionally similar to those of humans (7). Thus, studying sucrose interactions with *D. melanogaster* health can provide insights into the effects of sucrose on the human body (6,8).

Sucrose in moderation may be beneficial to *D. melanogaster* during infection, although the optimum concentration seems to vary based on variables such as the specific species of pathogen (7). To begin, sucrose may be beneficial as support to the microbiome. Beneficial gut bacteria such as *Acetobacter tropicalis* metabolize sugars consumed by their host (9). These gut microbes then assist their host in recovering from bacterial infections (10). For example, they release metabolites that are critical for maintaining gut integrity and preventing systemic infection (10). Further, gut bacteria assist in activating the host immune response once pathogens arrive (10). Therefore, a deficiency of dietary sugars such as sucrose may be detrimental to beneficial gut bacteria, leading to negative outcomes for *D. melanogaster* during infection.

In contrast, excessive sucrose intake can be detrimental to *D. melanogaster* during bacterial infection. Flies on high-sugar diets often reduce their overall food intake, leading to nutrient deficiencies (9). These deficits can compromise the immune system (11). Also, diets excessively high in sucrose have been linked to a decrease in translation of antimicrobial peptide (AMP) genes in infected *D. melanogaster*, further impairing the immune response (7). Additionally, there is evidence suggesting that insulin/insulin-like signaling (IIS) pathways may be involved in some of the detrimental effects of sucrose on infection outcomes (2). The IIS pathway regulates growth, metabolism, and energy utilization during infection, and research shows that suppression of insulin signaling in

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D. melanogaster fed high-sucrose diets may encourage the production of immune effectors such as AMPs, supporting *D. melanogaster* survival (2). Finally, high-sugar diets can induce hyperglycemia in flies, providing an abundant energy source for pathogens such as *S. marcescens* during systemic infection (7). *S. marcescens* is a bacterium that opportunistically infects humans and can cause lethal infections in *D. melanogaster* (7,8). One issue that remains unclear is how the effects of sucrose differ depending on the specific pathogen involved, and more research on the interactions between pathogen physiology, sucrose concentration, and host physiology are needed to better understand this topic (7).

Our study utilized the model organism *Drosophila melanogaster* and the pathogen *Serratia marcescens* to investigate how sucrose impacts the immune response during bacterial infection. Previous research demonstrated that flies fed a 0% added sucrose diet had significantly better survival after *S. marcescens* injection than those fed high added sucrose diets (16% or 24%, w/v) (7). Based on this study, we hypothesized that a 20% added sucrose diet would decrease the survival of *D. melanogaster* during *S. marcescens* infection when compared to a 0% added sucrose diet. We prepared low-sucrose (0%) and high-sucrose (20%) diets for two separate groups of flies inoculated with *S. marcescens*. We monitored each group to track the number of dead flies each day, using survival time as a rough indication of the efficacy of the immune responses generated on each diet. We found that flies fed high-sucrose diets survived significantly longer post-inoculation than flies fed low-sucrose diets, suggesting that sucrose may confer benefits to immunity in particular contexts. Overall, these findings show that the interactions between sucrose and immune function are complex and warrant further research.

RESULTS

To investigate the impact of a high-sucrose diet on *D. melanogaster* survival after a bacterial infection, we orally inoculated flies with the bacteria *S. marcescens* and monitored them daily as they fed on either a high-sucrose (20%) or low-sucrose (0%) diet. We recorded the number of fly deaths daily for six days.

To determine bacterial load post-inoculation, we performed a verification of infection procedure on one inoculated fly and one uninoculated fly. The inoculated fly yielded 21,000 total bacterial cells and the uninoculated fly yielded 8,700 total bacterial cells (Equation 1). Assuming the 8,700 cells detected in the uninoculated fly were nonpathogenic gut bacteria, we subtracted this value from 21,000 to approximate that 12,300 of the cells in the inoculated fly were *S. marcescens* cells **(Figure 1)**. Thus, we verified that the flies had received a high enough dose of bacteria to cause infection as determined in prior studies (7).

To determine survival after infection, we generated Kaplan-Meier curves. After six days, 30 out of 35 flies (85.71%) had died in the low-sucrose control group, while 15 out of 34 flies (44.12%) had died in the high-sucrose experimental group (**Table 1**, **Figure 2**). The experimental group consuming the 20% added sucrose diet had significantly longer survival times compared to the control group consuming the 0% added sucrose diet, according to the log-rank test (χ^2 = 13.72, df = 1, $p < 0.001$, $\alpha = 0.05$, **Figure 2**). The restricted mean survival time (RMST), which represents the average time to death, for the low-sucrose group was 78.57 hours, and the RMST for the high-sucrose group was 107.60 hours post-infection. Overall, these data demonstrate that the low-sucrose diet was associated with decreased survival times in *D. melanogaster* infected with *S. marcescens*.

DISCUSSION

Our study's objective was to explore how sucrose impacts the survival *of D. melanogaster* after a bacterial infection with the pathogen *S. marcescens*. From the log-rank test and the RMST values from each group, we found that providing flies with a 20% added sucrose diet improved survival outcomes when compared to flies with a 0% added sucrose diet. This suggests that under certain conditions, sucrose may be beneficial to the survival of infected flies. These unexpected findings may be the result of several factors that varied between our experimental design and that of a previous study that influenced our design (7). One such condition that may explain the conflicting results is the route of infection. In a previous study, bacteria were injected directly into the body of *D. melanogaster*, allowing pathogens immediate access to the entire fly and creating systemic infection (7). The route of *S. marcescens* infection greatly influences survival outcomes; an *S. marcescens* infection introduced directly into the body cavity can kill a fly in one day, while an infection introduced orally can create an enteric infection that may proliferate into the body cavity, taking six days to kill a fly (8).

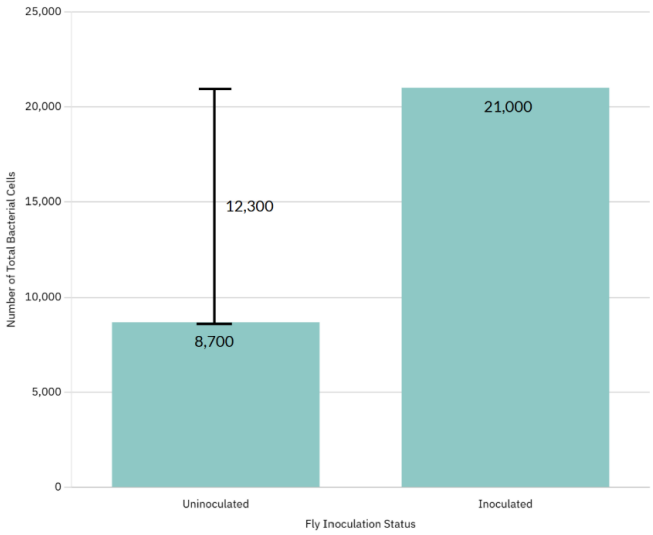

Figure 1: Bar graph displaying the total number of bacterial cells per fly estimated from the verification of infection procedure. The heights of the bars represent the number of bacterial cells estimated from one uninoculated fly and one inoculated fly after performing the verification of *S. marcescens* infection procedure. The inoculated fly was expected to contain both pathogenic *S. marcescens* cells and nonpathogenic bacterial cells, while the uninoculated fly was expected to only contain nonpathogenic bacteria. The uninoculated fly contained approximately 8700 total cells, while the inoculated fly contained approximately 21,000 total cells. By subtracting the total number of cells found in the uninoculated fly from the total number of cells found in the inoculated fly, we estimated that the inoculated fly contained 12,300 *S. marcescens* cells.

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Hours After Transfer to Maintenance Vials Post-Inoculation	Number of Dead Flies	
	Control Group (N=35) (0% added sucrose diet)	Experimental Group (N=34) (20% added sucrose diet)
0	0	0
20.167	3	4
47	17	6
68.25	19	9
93.75	25	9
121.167	28	12
138.5	30	15

Table 1: Death counts of *Drosophila melanogaster* over time after inoculation with *Serratia marcescens*. Table showing the number of dead *D. melanogaster* flies at each observation time point in both study groups. Flies were orally inoculated with a suspension of *S. marcescens* bacteria. The control group (N=35) was fed a 0% added sucrose diet, and the experimental group (N=34) was fed a 20% added sucrose diet. Counts were completed daily via thorough inspection of the maintenance vials.

Flies with systemic infections may be especially harmed by a high-sucrose diet because hyperglycemia is dangerous in such cases: the excess carbon circulating throughout the body can be used as an additional food source for pathogens (7). In contrast, our flies ingested the bacteria, resulting in enteric rather than systemic infections. For this reason, hyperglycemia may have been less detrimental to the flies, and the additional sucrose availability could have supported fly immune functions rather than feeding the infection. In the context of human infections caused by *S. marcescens* specifically, both enteric and systemic infection modeling with *Drosophila* is applicable because *S. marcescens* is known to cause a range of nosocomial infections, including enteric infections such as gastroenteritis as well as systemic infections such as sepsis (12,13).

Another factor that may have played a role in our results is the Target of Rapamycin (TOR) pathway, a nutrient-sensing pathway involved in growth and immunity (2). TOR activity increases when nutrients are abundant and is particularly important during enteric infections, when maintaining lipid stores via TOR activation can be critical for survival (14). In our study, access to sucrose may have supported TOR activity and, thus, survival. However, in the case of systemic infections such as those in the previous study, the role of TOR is poorly understood, as TOR inhibition during systemic infections in *D. melanogaster* after inoculation with different bacterial species has produced conflicting results (7,14). More research into the role of the TOR pathway during systemic and enteric infections caused by various pathogens with different physiologies is necessary.

Giving the flies time to adapt to a new diet prior to infection may also account for the variation between our findings and those of other studies (7). In a previous study, flies were reared on a 4% added sucrose diet and then switched to experimental diets with various sucrose concentrations of 0%, 2%, 4%, 8%, 16%, or 24% (7). Flies were allowed to feed on their experimental diets for three to five days before inoculation (7). In our study, the flies were first inoculated and then given a new diet out of necessity for a shorter experimental timeframe. Research shows that nutrient availability or lack thereof can induce metabolic remodeling (14). Increased TOR activity in response to increased nutrient availability and decreased TOR activity in response to decreased nutrient availability is an example of this remodeling (14). Thus, it is possible that the added stress of a sudden change in diet could have placed our low-sucrose control group of flies at a disadvantage as compared to the previous study's flies on low-sucrose diets.

Because *D. melanogaster* and humans have comparable genetics, inflammatory processes, and microbiota, sucrose might also positively influence human immune responses under certain conditions (6). However, it is important not to over-generalize these results, as results found in one organism do not always directly apply to another. Also, the experiment was limited to a single bacterial species, a single infection method, and one trial. A significant amount of evidence still indicates that sucrose has negative effects on immunity, so our results should not be misinterpreted as indicating that sucrose is universally beneficial during infection.

It is important to note that our study had limitations that warrant further replication. One major limitation includes the lack of two additional control groups, one given a 0% added sucrose diet and one given a 20% added sucrose diet, both without *S. marcescens* exposure, to observe if the experimental diets themselves impacted survival independently of infection. Additionally, there were inaccuracies in death counts because some flies appeared to be dead when they were caught in their food, and some dead flies were discovered later inside folds of the filter paper in the vials. Other limitations include variability in timing of death counts between days, an imprecise inoculation method that could result in differing bacterial loads between flies, and uncertainty regarding the ages of the flies. Future studies are needed to replicate these data and increase the statistical power of the results.

There are several ways to extend this research in the future. First, it would be interesting to test if sucrose significantly affects metabolic rates during bacterial infection. Future studies could look at changes in oxygen consumption rates of the flies over time. In addition to studying metabolic rates during infection, it could be helpful to test the impact of sucrose during enteric versus systemic infections. Based on our results combined with previous studies, we would expect that the infection route does affect metabolic rates differently. Next, it would be beneficial to use more sophisticated methods of verifying bacterial load and identity as well as assays of biomarkers associated with *Drosophila* immune responses. These would provide stronger verifications of infection. Further, a future experiment could test a greater number of sucrose concentrations. It is possible that if we had utilized a diet with a low but non-zero percentage of added sucrose as our control rather than the 0% added sucrose diet, the stress of adjusting to the new diet would be reduced for the flies, and survival may have improved and even exceeded that of the experimental group on a high-sucrose diet. Finally, future research could apply the findings gathered from *Drosophila*

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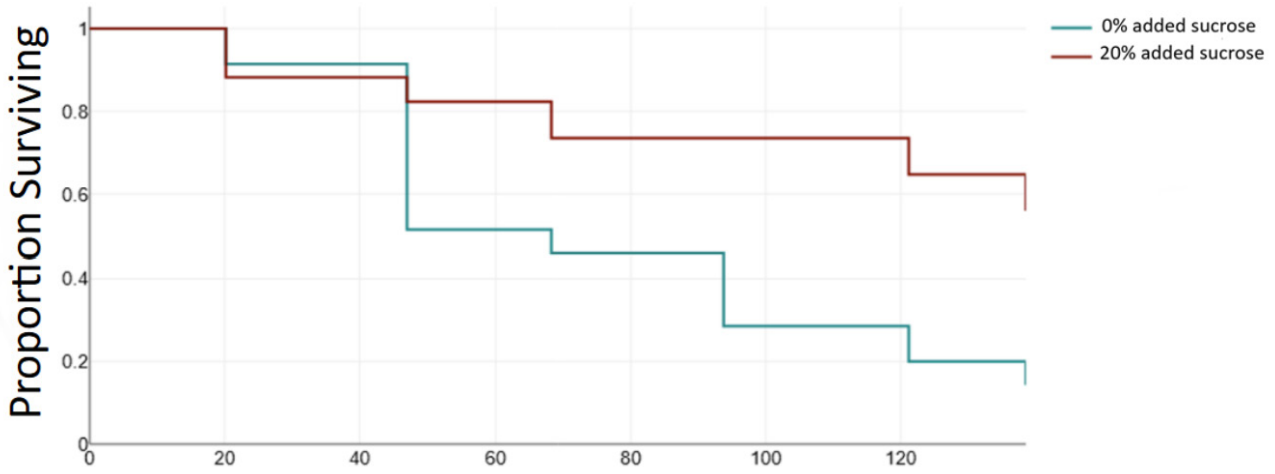

Figure 2: Kaplan-Meier curves for survival time analysis of *D. melanogaster* after inoculation with *S. marcescens*. Kaplan-Meier survival curves displaying the proportion of surviving *D. melanogaster* flies over an observation period of 138.5 hours after inoculation with *S. marcescens* bacteria. N=35 for flies fed 0% sucrose and N=34 for flies fed 20% sucrose. The number of deaths in each group per day were recorded and then plotted using Kaplan-Meier curves. The two curves were found to be significantly different by the log-rank test ($\chi^2 = 13.72$, df = 1, $p < 0.001$, $\alpha = 0.05$).

studies to humans, testing whether the impacts of sucrose observed in fruit flies apply to human biology. Using this research, we can expand the field of immunology and learn key insights that will benefit the ways in which we approach sucrose intake and healing from infections.

MATERIALS AND METHODS

Preparation of *Drosophila* food

A low-sucrose control batch and a high-sucrose experimental batch of fly food were prepared. The control food consisted of 12 g of cornmeal, 4 g of yeast, 2 g of agar, 2 g of methyl paraben (Josh's Frogs), and 100 mL of water. No added sucrose was present. 20 mL of this food was given to the control group of flies. The experimental food consisted of 12 g of cornmeal, 4 g of yeast, 2 g of agar, 30 g of added sucrose, 2 g of methyl paraben, and 100 mL of water. This food had a 20% (w/w) added sucrose concentration. 20 mL of this food was given to the experimental group of flies.

For both types of food, all dry ingredients were mixed with half of the total water content for the batch. This mixture was boiled until it thickened. Once it reached the boiling point, the heat was removed and the second half of the total water content was added. The mixture was then boiled again, removed from the heat, covered with aluminum foil, and stored in the refrigerator to gel. This food preparation method was adapted from procedures described in multiple online sources (15,16). Prior to the experiment, the two batches of food were reheated to liquid form and poured into separate BioChamber 250s (Vernier, Cat# BC-250). The BioChamber 250s were stored in the refrigerator so that the food could gel again before the flies were introduced.

Preparation of bacterial suspension

A stock culture of *Serratia marcescens* bacteria was grown out from an agar slant tube (Carolina, Cat# 155450). Our strain of *S. marcescens* was comparable to the *S. marcescens* subspecies *marcescens* strain BS 303 [CDC 813-60, NCIB 9155, NCTC 10211]. Using aseptic technique, bacteria were streaked from the slant tube onto LB agar plates (EZ BioResearch, Cat# KAP-010) using a flame-sterilized inoculation loop. The plates were placed in an incubator (21-26°C) (Carolina, Cat# 701298) in an inverted position until new bacterial growth was observed. Growth could usually be seen within 24 hours and was evident when dark pink colonies were visible. Every one to three weeks, bacteria were streaked from previously grown plates to new plates to ensure the health of the culture (17). Bacterial suspensions used in the experiment were prepared by swabbing colonies from an agar plate with a sterile cotton swab and suspending them in approximately 3 mL of a sterile 0.85% saline solution in a sterile spectrophotometer cuvette. The suspension was thoroughly homogenized by hand via agitation.

Calculation of bacterial cell counts

A Go Direct® SpectroVis® Plus Spectrophotometer (Vernier, Cat# GDX-SVISPL) was calibrated at a wavelength of 600 nanometers (OD600) using a blank solution of sterile 0.85% saline solution. The bacterial suspension's OD value was then measured to be approximately 1.0. To measure the number of bacterial cells per mL of the suspension, a series of dilutions ranging from 10^{-2} to 10^{-10} were prepared from 1 mL of the suspension by serial dilution. Using a flame-sterilized metal spreader, 1 mL of each dilution was evenly spread over separate LB agar plates, then incubated at 21°C for 23 hours and 20 minutes before colony counting. Only plates with 30 to 300 colonies were considered; a plate with fewer than 30 colonies may not adequately represent the sample, and a plate with more than 300 colonies is too crowded to obtain an accurate count from (18). Any roughly circular cluster of cells visible to the naked eye with clear borders was considered a colony. The number of bacterial cells was calculated using the following equation:

$$Number\ of\ bacteria = \left(\frac{Number\ of\ CFUs}{milliliters\ plated}\right)(dilution\ factor) \qquad \text{(Equation 1)}$$

where "number of CFUs" is the number of colonies or "colony-forming units" (CFUs) counted on the plate.

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Drosophila inoculation

1 mL of a suspension of *S. marcescens* in a 5% sucrose solution (OD ~1.0) was diluted by a factor of 10^4. 5 mL of this diluted solution were added to 5 mL of uninoculated 5% sucrose solution. This 10 mL mixture was absorbed by four pieces of filter paper. Four other pieces of filter paper were placed in 10 mL of uninoculated 5% sucrose solution. Based on the OD value of the original 1 mL *S. marcescens* solution, we estimated that there would be enough bacterial cells soaked into the papers for each fly to consume at least 3,000 cells.

Drosophila melanogaster flies (Carolina, Cat# 172781, Cat# 172100) were separated into groups using cold anesthesia. The age of the flies was unknown. One group contained about 100 flies, and one group contained 5 flies to be fed uninoculated sucrose solution to estimate the typical number of bacterial cells in a healthy fly's microbiome.

The two groups of flies were placed in separate temporary vials and starved for approximately one hour prior to inoculation. They were then anesthetized using cold anesthesia so that the filter paper could be introduced. Once the flies recovered from anesthetization, they could ingest the fluid from the papers. The vials were sealed with airtight caps for the duration of the inoculation period, which was 27.25 hours. During this time, the flies were stored in an incubator at a temperature of 26°C.

Fly maintenance

After the inoculation period and anesthetization procedure, the flies were separated into two groups of 38 flies. Each group was transferred to its own BioChamber 250 maintenance vial. In the low-sucrose group, 35 flies survived this transfer process while 34 flies survived in the high-sucrose group. To seal the vials, pieces of gauze were placed over the mouths of the vials and fastened with rubber bands. All flies were maintained at 26°C in an incubator for the duration of the experiment.

Verification of infection

One infected fly and one uninfected fly were euthanized in separate glass test tubes in a freezer immediately following the inoculation period. 0.1 mL of 95% ethyl alcohol were added to each tube to sterilize the surface of the flies. The alcohol was replaced with 0.1 mL of water to rinse the flies. The water was removed and replaced with 0.5 mL of sterile 0.85% saline solution.

The flies were homogenized in the saline solution using a glass stirring rod. Each homogenized fly mixture was serially diluted by factors of 10^1 and 10^2. 0.1 mL of each dilution, as well as 0.1 mL of an undiluted sample, were plated on separate LB agar plates. The plates were inverted and incubated at 25–26°C for approximately 48 hours. *Equation 1* was used to calculate how many total bacterial cells the flies consumed based on CFU counts from the agar plates. The typical number of *S. marcescens* bacteria in the infected flies was estimated by subtracting the total number of cells found in the uninfected fly from the total number of cells found in the infected fly, assuming the difference between these values would represent the number of *S. marcescens* cells. A bar graph representing the number of bacterial cells calculated for each group was generated in Draxlr.com.

Drosophila survival data collection

The number of dead flies in each vial was recorded daily over a six-day period. This timeframe was selected to be roughly consistent with the five-day timeframe of a prior study (7). Data were collected at varying times during the day. The standard deviation of the hours between observation points was 3.733 hours. At the end of the study, restricted mean survival times (RMSTs) were calculated for both groups using the following equation:

$$RMST = \int_0^\tau S(u)du \qquad \text{(Equation 2)}$$

where tau (T) is the last recorded data collection time point and S(u) is the survival function generated by the Kaplan-Meier curves. These curves and the statistical values used in this study were generated in Numiqo.com, where a log-rank test was performed on the Kaplan-Meier curve data. RMST values were used instead of means because some of the survival data were censored. Because some flies did not die during the observation period, we could not consider their times of deaths in a traditional mean calculation. For this reason, the data for those flies were "censored," and RMSTs were used to quantify typical survival times for each group while considering flies that did not die only up to a defined time point.

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