SCIENCE

Published by the **American Association for the Advancement of Science (AAAS)**, *Science* serves its readers as a forum for the presentation and discussion of important issues related to the advancement of science, including the presentation of minority or conflicting points of view, rather than by publishing only material on which a consensus has been reached. Accordingly, all articles published in *Science*—including editorials, news and comment, and book reviews—are signed and reflect the individual views of the authors and not official points of view adopted by the AAAS or the institutions with which the authors are affiliated.

Membership/Circulation

Director: Michael Spinella
Fulfillment: Marlene Zendell, *Manager*; Gwen Huddle, *Assistant Manager*; Mary Curry, *Member Service Supervisor*; Pat Butler, Helen Williams, Laurie Baker, *Member Service Representatives*
Promotions: Dee Valencia, *Manager*; Hilary Baar, Angela Mumeka, *Coordinators*
Research: Kathleen Markey, *Manager*; Robert Smariga, *Assistant*
Financial Analyst: Jacquelyn Roberts
Administrative Assistant: Nina Araujo de Kobes
***Science* Member Services**
Marion, Ohio: 800-347-6969;
Washington, DC: 202-326-6417

Advertising and Finance

Associate Publisher: Beth Rosner
Advertising Sales Manager: Susan A. Meredith
Recruitment Advertising Manager: Janis Crowley
Advertising Business Manager: Deborah Rivera-Wienhold
Financial Manager: Leslie Gelder
Marketing Manager: Laurie Hallowell
Traffic Manager: Tina Turano
Recruitment: Michele Pearl, *Operations Manager*; Dan Moran, *Traffic Manager*; Debbie Cummings, Millie Muñoz-Cumming, Angela Wheeler, *Sales*
Marketing Associate: Allison Pritchard
Reprints Manager: Corrine Harris
Permissions Manager: Arlene Ennis
Sales Associate: Carol Maddox

ADVERTISING SALES: East Coast/E. Canada: Richard Teeling, 201-904-9774, FAX 201-904-9701 • **Southeast:** Mark Anderson, 305-856-8567, FAX 305-856-1056 • **Midwest:** Donald Holbrook, 708-516-8882, FAX 708-516-8883 • **West Coast/W. Canada:** Neil Boylan, 415-673-9265, FAX 415-673-9267 • **UK, Scandinavia, France, Italy, Belgium, the Netherlands:** Andrew Davies, (44) 457-838-519, FAX (44) 457-838-898 • **Germany/Switzerland/Austria:** Tracey Peers, (44) 270-760-108, FAX (44) 270-759-597 • **Japan:** Mashy Yoshikawa, (3) 3235-5961, FAX (3) 3235-5852
Recruitment: 202-326-6555, FAX 202-682-0816
European Recruitment: AnneMarie Vis, (44) 0223-302067, FAX (44) 0223-302068
Send materials to *Science* Advertising, 1333 H Street, NW, Washington, DC 20005.

■

Information for Contributors appears on pages 40–42 of the 1 January 1993 issue. Editorial correspondence, including requests for permission to reprint and reprint orders, should be sent to 1333 H Street, NW, Washington, DC 20005. ***Science* Telephone:** 202-326-6500, TDD 202-408-7770. **Other AAAS Programs:** 202-326-6400.

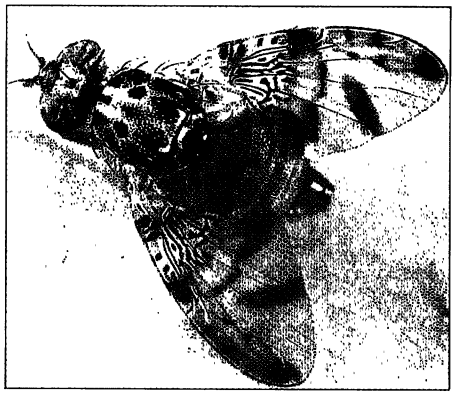
LETTERS

Fruit Fly Aging and Mortality

Two reports, "Slowing of mortality rates at older ages in large medfly cohorts" by James R. Carey *et al.* and "Demography of genotypes: Failure of the limited life-span paradigm in *Drosophila melanogaster*" by James W. Curtsinger *et al.* (16 Oct., pp. 457 and 461, respectively), discuss a phenomenon that was theoretically predicted long ago [see (*1*) for historical details] to be an inevitable feature of all stochastic models that consider aging as a progressive accumulation of random damage. We recently published the detailed mathematical proof of this prediction (*1*, pp. 246–276). In short, if destruction of an organism occurs not in one but in two or more sequential random stages, this is sufficient for the phenomenon of aging (mortality increase) to appear and then to vanish at older ages. Each stage of destruction corresponds to one of the organism's vitally important structures being damaged. In the simplest organisms with unique, critical structures, this damage usually leads to their deaths. This is why defects in such organisms do not accumulate and why the organisms themselves do not age. In more complexly structured organisms, where there are many vital structures with significant redundancy, every occurrence of damage does not lead to death. Defects do accumulate, however, giving rise to the phenomenon of aging (mortality increase). Thus, aging is a direct consequence of the increased reliability and life-span of organisms, which in turn result from the redundancy of vital structures. As defects accumulate, the redundancy in the number of key elements finally disappears. As a result, the organism degenerates into a system with no redundancy, that is, a system with elements connected in series, with the result being that any new defect leads to death. In such a state, no further accumulation of damage can be achieved, and the mortality rate levels off.

These explanations were published in our book (*1*), which was quoted in the reports by Carey *et al.* and by Curtsinger *et al.* in such a way as to possibly leave readers with the misimpression that the book is about limits to the life-span.

Leonid A. Gavrilov
Natalia S. Gavrilova
A. N. Belozersky Institute, Moscow State University, Moscow 119899, Russia



References

1. L. A. Gavrilov and N. S. Gavrilova, *The Biology of the Life Span: A Quantitative Approach* (Harwood Academic, Chur, Switzerland, 1991).

The reports by Carey *et al.* and by Curtsinger *et al.* represent a substantive contribution to the study of aging. The premise is simple—mortality rates at extreme old ages have not been reliably estimated because survival to extreme old age has always been a rare event. The solution was to place a huge cohort of a single species in a controlled environment and observe when they died.

These studies revealed non-Gompertzian mortality at older ages for fruit flies, thus indicating that programmed death does not exist. Programmed death is not consistent with evolutionary arguments about senescence (*1*). Current theories of senescence, based on principles of evolutionary biology, hypothesize that senescence occurs because the force of natural selection declines with age. Consequently, the maintenance and repair mechanisms necessary to ensure reproductive success early in life decline once reproduction begins. So, although we are not genetically programmed to die, neither are we programmed to survive much beyond the ages required to ensure reproductive success.

The Gompertz distribution is an empirical mathematical construct that describes mortality patterns for a genetically heterogeneous population. Once heterogeneity is reduced through differential mortality, a different mathematical function should apply to the surviving subgroup of Methuselahs. Carey's population of medflies was of sufficient size to reveal the heterogeneity not easily quantified in smaller study populations.