

ate, acetone, acetaldehyde, and formaldehyde, respectively. The estimated yield for all four products was 0.46%. This value probably underestimates the overall yield for these compounds because some organic matter that adsorbs to the oxides may not react, and some stable products may not desorb following formation. Also, our calculations almost certainly underestimate the overall formation of LMW organic products, because our analytical methods would not detect several major classes of likely products such as organic acids (other than α -ketoacids) and alcohols.

The dissolved NOC in most oxidizing environments consists primarily of biologically refractory compounds such as humic and fulvic acids. This material is composed of complex mixtures of moderately high-molecular-weight organics whose structural diversity defies both specific molecular identification and coherent enzymatic attack. Thus, the oxidative lysis of these compounds by Mn oxides represents a mechanism by which biologically refractory organic matter is converted into a suite of low-molecular-weight compounds that can be used as microbial substrates. Pyruvate, the major LMW product that we identified, is a major metabolic intermediary in the cell's TCA cycle, and as such, provides a ready source of both energy and carbon for growth. It is also a major product of the photolysis of humic compounds by ultraviolet light, and its photochemical production is closely coupled to its microbial use in sea water¹⁹.

In using LMW products, such as pyruvate, formed from photolysis of NOM¹⁹, microorganisms can be viewed as exploiting an external photochemical process that is otherwise beyond their control. But this is not true in the case of microbial use of the same or similar compounds formed from Mn oxide lysis of NOM because Mn oxides in most natural environments are formed by microbial catalysis³⁻⁶ and are found associated with the extracellular polymer sheath (glycocalyx) surrounding the

bacterial cells^{1,2}. Although several hypotheses have been proposed¹, the primary reason (or reasons) why the bacteria oxidize Mn (II) and deposit Mn oxides on their surfaces remains uncertain. We speculate that it may represent a means by which they can use the carbon contained in the large but biologically refractory pools of NOM, such as humic and fulvic acids. By mediating the production of Mn oxides these microbes also could play an important role in the biogeochemical cycling of organic carbon, particularly in surficial sediments where Mn oxide concentrations occur at much greater levels than those used in our experiments^{6,10}. □

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A genetic analysis of senescence in *Drosophila*

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Two attractive theories for the evolution of senescence are based on the principle that the force of natural selection decreases with age¹⁻⁵. The theories differ in the type of age-specific gene action that they assume. Antagonistic pleiotropy²⁻⁵ postulates that pleiotropic genes with positive effects early in life and negative effects of comparable magnitude late in life are favoured by selection, whereas genes with the reverse pattern of action are selected against. Mutation accumulation^{1,3-5} assumes that deleterious mutant alleles with age-specific effects will equilibrate at a lower frequency if their effects are expressed early rather than late in life. Explicit models demonstrate that both mechanisms can lead to the evolution of senescent life histories under reasonable conditions³⁻⁵. Antagonistic pleiotropy has gained considerable empirical support^{4,6}, but the evidence in support of mutation accumulation is more sparse^{4,5,7}. Here we report that the genetic variability of mortality in male *Drosophila melanogaster* increases greatly at very late ages, as predicted by the mutation accumulation hypothesis³⁻⁵. The rate of increase in mortality with age exhibits substantial genetic and environmental variability. This result provides a possible explanation for recent observations of non-increasing mortality rates in very old flies^{8,9}.

Forty wild-type third chromosomes were extracted from a large, outbred laboratory population of *D. melanogaster* (the IV

stock¹⁰). Wild-type male progeny produced by intercrossing the different extraction lines were assayed for age-specific survivorship (Table 1 and Fig. 1) to test a prediction of the mutation-accumulation theory of senescence^{1,3-5}. If deleterious mutant alleles have effects confined to specific ages, the equilibrium gene frequency for deleterious mutations should increase with the age of gene effect under a wide range of genetic and demographic conditions³⁻⁵. The mean value of fitness-related traits will therefore decrease with age and their genetic variability will increase. The decline in the mean of such a trait will contribute to senescence (defined here as a decline in fitness components with age). Both the additive genetic component of variance (V_A) and dominance component (V_D) are expected to increase with age under the mutation accumulation hypothesis (because for traits affected by rare deleterious alleles, both components increase with increasing mutant allele frequency^{11,12}).

Analyses of mortality over 3-week periods indicate that the genetic (V_A and V_D) and environmental (V_E) components of variance for this character increase dramatically with age (Table 1). All genetic components are statistically indistinguishable from zero during the early and intermediate periods, but are significantly greater than zero for the late time interval. It is clear from Table 1 that, at weeks 9 to 11, all five estimates of V_A , V_D and V_E from independent blocks of the experiment are much larger than corresponding estimates for earlier intervals. This result is not due to an inability to detect variation for low mortality rates, because the variance components for mortality during weeks 1 to 9 are very small compared to those for weeks 9 to 11, whereas the mean mortality rates over these two periods are comparable (for weeks 1 to 9, mean mortality rate $\mu = 14.6\%$, and the standardized variance components are $V_A = 3.94 \times 10^{-4}$, $V_D = 9.34 \times 10^{-4}$, $V_E = 14.7 \times 10^{-4}$). A large accumulation of mutational effects at very late ages, as exhibited by this population of flies, is predicted by theory¹³.

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Although there is ample evidence for genetic variation in lifespan and age-specific fertility^{4,5,14}, there appear to be no data on the extent of genetic variation in the rate of senescence itself. We used the Gompertz model of mortality to examine this question with our data. This model derives from the empirical tendency for mortality rates to increase exponentially with age in many animal species^{14,15}. Gompertz parameters are estimated as the slope and intercept of the regression line relating log mortality to age. The intercept of this line represents a baseline, or 'intrinsic' mortality rate for organisms that are not subject to the effects of senescence. The slope of the line estimates the rate of increase in mortality with age, and provides a measure of the rate of senescence. That is, the slope estimates the accelerating effect of senescence on the risk of dying in a given time interval¹⁴. Because the genotypes used in this experiment exhibited very low mortality before 6-weeks post-eclosion, and mortality generally increased monotonically after this period, the Gompertz parameters were estimated from the mortality rates observed after the first 42 days of adult life.

The additive genetic variance for the slope of the Gompertz curve was significantly different from zero, but that for the Gompertz intercept was not (see Fig. 1 legend). This suggests that

much of the heritable variability for lifespan in this population is due to variability in rates of senescence, and not to variability in baseline mortality rates. V_D estimates for the slope and intercept were not significant. Estimates of the coefficient of environmental variation were large and highly significant for both Gompertz parameters (0.210, $P < 0.001$, for the slope, and 0.270, $P = 0.03$ for the intercept). An increase in environmental variation with age has also been observed for female fecundity¹⁶.

These results represent the first demonstration to our knowledge of heritable variation in rates of senescence within a population of organisms. Even if the Gompertz parameterization is not a completely general description of patterns of mortality^{8,9}, the results strongly suggest that substantial heritable genetic variability exists for the change in mortality rates with age. The large amounts of both genetic and non-genetic variation in the rate of senescence provide a possible explanation for the results of two recent studies of mortality rates in very large cohorts of med flies and fruit flies^{8,9}. Both these studies suggest that cohort mortality rates cease to accelerate near the end of the lifespan of the oldest surviving flies. Individual variation in the rate of increase of mortality with age, whether genetic or environmental, would lead to the appearance of lower rates of increase at

FIG. 1 Regression lines of log (mortality + 1) on days since eclosion for 5 blocks, each consisting of 16 different sets of heterozygous third-chromosome genotypes. A third-chromosome balanced lethal system was constructed by placing *TM6/Sb* on an *IV* genetic background, producing a stock that is *+/+*, *+/+*, *TM6/Sb* for the X, second and third chromosomes, respectively. The *TM6* chromosome functions as an effective suppressor of crossing-over for the third chromosome²² and *Sb* is lethal in homozygous condition. The balancer stock is of the P/R cytotype, and should not induce hybrid dysgenesis²³ when crossed to flies from the *IV* stock. This balanced-lethal system was used to produce lines of flies carrying identical third chromosomes, heterozygous over *TM6*, as in ref. 20. Extraction lines were subdivided and maintained as two independent sublines. In any given experimental cross, flies from only one subline were used; the other subline was used in the replicate cross for that genotype. Common environmental effects and effects of common genetic background within a subline contribute to the variance within lines, not to the variance among lines. The 'environmental' variance may therefore be somewhat inflated. The effect is probably small, because many females from the balancer stock were used as parents in the first 2 generations of the extraction procedure (the variance due to the genetic background is $1/n$ times the corresponding genetic variance, where n is the number of maternally derived diploid genotypes per line). The 40 extraction lines were intercrossed in a North Carolina II crossing scheme²⁵, using 5 independent blocks of 4 maternal lines and 4 paternal lines (and reciprocal crosses) to produce 16 different heterozygous third-chromosome genotypes within each block. The additive genetic coefficient of variation (CV_A , equal to the ratio of $\sqrt{V_A}$ to the mean²⁴) for the slopes, pooled over 5 blocks, is 0.203, and is significantly greater than 0 ($P < 0.01$, $t = 4.12$). CV_A for the intercepts is 0.091 ($P = 0.16$, $t = 1.15$). Significance tests are based on 1-tailed t -tests of the variance components in 5 independent blocks.

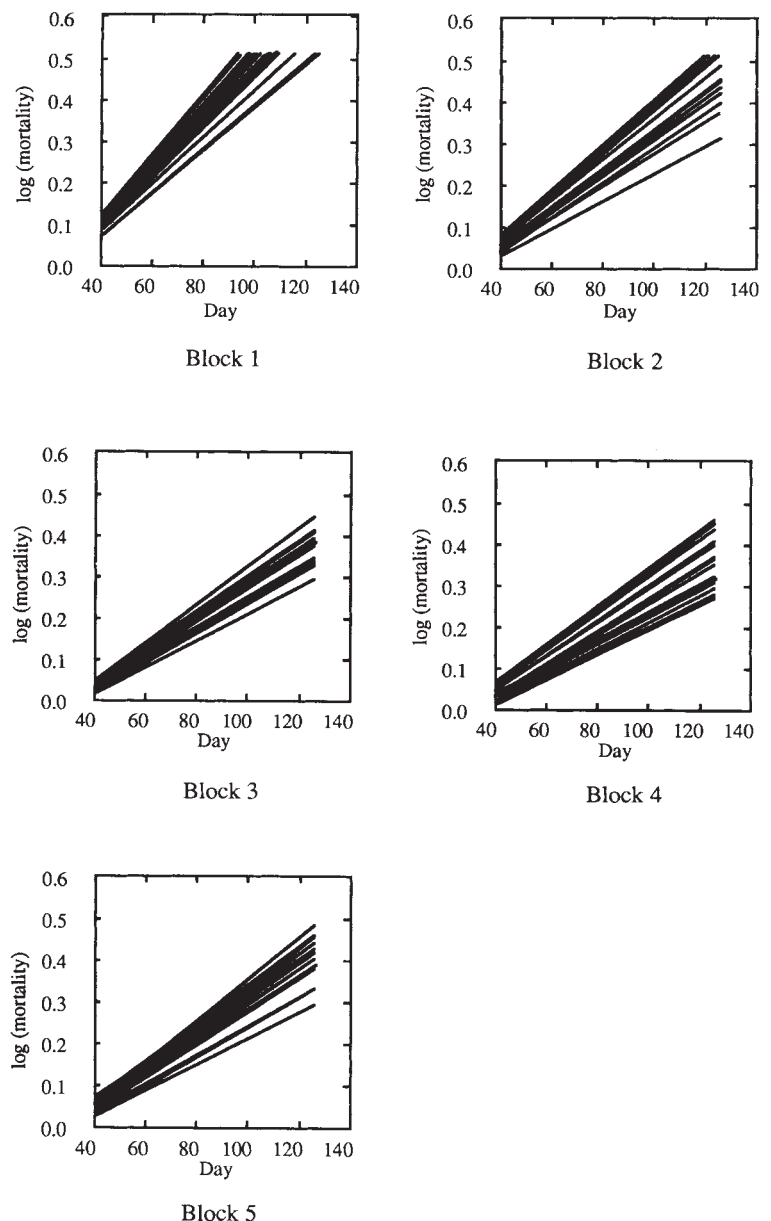


TABLE 1 Means and variance components for 3-week mortality rates at 3 ages

Block	Ecdysis to 3 weeks post-eclosion				5 to 7 weeks post-eclosion				9 to 11 weeks post-eclosion			
	μ (%)	$V_A \times 10^4$	$V_D \times 10^4$	$V_E \times 10^4$	μ (%)	$V_A \times 10^4$	$V_D \times 10^4$	$V_E \times 10^4$	μ (%)	$V_A \times 10^4$	$V_D \times 10^4$	$V_E \times 10^4$
1	0.52	0.7	-0.7	2.4	7.16	2.1	-0.2	15.6	81.5	91.4	40.2	489.6
		0.7	-0.6	2.1		2.0	-0.4	13.3		33.1	20.9	129.0
2	0.08	0.0	0.0	0.4	1.81	-0.1	1.1	9.7	19.2	60.7	37.8	64.7
		0.0	0.0	0.4		-0.1	1.3	8.2		44.0	27.3	41.7
3	0.47	-0.1	0.8	2.7	2.08	-0.5	2.4	9.6	25.1	53.8	35.9	69.1
		-0.1	0.8	2.5		-0.4	1.9	9.1		34.3	23.2	43.4
4	0.49	0.2	-0.4	2.5	0.92	0.3	-0.5	4.2	13.6	61.0	8.9	46.2
		0.1	-0.3	2.1		0.3	-0.4	3.9		47.1	7.6	34.0
5	0.48	0.0	-0.2	2.7	4.64	-0.5	2.5	21.0	27.8	34.5	31.4	251.1
		0.0	-0.2	2.4		-0.7	2.8	15.4		17.8	29.2	113.2
Pooled	0.41	0.1	0.1	1.3	3.32	0.2	1.7	7.1	33.4	59.4	30.5	89.7
		0.2	0.2	1.1		0.2	1.6	5.9		35.7	26.3	37.0
t-value		1.06	-0.20	4.89		0.73	1.65	4.96		6.88	5.70	3.59
Probability		n.s.	n.s.	<0.01		n.s.	n.s.	<0.01		<0.01	<0.01	<0.05

Blocks 1 to 5 represent partial-diallel crosses involving five independent sets of third-chromosome isogenic lines. μ is mean mortality rate, over a three-week period, of groups of flies representing many different heterozygous genotypes (see Fig. 1 legend). The mean is taken over all replicates in a block. Additive-genetic, dominance and environmental variance components for each block are given by V_A , V_D and V_E , respectively. These variance components were estimated by the synthesis method¹⁸, which produces unbiased estimates for random models¹⁹. No reciprocal effects were found, so reciprocal crosses were treated as simple replicates²⁰. Pooled estimates of variance components over all 5 blocks were calculated by the formula given in ref. 20. Within each block, the values for V_A , V_D and V_E reported on the upper line were calculated from unstandardized mortality rates; values appearing on the lower line were calculated from mortality rates that were first standardized to a block mean of 1. One-sample *t*-tests of the estimates from the 5 independent blocks indicate that, after correction for multiple tests, V_E is significantly greater than 0 at all ages, whereas V_A and V_D are significantly greater than 0 only for the latest time interval. Techniques used for estimating age-specific mortality are similar to those used in ref. 21. Males of each genotype were kept (without females) at a density of 20 per vial and were transferred weekly. Survivors were counted at each transfer until all flies were dead. To maintain constant density, dead flies were replaced by flies carrying a visible mutation. The proportion of flies dying from one transfer to the next gives an estimate of age-specific mortality for the genotype. These estimates were used to calculate mortality in 3 non-overlapping time intervals (all replicates contained some live flies throughout the experiment), and to compute Gompertz mortality parameters¹⁵ for each genotype.

advanced ages, because only individuals with low rates would survive to the end of the experiment. The large experiment in ref. 9 used highly inbred flies, which are known to be particularly sensitive to environmental effects^{11,17}. □

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Quantitative stereoscopic depth without binocular correspondence

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WHAT features in a stereogram define the disparities that lead to stereoscopic depth? The usual answer is that luminance-defined edges from the two eyes are matched and produce depth perception^{1,2}. But parts of an object may be occluded by other objects and absent from one eye's view. It was suggested that unpaired monocular elements might signal occlusion in depth, and the qualitative perception of depth associated with unmatched elements has been shown to be consistent with the geometry of occlusion³⁻⁵. We designed a stereogram that simulates a particular occlusion situation: an opaque white rectangle is stereoscopically

in front of a large black rectangle pasted on a white background. The position of the occluder is adjusted so that its left edge obscures the left-hand edge of the black rectangle in the right eye view and its right edge obscures the right-hand edge of the black rectangle in the left eye view. We report here that quantitative stereopsis can be seen from this stereogram, even though there are no binocular corresponding luminance edges to match.

The basic element of the stereogram is a square bracket pattern (Fig. 1a, b). When binocularly cross-fused, a white rectangle is seen floating in front of the image plane (Fig. 1a) or lying behind the image plane (Fig. 1b). In the fused image, the outside edges of the vertical bars of one eye's bracket are aligned with the short vertical edges above and below the openings of the other eye's bracket. Notice that neither the left nor the right edge of the middle parts of the vertical bars have corresponding luminance edges in the other eye's image.

Stereopsis without point to point correspondence has been reported previously. Monocular figures defined by either subjective contours or texture borders could be combined binocularly and produce illusory surfaces in depth as if they were defined by normal luminance contours⁶⁻⁹. But the phenomenon illus-